

Pediatric Education and Research

Editor-in-Chief

Pravakar Mishra

Professor and HOD Pediatrics,
PRM Medical College Hospital,
Baripada-757001, Odisha.

Past Editor-in-Chief

Surender N. Gupta, Kangra

Associate Editors

Jayendra R. Gohil, Bhavnagar

National Editorial Board

Bhawna Sharma, New Delhi

Daisy Khara, Jodhpur

H.P. Deshmukh, Pune

KS Venketraman, Mumbai

Rajniti Prasad, Varanasi

Ritu Rakholia, Haldwani

Shital Bhattad Gondhali, Latur

Sunil Mhaske, Ahmednagar

Usha Pranam, Raichur

Yogesh Kumar Sarin, New Delhi

RED FLOWER PUBLICATION PVT. LTD.

Managing Editor

A. Lal

Publication Editor

Manoj Kumar Singh & Dinesh Kr. Kashyap

Pediatric Education and Research (PER) (pISSN: 2321-1644, eISSN: 2455-4871) is a quarterly peer-reviewed journal. The journal is publishing original research, clinical observations, and special feature articles in the field of pediatrics, as broadly defined. Contributions pertinent to pediatrics are also included from related fields such as nutrition, surgery, dentistry, public health, child health services, human genetics, basic sciences, psychology, psychiatry, education, sociology, and nursing.

Abstracting and Indexing information: Index Copernicus, Poland; ProQuest, USA; Genamics JournalSeek.

Readership

Readership for The Indian Journal of Pediatric Education includes pediatricians, researchers, pediatric investigators, and all those who diagnose and treat infants, children, and adolescents.

Subscription rates worldwide: Individuals - contact on 91-11-22754205 or mail to redflowerppl@vsnl.net; Institutional (annual)- INR7500/USD586 Single issue INR2333/USD167. Payment methods: By Demand Draft/cheque should be in the name of **Red Flower Publication Pvt. Ltd.** payable at Delhi. By Bank Transfer/TT: **Bank name:** Bank of India, **IFSC Code:** BKID0006043, **Swift Code:** BKIDINBBDOS. **Account Name:** Red Flower Publication Pvt. Ltd., Account Number: 604320110000467, Branch: Mayur Vihar Phase-I, Delhi - 110 091 (India).

© 2018 Redflower Publication Pvt. Ltd. All rights reserved. The views and opinions expressed are of the authors and not of the **Pediatric Education and Research**. The **Pediatric Education and Research** does not guarantee directly or indirectly the quality or efficacy of any product or service featured in the the advertisement in the journal, which are purely commercial.


Printed at Mayank Offset Process, 794/95 Guru Ram Dass Nagar Extn, Laxmi Nagar, Delhi - 110092.

[←](#)
[→](#)
[↻](#)

[Secure](#) | <https://journals.indexcopernicus.com/search/form?search=Pediatric%20Education%20and%20Research>


[Apps](#)
[Red Flower Publicat](#)
[Inbox \(14\)](#) - author@
 [RED FLOWER PUBLIC](#)
[Google](#)
[Admin Panel](#)

[☆](#)


INDEX COPERNICUS
 I N T E R N A T I O N A L

[ICI World of Journals](#)
[ICI Journals Master List](#)
[Contact](#)

[Login/ Register](#)



Journal title: Pediatric Education and Research

ISSN: 2321-1644, 2455-4871

GICID: n/d

Country / Language: IN / EN

Publisher: Red Flower Publication Private Limited

Search Results

Citation:	N/A
MNISW 2016:	N/D
ICV 2016:	74.85
ICV 2015:	71.54

Pediatric Education and Research

May - August 2018
Volume 6 Number 2

Contents

Original Research Articles

- Effect of HAART on the Anthropometric Parameters and CD4 Counts in HIV/AIDS Affected Children** 45
Ritu Rakholia, Meher Bano, Vineeta Rawat
- A Comparative Study Based Grading of Malnutrition by WHO Z Score and IAP Classification** 53
Sanjeev Suman, Sonal Vyas, Swati Raipurkar
- Clinical Predictors of Metabolic Acidosis in Hospitalized Children of 6-60 Months with Severe Acute Malnutrition** 61
Saurabh Kumar Patel, Sunil Kumar Rao, Naresh Bajaj, Tej Bali Singh
- Role of Phototherapy in Neonatal Hyperbilirubinemia** 65
Amar Taksande, Rewat Meshram, Begam Rubia, Akshi Gandhi
- Study of The Quality of Life in Children with Chronic Diseases in A Tertiary Care Hospital in Mysuru** 73
Anitha C., Kavya Kurkal, Santhosh Kumar M.
- Clinical Profile of Hypoxic- Ischemic Encephalopathy in Neonates with Birth Asphyxia in A Rural Tertiary Care Hospital in Central India** 80
Kher Anjali M., Singh Kuldeep, Vagha Jayant
- Estimation Early Neonatal Hyperbilirubinemia Utilizing 24-hour Serum Bilirubin Level at Bhuj, Kutch** 87
Arun Parikh, Abhikumar Shingala
- Spectrum of Acute Kidney Injury and its Clinical Profile and Outcome in Children Admitted to PICU** 90
Basavaraj Patil, Savita Patil, Harsha S.
- Survival and Major Morbidity Pattern of Preterm Infants Admitted in a Level -3 NICU: A Retrospective Study** 95
N. Venugopalan, Salahudeen, Pavan Kumar D., Vidya Singaravelu, Manoj Narayanan K.A.
- Long Term Outcome of Treated Posterior Urethral Valve Patients at a Tertiary Care Center** 100
Neha Agarwal, Neeraj Agarwal, Megha Agarwal
- Clinical Correlates of Working Memory Deficits in School Going Children with and Without ADHD in Ahemdagar** 104
Prajakta Ghatage, Sunil Mhaske, Ramesh Kothari, Ganesh Misal

Craniofacial Anthropometric Measurement of Full Term Newborns in Tertiary Care Hospital	108
Punam Uke, Akash Daswaney, Sam Rodrigues, Sonal Gore, Amar Taksande	
Paediatric Museum: An innovative Teaching-Learning Method	112
Sunil Mhaske, Prajakta Ghatage, Ramesh Kothari	
Screening of School Children for Attention Deficit Hyperactivity Disorder using Vanderbilt ADHD Diagnostic Teacher Rating Scale	116
Jayendra R. Gohil, Mrigank N. Gupta, Shruti U. Patel, Shreya A. Patel	
Rebound Hyperbilirubinaemia Following Intensive Phototherapy	120
Basavaraj M. Patil, Harsha A. Sangolgikar	
 <i>Review Article</i>	
Rotavirus Vaccine	124
Sunil Mhaske, Vineetranjan Gupta	
 <i>Case Report</i>	
Neonatal Head Injury as a Potential Etiology for Autism Spectrum Disorders and Attention Deficit Hyperactivity Disorder (ADHD)	127
Santosh Kondekar, Amit Rudrakar, Sharan Subramanian, Surbhi Rath	
Guidelines for Authors	129

Subscription Information

India

Institutional (1 year) (Print+Online): INR7500

Rest of the World

Institutional (1 year) (Print+Online): \$586

Payment instructions

Online payment link:

<http://rfppl.co.in/payment.php?mid=15>

Cheque/DD:

Please send the US dollar check from outside India and INR check from India made.
Payable to 'Red Flower Publication Private Limited'. Drawn on Delhi branch

Wire transfer/NEFT/RTGS:

Complete Bank Account No. 604320110000467
Beneficiary Name: Red Flower Publication Pvt. Ltd.
Bank & Branch Name: Bank of India; Mayur Vihar
MICR Code: 110013045
Branch Code: 6043
IFSC Code: BKID0006043 (used for RTGS and NEFT transactions)
Swift Code: BKIDINBBDOS

Send all Orders to: Subscription and Marketing Manager, Red Flower Publication Pvt. Ltd., 48/41-42, DSIDC, Pocket-II, Mayur Vihar Phase-I, Delhi - 110 091(India), Phone: 91-11-45796900, 22754205, 22756995, E-mail: sales@rfppl.co.in, Website: www.rfppl.co.in

Effect of HAART on the Anthropometric Parameters and CD4 counts in HIV/AIDS Affected Children

Ritu Rakholia¹, Meher Bano², Vineeta Rawat³

¹Associate Professor, Department of Pediatrics ²Assistant Professor, Department of Community Medicine ³Associate Professor, Department of Microbiology, Government Medical College, Haldwani, Uttarakhand 263129, India.

Abstract

Background: Role of Highly Active Retro Viral Therapy (HAART) in improving the nutritional status and immunity in poorly accessible areas of developing countries has been inadequately documented. This study aims to assess the effect of HAART on weight, height and CD4 counts of Human Immune Deficiency Virus (HIV) infected children and analyze factors associated with the effect. **Methods:** Anthropometric measurements and CD4 counts were taken on registration and follow up and duly recorded. **Results:** 107 children were studied for one year out of these 63 received ART on the basis of CD4 counts or symptoms. The mean weight improved from 16.67 ± 7.64 kg to 19.63 ± 9.15 kg in boys and from 18.45 ± 9.77 kg to 21.7 ± 10.89 kg in girls. The mean height increased from 105.82 ± 21.5 cm to 108.75 ± 23.18 cm in boys and from 107.7 cm to 108.58 ± 24.54 cm in girls. The percentage of underweight, stunted and wasted children decreased after the start of HAART from 58.7% to 32.6%, 69.8% to 60.5% and 21.7% to 11.4% respectively. Weight for age and weight for height z scores improved significantly while height for age showed a small increase. No effect on CD4 counts was seen. **Conclusions:** HAART has a positive effect on growth in HIV1 infected children irrespective of nutritional supplementation. The change is more significant in severe forms of malnutrition. We strongly recommend viral load estimation for better monitoring and prompt and universal initiation of HAART in children with HIV infection to prevent growth failure.

Keywords: Malnutrition; Children with HIV/AIDS; HAART; CD4 Count.

Introduction

Globally the AIDS epidemic has slowed down and the number of new patients has declined or stabilized. Adult HIV prevalence in India too has declined to 0.31% in 2009 against 0.36% in 2006. Estimated number of people with HIV/AIDS in India is 2.40 million in 2009 against 2.44 million in 2008 out of which 3.5-4.4% is children less than 15 years [1]. Nutritional disorders may decide the course of HIV infection and timely management of these conditions may help in recovery and maintenance of health status [2,3].

Uttarakhand is a small hilly state with 80% of population scattered through small rural hamlets which are economically poor and geographically difficult to access due to hilly terrain and frequent

natural disasters. Unlike the trend of HIV/AIDS internationally as well as in high prevalence areas of India, where new cases and prevalence are showing a decline, in low prevalence state of Uttarakhand. However, adult HIV prevalence has increased in the last few years from 0% in 2007 to 0.1% in 2010 [3,4].

With the increasing number of children surviving into adulthood and taking long term HAART its response on nutritional status needs greater importance. Malnutrition is rampant in Indian children with HIV/AIDS as seen in studies from Uttarakhand [5] and South India [6]. Malnutrition is an independent risk factor for death [7]. Role of HAART alone in alleviation of malnutrition is not well documented. This study was conceived with an aim to study the baseline profile of these children. To determine the effect of HAART on the under

Corresponding Author: Ritu Rakholia, Associate Professor, Department of Pediatrics, Government Medical College, Haldwani, Uttarakhand 263129, India.

E-mail: lalitriturakholia@rediffmail.com

Received on 03.08.2018, Accepted on 31.08.2018

nutrition and anthropometric measurements in the children and to ascertain the effect of HAART on the CD4 count of the children.

Materials and Methods

This is a retro prospective record based study carried out for a duration of 1 year (Jan 2013 to December 2013) in the ART Centre of a tertiary Centre in Kumaon region of Uttarakhand. The recorded data of the children (less than 15 years of age) affected with HIV/AIDS, either born in the medical college hospital or coming from peripheral areas of Uttarakhand for taking HAART or follow up was obtained from the ART Centre of the hospital after completing permission and ethical formalities. The anthropometric measures of all children including height (in centimeters), weight in kilogram was taken. The CD4 count was also taken at the time of registration and every 6 monthly as per earlier protocols [8]. Children were also examined clinically for nutritional deficiencies and opportunistic infections like tuberculosis.

Height and weight were obtained upon enrolment. The z scores for weight, height, and BMI were computed based on child's age and gender using the standard [9] method and references [10,11]. The WHO recommends a cut off z score of <2 weight for age to classify underweight, low height for age (stunting) and low weight for height (wasting) and a z score of <3 SD to define severe under nutrition. A z score of <2 indicates that a child's height for age (HAZ), weight for age (WAZ) and weight for height (WHZ) is 2 SD below the age and gender specific median for the normal population. Valid z scores were obtained for only 46 out of 107 children. The CD4 counts of all patients were also recorded and correlated with under nutrition, wasting and stunting.

The children once found to be HIV positive were registered in the ART Centre of the Medical College and kept on follow up. HAART was started on the basis of symptoms or CD4 counts as per prevalent guidelines [8]. The counts and anthropometric measurements were repeated on follow up 6 monthly.

Statistical Analysis

The obtained information was checked for missing data, coded properly and entered in MS excel 2007. The data was analyzed using SPSS -18 for carrying out chi-square test of association. WHO z score value was calculated using WHO

Antroplus software to determine the Under nutrition in the children.

Results

The total number of children infected with HIV attending ART Centre during the study period was 107 out of which 63 children on HAART were studied. All of them were infected by the parent to child transmission and all of them were found to be infected by HIV1. Of the 107 children there were 67 male and 40 female children. Mean age of the children was 7.15 ± 3.8 years (range 2 to 15 years). 4 cases were lost to follow up over the one year study period one each at 2yr, 5yr, 8yr and 10 yrs. An 8 year old child died due to severe pneumonia in respiratory failure during the study period and another died at home due to unknown cause. The HAART regime given to the children is shown in Table 1.

The change in under nutrition, wasting and stunting after use of HAART is depicted in Table 2.

Table 1: HAART regime given to children (n=63)

HAART Regime	Number of children receiving HAART
Abacavir+ Nevarapine (ABC+NVP)	6
Stamivudine (SLE)	3
Stamivudine/ Abacavir (SLE/ ABC)	1
Stamivudine/ Zidovudine (SLE/ ZLN)	1
Stamivudine/ Abacavir +3TC+Efavirenz+ Zidovudine (SLE/ ABC+3TC+EFV+ZLN)	1
Tenofovir	2
Zidovudine (ZLN)	45
Lost to follow up (LFU)	4

The comparison of mean weight and height of children before and after HAART with WHO reference is shown in Figure 1-4. The percentage of underweight, stunted and wasted children has decreased after the start of ART from 58.7% to 32.6%, 69.8% to 60.5% and 21.7% to 11.4% respectively. There is a marked reduction in underweight, wasting and a minimal improvement in stunting. There is also a greater improvement in more severe forms of underweight, wasting and stunting as compared to milder forms.

In the present study the valid z score values could be calculated only for 46 children aged 2 to 5 years.

As per WHO the WHZ parameter is not used in children 5-14 years of age because of puberty changes hence to assess wasting BMI/age is used. But in the present study the valid z score could not be obtained for children beyond 5 years. Hence, both WHZ and BMI/age is used in the children 2-5 years of age. After the start of HAART, the children were gaining weight as shown by increased percentage of 56.8% in the BMI/age category of > +1SD and 22.7% in > +2SD category. BMI/age helps us identify not only a decrease in thinness but also appreciate an increase in overweight from 19.6% to 56.8% and obesity from 10.9% to 22.7% with the institution of HAART (Table 3).

Mean WAZ (weight for age) z score HAZ (height for age) z score and WHZ (weight for height) z score increased after institution of HAART (Table 4). The mean weight and height of all children

has shown an increasing trend with institution of HAART (Table 5).

CD4 count was available for only 44 children before HAART was initiated and for 35 children after the start of HAART out of the 46 children had valid z score values. Median CD4 count in terms of median \pm inter-quartile range before HAART was 790 \pm 504 cells/ μ l (Range 179 to 1895) and after HAART was 586 \pm 383 cells/ μ l (range 207-1280). (Table 6)

The Z score value of different anthropometric parameters represent the deviation from the median value of the WHO reference population. After HAART, the mean Z score value of different anthropometric parameters decreased clearly indicating a decrease in variation from normal (Table 7).

Table 2: Comparison of under nutrition in the children before and after the HAART using WHO z score

Children	Number	Before HAART % (95% C.I)	Number	After HAART % (95% C.I)
Underweight(n=46)	27	58.7% (95% C.I is 43.4% to 74%)	15	32.6% (95% C.I is 18% to 47.2%)
Severe underweight(n=46)	18	39.1% (95% C.I is 23.9% to 54%)	10	21.7% (95% C.I is 8.7% to 34%)
Stunted(n=43)	30	69.8% (95% C.I is 54.9% to 84%)	26	60.5% (95% C.I is 44.7% to 76%)
Severe stunted(n=43)	20	46.5% (95% C.I is 30.4% to 62%)	14	32.6% (95% C.I is 17.4% to 47%)
Wasted(n=46)	10	21.7% (95% C.I is 8.7% to 34%)	5	11.4% (95% C.I is 0.8% to 21.9%)
Severe wasted(n=46)	6	13% (95% C.I is 2.2% to 23.9%)	1	2.3% (95% C.I is 0% to 7.8%)

Table 3: Anthropometric profile of children before and after HAART

Anthropometric parameters	Before HAART (n=46)		After HAART (n=46)	
	Number	%	Number	%
WAZ (<-2SD) (underweight)	27	58.7%	15	32.6%
HAZ (<-2SD) (Stunted)	30	69.8%	26	60.5%
WHZ (<-2SD) (wasted)	10	21.7%	5	11.4%
BMI/age	Number	%	Number	%
Thinness (<-2SD)	10	21.7%	2	4.5%
Severe Thinness (<-3SD)	3	6.5%	1	2.3%
Overweight (>+1SD)	9	19.6%	25	56.8%
Obesity (>+2SD)	5	10.9%	10	22.7%

Table 4: Mean Z score value of different anthropometric parameters before and after HAART

Anthropometric parameters	Before HAART (n=46)			After HAART (n=46)		
	Number	Mean value	SD	Number	Mean value	SD
WAZ	n=46	-2.25	1.31	n=46	-1.0	1.67
Length/HAZ	n=43	-2.88	1.82	n=43	-2.34	1.95
WHZ	n=46	-0.66	1.8	n=44	0.61	1.72
WAZ in males	n=32	-2.39	1.4	n=32	-1.29	1.67
Length/HAZ in males	n=29	-2.89	1.87	n=29	-2.31	2.05
WHZ in males	n=32	-0.8	1.68	n=31	0.41	1.64
WAZ in females	n=14	-1.91	1.01	n=14	-0.32	1.52
Length/HAZ in females	n=14	-2.87	1.77	n=14	-2.41	1.8
WHZ in females	n=14	0.33	2.08	n=13	1.09	1.89

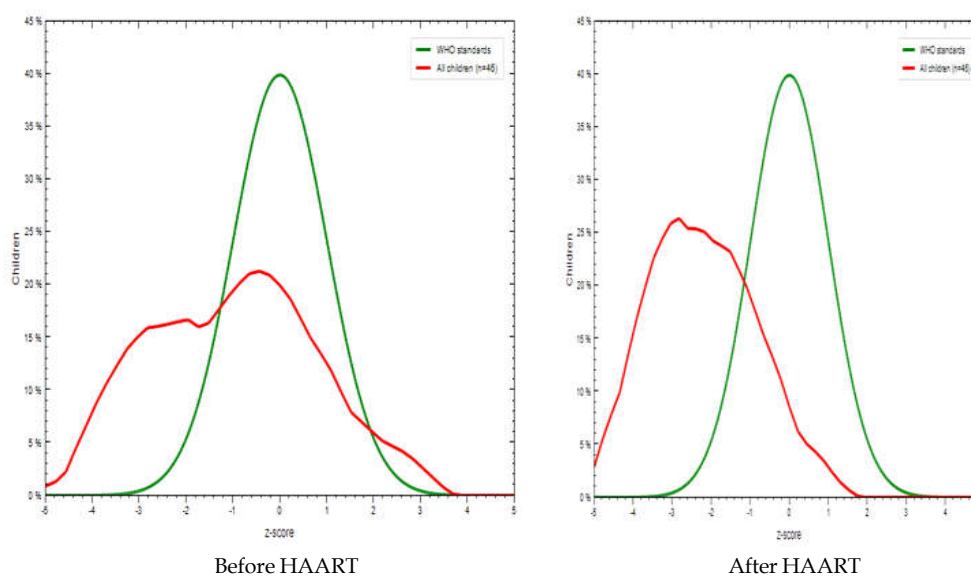
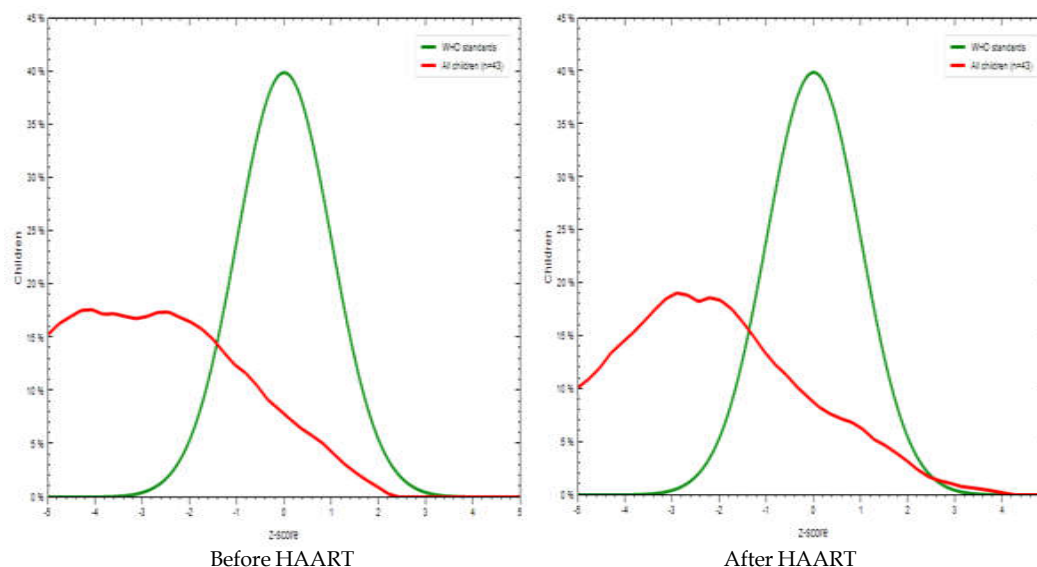
Table 5: Effect of ART on the weight of children and CD4 count

CD4 count (per microlitre of blood)	Normal weight		Moderate underweight		Severe underweight children	
	Before HAART *	After HAART#	Before HAART*	After HAART#	Before HAART*	After HAART#
>=1000/ulit (No suppression level)	6 (33.3%)	3 (11.1%)	3 (37.5%)	1 (50%)	2 (11.1%)	0 (%)
500-999/ulit (Moderate suppression level)	6 (33.3%)	13 (48.1%)	3 (37.5%)	0 (%)	11 (61.1%)	4 (66.6%)
< 500/ulit (Severe suppression level)	6 (33.3%)	11 (40.7%)	2 (25%)	1 (50%)	5 (27.8%)	2(33.3%)

*BeforeHAART (n=44), # After HAART(n=35)

On using HAART the WAZ, HAZ, WHZ curves were shifting towards right side depicting positive improvement in all growth parameter with HAART.

There was no significant difference between male and female children (Fig. 4-12).

**Fig. 1:** Comparison of WAZ z score value in the studied children with the WHO reference WAZ z score value(original)**Fig. 2:** HAZ z score value comparison in the studied children with the WHO reference HAZ (original)

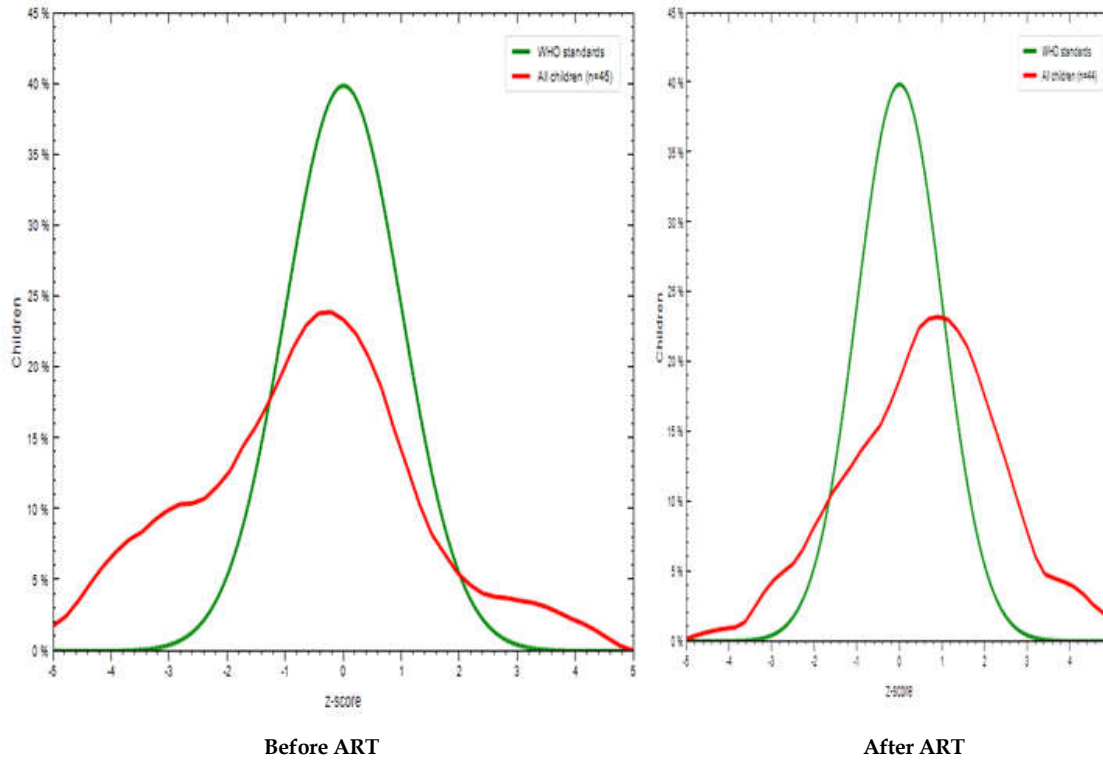


Fig. 3: Comparison of weight for height z score value with the WHO reference median population(original)

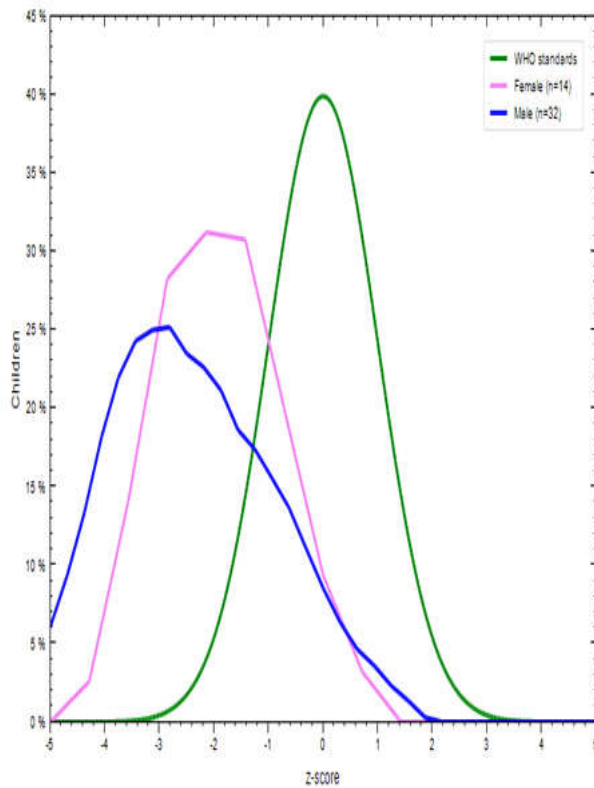


Fig. 4: Weight for age z score curve according to sex (Before ART)(original)

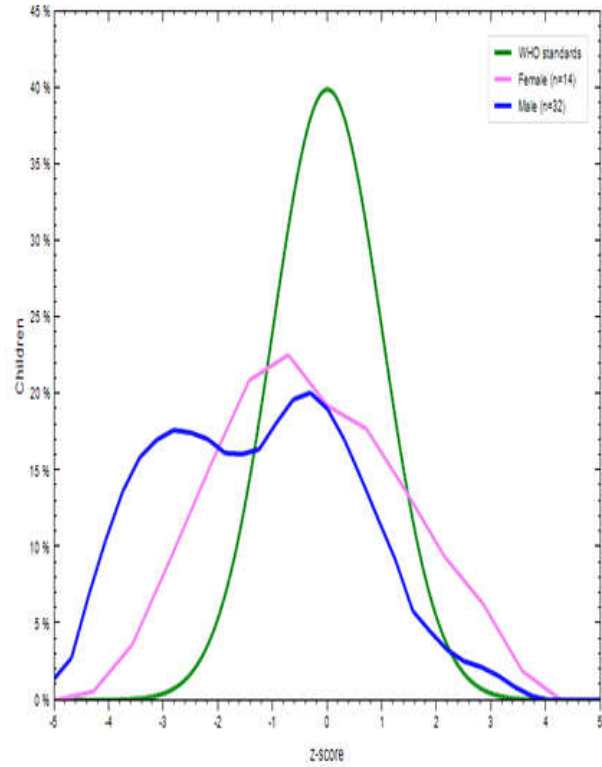


Fig. 5: Weight for age z score curve according to sex (After ART) (original)

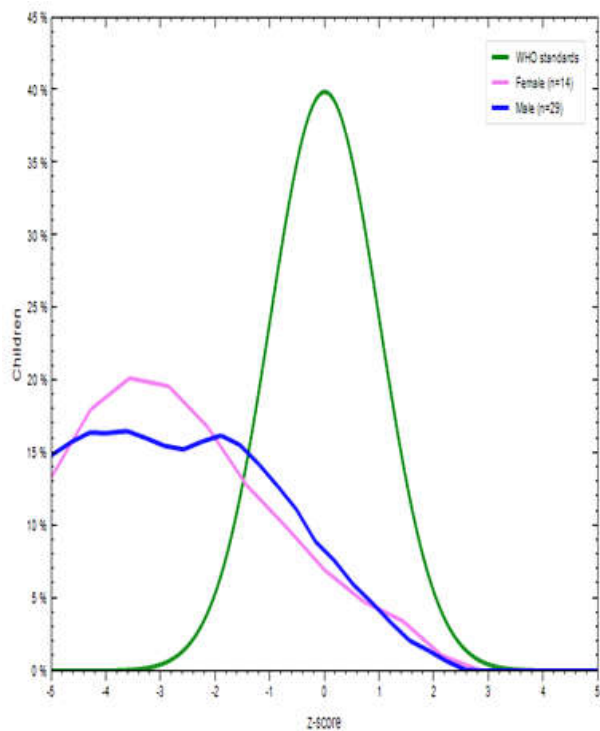


Fig. 6: Height for age z score curve according to sex (Before ART) (original)

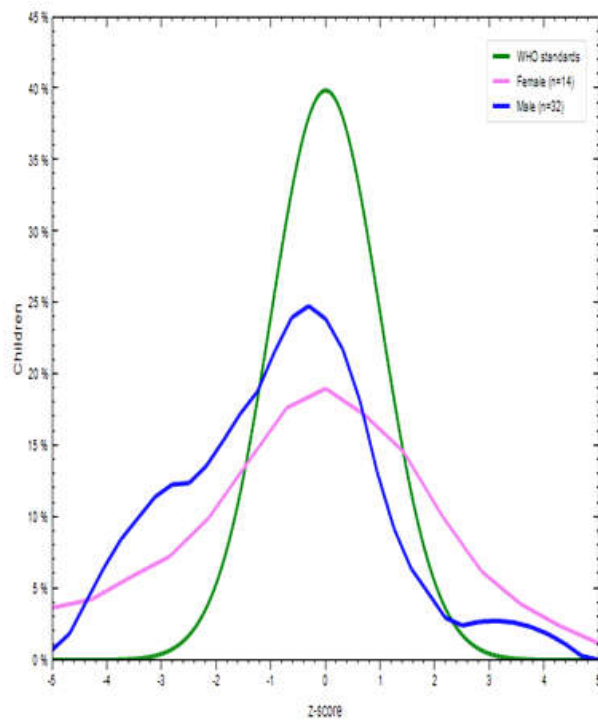


Fig. 8: Weight for height z score curve according to sex (Before ART) (original)

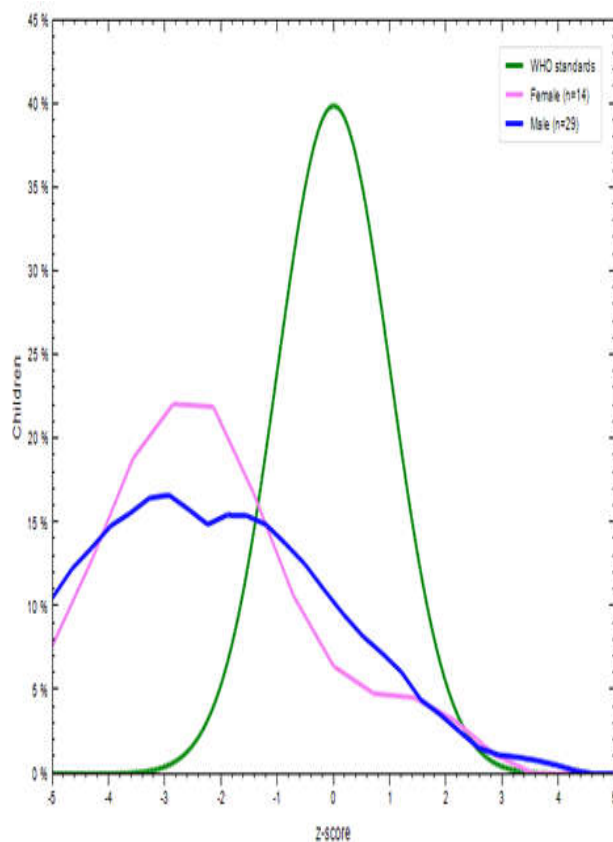


Fig. 7: Height for age z score curve according to sex (After ART) (original)

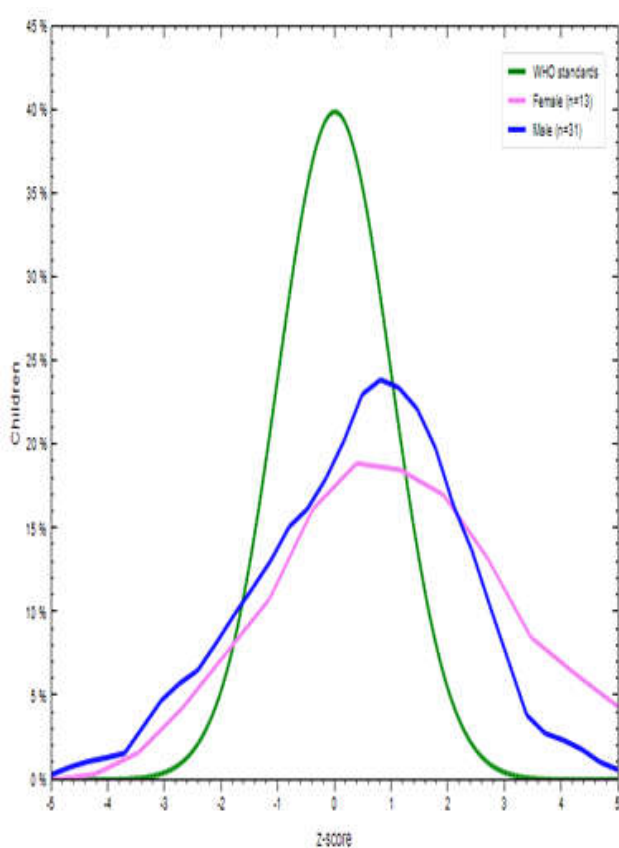


Fig. 9: Weight for height z score curve according to sex (After ART) (original)

Discussion

Malnutrition accelerates the progression of HIV/ AIDS and chances of acquiring other opportunistic infections by lowering immunity and also causes growth failure i.e short stature or stunting with its permanent psychological and functional consequences [12]. Role of HAART alone in alleviation of malnutrition (measured by anthropometric measurements like weight, height) is not well documented as developed countries follow enteral or parenteral support along with HAART and some studies show contradictory findings [13].

In the present study we found that height and weight of children infected with HIV1 improved after HAART. There was a marked reduction in underweight (WAZ z score -2.25 to -1.0), wasting (WHZ z score -0.66 to +0.61) and stunting (HAZ z score -2.88 to -2.34) and the effect is more marked in children with severe forms of malnutrition as seen by a greater reduction in severe underweight (31.9% to 21.7%), severe wasting (13% to 2.3%) and severe stunting (46.5% to 32.6%). There is also a more marked improvement in weight than height as indicated by a greater reduction in wasting than stunting. We found that height did improve on follow up though not as significantly as weight. This is understandable as in all chronic infections height and weight do not accelerate simultaneously. An increase in weight is followed by an increase in height. Similar results have been seen in Indian studies [14,15,16], resource poor African countries [17] and developed countries with easy accessibility to HAART like USA [18,19]. The difference in the increase is probably due to the different durations of follow up. Our study has a short follow up of 1 year while other studies have 6 months to 6 years. Buchacz et al. [20] found a per year gain of 0.13z score in height and 0.05 z score in weight on protease inhibitor based HAART. Why HAART improves the nutritional status can be due to the energy used for immunologically fighting the virus being better utilized for growth after institution of HAART along with lesser number of opportunistic infections and decreased gut infections hence decreased malabsorption.

The change in nutritional status in the present study was not affected by CD4 counts as also seen by Miller TZ et al. [19] in contrast to other studies from India [14,15] and Nepal [21] who found HAART to have a simultaneous effect on CD4 counts and WAZ z scores. Other studies [22,23] found that the children on HAART who were viral responders had

significant increases in height and weight while the non-responders did not. We could not do viral load estimation due to economic constraints but CD4 counts did not correlate to nutritional status and did not show short term response to HAART.

We have not studied changes in fat distribution though the percentage of overweight and obese children increased. Adult HIV patients receiving protease inhibitor therapy have described a metabolic syndrome with peripheral insulin resistance, hyperlipidemia, lipodystrophy, truncal obesity. They have not been widely described in children (39% lipodystrophy seen by Guillen et al. [18] but a longer and larger study would be needed to rule it out.

We have not used control of similar children due to ethical reasons but compared the growth parameters before and after HAART. Longer duration studies are needed to conclusively study the effect of HAART on CD4 counts.

Conclusion

This study strongly supports the institution of HAART at the earliest to all children with HIV/ AIDS for its positive effect on nutritional status which in turn is responsible for the growth and immunity of children and lower morbidity and mortality. We also recommend viral load estimation rather than CD4 counts for assessing response to HAART and immunological status as CD4 counts did not show a good correlation in this study. This study also serves to alert health workers to the importance of routine monitoring of nutritional status by anthropometry so that not only malnutrition but also potential metabolic problems which may arise in a small subset of children with HAART suggested by an increase in obese and overweight children may be timely diagnosed and remedial measures may be instituted at the earliest.

References

1. National AIDS Control Organisation, Ministry of Health and Family Welfare Government of India. NACO press release on HIV estimates 2009. Available at <http://naco.gov.in/NACO/>.
2. Liu L, Johnson HL, Cousens S, Perin J, Scott S, Lawn JE. Global, regional and national causes of child mortality: an updated systematic analysis for 2010 with time trends since 2000. *Lancet* 2012;379:2151-61.
3. Pandey A, Sahu D, Bakkali T, Reddy DCS, Venkatesh S, Kant S et al. Estimate of HIV prevalence and

- number of people living with HIV in India 2008-2009. *BMJ Open*. 2012;2:e000926. doi:10.1136/bmjopen-2012-0009.
4. Kumar R, Sharma SK. Trends of Communicable and Non-Communicable Morbidities in Uttarakhand state: a systemic review. *Indian J Community health* 2013;25(2):178-87.
 5. Rakholia R, Bano M, Rawat V. Malnutrition among HIV- infected Children by Anthropometric Measures in Poor Outreach Area of a Developing Country and its Relationship with CD4 Counts. *Int J Pediatr* 2016; 4(4):1643-54.
 6. Padmapriyadarsini C, Pooranagangadevi N, Chandrasekaran K, Subramanyan S, Thiruvalluvan C, Bhavani PK et al. Prevalence of underweight and wasting among children infected with Human Immunodeficiency Virus in South India. *Int J Pediatr* 2009;83:7627.
 7. Arpadi S. Growth Failure in HIV infected children. *J Acquir Immune Defic Syndr* 2000;25 Suppl 1:S37-42.
 8. National Pediatric and Family HIV Resource Centre(NPHRC). The health Resources and services Administration (HRSA) and the National Institute of Health(NIH). Guidelines for the use of Antiretroviral agents in Pediatric HIV infection. November 2003. Available at: <http://AIDSinfo.nih.gov>.
 9. Lohman T, Roche AF, Martorell R. Anthropometric standardization reference manual. Champaign, IL: Human Kinetics Books; 1988.
 10. World Health Organization. Physical status: the use and interpretation of Anthropometry -report of a WHO expert Committee. Geneva; 1995. (WHO Technical Report Series, 854). Available at http://www.who.int/child growth/publications/physical_status/en/.
 11. World Health Organization. Multicentre Growth References Study Group. WHO Child Growth Standards based on length/height, weight and age. *Acta Paediatr Suppl* 2006;450:76-85.
 12. Gillespie S, Kadiyal S. HIV/ AIDS and food nutrition security-From Evidence to action. *LSHTM Research Online* 2005;24(4):352-357.
 13. Nachman SA, Sharon A, Lindsey JC, Moye J, Stanley KE, Johnson GM, Krogstad PA, Andrew A. Growth of Human Immunodeficiency Virus-Infected Children Receiving Highly Active Antiretroviral Therapy. *Pediatric Infect Dis Journal*. 2005;24(4):352-57.
 14. Banerjee T, Pensi T, Banerjee D, Grover G. Impact of HAART on survival, weight gain and resting expenditure in HIV infected children in India. *Annals Trop Peds*. 2010;30(1):27-37.
 15. Savakar S, Kachrul S. Effect of institution of HAART in correlation with various anthropometric indices in paediatric HIV patients. *Medical Journal* March 2014; 1(3):110-114.
 16. Lodha R, Upadhyay A, Kapor V, Kabra SF. *Indian J Pediatr*. 2006;73(3):201-204.
 17. Ellis J, Molyneux EM. Experience of anti retroviral treatment for HIV -infected children in Malawi: the 1st 12 months. *Annals Trop Pediatr*. 2013;261-267.
 18. Guillen S, Ramos JT, Resino R, Bellon JM, Munoz MA. Impact on Weight and Height with the Use of HAART in HIV Infected Children. *Pediatric Infect Dis Journal*. 2007;26(4):334-338.
 19. Miller TL, Mawn BE, Orav EJ, Wilk D, Weinberg GA, Nicchitta J et al. The Effect of Protease Inhibitor Therapy on Growth and Body Composition in Human Immunodeficiency Virus Type 1-Infected Children. *Pediatrics*. 2001;107:e77.
 20. Buhacz K, Cervia JS, Lindsay JC, Hughes MD, Seage III GR, Dankner WM, Oleske JM. Impact of protease inhibitor -containing antiretroviral therapies on height and weight growth in HIV infected children. *pediatrics*; 2001;108(4) for Pediatric AIDS Clinical Trial Group 219 study team.
 21. Poudal P, Pokharal R, Chittangia M, Chaudhary S. Profile of HIV infected children: A hospital based study at Eastern Nepal, *Asian Pac Trop Dis*; 4(3):169-75.
 22. Verweel G, Van Rossum AN, Hartwig HJ, de Groot R. Treatment with highly active antiretroviral therapy in human immunodeficiency virus type 1-infected children is associated with a sustained effect on growth. *Pediatrics*. 2002;109(2):E25.
 23. N Kumarswamy, Venkatesh KK, Devaleenol B, Poongulali S, Mothi SN, Solomon S. Safety and Effectiveness of generic HAART in HIV infected children in South India. *Journal of Trop Pediatr*. 2008; 55(3):155-59.

A Comparative Study Based Grading of Malnutrition by WHO Z Score and IAP Classification

Sanjeev Suman¹, Sonal Vyas², Swati Raipurkar³

¹Senior Resident, Department of Pediatrics, K.D. Medical College, Hospital & Research Center, Mathura, Uttar Pradesh 281406, India. ²Professor and Head, Department of Pediatrics ³MD, Department of Pathology, Index Medical College, Hospital & Research Centre, Indore, Madhya Pradesh 452001, India.

Abstract

Serious malnutrition is all around us and yet it is not obvious, for it exhibits the Iceberg phenomenon". By Anthropometric assessment one can detect the sub-clinical malnutrition very easily. Anthropometric measurements obtained in children are usually compared with that of a "reference standard". In developing countries anthropometry despite its inherent limitations still remains the most practical tool for assessing the nutritional status of the children. Biological, epidemiological and statistical evidence suggests that wasting and stunting represent different processes of malnutrition. Low height-for-age (stunting) is a principal indicator of long term growth impairment caused by malnutrition in the past. The present study aims at comparing the prevalence of malnutrition in WHO Z Score classification and IAP classification. *Method:* About 400 children in the age group of 1-5 years were studied for their Anthropometric indices. The anthropometric measurements were performed using the standard WHO anthropometric measuring tools. Standardized methods were used in all measurements and were compared with standard HARVARD classification of IAP and WHO Z Score classification. *Result:* Overall prevalence of malnutrition was found to be 67.5% according to IAP. Among 67.5% malnourished children the prevalence of underweight was more in girls (51.8%) than boys (48.1%). According to WHO classification prevalence of malnutrition was 68.7% of which 27.5% had severe malnutrition. Among 68.7% of underweight children about 25.7% were found to be below -3SD. Among 59.2% of stunted children about 26.5% below -3SD were observed. Among 13.5% of wasted children about 2.6% below -3SD were observed. According to IAP criteria among underweight (67.5%), preschool children, Grade I were 55.9%, Grade II 23.7%, Grade III- 16.2% and Grade IV were 4.07%. *Conclusion:* In the current study it was observed that the Z Score system obtained the result with 1.2% precision as compared to that of IAP classification. Thus it can be concluded that Z Score system is more accurate in early diagnosis of malnutrition.

Keywords: Malnutrition; IAP; Anthropometric; Anemia.

Introduction

Optimal nutrition during early childhood lays a strong foundation for growth and development as well as long term health.

In children normal growth and development are signs of good health and nutrition, one of the best ways to measure child's health is to measure growth. One of the easiest way to do so is to weigh child regularly and to note his body weight increment with age in comparison with standard weight to the healthy children of the same age. In general, the growing child requires a high calorie

intake because of activity and abundant good quality protein and minerals because of their rapid growth.

Deficiency diseases occur when food is not provided in sufficient quality and quantity for growth and development. One means of judging adequacy of diet in childhood is the plotting of height against corresponding weight with reference to age. Such record also is helpful in the clinical evaluation of general health. Since childhood is the period of active growth a well-nourished child can be expected to have a growth pattern characterized by predictable increments in both height and weight. Physical growth has become a readily

Corresponding Author: Sonal Vyas, MD, Department of Pathology, Index Medical College, Hospital & Research Centre, Indore, Madhya Pradesh 452001, India.

E-mail: vyas_sp@rediffmail.com

Received on 02.05.2018, Accepted on 22.05.2018

©Red Flower Publication Pvt.Ltd

available standard to assess the nutritional status. Of all population groups affected by malnutrition, young children need the most attention.

To get a measure of malnutrition in a population, young children can be weighed and their height measured and the results compared to those of a reference population known to have grown well. Measuring weight and height is the most common way of assessing malnutrition in populations [1]. One out of every three children under five in developing countries is malnourished [2]. This unacceptable state of affairs leads to a great deal of human suffering both physically and emotionally. It is a major drain on developing countries' prospect for development because malnourished children require more intense care from their parents and are less physically and intellectually productive as adults [2].

Growth faltering can be detected in children long before any observable signs or symptoms of malnutrition become evident. Growth monitoring can therefore enable early diagnosis of health problems to be made and corrective measures to be taken [3].

Method and Material

The present study was a prospective study conducted for two years at Index medical college hospital and research centre between Jan 2016 to June 2017.

This study was undertaken with the aim of evaluating the nutritional status of malnourished pre-school children (1-5 years) in relation to anthropometric measurements using WHO Z Score classification and IAP Classification in comparison to each other.

About 400 children in the age group of 1-5 years were studied for their Anthropometric indices. All children aged 1-5 years attending indoor outdoor clinic at Index Hospital and of nearby areas of Index hospital were included in the study. Non-consenting children diagnosed with congenital disorders, major illness and non-consenting parents were excluded from the study. The anthropometric measurements were done to assess the nutritional status wherein age was the only criteria used. Age was recorded by interviewing the parents or by the birth record of the child. Growth pattern of children was worked out for boys and girls separately, in respect of different body measurements were compared with international and national standards.

Methodology

Complete nutritional status and clinical status has been assessed using the questionnaire and clinical examination. The anthropometric measurements were performed using the standard WHO anthropometric measuring tools. For Anthropometric measurement Electronic weighing machine, WHO recommended measuring tape and Infantometer / stadiometer were used.

The anthropometric data in present study were compared with the National Growth Data and with the international NCHS standard data. Literacy status of mother was recorded. Nutritional status was graded according to Z-score classification and I.A.P classification. The reference standard used was National Centre of Health Statistics (NCHS) for Z score classification and Harvard Standard for I.A.P. classification. In this study Z-scores were calculated for all three indices, weight-for-age (underweight), height-for-age (stunting) and weight-for-height (wasting) by using NCHS reference standard.

Reference Standards

According to the NCHS/WHO standards Wasting, stunting, and underweight is defined as low weight for height, height for age and weight for age respectively. For reporting of these indices, Z-scores (standard deviation scores) are used. The Z-score or standard deviation unit (SD) is defined as the difference between the value for an individual and the median value of the reference population for the same age or height, divided by the standard deviation of the reference population. This can be written in equation form as: Z-score (or SD-score) = (observed value) - (median reference value) / Standard deviation of reference population 2.10 Cut-Offs.

Cut-off based prevalence for the indicators were used in this study. The use of cut-off enables the different individual measurements to be converted into prevalence statistics. The cut-off used in this study with Z-scores is minus two standard deviations. The cut-off points for WHO classification (Z-Scores) was adopted for this study. Broadly children are considered normal if the Z-Scores are above -2 and malnourished if Z-Scores below -2. Mid upper arm circumference cut-offs are somewhat arbitrary due to its lack of precision as a measure of malnutrition. A cut-off of 11.0 cm is used for severely malnourished children. Those children below 12.5 cm are classified as moderate and severe.

Statistics

The data generated from the study was entered into SPSS, Epi info software and anthropus software. Assessment of the children's nutritional status was done using the Nutritional Anthropometry software (Epi Info 2002 system) from the division of Nutrition, CDC, Atlanta. Univariate, bivariate and multivariate analyses were done to infer from the study data and anthropus software.

Result

The prevalence of underweight and stunting was observed as very high in our study 67.5%, 59.2%, whereas prevalence of wasting was high according to criteria for assessing severity of under nutrition in a population proposed by grostein et al. (1994) and WHO criteria 1995.

In the present study it was observed that there is an increase in prevalence maximally in between 1-2 years both for underweight and wasting whereas for stunting it was found that highest prevalence

Classification	Low (%)	Medium	High	Very High
Under weight	Less than 10	10-19	20-29	>30
Stunting	Less than 20	20-29	30-39	>40
Wasting	Less than 5	5-9	10-14	>15

occurred in 2-4 years of age group. The prevalence of underweight and wasting were higher in girls in comparison to boys (underweight- 52% stunting - 50.8%, wasting - 62.9% for girls and for boys underweight- 47.2% stunting - 49.1%, wasting - 29.6%). According to IAP classification it was observed that 67.5% of preschool children were underweight as per grading- Grade I - 55.9%, II - 23.7%, III - 16.2% and IV - 4.07% were recorded. Age wise prevalence of PEM showed that there was a gradual decrease with age this relationship was significant ($p < 0.05$).

Discussion

In developing countries anthropometry despite its inherent limitations still remains the most practical tool for assessing the nutritional status of the children. Biological, epidemiological and statistical evidence suggests that wasting and stunting represent different processes of malnutrition. Low height-for-age (stunting) is a principal indicator of long term growth

impairment caused by malnutrition in the past. Wasting indicates a deficit in tissues and fatness compared with the amount expected for a child of the same height or length and may result either from weight loss or failure to gain weight. Thus low weight for height commonly used to assess acute or recent malnutrition. Low weight-for-age (underweight) is a combined index that reflects both height for-age and weight-for-age data. The following studies are in support of our observations.

Nutritional status of preschool children residing in Coimbatore slums was assessed using the z-score system of classification and compared the z-score with IAP classification [17]. Seetharaman and co-workers, 2007). A total of 625 children were selected for the study, among those 31.40 per cent were normal, 68.60 per cent were in a state of anthropometric failure. As per the z score system, 49.60 per cent were underweight (21.70% severely), 48.40 per cent were stunted (20.30% severely) and 20.20 per cent were wasted (6.90% severely). Whereas, as per IAP criteria, 51.40 per cent were undernourished and 3.20 percent were severely undernourished.

Vijayashree Mathad (2011) assessed the nutritional status of under-five years of age as a cross sectional study conducted in Kakati-A sub-centre, under the Primary Health Centre at Vantamuri in Belgaum district. The sample size was 290. The prevalence of underweight, stunting and wasting was observed to be 26.55%, 31.38% and 7.59%, while severe degree of underweight, stunting and wasting was observed in 5.86%, 27.24% and 6.51%, respectively, in terms of World Health Organization (WHO) 2006 classification. According to the Indian Academy of Paediatrics (IAP) classification, the prevalence of Grade I malnutrition was 121 (47.10%), Grade II was 29 (10.00%), and Grade III and IV were 4 (1.40%).

Growth in the first year of life is particularly vulnerable to environmental stresses. There is usually delay in starting weaning and if started otherwise with improper feeding. This results in failure to thrive. During first 4-5 months of life the child's requirements are met adequately by breast milk. However after 5 months the breast milk is insufficient and weaning is delayed. This leads to energy deficit in the child's diet.

The prevalence of malnutrition was significantly higher among female children than male children. This observation is also comparable with observations reported by Kapil, U, Bali, P (1989) [4], Luwang WC (1980) [5], Kumar, Rajesh [6], Aggarwal & Iyengar (1996), and Yadav, RJ and Singh, P (1999) [7]. Child rearing for male children is more

careful than female children in our male dominated society. The preferential treatment and feeding of male children over female has been commonly observed in India.

This Table 2 shows prevalence of underweight was significantly higher in girls 52% than boys 47.9% ($p \leq 0.05$). The prevalence of wasting was higher in girls 50.8% as compared to boys 49.1%. $p \leq 0.001$ Prevalence of stunting was similar among boys and girls ($p \leq 0.05$)

This table 3 shows prevalence of malnutrition in different ages according to weight for height criteria. It was observed that the highest prevalence was found

in age group of 1-2. This association was highly significant ($p \leq 0.001$).

This table 4 shows prevalence of malnutrition in different ages according to height for age criteria. It was observed that the highest prevalence was found in age group of 3-4 yrs. This association was highly significant ($p \leq 0.05$).

This table 5 shows prevalence of malnutrition in different ages according to weight for age criteria. It was observed that the highest prevalence was found in age group of 1-2. This association was highly significant ($p \leq 0.001$).

Table 1: Nutritional status of the Pre-school children according to three basic indices: Number of Subjects (%)

Index	Normal	Grade I	Grade II	Total
Weight-for-age	125 (31.2%)	172 (43%)	103 (25.7%)	275 (68.7%)
Height-for-age	164 (41%)	130 (32.7%)	106 (26.5%)	236 (59.2%)
Weight-for-height	346 (86.5%)	44 (10.9%)	10 (2.6%)	54 (13.5%)

Table 2: Malnutrition Prevalence % in Pre-school children in Relation to Sex

Index	Boys			Girls		
	No. (%)	Grade I (%)	Grade II (%)	No. (%)	Grade I (%)	Grade II (%)
Weight for age	132 (47.2%)	82 (29.8%)	50 (18.1%)	143 (52.0%)	75 (27.2%)	68 (24.7%)
Height for age	116 (49.1%)	70 (29.6%)	46 (19.7%)	120 (50.8%)	64 (27.1%)	56 (23.7%)
Weight for Height	20 (37.0%)	16 (29.6%)	04 (7.4%)	64 (62.9%)	28 (51.8%)	06 (11.1%)

Table 3: Malnutrition Prevalence % in Pre-school Children In Relation to Age using Weight for Height (Wasting) criteria

Age in Years	Number	Grade I	Grade II
1 - 2	116	11.2	5.12
2 - 3	128	10.9	1.56
3 - 4	72	11.11	1.38
4 - 5	84	10.71	1.19
All Age	400	11.0	2.5

Table 4: Malnutrition Prevalence % in Pre-school Children In Relation to Age using Height for age (Stunting) criteria

Age in Years	Number	Grade I	Grade II
1 - 2	116	30	24.5
2 - 3	128	32.2	23.1
3 - 4	72	42.8	30.4
4-5	84	32.2	28.9
All age	400	32.7	26.5

Table 5: Malnutrition Prevalence % in Pre-school Children In relation to age using Weight (Underweight) for age criteria

Age In Years	Number	<-2SD Grade I	<-3SD Grade II
1 - 2	116	50.0 %	27.5 %
2 - 3	128	38.2 %	28.9 %
3 - 4	72	41.6 %	25.0 %
4 - 5	84	22.6 %	32.1 %
All Age group	400	43.0 %	25.7 %

Table 6: Nutritional Status as according to IAP Classification

Grades	No. of cases	Percentage in Malnourished cases (%)	Percentage in total
I	151	55.9%	37.7%
II	64	23.7%	16.0%
III	44	16.2%	11.0%
IV	11	4.07%	2.75%

From the observation it was seen like grade 1 malnutrition was most prevalent followed by grade II, III and IV (Table 6).

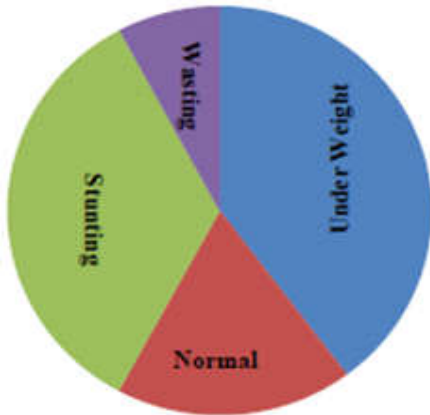


Fig. 1: Pie Chart showing malnutrition according to three basic indices (WHO)

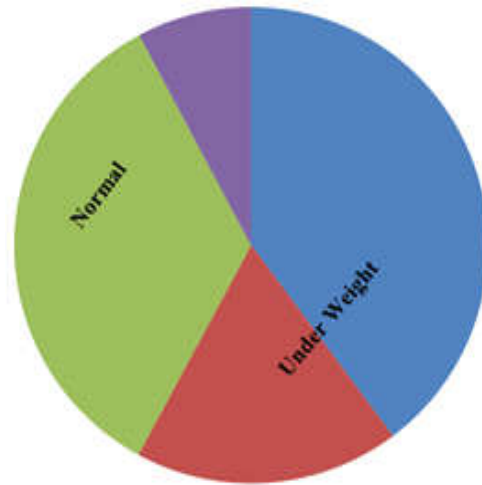


Fig. 3: Pie chart showing prevalence of malnutrition according to WHO

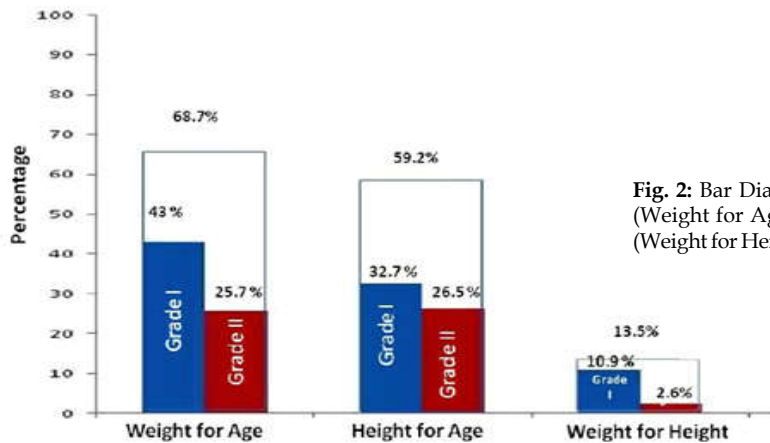


Fig. 2: Bar Diagram Showing Prevalence of Underweight (Weight for Age), Stunting (Height for Age) and Wasting (Weight for Height)

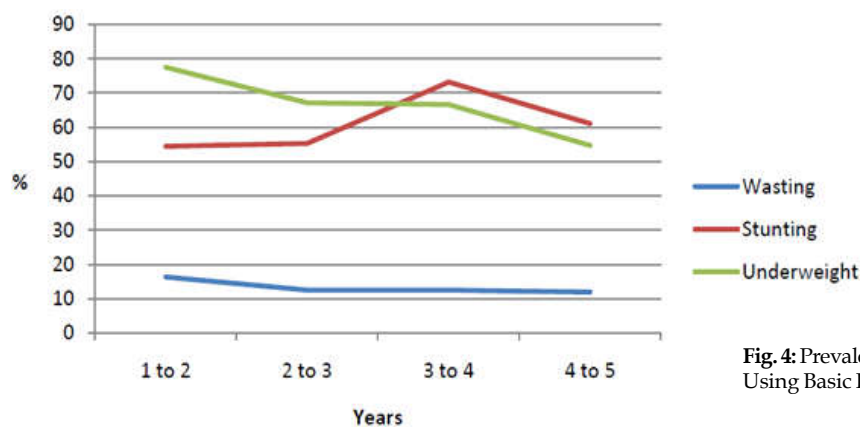


Fig. 4: Prevalence of Malnutrition In Different Age group Using Basic Indices Line Diagram

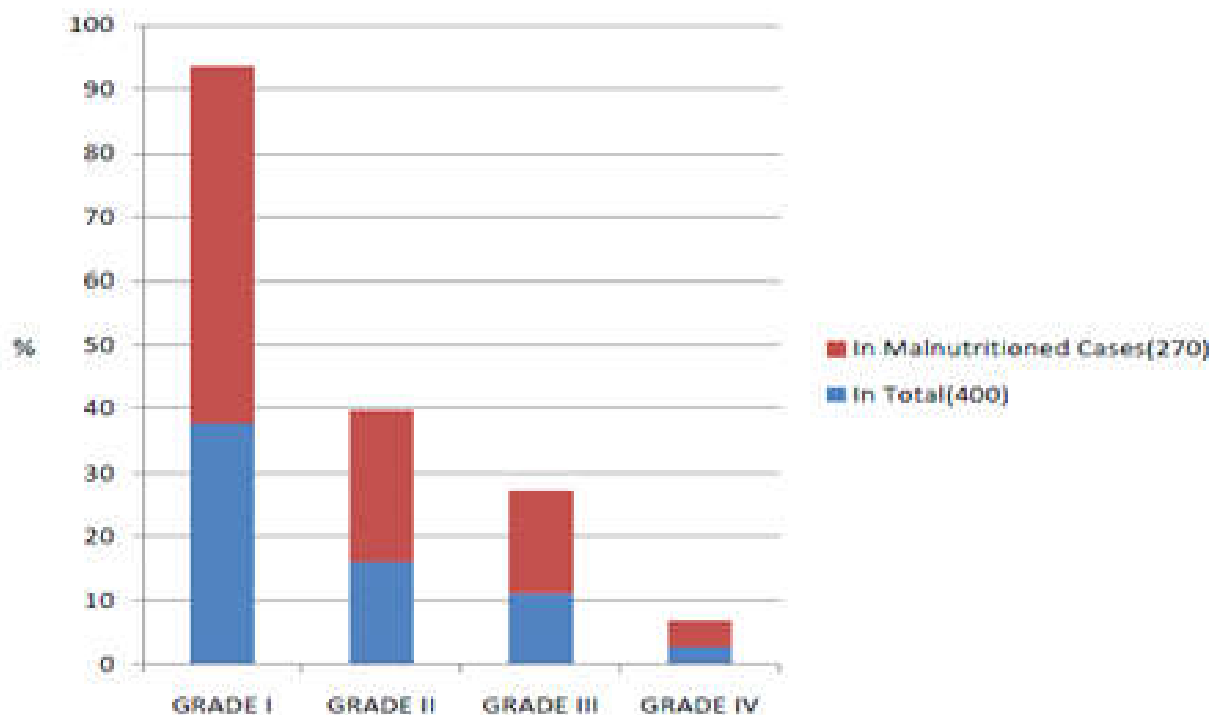


Fig. 5: Bar Diagram Showing Grading of Malnutrition (Using IAP Classification)

Conclusion

To conclude, in the present study high prevalence of the under nutrition and its relation with age, sex among the under 5 children was documented significantly higher and much earlier by the WHO Z Score classification as compared to IAP Classification.

Changing the focus of nutrition from treatment to prevention will definitely take some time but this will provide more value for money spent. Efforts are needed to encourage the concept of preventive nutrition across population segments and not just 'high-risk' groups.

Pediatric healthcare is an apt segment to implement preventive nutrition behaviors because eating preferences are established early in life [8].

The present study is limited by its small sample size being only from one area of India. These results may therefore only be representative of a small community and not representative of the state or country.

The cost impact of measurements as effective screening measures would be phenomenal and this era of economic prudence such potential benefit warrants investment in research in this field.

References

1. UNICEF. The state of the world's children 1998. Oxford university press, UK. 1998.
2. Smith L.C. and Hadad L. Explaining child malnutrition in developing countries: Across-country analysis. International Food Policy Institute. Research Report 111. Washington. 2000.
3. WHO. The growth chart a tool for use in infants and children health cares, WHO. Geneva. 1986.
4. Kapil U, Bali P. Nutritional Status of Preschool Children of urban communities in Delhi, Ind. *Pediatr.* 1989;26:338-42.
5. Luwang WC. Protein energy malnutrition among preschool children in a rural community of Manipur. *Ind. Pediatr.* 1980;17:879-882.
6. Kumar Rajesh, Aggarwal K. Arun and Iyenger D. Sharad, Nutritional status of preschool children, *Ind. Pediatr.* 1996;33:189-96.
7. Yadav R.J. and Singh P. et al, Nutritional status of preschool tribal children of Bihar, *Ind. Pediatr.* 1999;36:37-42.
8. Bendich, A Deckelbaum R.J. Health Economics of Preventive Nutrition. In: Bendich A, Deckelbaum R.J. (eds). Preventive Nutrition, Nutrition and Health. Humana Press. Springer Science and Business Media 2010.
9. Mittal A., Singh J. and Ahluwalia S.K., Effect of maternal factors on nutritional status of 1-5 year old

- children in urban slum population. *Indian J. Comm. Med.*, 2007;32(4):264-268.
10. Nale T., Chavan M. K., Mahajan H. and Mahajan A. Primary care based interventions are associated with improvement in nutritional status of children: evidence from community based study in India. *International J. Sci. Res. Pub.*, 2013;3(2):2250-58.
11. Uppal M., Kumari K. and Sidhu S. Clinical assessment of health and nutritional status of scheduled caste preschool children of Amritsar. *Anthropologist*, 2005;7(3):169-171.
12. Kaur G., Kang H.S., Singal P. and Singh S.P. Nutritional status: Anthropometric perspective of pre-school children. *Anthropologist*, 2005;7(2):99-103.
13. Kapur D., Sharma S. and Agarwal K.N. Dietary intake and growth pattern of children 9-36 months of age in an urban slum in Delhi. *Indian Pediatrics*, 2005;42:351-56.
14. Kumar D., Goel N.K., Mittal P.C. and Misra P. Influence of infant feeding practices on nutritional status of under-five children. *Indian J. Pediatr.*, 2006;73(5):417-21.
15. Seetharaman N., Chacko T.V., Shankar S.L. and Mathew A.C. Measuring malnutrition- the role of Z scores and the composite index of anthropometric failure. *Indian J Comm. Med.*, 2007;1(1):35-39.
16. Luthra M., Kishor S. and Jain K. Epidemiology of under-nutrition in children between 0-5 years from rural areas of Dehradun. *Indian J. Comm. Health*, 2010;21(2):18-21.
17. Vinod N., Swarnakanta L., Smita P. and Pushpa D. Nutritional status and dietary pattern of under five children in urban slum area. *National Journal of Comm. Med.*, 2011;2(1):143-48.
18. Meshram I.I., Laxmaiah A., Reddy G., Ravindranath M., Venkaiah K. and Brahmam G.N. Prevalence of under-nutrition and its correlates among under 3 year-old children in rural areas of Andhra Pradesh, India. *Ann. Human Biol.*, 2011;38(1):93-101.
19. Ahmed E., Khalil S. and Khan Z. Nutritional status in children (1-5 yrs)- a rural study. *Indian J. Comm. Health*, 2011;23(2):84-86.
20. Mathad V., Metgud C. and Mallapur M.D. Nutritional status of under-fives in rural areas of south India. *Indian J. Med. Sci.*, 2011;65(4):151-156.
21. Amosu A. M., Degun A.M., Atulomah N.O. and Olanrewaju M.F. A study of the nutritional status of under-5 children of low income earners in a south-western Nigerian community. *Curr. Res. J. Biol. Sci.*, 2011;3(6):578-85.
22. Singh A.K., Jain S., Bhatnagar M., Singh J.V., Garg S. K., Chopra H. and Bajpai S.K. Socio-demographic determinants of malnutrition among children of 1-6 years of age in rural Meerut. *J. Dental and Med. Sci.*, 2012;3(3):37-40.
23. Bant D.D. Prevalence of protein energy malnutrition among anganwadi children of Hubli, Karnataka. *J. Nut. Res.*, 2013;1(1):11-13.
24. Indu, Kumar N., Kumar A. and Kumari M. Prevalence of malnutrition among preschool children of Khagaria district of Bihar. *Food Sci. Res. J.*, 2013;4(2): 164-66.
25. Lakshmi J. A., Begum K., Saraswati G. and Jamuna P. The prevalence of anemia in Indian rural preschool children. *Indian J. Nutr. Dietet.*, 2001;38:182-90.
26. Singh M.B., Haldiya K.R. and Lakshminarayana J. Morbidity pattern and its association with malnutrition in preschool children in desert areas of Rajasthan. *J. Arid Envi.* 2002;51(3):461-68.
27. Lakshmi J. A., Begum K., Saraswati G. and Jamuna P. Influence of nutrition and environment on morbidity profile of Indian preschool children. *Mal. J. Nutr.*, 2005;11(2):121-32.
28. Vaid S. and Vaid N. Nutritional status of ICDS and non-ICDS children. *J. Hum. Ecol.*, 2005;18(3):207-12.
29. Mitra M., Sahu P. K., Chakrabarty S., Bharati S. and Bharati P. Nutritional and health status of Gond and Kavar tribal preschool children of Chattisgarh, India. *J. Hum. Ecol.*, 2007;21(4):293-99.
30. Prabhakar S.C. and Gangadhar M.R. Nutritional status of jenukuruba tribal children in Mysore district, Karnataka. *Anthropologist*, 2009;11(2):83-88.
31. Bhavsar S., Hemant M. and Kulkarni R. Maternal and environmental factors affecting the nutritional status of children in Mumbai urban slum. *IJSR*, 2012;2(11):1-9.
32. Patro S. and Sahu R. Clinical profile of tribal children of Koraput district of Odisha. *Asian J. Home Sci.*, 2012;7(1):55-58.
33. Kubde, S.R. and Kokiwar, P.R. A comparative study of morbidity pattern among children of 0- 6 years in ICDS and non ICDS area. *J. Health Sci.*, 2013;1(1):30-33.
34. Bansal, R. Health profile of underfive migrant tribal children. *Ind. Med. Gazette*, 1992;16(5):137-38.
35. Pius C Manyike, Josephat M Chinawa, Agozie Ubesie, Herbert A Obu, Odutola I Odetunde, and Awoere T Chinawa. Prevalence of malnutrition among pre-school children in, South-east Nigeria, *Ital J. Pediatr.* 2014;40:75-77.
36. I.V. Mamatha, N. Konda Reddy. Nutritional Status of Pre-School Children Attending Anganwadi Centres in Tirupati, Andhra Pradesh, India, *IOSR Journal of Nursing and Health Science (IOSR-JNHS)*, 2015;4(5):139-43.
37. Priyanka R., Vivin Vincent, Jini M.P., C.R. Saj. An assessment of the nutritional status of underfive children in a rural area of Thrissur district, Kerala, India, 2016;3(12):235-40.
38. Vijayaraghavan K. Anthropometry for assessment of nutritional status. *Indian J. Pediatr* 1987;54(4): 511-20.
39. Currimbhoy Z. Growth and development of Bombay children. *Indian d. Child Hlth.* 1963;12:626.

40. Udani, P.M. Physical growth of children in different socio-economic groups in Bombay. Indian Journal of Child Health. 1963;12:593-61.
 41. Shah PM, Udani PM. Medical examination of rural school children in Palghar Taluk. Indian Pediatr 1968;5:343-61.
 42. Gangadharan M. School health service programme in Kerala, a rural study. Ind Pediatr, 1977;14:603-13.
 43. Rath B. Ghosh G., Manmohan et al. Anthropometric indices of children (5 to 15 years) of a privileged community. Indian Pediatrics. 1978;15:653-665.
-

Clinical Predictors of Metabolic Acidosis in Hospitalized Children of 6-60 Months with Severe Acute Malnutrition

Saurabh Kumar Patel¹, Sunil Kumar Rao², Naresh Bajaj³, Tej Bali Singh⁴

¹Assistant Professor ³Professor, Department of Pediatrics, Shyam Shah Medical College, Rewa, Madhya Pradesh 486001, India. ²Associate Professor, Department of Pediatric, ⁴Professor, Division of Biostatistics, Department of Community Medicine, Institute of Medical Science, Banaras Hindu University, Varanasi, Uttar Pradesh 221005, India.

Abstract

Context: Malnourished children are at increased risk of many common infections and their complications. In those hospitalized with Severe Acute Malnutrition (SAM), concomitant infections and diarrhoea are frequent complications resulting in adverse outcome. Metabolic acidosis is a consequence of dehydration in diarrhoea, and the diagnosis of dehydration is based on history of fluid loss. Present study carried out to find the predictors of metabolic acidosis among children hospitalized with SAM. *Aims:* To study the predictors of metabolic acidosis among children hospitalized with severe acute malnutrition. *Settings and Design:* Department of Paediatrics Shyam Shah Medical College Rewa, Prospective cross sectional study. *Methods and Material:* Children between 6-60 months fulfilling the WHO criteria of SAM were enrolled. Variables recorded were sociodemographic, anthropometry, history, clinical sign and laboratory results. Study population was grouped in children with (case) and without metabolic acidosis as (control). WHO guidelines for management of SAM was used to treat the morbidities. *Statistical analysis used:* Data was analyzed by using SPSS 16, variables were initially analyzed in univariate model, and then independently associated factors with metabolic acidosis were identify using logistic regression analysis. A probability of less than 0.05 was considered statistically significant ($p < 0.05$). *Results:* Out of total 202 children, 91 (45%) children developed metabolic acidosis, 110 (54%) out of 202 children were presented with either acute watery diarrhoea or vomiting or both; out of which 69 (62%) developed metabolic acidosis and association of fluid loss was significant ($p < 0.001$). Septic shock, watery diarrhoea and vomiting were independently associated with metabolic acidosis in SAM children. *Conclusions:* History of fluid loss either in form of loose motion or loose motion with vomiting and septic shock were independently predict metabolic acidosis in SAM children.

Keywords: Metabolic Acidosis; Severe Acute Malnutrition; Dehydration; Shock.

Introduction

Malnourished children are at increased risk of many common infections and their complications. In those hospitalized with SAM, concomitant infections and diarrhoea are frequent complications resulting in adverse outcome. Metabolic acidosis is a consequence of dehydration in childhood diarrhoea [1], it may be because of severe sepsis and/or pneumonia without dehydration [2].

Faecal loss of bicarbonates in dehydrating diarrhoea [3] and anaerobic cellular respiration in sepsis [4] are responsible for development of

metabolic acidosis. The clinical diagnosis of dehydration in severe acute malnutrition (SAM) is based on definitive history of diarrhoea or vomiting and history of a recent change in eye status [5] Therefore SAM children with definitive history of fluid loss could have dehydration and severe dehydration is diagnosed as history of fluid loss and sign of poor perfusion (cool extremities, prolonged capillary refill and weak and fast pulse for age cut off). However, there is knowledge gap about the correlation between history of fluid loss and development of metabolic acidosis in SAM. So we did this study to identify predictors of metabolic acidosis among children hospitalized with SAM.

Corresponding Author: Sunil Kumar Rao, Associate Professor, Department of Pediatrics, Institute of Medical Science, Banaras Hindu University, Varanasi, Uttar Pradesh 221005, India.
E-mail: drsunilrao21@gmail.com

Received on 03.08.2018, **Accepted on** 31.08.2018

Materials and Methods

This is a prospective cross sectional study on SAM children recruited from august 2012 to July 2013 over one year period at severe malnutrition treatment unit (SMTU) of Medical college located in Central India. The inclusion criteria were children between 6-60 months of age fulfilling WHO criteria for SAM [6]. The exclusion criteria were children with congenital malformations, cerebral palsy and inborn error of metabolism. The ethical committee of the college approved the study and informed consent was obtained from the parents of all patients before the study. Detailed history, clinical examination, sociodemographic variables, anthropometry, laboratory results and arterial blood gas results were recorded in predesigned case report forms. Metabolic acidosis was defined as $\text{pH} < 7.35$ with $\text{HCO}_3^- < 22 \text{ mmol/l}$ in blood gas analysis. Laboratory test results such as severe anaemia ($\text{Hb} < 4 \text{ gm/dl}$), hypokalemia (serum $\text{K} < 3.5 \text{ mmol/l}$), hyperkalemia (serum $\text{K} > 5.5 \text{ mmol/l}$), hyponatremia (serum $\text{Na} < 130 \text{ mmol/l}$) and hypernatremia (Serum $\text{Na} > 146 \text{ mmol/l}$) were used to diagnosed severe anaemia and electrolyte imbalance respectively. Diarrhoea was defined as the passage of three or more abnormally loose or watery stools in the previous 24 hours [7]. Pneumonia was diagnosed based on radiological evidence of consolidation or patchy opacities [8] and clinical criteria defined by WHO [9]. Severe dehydration was differentiated from septic

shock based on clinical features response to administration of 15 ml/kg body weight fluid over an hour [10] and respond to fluid. WHO guidelines for the management of SAM [10] was use to treat the morbidities.

All data were entered into excel sheet and analyzed by using SPSS 16. Comparison was made between SAM with (case) and without metabolic acidosis (control). Differences in proportion were compared by the χ^2 or Fisher Exact test when applicable and difference of means were compared by Student t test. A probability of less than 0.05 was considered statistically significant ($p < 0.05$). Strength of association was determined by a calculating odds ratio and its 95% confidence interval (95% CI). Variables were initially analyzed in univariate model, and then independently associated factors with metabolic acidosis were identify using logistic regression analysis.

Results

Out of total 202 children, 91 (45%) children developed metabolic acidosis. SAM children with metabolic acidosis more often presented with dehydration, septic shock, acute watery diarrhoea and vomiting and its association was significant ($p < 0.05$) (Table 1). The distribution of sex, age, severe stunting, severe wasting, oedema, dyselectrolymia and MUAC $< 11.5 \text{ cm}$ were not different between the

Table 1: Characteristic of SAM children between 6-60 months of age with(case) and without metabolic acidosis (control)

Variables	Metabolic Acidosis(n=91)	Non Metabolic Acidosis(n=111)	OR	95% CI	P value
Age(months) Median IQR	10(8,18)	10(8,16)			0.335
Male Sex	45	59	1.160	0.67-2.020	0.6
Severe Stunting $< 85\%$	89	109	1.011	0.424-2.410	0.98
Severe wasting $< 3 \text{ SD}$	84	95	2.380	0.89-6.366	0.084
MUAC $< 115 \text{ mm}$	81	98	0.98	0.48-1.98	0.95
Fever	66	86	1.303	0.68-2.47	0.42
Cough	12	32	2.66	1.28-5.551	0.009
Rapid Breathing	8	25	3.051	1.30-7.151	0.01
Lethargic	5	16	2.93	1.03-8.33	0.04
Watery diarrhoea	26	16	5.170	2.35-11.344	< 0.001
Vomiting	5	6	2.652	0.737-9.535	0.135
Diarrhoea & Vomiting	38	19	6.364	3.067-13.204	< 0.001
Some Dehydration	29	25	2.96	1.491-5.877	0.002
Severe Dehydration	33	12	7.017	3.191-15.43	< 0.001
Hypothermia	3	12	3.56	0.972-13.12	0.05
Oedema	21	32	1.350	0.714-2.554	0.36
Septic shock	23	8	4.355	1.84-10.29	0.001
Pneumonia	16	31	1.816	0.086-3.588	0.08
Severe Anaemia	6	14	0.379	0.125-1.149	0.08
Moderate Anaemia	59	74	0.705	0.36-1.36	0.298
Hypokalemia	42	44	1.37	0.0778-2.25	0.99
Hyponatremia	12	15	0.995	0.439-2.2588	0.08

Table 2: Clinical predictors for metabolic acidosis in children with SAM

Variables	OR	95%CI	P Value
Rapid Breathing	0.473	0.153-1.457	0.192
Lethargic	0.765	0.226-2.594	0.76
Cough	1.180	0.439-3.172	0.74
Acute Watery diarrhoea	6.114	1.324-27.85	0.019
Vomiting	3.852	0.788-18.835	0.09
Diarrhoea & Vomiting	6.924	1.398-34.303	0.018
Some Dehydration	0.639	0.162-2.510	0.52
Severe Dehydration	1.599	0.375-6.814	0.526
Septic shock	7.139	2.691-18.934	<0.001

Table 3: Predictors for metabolic acidosis in SAM children with diarrhea and vomiting

Variables	OR	95%CI	P value
Age 6-12 months	2.954	1.134-7.69	0.02
Hypothermia	6.867	1.232-38.262	0.02

groups (Table 1). In logistic regression analysis, SAM children with metabolic acidosis were independently associated with septic shock (severe sepsis), acute watery diarrhoea and vomiting (Table 2). Out of 202 children, 110 (54%) children were presented with either acute watery diarrhoea or vomiting or both; out of which 69 (62%) developed metabolic acidosis, which was significant ($p < 0.001$) with odd ratio 5.355 and 95%CI (2.894-9.910). In children with diarrhoea or vomiting or both, age group of 6-12 months ($p < 0.03$) and hypothermia ($p < 0.02$) had significant independent association with development of metabolic acidosis (Table 3).

Discussion

Diarrhoea still plays key role in both morbidity and death among under-5 children and accounts for 9% of global under-5 deaths in 2015 [11]. The observation of this study was independent association of septic shock and history of fluid loss with metabolic acidosis. Either acute watery diarrhoea or vomiting or both were risk factors for metabolic acidosis in SAM. Age between 6 to 12 months and hypothermia were two independent risk factors for development of acidosis in children with SAM who had presented with history of fluid loss. Children with diarrhoea developed metabolic acidosis due to loss of bicarbonates in faeces [3] similar observation was found by Chisti MJ et al. [12]. Vasodilatation and capillary leakage in septic shock leads to microcirculation derangement as results by product of anaerobic cellular respiration produces lactate which is responsible for acidosis [13,14], similar observation was found by Sharifuzzaman et al [15].

Our observation of younger age (6-12 months) among diarrheal children with SAM and metabolic acidosis compared to those without metabolic acidosis can be explained as dehydration at younger age has severe presentation. Hypothermia is one of the clinical sign of sepsis in SAM, therefore its association with acidosis is self explanatory. WHO guideline recommends definite history of fluid loss as evidence of dehydration, but in this present study we found that definite history of fluid loss is also a predictor of metabolic acidosis in SAM and this is the strength of our study. The limitation of present study was that we only analyzed the children at time of admission and not after their stabilization. So could not find weather reductive adaptation in SAM is contributing to the acid base imbalance. Also Clinical features of metabolic acidosis and pneumonia frequently overlap in young diarrheal children, resulting in differentiation from each other very difficult.

Conclusion

Severe acute malnutrition increases 9 fold chance of mortality, so it necessary to identified complications in SAM. This present work emphasises that recent history of fluid loss and clinical sign of shock independently predict metabolic acidosis in SAM.

Acknowledgement

Nil

Conflict of Interest: Nil

Key Messages

History of fluid loss either in form of loose motion or loose motion with vomiting and septic shock were independently predict metabolic acidosis in SAM children.

References

1. Okposio MM, Onyiriuka AN, Abhulimhen-Iyoha BI. Point-of-admission serum electrolyte profile of children less than five years old with dehydration due to acute diarrhoea. *Trop Med Health*. 2015;43: 247-252.
2. Chisti MJ, Salam MA, Bardhan PK, et al. Severe sepsis in severely malnourished young Bangladeshi children with pneumonia: a retrospective case control study. *PLoS One*. 2015;10:e0139966.
3. Richards L, Claeson M, Pierce NF. Management of acute diarrhoea in children: lessons learned. *Pediatr Infect Dis J*. 1993;12:5-9.
4. Chisti MJ, Duke T, Robertson CF, Ahmed T, Faruque AS, et al. Clinical predictors and outcome of hypoxaemia among under-five diarrhoeal children with or without pneumonia in an urban hospital, Dhaka, Bangladesh. *Trop Med Int Health*. 2012.
5. Facility based management of severe acute malnutrition (SAM) in children: reference manual for medical and para medical staff of NRC Madhya Pradesh. 2011.
6. WHO child growth standards and the identification of severe acute malnutrition in infants and children: A Joint Statement by the World Health Organization and the United Nations Children's Fund: WHO, Unicef.
7. Chisti MJ, Ahmed T, Bardhan PK, Salam MA. Evaluation of simple laboratory investigations to predict fatal outcome in infants with severe malnutrition presenting in an urban diarrhoea treatment centre in Bangladesh. *Trop Med Int Health*. 2010;15:1322-25.
8. Chisti MJ, Ahmed T, Faruque AS, Abdus Salam M. Clinical and laboratory features of radiologic pneumonia in severely malnourished infants attending an urban diarrhoea treatment centre in Bangladesh. *Pediatr Infect Dis J*. 2010;29:174-77.
9. Chisti MJ, Tebruegge M, La Vincente S, Graham SM, Duke T. Pneumonia in severely malnourished children in developing countries – mortality risk, aetiology and validity of WHO clinical signs: a systematic review. *Trop Med Int Health*. 2009;14:1173-89.
10. Ashworth, A., Jackson, A., Khanum, S., Schofield, C. Ten steps to recovery. *Child Health Dialogue* 1996; 10-12.
11. UNICEF. Committing to child survival: a promise renewed. Progress report 2015. http://www.apromiserenewed.org/wp-content/uploads/2015/09/APR_2015_8_Sep_15.pdf. Published September 2015.
12. Chisti MJ, Ahmed T, Ashraf H, et al. Clinical predictors and outcome of metabolic acidosis in under-five children admitted to an urban hospital in Bangladesh with diarrhoea and pneumonia. *PloS One*. 2012;7:e39164
13. Ebrahim GJ. Sepsis, septic shock and the systemic inflammatory response syndrome. *J Trop Pediatr*. 2011;57:77-79.
14. Sarmin M, Ahmed T, Bardhan PK, Chisti MJ. Specialist hospital study shows that septic shock and drowsiness predict mortality in children under five with diarrhoea. *Acta Paediatr*. 2014;103:e306-e311.
15. Sharifuzzaman, Sarmin M, Ahmed T, et al. Determinants and Outcome of Metabolic Acidosis in Diarrheal Children Under 5 Years of Age in an Urban Critical Care Ward in Bangladesh. *Global Pediatric Health*. 2017;4.

Role of Phototherapy in Neonatal Hyperbilirubinemia

Amar Taksande¹, Rewat Meshram², Begam Rubia³, Akshi Gandhi⁴

¹Professor ²Associate Professor ^{3,4}Resident, Department of Pediatrics, Jawaharlal Nehru Medical College, Sawangi Meghe, Wardha, Maharashtra 442102, India.

Abstract

Phototherapy is a simple treatment for neonatal hyperbilirubinemia. During phototherapy, the bilirubin in the neonatal body is changed into another form that can be easily excreted in the stool and urine. Phototherapy devices include halogen lamp, fluorescent, light emitting diode light sources. It is important that phototherapy is applied correctly to combat increased bilirubin production. Light in the wavelength range of 450–475 nm (blue) is the most effective because it overlaps the peak absorption spectrum of bilirubin. The effectiveness of phototherapy is dependent upon four major factors: Color of the light, Intensity of the light, Exposed body surface area and Duration of exposure. It is a safe and easily available treatment worldwide and the side effects of phototherapy are discussed.

Keywords: Phototherapy; Hyperbilirubinemia; Neonate; Irradiance.

Introduction

Phototherapy is considered to be a safe and effective method for treatment of neonatal unconjugated hyperbilirubinemia [1]. Neonatal jaundice is a common condition in newborn babies, affecting about 50% of term and 80% of preterm babies in the first week of life. In physiological jaundice bilirubin levels do not increase to a level that requires treatment. However, in exaggerated physiological jaundice, and infants with pathological jaundice, bilirubin in the blood reaches very high levels that put the infant at risk for acute encephalopathy. The effective treatments to decrease serum bilirubin levels include phototherapy and exchange transfusion [2]. The effect and the ability of light to decrease serum bilirubin levels, was first described by Cremer et al. in 1958 [3]. This observation led to the development of phototherapy for use in the treatment of neonates with hyperbilirubinemia. The exchange transfusions are used as a rescue therapy to avoid kernicterus in newborns with severe jaundice when phototherapy is inadequate [4].

Mechanism of Action

The basic mechanism of action of phototherapy is the use of phototherapy to transform bilirubin into more hydrosoluble products that can be excreted by the body. When bilirubin absorbs light, three types of photochemical reactions occur [3-5].

1. *Photoisomerization* occurs in the extravascular space of the skin. The natural isomer of unconjugated bilirubin (UCB) (4Z,15Z) is rapidly converted to a less toxic polar isomer (4Z,15E) that diffuses into the blood and is excreted into the bile without conjugation. However, excretion is slow, and the photoisomer is readily converted back to UCB, which is resorbed from the gut if the baby is not having stools. Configurational isomerization is reversible. The configurational isomers of bilirubin are less lipophilic than normal bilirubin and can be excreted into bile without undergoing glucuronidation in the liver. Some of the configurational isomers of bilirubin, however, revert back to the native form after excretion into bile and can be reabsorbed via enterohepatic circulation in the gut. After

Corresponding Author: Amar M. Taksande, Professor, Department of Pediatrics, Jawaharlal Nehru Medical College, Sawangi Meghe, Wardha, Maharashtra 442102, India.
E-mail: amar.taksande@gmail.com

Received on 17.02.2018, **Accepted on** 02.04.2018

approximately 12 hours of phototherapy, the photoisomers make up approximately 20% of total bilirubin. Standard tests do not distinguish between naturally occurring bilirubin and the photoisomer, so bilirubin levels may not change much although the phototherapy has made the bilirubin present less toxic.

2. *Structural isomerization* is the intramolecular cyclization of bilirubin to lumirubin. Lumirubin makes up 2% to 6% of serum concentration of bilirubin during phototherapy and is rapidly excreted in the bile and urine without conjugation. Unlike photoisomerization, the conversion of bilirubin to lumirubin is irreversible, and it cannot be reabsorbed. Structural bilirubin isomers, like Z-lumirubin, can also be excreted in the urine.
3. The slow process of photo-oxidation converts bilirubin to small polar products that are excreted in the urine. The absorptions of light by bilirubin also results in the generation of excited-state bilirubin molecules that react with

oxygen to produce colorless oxidation products, or photooxidation products. It is the least important reaction for lowering bilirubin levels. This process occurs more slowly than configurational or structural isomerization. Photooxidation products are primarily excreted in the urine.

Indications for Phototherapy

The indication of phototherapy is only in the treatment of unconjugated hyperbilirubinemia although it has not reached levels requiring exchange transfusion. If indirect bilirubin level is high in the presence of conjugated hyperbilirubinemia, exchange transfusion is the indication. Prophylactic phototherapy may be indicated in special circumstances, such as with extremely low birth weight babies, when the TSB is anticipated to increase rapidly. A commonly used rule of thumb in the Special Neonatal Care Unit (SNCU) is to start phototherapy when the total serum bilirubin level is greater than 5 times the birth weight [3,6].

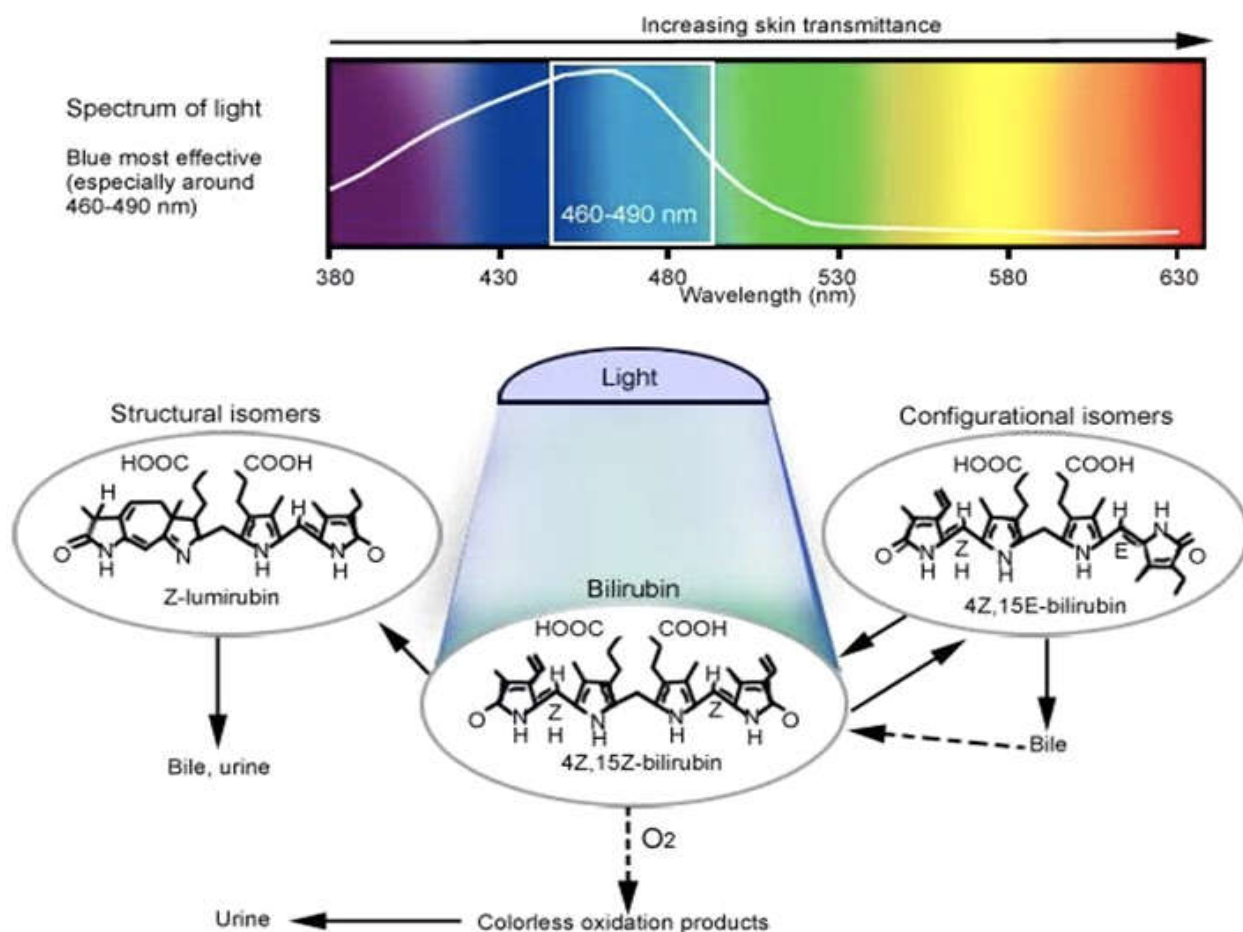


Fig. 1: Shows the Mechanism of phototherapy

Contraindications for Phototherapy

The concomitant use of photosensitizing medications, congenital erythropoietic porphyria, or a family history of porphyria are contraindications to phototherapy. It is usually contraindicated in infants with direct hyperbilirubinemia caused by liver disease or obstructive jaundice. Because it may lead to the "bronze baby" syndrome [7].

Ceasing Phototherapy

Phototherapy should be given until serum bilirubin comes down to safe level i.e. below 10 mg/dl. Rechecking the bilirubin level after cessation of phototherapy is not usually required unless increased risk of significant rebound is there as in haemolytic disease. In these circumstances, a bilirubin level is to be checked 12 hours after cessation of phototherapy [5-8].

The Effectiveness of Phototherapy is dependent upon four major factors: color of the light, intensity

of the light, exposed body surface area and duration of exposure.

a. Color & Wavelength of the Light

Color of the Light: The human eye is sensitive to light which lies in a very small region of the electromagnetic spectrum labeled "visible light". This "visible light" corresponds to a wavelength range of 400-700 (nm) and the visible colors from shortest to longest wavelength are: violet, blue, green, yellow, orange, and red; e.g. blue and green lights have a wavelength of about 475 nm (450-495 nm) and 510 nm (495-570 nm).

- *The white light* is a mixture of the colors of the visible spectrum.
- *Blue light* in the narrow wavelength band of 450-475 nm is the most closely matched and hence the most effective type of light in degrading bilirubin [6,7].

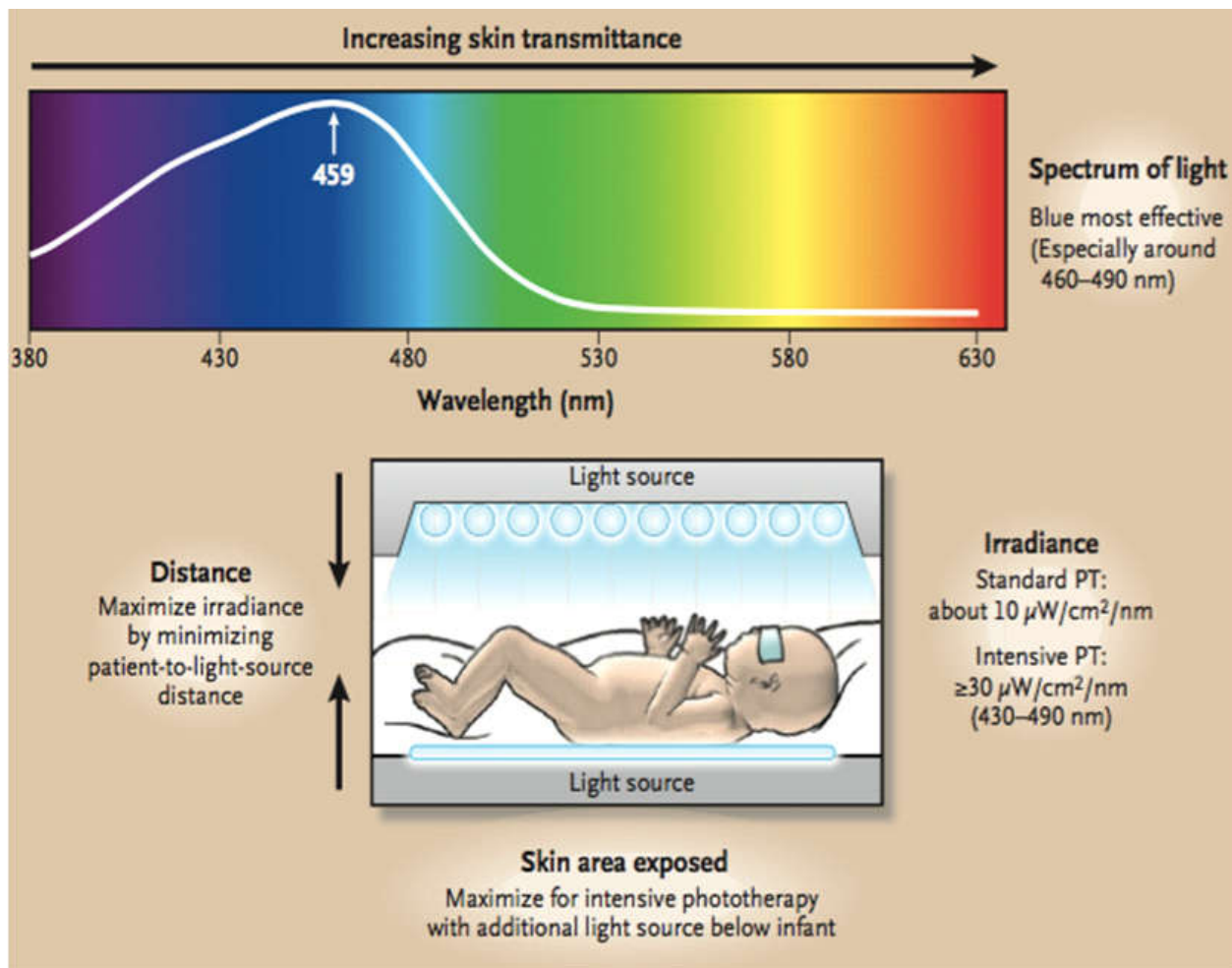


Fig. 2: Shows the effectiveness of phototherapy

Turquoise (blue-green) light has longer wavelengths than blue light. Deeper penetration into the skin, and greater production of E,Z-bilirubin and lumirubin, in infants under turquoise light has a greater bilirubin reducing effect than blue light with equal irradiance.

Light Wavelengths: Bilirubin is a yellow pigment absorbs visible light with wavelengths of approximately 400 to 500 nm, the most effective lights for phototherapy are those with high energy output near the maximum absorption peak of bilirubin (450–460 nm).

Special blue lamps with a peak output at 425 to 475 nm are the most efficient for phototherapy. Blue fluorescent lamps have been used worldwide with peak emission at 452 nm providing more photoisomerization than green light or daylight

Cool white lamps with a principal peak at 550 to 600 nm and a range of 380 to 700 nm are usually adequate for treatment.

The most effective light in vivo is probably in the blue to green region (460-490nm). A turquoise fluorescent source with peak intensity near 490 nm is significantly more effective than blue or green fluorescent lamps in reducing the duration of phototherapy [7-13].

b. Light Irradiance

Light intensity or energy output is defined by irradiance and refer to the number of photons (spectral energy) that are delivered per unit area (cm²) of exposed skin. The dose of phototherapy or "irradiance" which determines the effectiveness of treatment. It is reported in watts per square meter (irradiance) or in microwatts per square cm per nm ($\mu\text{W}/\text{cm}^2/\text{nm}$) over a certain wavelength band (spectral irradiance). The higher the irradiance the larger the rate of serum total bilirubin (STB) decline. There may be a saturation point at 30 mW/cm²/nm where an increase in irradiance has no benefit in decreasing STB levels. Therefore phototherapy devices should be used to deliver at least 30 mW/cm²/nm. The flux or irradiance must be checked once a week with radiometer or spectroradiometer and tubes should be replaced when their flux falls below 15mW/cm²/nm. Conventional daylight phototherapy lamps deliver an irradiance of approximately 8-10 $\mu\text{W}/\text{cm}^2/\text{nm}$. Special blue fluorescent lamps irradiance of 30-40 $\mu\text{W}/\text{cm}^2/\text{nm}$. The irradiance of different phototherapy devices varies widely and is dependent on the number and distance of the light source from the neonate [14-15].

c. Exposed Surface Area

Effective treatment for hyperbilirubinemia is dependent on exposing the infant's surface area to phototherapy as possible. The greater the area exposed, the greater the efficacy of phototherapy. When using spotlights, multiple devices may be necessary to ensure proper surface area coverage. When using banks of lights, caregivers must ensure that the intensity delivered to the entire surface area is within the effective intensity range.

d. Duration

More effective phototherapy treatments will degrade bilirubin to safe levels faster resulting in shorter treatment times, especially with dangerously high bilirubin levels. During phototherapy, neonate's temperature will be monitored to ensure they are not getting too hot and they will be checked for signs of dehydration. Neonate's need to have intravenous fluids if they are becoming dehydrated and are not able to drink a sufficient amount. The bilirubin levels will be tested every four to six hours after phototherapy has started. Once levels start to fall, they will be checked every 6 to 12 hours. Phototherapy will be stopped when the bilirubin level falls to a safe level, which usually takes a day or two. The longer an infant is exposed to the phototherapy light, will increase the effectiveness of the treatment [14-18].

Administering Phototherapy

The room temperature is optimum to prevent hypothermia or hyperthermia. Neonates should be nursed naked apart from a nappy under phototherapy. Place the neonate below the phototherapy unit. The distance from the phototherapy units to the infants is 30-45 cm because the heat formation from the fluorescent tubes risked overheating the infants at reduced distance. Expose as much of the skin surface as possible to the phototherapy light. Check flux with help of fluxmeter. Ideal 6-8 mw/cm²/nm. Cover the eyes with appropriate opaque eye covers. Phototherapy is switched on. Baby is turned every two hours or after each feed. Ensure optimum breastfeeding. Baby can be taken out for breastfeeding and the eye patch can be removed for better mother-infant interaction. Monitor vital signs and temperature at least 4 hourly, more often if needed. Ensure that phototherapy unit is turned off during collection of blood for TSB levels, as both conjugated and unconjugated bilirubin are photo-oxidized when exposed to white or

ultraviolet light. Observe for signs of potential side effects. Serum bilirubin is monitored at least every 12 hours. Phototherapy is discontinued if two serum bilirubin values are < 10 mg/dl. Rebound bilirubin is measured 6-8 hours after stopping phototherapy [2-7].

Phototherapy Devices & Light Source

In the management of neonatal jaundice, different phototherapy devices are being used worldwide. Two types of phototherapy devices are currently available: Conventional phototherapy device and Fiberoptic phototherapy device.

A. Conventional phototherapy devices

The common light sources used in these devices are conventional long Fluorescent-tube devices, halogen lamps with wide emission spectrum, Light-emitting diodes (LEDs) lights.

1. Conventional long Fluorescent-tube devices are the most common type of light source used. These tubes have the advantage of being inexpensive but their light intensity and irradiance reduces with time and needs to change after 1,000-1,500 hours.
2. Halogen bulbs / Spotlight Spotlight phototherapy units generally use a 150 Watt, 21V halogen bulb with a specially coated reflector which absorbs infrared wave length. A fan continuously cools the hot bulb. Options for varying aperture diameter and different filters are available. Positioning of the light on the baby is critically important in maximizing the spotlight's effectiveness. They are most effective when located directly above the infant at a distance of 45-50cm. A few halogen spotlights incorporate a dosimeter which depicts how much dose of phototherapy the baby has received. It considers the total irradiance received by the baby and multiplies this by the duration in hours.
3. Compact Fluorescent Tubes These are short (approx. 5 to 7 inch) double folded tubes (9-18 Watts) that emit blue or white light. Several tubes [6-8] are housed in a panel with reflectors. As they do not produce much heat the distance to baby can be relatively short thus increasing the irradiance delivered. Most of them produce an irradiance of 20-30 W/cm²/nm when placed close to the baby.
4. Light-emitting diodes (LEDs) a special type of semiconductor diode which emits light when connected to an electrical circuit. The light

produced is of narrower bandwidth, and the colour depends on the semiconductor utilized. LED devices usually contain indium or gallium nitrate or nitride as semiconductor element. Such light sources emit high-intensity light while generating little heat, and can be placed closer to the infant, increasing spectral irradiance. It does not appears to have a significant effect on transepidermal water loss. It has useful features such as light weight, compact size, non-fragile and an ability to be focused with a lens or through spatial orientation, in addition lasting durability without decreasing in intensity with age (at least 3,000 hours). LED phototherapy might be clinically more effective than conventional phototherapy with blue-white or green fluorescent tubes as judged by the production of lumirubin in vitro studies [15-19].

B. Fiberoptic Phototherapy Devices / Fiber-Optic Pads

In Fiberoptic phototherapy, use a standard light source, usually a quartz halogen bulb. The light from the bulb passes through a fiberoptic bundle into a pad of woven optic fibers. The pad can then be placed next to the neonate's skin. Thus, infants under fiberoptic phototherapy can be nursed close to their parents without mother-infant separation. It is a safe alternative to conventional phototherapy although it has a low spectral irradiance and a lower spectral power, as it irradiates a minor body surface. In recent models, the halogen light source has been replaced by high intensity high power LED bulbs. This increases the irradiance delivered by the pads [20].

Filtered Sunlight

Using direct sunlight for phototherapy has a number of clinical and practical drawbacks that could make its use undesirable. Sunlight contains harmful ultraviolet A, B, and C radiation, which can cause a serious and permanent damage to human skin. It also contains significant levels of warming infrared radiation, which, in the absence of sufficient cooling, could raise core body temperatures to unsafe levels. It must be underlined that the use of sunlight, when filtered to exclude the harmful spectral radiation, is a novel, practical, and inexpensive method of phototherapy that potentially offers safe and efficacious treatment strategy for management of neonatal jaundice in tropical countries where conventional phototherapy treatment is not available [21].

Double or Triple Phototherapy

Despite significantly higher irradiance in the double (fiber optic plus conventional, or both conventional) or triple phototherapy, there is no statistically significant differences in the treatment [22-23]. Although STB values decrease significantly more slowly in infants who received single phototherapy than the double or triple phototherapy, the actual difference in 0-4 h decrease is small [24].

Reflecting lights

Aluminum foil or white cloth placed on either side of the infant to reflect light will increase irradiance. Though hanging of white reflective sling on sides of fluorescent phototherapy equipment results in marginal increase in irradiance, it does not decrease the duration of phototherapy. Use of mirrors behind the bulbs in tunnel phototherapy units may lower STB levels earlier [25-26].

Intermittent Vs Continuous Phototherapy

Phototherapy does not need to be continuous. Phototherapy may be interrupted during feeding or brief parental visits. If the infant's bilirubin level is approaching the exchange transfusion level, phototherapy should be administered continuously until a satisfactory decline in the serum bilirubin level occurs or exchange transfusion is initiated [2-8].

Intensive Phototherapy

Intensive phototherapy is recommended for those with "higher risk" based on age specific nomograms. It implies the use of high levels of irradiance in the 430 to 490 nm band (usually 30 microwatt/cm²/nm or more) with the aid of two or more light sources (combination of overheads, spots, biliblankets) delivered to as much of infant's surface area as possible. The overheads should be placed as close as to the baby as possible without causing hyperthermia or burn. Normally, bilirubin should decline by 1-2 mg/dl within 4-6 hours of intensive phototherapy and continue to decline and remain below the threshold level for exchange transfusion. Failure of intensive phototherapy is said to occur and hence an indication for exchange transfusion when this predicted normal fall in serum bilirubin does not occur. Thermal-neutral lights, such as LEDs, can be placed to provide intensive phototherapy while reducing the potential risk of thermal injury or fluid loss [5-7].

Side Effects of Phototherapy [27-35]

1. *Insensible water loss* is increased in infants undergoing phototherapy, especially those under radiant warmers.
2. *Redistribution of blood flow*. In term neonates, left ventricular output and renal blood flow velocity decrease, whereas left pulmonary artery and cerebral blood flow velocity increase. After discontinuation of phototherapy, all velocities return to baseline. In preterm neonates, cerebral blood flow velocity also increases and renal vascular resistance increases with a reduction of renal blood flow velocity. In addition, in preterm neonates under conventional phototherapy, it has been shown that the usual postprandial increase in superior mesenteric blood flow is blunted. Although the changes in cerebral, renal, and superior mesenteric artery blood flow with phototherapy treatment in preterm infants is of potential concern, no detrimental clinical effects due to these changes have been determined.
3. *Watery diarrhea and increased fecal water loss* may occur. The diarrhea may be caused by increased bile salts and unconjugated bilirubin in the bowel.
4. *Retinal damage* has been described in animals whose eyes have been exposed to phototherapy. The eyes should be shielded with eye patches.
5. *Tanning* of the skin of infants. During exposure to light, infants skin gets bleached. The clinical evaluation of jaundice severity becomes unreliable in babies receiving phototherapy and hence serum bilirubin level should be monitored every 6-8 hourly. Erythema and increased skin blood flow may also be seen.

Hyperthermia

LED phototherapy with low irradiances does not cause significant hyperthermia similar to conventional phototherapy with blue fluorescent light. LED phototherapy with high irradiances (60-120 μ W/cm²/nm) significantly increases body temperature in hyperbilirubinemic newborns compared to infants who received conventional phototherapy with fluorescent lamps (10-15 μ W/cm²/nm) or LED phototherapy (26-60 μ W/cm²/nm). Thus the increase in body temperature is a function of increase of irradiance rather than the type of the light source. Hyperthermia might be related to release of pyrogenic cytokines, although effects of

light with different wave-lengths and irradiances on serum cytokine levels are not known.

Skin Rashes

Skin rash were noted in the super (high-intensity, or high-irradiant LED) group compared with the fluorescent tubes-treated group. Erythema and increased skin blood flow may also be seen.

Bronze baby syndrome (BBS) The bronze-baby syndrome (BBS) is a rare side-effect of phototherapy which causes the appearance of grey-brown discoloration of skin. It is harmless, and pigmentation returns slowly to normal if phototherapy is discontinued. Infant with parenchymal liver disease with biliary obstruction may develop peculiar bronzed skin due to excessive accumulation of one of the photoisomers designated as lumirubin retained and polymerized to Bilifuscin imparting brownish discoloration to skin.

Hypocalcemia

Phototherapy can lead to decreased total and ionized calcium levels of neonates, especially in preterm neonates. This effect might be attributable to increased urinary calcium excretion. In addition, light can affect calcium homeostasis by inhibiting pineal secretion of melatonin and consequently leading to hypocalcemia.

Patent Ductus Arteriosus

It is hypothesized that light can penetrate the thin chest wall of extremely preterm infants, and causes the relaxation of aortic smooth muscle through the activation of the nitric oxide-cyclic GMP pathway and Ca^{2+} -dependent K^{+} ion channels. Therefore, neonatal phototherapy may exert a relaxing effect on the smooth muscles of the ductus arteriosus in neonates, thus prevents the closure of patent ductus arteriosus (PDA) and may cause the reopening the ductus arteriosus. Phototherapy has been reported to increase the heart rate, diminish the mean arterial blood pressure and increase peripheral blood flow. These alterations may also affect the closure of PDA, and it has been speculated that phototherapy may be a risk factor for PDA, especially in the VLBW infants.

Mutations, sister chromatid exchange, and DNA strand breaks have been described in cell culture.

Tryptophan is reduced in amino acid solutions exposed to phototherapy

Maternal-infant separation – this can interfere with bonding and breastfeeding but can be minimised by utilising Bilibeds on the postnatal wards when appropriate.

Ileus during conventional phototherapy due to photorelaxation of gut smooth muscle as in vascular smooth vessels possibly either by nitric oxide-cyclic guanosine monophosphate pathway or direct photorelaxation is thus biologically plausible. Changes in peripheral blood flow and cardiac output during conventional phototherapy may also contribute to ileus during conventional phototherapy in preterm neonates.

In some infant, platelet turnover may be increased resulting in lower mean platelet count but bleeding does not occur.

Recent studies have suggested that phototherapy is associated with an increased risk for childhood bronchial asthma. Allergic rhinitis are also more common in children who have history of jaundice and/or phototherapy during neonatal period.

Conclusion

Phototherapy is the use of visible light which is safe and easy to use for the treatment of hyperbilirubinemia in the newborn.

References

1. Singh M. Jaundice. In Care of the Newborn. Eds: Singh M. 8 th edn; Sagar Publications, New Delhi 2004.
2. Maisels MJ, Baltz RD, Bhutani V, et al. Management of hyperbilirubinemia in the newborn infant 35 or more weeks of gestation. Pediatrics. 2004;114:297-316.
3. American Academy of Pediatrics, Subcommittee on Hyperbilirubinemia. Management of hyperbilirubinemia in the newborn infant 35 or more weeks of Pediatrics. 2004;114(1):297-316.
4. Cremer RJ, Perryman PW, Richards DH. Influence of light on the hyperbilirubinaemia of infants. Lancet. 1958;1(7030):1094-7.
5. Maisel MJ, McDonagh AD. Phototherapy for Neonatal Jaundice. N Engl J Med. 2008;358:920-928.
6. Muchowski KE. Evaluation and treatment of neonatal hyperbilirubinemia. Am Fam Physician. 2014 Jun 1; 89(11):873-8.
7. Martin CR, Cloherty JP. Neonatal hyperbilirubinemia. In Manual of Neonatal Care. In Cloherty JP, Eichenwald EC, Stark AR. (eds) 8 th edn; Philadelphia, Lippincott Williams & Wilkins.2004 .

8. Ennever JF, Sobel M, McDonagh AF, Speck WT. Phototherapy for neonatal jaundice: in vitro comparison of light sources. *Pediatr Res*. 1984;18(7):667-70.
9. Costarino AT Jr, Ennever JF, Baumgart S, Speck WT, Polin RA. Effect of spectral distribution on isomerization of bilirubin in vivo. *J Pediatr*. 1985;107(1):125-8.
10. Ebbesen F, Madsen P, Stovring S, Hundborg H, Agati G. Therapeutic effect of turquoise versus blue light with equal irradiance in preterm infants with jaundice. *Acta Paediatr*. 2007;96(6):837-41.
11. Donzelli GP, Pratesi S, Rapisardi G, Agati G, Fusi F, Pratesi R. 1-Day phototherapy of neonatal jaundice with blue-green lamp. *Lancet*. 1995;346(8968):184-5.
12. Seidman DS, Moise J, Ergaz Z, Laor A, Vreman HJ, Stevenson DK, Gale R. A prospective randomized controlled study of phototherapy using blue and blue-green light-emitting devices, and conventional halogen-quartz phototherapy. *J Perinatol*. 2003;23(2):123-7.
13. Ebbesen F, Madsen P, Støvring S, Hundborg H, Agati G. Therapeutic effect of turquoise versus blue light with equal irradiance in preterm infants with jaundice. *Acta Paediatr*. 2007;96(6):837-41.
14. Vandborg PK, Hansen BM, Greisen G, Ebbesen F. Dose-response relationship of phototherapy for hyperbilirubinemia. *Pediatrics*. 2012;130(2):e352-7.
15. Bhutani VK; Committee on Fetus and Newborn; American Academy of Pediatrics. Phototherapy to prevent severe neonatal hyperbilirubinemia in the newborn infant 35 or more weeks of gestation. *Pediatrics*. 2011;128(4):e1046-52.
16. Agati G, Fusi F, Pratesi R, McDonagh AF. Wavelength dependent quantum yield for Z'E isomerization of bilirubin complexed with human serum albumin. *Photochem Photobiol*. 1992;55(2):185-90.
17. Zonios G, Bykowski J, Kollias N. Skin melanin, hemoglobin and light scattering properties can be quantitatively assessed in vivo using diffuse reflectance spectroscopy. *J Invest Derm*. 2001;117(6):1452-57.
18. Lamola AA, Bhutani VK, Wong RJ, Stevenson DK, McDonagh AF. The effect of hematocrit on the efficacy of phototherapy for neonatal jaundice. *Pediatr Res*. 2013;74(1):54-60.
19. Okada H, Abe T, Etoh Y, Yoshino S, Kato I, Iwaki T, Okubo K, Yasuda S, Kawada K, Kusaka T, Namba M, Nishida T, Imai T, Isobe K, Itoh S. In vitro production of bilirubin photoisomers by light irradiation using neoBLUE. *Pediatr Int*. 2007;49(3):318-21.
20. Mills JF, Tudehope D. Fiberoptic phototherapy for neonatal jaundice. *Cochrane Database Syst Rev*. 2001;(1):CD002060.
21. Slusher TM, Vreman HJ, Olusanya BO, Wong RJ, Brearley AM, Vaucher YE, Stevenson DK. Safety and efficacy of filtered sunlight in treatment of jaundice in African neonates. *Pediatrics*. 2014;133(6):e1568-74.
22. Nuntnarumit P, Naka C. Comparison of the effectiveness between the adapted-double phototherapy versus conventional-single phototherapy. *J Med Assoc Thai*. 2002;85(Suppl 4):S1159-66.
23. Romagnoli C, Zecca E, Papacci P, Vento G, Girlando P, Latella C. Which phototherapy system is most effective in lowering serum bilirubin in very preterm infants? *Fetal Diagn Ther*. 2006;21(2):204-9.
24. Boonyarittipong P, Kriangburapa W, Booranavanich K. Effectiveness of double-surface intensive phototherapy versus single-surface intensive phototherapy for neonatal hyperbilirubinemia. *J Med Assoc Thai*. 2008;91(1):50-5.
25. Hansen TW. The role of phototherapy in the crash-cart approach to extreme neonatal jaundice. *Semin Perinatol*. 2011;35(3):171-4.
26. Sivanandan S, Chawla D, Misra S, Agarwal R, Deorari AK. Effect of sling application on efficacy of phototherapy in healthy term neonates with nonhemolytic jaundice: a randomized controlled trial. *Indian Pediatr*. 2009;46(1):23-8.
27. Aydemir O, Soysaldý E, Kale Y, Kavurt S, Bas AY, Demirel N. Body temperature changes of newborns under fluorescent versus LED phototherapy. *Indian J Pediatr*. 2014;81(8):751-4.
28. Kurt A, Aygun AD, Kurt AN, Godekmerdan A, Akarsu S, Yilmaz E. Use of phototherapy for neonatal hyperbilirubinemia affects cytokine production and lymphocyte subsets. *Neonatology*. 2009;95(3):262-6.
29. Clark CF, Torii S, Hamamoto Y, Kaito H. The "bronze baby" syndrome: postmortem data. *J Pediatr*. 1976;88(3):461-4.
30. Eghbalian F, Monsef A. Phototherapy-induced hypocalcemia in icteric newborns. *Iran J Med Sci*. 2002;27(4):169-71.
31. Hooman N, Honarpisheh A. The effect of phototherapy on urinary calcium excretion in newborns. *Pediatr Nephrol*. 2005;20(9):1363-4.
32. Venturini CM, Palmer RM, Moncada S. Vascular smooth muscle contains a depletable store of a vasodilator which is light-activated and restored by donors of nitric oxide. *J Pharmacol Exp Ther*. 1993;266(3):1497-500.
33. Benders MJ, Van Bel F, Van de Bor M. Cardiac output and ductal reopening during phototherapy in preterm infants. *Acta Paediatr*. 1999;88:1014-9.
34. Bader D, Kugelman A, Blum DE, Riskin A, Tirosh E. Effect of phototherapy on cardiorespiratory activity during sleep in neonates with physiologic jaundice. *Isr Med Assoc J*. 2006;8(1):12-6.
35. Liu GS, Wu H, Wu BQ, Huang RZ, Zhao LH, Wen Y. Effect of phototherapy on blood endothelin and nitric oxide levels: clinical significance in preterm infants. *World J Pediatr*. 2008;4(1):31-5.

Study of The Quality of Life in Children with Chronic Diseases in A Tertiary Care Hospital in Mysuru

Anitha C.¹, Kavya Kurkal², Santhosh Kumar M.³

¹Associate Professor ²Post Graduate ³Assistant Professor, Department of Pediatrics, JSS Medical College Hospital, JSSAHER, Mysore, Karnataka 570004, India.

Abstract

Introduction: The Quality of Life in Children with Chronic diseases is a crucial, yet often neglected part of child's overall health. Chronic diseases and their treatment present children and their parents with significant chronic stress that lead to emotional and behavioural problems compromising adherence to treatment. WHO defines Quality of life as an individual perception of their position in life in context of the culture and value systems in which they live and in relation to their goals, expectations, standards and concerns [1]. **Method:** Cross-Sectional Study conducted in the Department of Paediatrics, JSS Hospital Mysuru from 1-10-2014 to 1-8-2016. 60 Children aged 7 to 18 years, suffering from chronic disease since at least 3 months were selected at random. Interviewing was done using 2 Questionnaires namely EQ-5DY administered to children and CPMS Questionnaire given to Parents/Guardians, after obtaining valid consent from authorities and Parents. Data was entered in Excel and analysed using SPSS Software. **Results:** There was no association between the Socio-Demographic factors and Quality of Life or Psychological issues in the children. In the EQ-5DY Questionnaire, the children who had some pain and discomfort also had significant Psychopathology and P Value being 0.03 was significant. 95% of our population had possible Psychopathology indicating that Mental Health is affected in children suffering from various chronic diseases. Children with seizure disorder had problems in daily activities, had some pain and discomfort and all were worried/sad. **Conclusions:** All children with chronic disease, irrespective of age, gender and socio-economic class have high risk for impaired Quality of Life and Mental Health problems.

Keywords: Quality of Life; Chronic Diseases; EQ-5D-Y IS A; Childhood Psychopathology Measurement Schedule (CPMS).

Background

Role of a Paediatrician is significant in detecting early signs of Psychopathology and also to extend holistic treatment to the Children suffering from chronic diseases in addition to the management of the disease per se. Quality of Life in children with chronic diseases is a crucial yet often neglected part of a child's overall health.

Many studies conducted in India and at abroad have concluded that children with chronic diseases are unable to lead normal healthy lives due to physical, emotional, psychological and mental health issues.

WHO defines Quality of Life as an individual perception of their position in life in context of the culture and the value systems in which they live and in relation to their goals, expectations, standards and concerns [1].

Chronic illness is defined as a condition that either interferes or is likely to interfere with an individual's daily functioning for at least 3 months of a year or a condition that will require hospitalization for more than 1 month in a year [2].

Developmental changes make it difficult to apply any single measure to all age groups and Child QOL should be developed within the context of a developmental framework. Child's understanding of the illness is an important determinant of their QOL.

Corresponding Author: Santhosh Kumar M., Assistant Professor, Department of Pediatrics, JSS Medical College Hospital, JSSAHER, Mysore, Karnataka 570004, India.
E-mail: santhosh.kumar94@yahoo.com

Received on 09.08.2018, **Accepted on** 31.08.2018

Illness in Adolescents represents a major barrier to attainment of autonomy and chronic diseases in them can be associated with changes in mobility, energy level, social and peer interactions, physical appearance, self esteem and cognitive function [3].

Family is an influential mediator of the chronically ill child's adjustment which deserves a primary place in future research efforts [4,5,6].

Method

This is a cross-sectional interview based study and was conducted in JSS Hospital Mysuru between 1-10-2014 to 1-08-2016.

Taking the prevalence of anxiety related symptoms in children with chronic diseases as 67% [from a Nimhaans based study], the sample size was calculated to be 60. Formula- $S = Zpq/d^2 = 4pq/d^2$.

Children aged 7 to 18 yrs who have been diagnosed with Thalassemia Major, Nephrotic Syndrome, Rheumatic heart disease, Asthma, Type 1 Diabetes Mellitus, and Seizure Disorder, since at least 3 months and who visit JSS Hospital on either OPD or IPD basis were included in the study and excluded Children with impaired intelligence or severe Developmental Delay. The selection was done at convenience and all these children have been on continuous or regular treatment for their disease.

After obtaining valid informed consent from parents/guardians, 2 Questionnaires were administered to children and parents respectively. Ethical clearance was obtained from the Medical College authorities. The nature of the study and the contents of the questionnaires were explained to parents /guardians in their own language and their informed written consent was obtained.

The first Questionnaire i.e the EQ-5D-Y IS A Health related Quality of Life questionnaire developed by the Euroqol group [7]. The Euroqol group is a network of international multidisciplinary researchers devoted to the measurement of health status.

EQ-5D-Y provides a simple descriptive profile and a single index value for health status that can be used in clinical and economic evaluation of health care. The EQ-5D-Y has 2 portions. The EQ-5D-Y Descriptive system and the EQ-Visual Analogue state. (EQVAS). The descriptive system has 5 dimensions/domains, using child friendly wordings. (Mobility, Looking after self, Doing usual activities, Having pain and discomfort, Feeling worried/

unhappy/sad.). Each domain has 3 levels—No Problem, Some Problem, Lot of problems. The Child has to tick a cross against the statement. EQVAS records the child's self rated health on a Vertical Analogue Scale marked from 0 to 100 and end points labelled "The Best Health You can imagine" rated 100 and "The Worst Health You can imagine" rated 0.

The Second Questionnaire is the Childhood Psychopathology Measurement Schedule (CPMS) [8], which is an Indian adaptation of the child behavior checklist by Achenbach and Edelbrock [9]. It has 75 items both in Hindi and English rated as yes or No and this questionnaire is answered by parents. High score indicates higher the psychopathology. Eight Subscales are present namely low intelligence with behaviour problems, anxiety, depression, psychotic symptoms, special symptoms, physical illness with emotional problems and somatization. Total score being the sum total of all the factor scores.

Socio-Demographic data was also collected in the form of name, age, sex, education, and occupation of parent. monthly income, Socioeconomic status and residence. Modified BG Prasad Social classification revised for 2016 was used [10].

Statistical Analyses

Data was entered in Excel format and analysed using SPSS Software.

Results

In this study 60 children and their Parents were enrolled for the interview using the 2 Questionnaires.

Table 1 shows demographic details of children enrolled in the study along with the type of chronic diseases in them.

Table 2 shows EQ-5D-Y Descriptive Questionnaire where in 40% of Nephrotics had problems with Mobility. 66.7% of Seizure Disorder children had problems in looking after self, in daily activities, and had some pain and discomfort. 100% of Seizure Disorder Children, 90% of Asthmatics, 81.3% of Diabetics and 80% of Nephrotics were worried, sad or unhappy.

Table 3 shows the Visual Analogue Scale which is a part of EQ-5D-Y wherein the VAS Score was highest in Class 5 of SEC, more in males and in rural areas, but not statistically significant.

Table 1: Demographic/ baseline data

		No.	%
Age	<10	29	48.3%
	11-15	28	46.7%
	>16	3	5.0%
Gender	Male	30	50%
	Female	30	50%
Sec	CLASS 1	0	0%
	CLASS 2	3	5.0%
	CLASS 3	23	38.3%
	CLASS 4	30	50.0%
	CLASS 5	4	6.7%
Type of chronic Disease	Thalassemia major	17	28.3%
	Type 1 diabetes mellitus	16	26.7%
	Asthma	10	16.7%
	Rheumatic heart disease	9	15.0%
	Nephrotic syndrome	5	8.3%
	Seizure disorder	3	5.0%

CPMS Score were highest in females, urban residents and in children with Thallasemia, but again was not statistically significant.

Table 4 Shows the subsets of CPMS Score comparing to gender and residence. Anxiety was significantly higher in females and statistically significant. p value=0.03. Mean CPMS Score, Depression and Somatization were high in females while conduct disorders and psychotic illness were high in males but not statistically significant.

CPMS Score was more in urban than in Rural but again not statistically significant.

Table 5 Using Pearsons correlation, there was a negative correlation between duration of illness and VAS i.e longer the duration lower was the mean VAS Score, p=0.7, however, there was a positive correlation

Table 2: Comparision between types of Chronic Diseases and Different Domains of the EQ-5D-Y Descriptive Questionnaire

Diagnosis	Mobility Some problems	Looking after self Some problems	Problems in Daily Activity Some problems	Pain & discomfort Some problems	Worried Sad/unhappy Some problems
Asthma	0(0%)	2(20%)	5(50%)	5(50%)	9(90%)
Nephrotic Syndrome	2(40%)	2(40%)	2(40%)	2(40%)	4(80%)
RHD	1(11.1%)	2(22.2%)	1(11.1%)	1(11.1%)	5(55.6%)
Seizure Disorder	1(33.3%)	2(66.7%)	2(66.7%)	2(66.7%)	3(100%)
Thalassemia Major	1(5.9%)	0(0%)	3(17.6%)	6(35.3%)	11(64.7%)
Type 1 Diabetes Mellitus	0(0%)	1(6.2%)	8(50%)	7(43.8%)	13(81.3%)
Total	5(8.3%)	9(15%)	21(35%)	23(38.3%)	55(75%)

Mobility problems are more in nephrotic syndrome (40%) and 66.7% of seizure disorder had problems looking after self. 66.7% of seizure disorder had problems indaily activities. 50% of Asthma patients and diabetic patients had problems in daily activities. 66.7% of seizure disorder had pain and discomfort, while 50% of asthmatics had pain and discomfort. 100% of seizure disorder, 90% of asthmatics, 81.2% of diabetics and 80% of nephrotics were worried/unhappy/sad.

Table 3: Comparision of VAS and CPMS with Socio-economic class,Gender, Residence and Diseases

		VAS		CPMS	
		Mean	SD	Mean	SD
SEC	Class 1	0	0	0	0
	Class 2	65	13.23	16.67	8.96
	Class 3	64.65	19.84	17.91	5.46
	Class 4	69.33	17.99	16.07	4.48
	Class 5	73.75	11.09	20.00	2.45
Gender	Male	70.07	17.87	16.63	5.22
	Female	65.17	18.26	17.50	4.90
Residence	Rural	68.45	17.72	16.94	4.01
	Urban	66.72	18.67	17.21	5.54
Diagnosis	Asthma	66.20	12.47	16.80	3.74
	Nephrotic Syndrome	65.00	21.79	17.80	3.42
	RHD	70.00	21.21	15.22	2.77
	Seizure	56.67	5.77	16.33	2.08
	Thalassemia	65.88	20.02	18.35	7.12
	Type 1 DM	71.88	18.34	16.81	5.02

VAS scoreis highest in class 5 and lowest in class 3, VAS score high in males, in rural areas and in Diabetics.

CPMS Scores high in class 5, in females, in urban areas and in Thallasemia patients.

between duration and CPMS Scores i.e longer the duration higher the Psychopathology. $p=0.3$. CPMS and VAS were negatively correlated. The mean duration of illness was 4yrs and the 25th and 75th centile was 1.5yrs and 7.5yrs respectively.

Table 6. Out of a total CPMS score of 75, 10 or more was considered significant indicating the presence

of some Psychopathology. 95% Of Children had some psychopathology.

Table 7 is a comparison between Domains of QOL and the Mean CPMS score. The domain of Pain and Discomfort had a higher CPMS score and $p=0.03$ was significant.

Table 4: Depicts the various parameters of CPMS in comparison with Gender and Residence.

CPMS	Gender				Residence			
	Male Mean	Male SD	Female Mean	Female SD	Rural Mean	Rural SD	Urban Mean	Urban SD
Low intelligence With behavioural problems	4.40	1.92	4.43	1.72	4.45	1.79	4.38	1.86
Conduct Disorder	4.60	2.62	3.90	2.28	4.16	2.48	4.34	2.48
Anxiety	0.90	1.03	1.50	1.04	1.13	1.09	1.28	1.07
Depression	2.50	1.46	3.03	1.61	2.87	1.80	2.66	1.23
Psychotic Symptoms	1.30	1.15	1.20	0.96	1.13	1.12	1.38	0.98
Special Symptoms	0.33	0.61	0.47	0.78	0.35	0.61	0.45	0.78
Physical illness With emotional Problems	1.10	0.99	1.23	0.94	1.32	0.98	1.00	0.93
Somatisation	1.50	1.07	1.73	1.11	1.52	1.12	1.72	1.07
Total CPMS score	16.63	5.22	17.50	4.90	16.94	4.61	17.21	5.54

Anxiety was found to be significantly higher in females. ($p=0.03$). Depression, somatization and CPMS Scores higher in females. Conduct disorders and Psychotic illness were higher in males. Psychotic symptoms, CPMS Scores were more in urban areas

Table 5: Percentage of children with possible psychopathology (as per cpms)

Psychopathology	Number	Percentage
Yes	3	5
No	57	95

Out of a total score of 75, 10 or more was considered significant i.e, it indicated the presence of some psychopathological problem. 95% of children with chronic disease had some psychopathology.

Table 6: Correlation between duration of illness and VAS and Total CPMS Score

		Duration of Illness	VAS	Total CPMS Score
Duration of Illness	Pearson Correlation (r)	1	-0.037	0.113
	P		0.781	0.392
	N	60	60	60

The median duration of illness was 4 years and the 25th and 75th centile was 1.5 years and 7.5 years respectively. Longer duration of illness decreased VAS score and increased CPMS scores.

Table 7: Comparison between Domains of Quality of life with the Mean CPMS Scores

		Total CPMS		Probability
		Mean	SD	
Mobility	No Problem	16.89	5.19	0.4
	Some Problem	19.10	2.35	
Looking After self	No Problem	16.88	5.24	0.5
	Some Problem	18.11	3.72	
Daily Activities	No Problem	16.44	5.29	0.4
	Some Problem	18.05	4.56	
Pain Discomfort	No Problem	15.95	5.02	0.03
	Some Problem	18.87	4.62	
Worried/ Sad	No Problem	15.07	4.79	0.1
	Some Problem	18.13	5.22	

Pain and discomfort had a significant association ($p=0.03$) which means children who had some pain and discomfort had a higher CPMS score. Other Domains had no significant correlation

Discussion

EQ-5D-Y and CPMS Questionnaires were applied to 60 Children aged 7 to 18 yrs and to their parents respectfully.

By applying the EQ-5D-Y Descriptive to our study population, it was found out that 8.3% had some problems in mobility, 15% had some problems in looking after self, 35% had some problems in daily activity, 38.3% had pain and Discomfort and 75% were worried/sad/unhappy. This is comparable to a study conducted by M.A Grootenhuys et al. [11]. wherein by administering questionnaire related to 7 domains of quality of life, they found significant differences in motor functioning, autonomy, social and cognitive functioning in children with chronic diseases as compared to normal children.

Domains of the QOL was compared with the diagnosis using Chi-Square test and there was a significant association ($p=0.04$) between mobility and type of disease. In our study, 40% of nephrotics, 33.3% of Children with Seizure Disorder, 11.1% of children with Rheumatic Heart Disease and 5.9% of Thalassemics had some problem with mobility.

Significant association ($p=0.02$) was seen between the type of Chronic Disease and Ability to look after themselves. 66.7% of children with Seizure Disorder, 40% of Nephrotics, 22.2% of Children with RHD, 20% of Asthmatics and 6.3% of Diabetics had some problems in looking after themselves.

In a Study by D Shaligram, S.C. Girimaji and S.K. Chaturvedi [12] on children with Thalassemia, 74% had poor QOL AND 44% had Psychological problems. In the QOL domains, 64% had Pain and Discomfort, 33% had problems with mobility and were also depressed. In our Study 35.3% of Thalassemics had Pain and Discomfort, 5.9% had mobility problems, 17.6% had problems in Daily Activities, where as 64.7% of them were worried or sad.

In a Study by Prabhjoth Malhi, Lata kumar and Meenu Singh [13], Children with Asthma were found to be more at risk for emotional and behavior problems than healthy controls where as in our study, 90% of the Asthmatics had some worry or sadness, 50% of them had problems in daily activities and also experienced pain and discomfort.

In a study by Jayashree Nadkarni, Arti Jain, Rashmi Dwivedi [14] on children with epilepsy, they found that they had compromised QOL in psychological, social and behavioural functioning but physically

unaffected. In our study 100% of the epileptics were sad/worried/unhappy, while 66% had some problems in daily activities, pain and discomfort and in looking after themselves.

In a study conducted by OP. Mishra, Biswanath Basu, Shashi K, et al. [15] on the behavior problems in children with Nephrotic syndrome, they found clear evidence of behavioural changes across all age groups, where as in our study. 40% of Nephrotics had some problems in mobility, looking after self, daily activities and 40% experienced pain and discomfort. 80% were worried/sad.

In a study on children with Type 1 Diabetes Mellitus by M. Abdul-Rasoul, F. Alotaibi, A. Abdulla, Z. Rahme and F. Alshawaf [16], showed that QOL scores were low in emotional and physical domains. In our study 81.2% of Diabetics had some worry/sadness, 50% had problems in Daily Activities, 43.8% had some Pain and Discomfort.

In our study the Visual Analogue Scale which is 2nd part of EQ-5D-Y when applied was found to be highest in class 5 SEC i.e 73.75% and lowest in class 3 SEC i.e 64.65%. ($p=0.7$ was not significant). Mean VAS was more in males (70.07) and rural children (68.45) but p value was not significant in both. Mean VAS score was highest (71.88) in Type 1 Diabetics and lowest in Nephrotics [65] and p value was 0.8 and hence statistically insignificant.

The Ontario Child Health Study conducted by David Cadman, Michael Boyle, Peter Szatmari and David R. Offord [17] demonstrated that in a representative general population sample of children, 22% of those with chronic illness with disability and 31% of those with chronic illness without disability had Psychiatric problems. In our study, using the CPMS questionnaire 57 out of 60 children (95%) were found to have possibility of some psychopathology. Since CPMS Questionnaire is a screening tool, these children need to be subjected to detailed psychiatric evaluation.

In the CPMS Questionnaire males scored a mean of 16.63 while females scored 17.5 and $p=0.5$. Anxiety was found to be significantly higher among girls with chronic illness ($p=0.03$). Here, independent 't' test was used to study correlation between gender and CPMS.

The Mean CPMS Scores when correlated with different age groups, the score was maximum below 10 yrs of age (17.86), and minimum in children above 16 yrs of age (14.67). Using the Anova test, the P value was 0.4. It shows that younger children had a higher CPMS Score which reduced with increasing age. This finding is similar to a study conducted by Anna

Mikelli and John Tsiantis [18], on the depressive symptoms and QOL in Adolescents with Beta Thalassemia which revealed older adolescents reporting significantly fewer depressive symptoms than younger ones.

In our study, age, sex, and socioeconomic status of the child has no bearing on the quality of life and mental health as perceived by child and parent, and this finding is consistent with the study conducted by Prakash V, Pradhan, Henal Shah, Pradeep Rao, Dhananjay Ashturkar and Pradnya Ghaisas (19), where 60 children were studied with 30 Epileptics and 30 Thalassemics and there was no correlation between Socio-Demographic Data and Psychopathology.

Using Pearson correlation, it was found that longer the duration of illness lower was the Mean VAS Score ($p=0.7$) was not significant. On the other hand, longer the duration of illness higher the CPMS Score. Indicating higher psychopathology, ($p=0.3$) was not significant. As per the study done by Prakash et al. [19], they found that chronicity of the disease did impact the QOL.

Majority of children (95%) in our study had possible Psychopathology which infers that Mental Health is affected in children suffering from various chronic diseases.

Conclusion

From all these studies, we can see that though there are minor differences in the results, the common consensus is that the overall Quality of Life in children suffering from chronic diseases is definitely hampered.

Duration of illness had an impact on Quality of Life depicted by low VAS scores and the higher CPMS Scores indicated that some Psychopathological problems are present in children with chronic diseases which further requires a detailed evaluation.

QOL and Mental Health of children with chronic diseases are not affected by age, gender or social background.

Chronic diseases in children does affect the QOL and Mental Health and goes often neglected by Parents and Paediatricians alike. The first step towards bettering their Lives is to acknowledge that a problem exists, the rest is the cohesive efforts of Child, Parent and team of Health Care Givers.

Pain and discomfort had a significant association ($p=0.03$) which means children who had some pain and discomfort had a higher CPMS score. Other Domains had no significant correlation.

References

1. WHO. Development of the World Health Organisation WHOQOL-BRIEF quality of life assessment. The WHOQOL Group. Psychol Med. 1998;28(3):551-8.
2. Pless IB, Pinkerton P. Chronic childhood disorder: promoting patterns of adjustment. Chicago: Year Book Medical; 1975.
3. Coupey S. Chronic illness. In: Friedman SB, Fisher M, and Alderman EM (eds). Comprehensive Adolescent Health Care. St Louis, MO: Mosby-Year Book; 1998. pp. 132-39.
4. Caplan G. & Killea M. Support systems and mutual help: Multidisciplinary explorations. New York: Grune & Stratton, 1976.
5. Litman, T.J. The family as a basic unit in health and medical care: A social-behavioural overview. Social Science and Medicine, 1974;8:495-519.
6. Litman T.J. & Venters M. Research on health care and the family: A methodological overview. Social Science and Medicine, 1979;13A:379-85.
7. Reenen van M, Janssen B, Oppe M, Kreimeier S, Greiner W. EQ-5D-Y User Guide, basic information on how to use the EQ-5D-Y instrument 2014; (August):1-23.
8. Malhotra S, Verma VK, Verma SK, Malhotra A. Childhood Psychopathology Measurement Schedule: Development and Standardization. Indian Journal of Psychiatry. 1988;30(4):325-31.
9. Achenbach T.M. The child behaviour profile in empirically based system for assessing children's behavioural problems and competencies, International J. On Mental Health 1979;7(3).
10. Sharma R. Revision of Prasad's social classification and provision of an outline tool for real-time updating. South Asian J CANCER 2013;2(3):157.
11. Grootenhuis M, Koopman H, Verrips E, Vogels A, Last B. Health-related quality of life problems of children aged 8-11 years with a chronic disease Developmental Neurorehabilitation. 2007;10(1):27-33.
12. Shaligram D, Girimaji SC, Chaturvedi SK. Psychological problems and quality of life in children with thalassemia. Indian J Pediatr. 2007;74(8):727-30.
13. L.K.M.S. Prabhjot malhi, Screening for psychological problems in children and adolescents with asthma, Indian Pediatrics, 2001;38:524-530.
14. Nadkarni J, Jain A, Dwivedi R. Quality of life in children with epilepsy. Ann Indian Acad Neurol. 2011;14:279-82.

15. Mishra OP, Biswanath B, Upadhyay SK. Behavioural abnormalities in children with nephrotic syndrome. *Nephrology Dialysis Transplantation*. 2010;25: 2537-41.
 16. Abdul- Rashid M, ALOtaibi F, Abdulla A, Rahme Z, AlShawaf F. Quality of life of children and adolescents with type 1 diabetes in Kuwait. *Med Princ Pract*. 2013;22:379-384.
 17. David Cadman, Michael Boyle, Peter Szatmari and David R. Offord. Chronic Illness, Disability, and Mental and Social Well-Being: Findings of the Ontario Child Health Study *Pediatrics*. May 1987;79(5): 805-13.
 18. Mikelli A, Tsiantis, J. Brief report: Depressive symptoms and quality of life in adolescents with β -thalassemia. *Journal of Adolescence*, 2004;27(2): 213-16.
 19. Pradhan PV, Shah H, Rao P, Ashturkar D, Ghaisas P. Psychopathology and Self-esteem in Chronic Illness. *Indian Pediatrics* 2003;70(20):135-38.
-

Clinical Profile of Hypoxic- Ischemic Encephalopathy in Neonates with Birth Asphyxia in a Rural Tertiary Care Hospital in Central India

Kher Anjali M.¹, Singh Kuldeep², Vagha Jayant³

¹Associate Professor ²Junior Resident ³Professor, Department of Pediatrics, Jawaharlal Nehru Medical College, DMIMS, Sawangi (M), Wardha, Maharashtra 442004, India.

Abstract

Context- Perinatal asphyxia and Postasphyxial hypoxic-ischemic encephalopathy (HIE) is a major cause of early neonatal mortality in India accounting for 24.3% of neonatal deaths. **Aim-** To study risk factors, clinical, biochemical, Neurosonography parameters and short term outcome of neonates with HIE. **Setting and Design-** Study conducted at AVBRH, Sawangi (M) after seeking IEC approval. It was a prospective observational study. **Material and method-** All neonates delivered in AVBRH and admitted to NICU with birth asphyxia and HIE were included in the study. A detailed history, examination, HIE staging, cord blood ABG, Neurosonography, other investigations and short term outcome were recorded in prevalidated proforma and data was statistically analyzed using SPSS 22.0. **Results-** Incidence of Birth asphyxia was 2.97%, incidence of HIE was 1.76%. Of all neonates with HIE (study group n=50) 26% were in HIE stage I, 54% were in stage II and 20% were in stage III. On umbilical cord ABG 20% neonates had severe acidemia and 80% had moderate acidemia. Neurosonography changes were seen in 4% neonates, mortality rate was 18%. All 9 neonates who died were in HIE stage III. **Conclusion-** Birth asphyxia and HIE are still a major cause of neonatal morbidity and mortality. HIE stage III, severe acidemia are associated with high mortality. Preclampsia, oligohydramnios and MSAF are risk factors associated with HIE.

Keywords: Birth Asphyxia; HIE; Incidence; Mortality and Risk Factors.

Introduction

India contributes to one-fifth of global live births and more than a quarter of neonatal deaths [1]. The neonatal mortality rate (NMR) in India was 28 per 1000 live births with perinatal mortality rate (PMR) of 26 per 1000 births and the early NMR of 22 per 1000 live births in 2013. Deaths in the first week alone account for approximately 45% of total under-five deaths [2].

The true burden of birth asphyxia is difficult to estimate because of the different definitions used in the studies. According to World Health Organization (WHO) in the developing countries 3% of all infants suffer from moderate to severe birth asphyxia, of which 23% die and approximately the same numbers develop serious sequelae. Asphyxia accounts for 23% of neonatal

deaths globally, and 8% of all deaths in children under five years of age [3].

Birth asphyxia is the cause of 20% of neonatal deaths in India. About 2.8 and 5.6% of all live births had moderate and severe asphyxia, respectively, in a large hospital-based study; the case fatality rate was relatively low at ~8.7% [4].

Perinatal asphyxia is one of the major causes of early neonatal mortality in India. Among the institutional births, incidence is 5% and accounts for 24.3% of neonatal deaths [5].

Postasphyxial Hypoxic Ischemic Encephalopathy (HIE) occurs in approximately 1 to 2 infants per 1000 live term births in developed countries but in developing countries its incidence is expected to be much higher [6]. Among term infants, 6% to 23% of cases of cerebral palsy (CP) are attributable to intrapartum asphyxia [7]. Predictions of long-term

Corresponding Author: Anjali Kher, Associate Professor, Department of Pediatrics, Jawaharlal Nehru Medical College, DMIMS, Sawangi (M), Wardha, Maharashtra 442004, India.
E-mail: anjalikher@gmail.com

Received on 10.08.2018, **Accepted on** 27.08.2018

out come in the immediate neonatal period are based on clinical, biochemical, electrophysiological and imaging findings.

HIE is characterized by clinical and laboratory evidence of acute or subacute brain injury due to asphyxia.

Systemic hypoxemia and/or reduced cerebral blood flow (CBF) in utero or postnatally are primary causes of HIE.

Perinatal asphyxia is an insult to fetus or newborn due to lack of oxygen (hypoxia) or lack of perfusion (ischemia) to various organs of sufficient magnitude and duration.

In a study conducted at Thailand, significant risk factors for HIE were inappropriate antenatal care, vacuum extraction, male sex, prolapsed cord and 1 and 5-minute low Apgar scores [8].

In HIE neonate may have low Apgar scores at delivery and metabolic acidosis is documented in the cord blood. Within the first 24 hours of life, the infant may develop symptoms of apnea and seizures. Neurosonography (NSG) provides a convenient, non invasive screening examination of the hemodynamically unstable neonate at the bedside. NSG findings suggestive of HIE include hyperechogenicity of involved structures and/or abnormal Resistive Index (RI) on duplex Doppler images. But NSG is operator dependent and less sensitive to structural abnormalities in the cerebral convexity and in the brainstem. CT brain is the least sensitive modality for evaluation of HIE and most sensitive and specific is MR imaging, diffusion-weighted MR imaging and MR spectroscopy [9].

Perinatal asphyxia and HIE are still a major contributor to neonatal mortality and morbidity irrespective of improved obstetric and perinatal care. In view of paucity of studies on perinatal asphyxia and HIE in central rural India this study was undertaken with following objectives:

1. To study clinical, biochemical and NSG parameters of neonates with HIE.
2. To study the risk factors for HIE.
3. To study the short term outcome of neonates with HIE.

Materials and Method

After seeking approval from IEC (Ref.No. DMIMS (DU) / IEC/2016-15/5099) this study was undertaken in NICU (Inborn), Department of Pediatrics AVBRH, Sawangi (M), Wardha. It was a

prospective observational case study conducted from December 2016 to December 2017. All neonates delivered in our hospital and admitted to NICU (Inborn) with birth asphyxia and HIE were included in the study.

The neonates included in study fulfilled following criteria [10,11]:

1. Moderate birth asphyxia: Slow/gasping breathing or an Apgar score of 4 to 6 at 1 minute
2. Severe birth asphyxia: No breathing or an Apgar score of 0-3 at 1 minute of age
3. Umbilical cord blood gas analysis within 1st hour of birth with ph <7.2
4. HIE stage I, II & III according to Sarnath & Sarnath clinical staging of HIE [12].

Exclusion Criteria

1. Neonates with congenital malformations, infections, chromosomal abnormalities, inborn errors of metabolism, dysmorphic syndromes and still born.
2. Neonates who died or were discharged within 24 hours of birth.

A detailed maternal obstetric history was taken in a predesigned prevalidated proforma, to assess the role of maternal factors along with detailed history and examination of all neonates included in the study. The neonates were assessed and followed closely to assign a stage of HIE as stage I, II and III (mild, moderate and severe) according to the Sarnath and Sarnath clinical staging of HIE. The clinical course, complete blood count (CBC), C- reactive protein (CRP), Blood culture along with cord blood pH and gases and NSG were done and reports were entered in the pre-validated proforma. On discharge, babies were assessed for abnormal neurological signs like tone abnormalities, Moro reflex, feeding difficulty. The short term outcome and condition of neonates at discharge were noted. Birth asphyxia was considered to be severe if cord blood pH was < 7.

Statistical analysis were performed on Microsoft excel and SPSS software. Data was summarized using descriptive statistics. Categorical variables are presented as number and percentage. Chi-square test was used to compare the association among two or more categorical variables. All statistical tests were two-tailed and alpha level of significance was set less than 5%. A p value of <0.05 was taken as statistically significant.

Results

Total number of live births in our hospital during study period were 2830, out of them 84 (2.97%) neonates had birth asphyxia that is incidence of birth asphyxia was 29.7 per 1000 live births. Out of 2830 live births 50 (1.76%) had HIE which means 17.6 per 1000 live births was incidence of HIE. Birth asphyxia accounted for 14.7% of admissions to NICU, out of 571 admissions in inborn NICU 84 had birth asphyxia. Out of 84 birth asphyxia neonates 50 (59.5%) developed HIE (Table 1 Graph 1).

Out of 2830 live births 1367 (48.3%) were normal vaginal deliveries (NVD) and 1463 (51.7%) were lower segment cesarean section (LSCS). In the study group 50 neonates with HIE were included, 32 (64%) were males and 18 (36%) were females with M:F ratio of 1.8:1, of the study group 26 (52%) were born by LSCS and 24 (48%) were born by NVD.

In study group 35 (70%) were full term (FT), 15 (30%) were preterm, 30 FT were AGA, 5 were SGA and all 15 preterm neonates were AGA thus 90% were AGA and 10% were SGA.

In cases maternal risk factors associated were, 5 (10%) had preeclampsia, 5 (10%) had oligohydramnios,

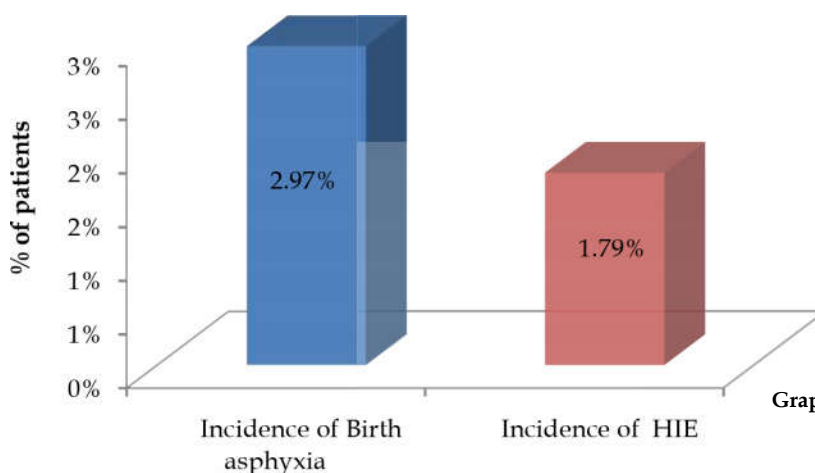
4 (8%) had meconium stained amniotic fluid (MSAF), 2 (4%) had polyhydramnios, 2 (4%) had gestational diabetes mellitus (GDM), 2 (4%) mothers had severe anemia, 1 (2%) had premature rupture of membranes (PROM) > 12 hours and 1 (2%) had prolonged labor.

Out of 50 neonates with HIE 13 (26%) were in stage I, 27 (54%) were in stage II and 10 (20%) were in stage III. Umbilical cord pH showed that 10 (20%) neonates had severe acidemia and 40 (80%) had moderate acidemia. Out of 10 neonates with severe acidemia 5 (50%) died. In 20 (40%) cases CRP was positive and blood culture was positive in 6 (12%) of cases. NSG changes were seen in 2 (4%) neonates, out of 2 neonates with mild prominence of bilateral lateral ventricles on NSG one died. In short term outcome 41 (82%) were discharged and 9 (18%) died, mortality rate was 18%. All 9 neonates who died were in HIE stage III. One newborn with HIE stage III was discharged, in neonates with HIE stage II out of 27 neonates 13 had good cry, tone activity at discharge and 14 had fair cry tone activity at discharge. All 13 cases with HIE stage I at discharge had good cry, tone and activity. Out of these 9 deaths 3 (33%) neonates had associated culture positive sepsis of which 2 (66%) had

Table 1: Incidence of Birth Asphyxia & HIE

Variables	Frequencies
Live births during study period	2830
Neonates with Birth asphyxia	84 (2.97%)
Neonates with HIE secondary to birth asphyxia and admitted to NICU (study population)	50 (1.76%)
Incidence of Birth asphyxia	29.7/1000 live births or 2.97%
Incidence of HIE	17.6/1000 live births or 1.76%

Abbreviations: HIE- Hypoxic Ischemic Encephalopathy
NICU- Neonatal Intensive Care Unit



Graph 1: Incidence of Birth Asphyxia & HIE-

Table 2: Clinical Profile of HIE associated with Birth Asphyxia

	Number	Percentage
Sex		
Male	32	64%
Female	18	36%
Mode of Delivery		
NVD	24	48%
LSCS	26	52%
Gestational age		
SGA	5	10%
AGA	45	90%
Risk factors		
MSAF	4	8%
Preeclampsia	6	12%
Oligohydramnios	5	10%
Polyhydramnios	2	4%
GDM	2	4%
Severe Anemia	2	4%
PROM>12 hrs	1	2%
HIE Stages (Sarnath & Sarnath)		
HIE Stage I	13	26%
HIE Stage II	27	54%
HIE Stage III	10	20%
Umbilical cord blood pH		
pH 7 to 7.2	40	80%
pH < 7	10	20%
NSG		
Normal	48	96%
Abnormal	2	4%

Abbreviations:

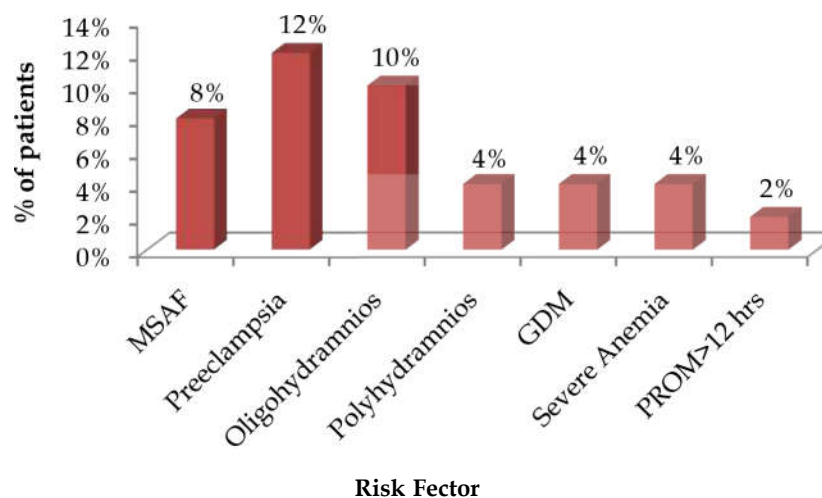
NVD- Normal Vaginal Delivery
 LSCS- Lower Segment Cesarean Section
 SGA- Small for Gestational Age
 AGA- Appropriate for Gestational Age
 MSAF- Meconium Stained Amniotic Fluid
 GDM- Gestational Diabetes Mellitus
 PROM- Premature Rupture of Membranes
 HIE- Hypoxic Ischemic Encephalopathy
 NSG- Neurosonography

Pseudomonas and 1 (33%) had *Klebsiella* in their blood culture (Table 2 Graph 2).

When the two groups discharged and expired were compared there was no significant difference between the two groups regarding gender, parity, gestational age and maternal risk factors (p value>0.05). But there was significant difference in staging of HIE and short term outcome, all neonates in expired group were in HIE stage III and all those who were discharged were in HIE stage I and II (p value = 0.0001, S).

Severe acidemia in cord blood (pH<7) was associated with increased mortality and moderate acidemia (pH 7 -7.2) neonates had better short term outcome (p=0.0032, S).

Anemia and high mortality had significant correlation (p=0.0009, S) and positive blood culture also was associated with mortality (p=0.029, S) (Table 3 Graph 3).

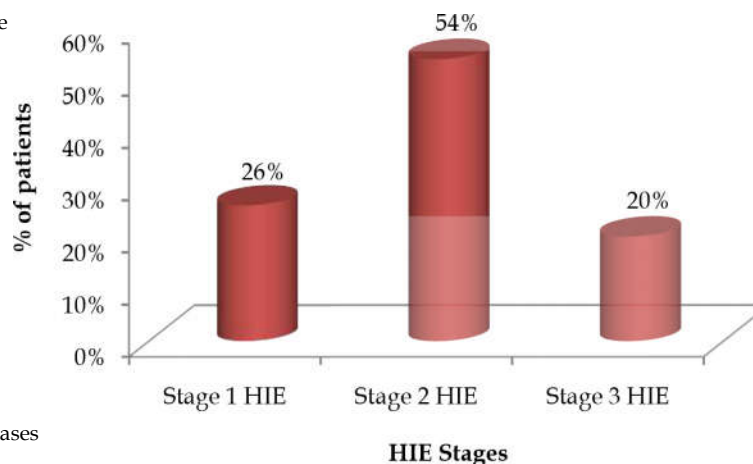


Graph 2: Maternal Risk factors in HIE with birth asphyxia

Table 3: Risk factors, HIE staging, clinical profile and their distribution in 2 groups (Discharged & Expired)

	Discharged (n=41)	Expired n=9)	p- value
Gender	24	6	0.94,NS
Male	17	3	
Female			
Parity	22	4	0.61,NS
Primipara	19	5	
Multipara			
Gestational Age	28	7	0.57,NS
Full Term	13	2	
Preterm			
Maternal Risk factors-			
MSAF	3	1	0.56,NS
Pre-eclampsia	5	1	1.00,NS
Oligohydramnios	3	2	0.21,NS
Polyhydramnios	1	1	0.33,NS
GDM	2	0	1.00,NS
Severe Anemia	1	1	0.33,NS
PROM>12 hrs	1	0	1.00,NS
Prolonged Labour	0	1	0.18,NS
HIE Stage I	13	0	0.0001,S
HIE Stage II	27	0	
HIE Stage III	1	9	
Umbilical cord blood pH			
pH 7 to 7.2	36	4	0.0032,S
pH < 7	5	5	
NSG			
Normal	40	8	0.22,NS
Abnormal	1	1	
Hb%	2	4	
Anemia (Hb% < or = 13.6 gm %)	4	0	
Septic screen	0	0	0.0009,S
Leucopenia (< 9000/ mm ³)	7	2	0.23,NS
Leucocytosis (> 30,000/mm ³)	1	0	0.029,S
Thrombocytopenia (<1,50,000)	14	6	
Thrombocytosis (> 4,00,000)	3	3	
CRP	(Micrococci-2	(Psuedomonas-2	
Blood Culture	Psuedomonas-1)	Kliebsiella-1)	

Abbreviations: MSAF- Meconium Stained Amniotic Fluid
 GDM- Gestational Diabetes Mellitus
 PROM- Premature Rupture of Membranes
 HIE- Hypoxic Ischemic Encephalopathy
 NSG- Neurosonography
 Hb%- Hemoglobin percentage
 CRP- C Reactive Protein

**Graph 3:** Staging of HIE in cases

Discussion

This study was undertaken to know the clinical, biochemical and neurosonographic parameters, risk factors and short term outcomes of neonates with HIE secondary to birth asphyxia in NICU of our hospital. The study group consisted of 50 neonates with HIE secondary to birth asphyxia admitted in inborn NICU over a period of 12 months. The incidence of birth asphyxia was 29.7 per 1000 live births or 2.97%. The incidence of HIE secondary to birth asphyxia was 17.6 per 1000 live births or 1.76%. This was similar to a study by Bhunia NS [13] in which incidence rate of perinatal asphyxia and HIE was 28.5 /1000 live births and 19.97/1000 term live births respectively. Padayachee N [14] in his study found that incidence of perinatal asphyxia was 4.7/1000 live births, and that of HIE was 3.6/1000 live births which was much less as compared to this study. In Siva Saranappa SB [15] study frequency of perinatal asphyxia was 5.1% which was slightly high than our study and this could be due to inclusion of neonates born outside their hospital and referred within one hour of birth. However this incidence does not reflect the true incidence as most of the data were hospital based and was of single centre, which does not reflect the true incidence in general population. In this study 59.5% of birth asphyxia neonates developed HIE these findings are similar to study by Lohan R [16] in which 42% of birth asphyxia neonates developed HIE but in study by Siva Saranappa SB [15] 31.66% developed HIE, whereas in a study by Thakkar PA [17] 77.7% of full term neonates with severe birth asphyxia developed HIE and 83% developed HIE of all full term birth asphyxia cases in study by Bhunia NS [13] which are not similar to our findings. In this study according to Sarnath & Sarnath staging for HIE 26% were in HIE stage I, 54% were in HIE stage II & 20% in HIE stage III this distribution was not similar to study by Lohan R [16] where 23.81% were HIE-I, 33.33% HIE-II and 42.86% HIE-III and in study by Bhunia NS [13] 43.42% cases were in Stage I HIE, 28.07% were in stage II HIE and 28.91% were in stage III HIE.

Maternal risk factors associated with birth asphyxia were preclampsia, oligohydramnios, meconium stained liquor but were not statistically significant. Thakkar PA [17] in his study found that maternal risk factors associated with increased neonatal mortality were prolonged labour, oligohydramnios, polyhydramnios and severe anaemia. Whereas MSAF was significant risk factor associated in study by Siva Saranappa SB [15] and in study by Bhunia NS [13] prolonged labour,

primiparity, fetal distress were significant risk factor.

In this study 20% neonates had severe acidemia ($\text{pH} < 7$) and 50% of these neonates expired, 88% of discharged neonates had moderate acidemia (pH 7-7.2) and these findings were statistically significant. Siva Saranappa SB [15] study 35% had severe acidemia and out of them 23.8% expired.

In this study overall mortality rate was 18% which is similar to Padayachee N [14] in which overall mortality rate was 13.3% and to study by Lohan R¹⁶ in which overall mortality was 14% but it was higher the Siva Saranappa SB [15] study in which overall mortality rate was 8% but was less as compared with Bhunia NS [13] study in which overall mortality rate was 31%. Mortality was higher in HIE III which was 90% whereas in HIE stage I & II there was no mortality in present study and these findings are similar to Bhunia NS [13] study where in HIE stage III mortality was 93.84% and in HIE II it was 15.62% and in Siva Saranappa SB [15] study mortality in HIE stage III neonates was 100%.

Conclusion

Birth asphyxia and HIE still are one of the major causes for admission of neonates to NICU and one of the leading causes which contribute to early neonatal mortality and morbidity. Stringent monitoring for well being of mother and baby throughout entire pre-conceptional and antenatal period right through labour and during early neonatal period with early efficient management will definitely help to further reduce the incidence of HIE and will improve the outcome of these newborns. Further research is needed for monitoring, diagnosis and newer modalities of treatment for HIE in neonates and more long term follow up studies should be undertaken in future.

Acknowledgement

Nil

Conflict of Interest: Nil

Key Message

Birth asphyxia and HIE are still a major cause of neonatal morbidity and mortality, research should be undertaken in preventive and treatment modalities for HIE and Birth Asphyxia.

References

1. Liu L, Oza S, Hogan D, Perin J, Rudan I, Lawn JE et al. Global, regional, and national causes of child mortality in 2000–13, with projections to inform post-2015 priorities: an updated systematic analysis. *Lancet* 2015;385(9966):430–440 [PubMed].
2. Registrar General of India. Sample registration system (SRS) statistical report 2013. New Delhi: 2013.
3. Lawn JE, Cousens S, Zupan J 4 million neonatal deaths: when? where? why? *Lancet*. 2005;365:891-900.
4. National Neonatal Perinatal Database. Report for the year 2002–03. Available at http://www.newbornwhocc.org/pdf/nnpd_report_2002-03.PDF (accessed on 2 April 2016).
5. Neonatal Morbidity and Mortality. Report of the National Neonatal Perinatal Database. *Indian Pediatr* 1999;36:167-69.
6. Low JA, Lindsay BG, Derrick EJ Threshold of metabolic acidosis associated with newborn complications. *Am J Obstet Gynecol*. 1997;177:1391-94.
7. Volpe JJ. Hypoxic-ischemic encephalopathy: intrauterine assessment. In: Volpe JJ, ed. *Neurology of the Newborn*. Philadelphia, Pa: WB Saunders Co; 2000, pp.277-95.
8. Futrakul S, Praisuwanna P, Thaitumyanon P. Risk factors for hypoxic ischemic encephalopathy in asphyxiated newborn infants. *J Med Assoc Thai*. 2006; 89(3):322-328.
9. Christine P. Chao, Christopher G. Zaleski, Alice C. Patton. Neonatal Hypoxic-Ischemic Encephalopathy: Multimodality Imaging Findings. *Radio Graphics*. 2006; 26:S159–S172.
10. World Health Organization. Perinatal mortality: a listing of available information. *FRH/ MSM*. 96.7. Geneva: WHO, 1996 & Report of the National Neonatal Perinatal Database (National Neonatology Forum, India) 2003.
11. Committee on fetus and newborn, American Academy of Pediatrics and Committee on obstetric practice, American College of Obstetrics and Gynecology. Use and abuse of the APGAR score. *Pediatr* 1996;98: 141-142.
12. Sarnat HB, Sarnat MS: Neonatal encephalopathy following fetal distress: A clinical and electroencephalographic study. *Arch Neurol* 1976;33: 695-706.
13. Bhunia NS, Saharia NP. A Profile of Hypoxic Ischaemic Encephalopathy in Neonatal Intensive Care Unit, Gauhati Medical College and Hospital, Guwahati. *Curr Pediatr Res* 2015;19(1&2):73-78.
14. Padayachee N, Ballot DE. Outcomes of neonates with perinatal asphyxia at a tertiary academic hospital in Johannesburg, South Africa. *S Afr J CH* 2013;7(3):89-94. DOI:10.7196/SAJCH.574.
15. Siva Saranappa SB, Nair CC, Madhu G N, Srinivasa S, Manjunath MN. Clinical profile and outcome of perinatal asphyxia in a tertiary care centre. *Curr Pediatr Res* 2015;19(1 & 2):9-12.
16. Lohan R, Chauhan G, Dalal A, Gupta SP, Bhardwaj AK, Ahlawat M. A Study on Incidence of Clinical Hypoxic Ischemic Encephalopathy Injury in Newborns with Perinatal Asphyxia. *Int J Pediatr Child Care* 2016;1(1):4.
17. Thakkar PA, Valia P, Parmar N, Javadekar B, Ukti P. Clinical profile, outcome and clinical indicators for poor prognosis in full term babies born with severe birth asphyxia: study from tertiary care hospital from western India. *Int J Contemp Pediatr* 2017 Mar; 4(2):470-76.

Estimation Early Neonatal Hyperbilirubinemia Utilizing 24-hour Serum Bilirubin Level at Bhuj, Kutch

Arun Parikh¹, Abhikumar Shingala²

¹Associate Professor, Dept. of Paediatrics, Gujarat Adani Institute of Medical Science, Bhuj, Kutch, Gujarat 370001, India. ²MPH candidate, Class of 2018, Tennessee State University.

Abstract

Background and Aim: Jaundice is the frequent irregular physical finding in the first week of life. Jaundice is the noticeable form of hyperbilirubinemia. The present study was performed with an aim to evaluate the predictive value of TSB=6 mg/dl at 24±6 hours of age in identifying infants. **Material and Methods:** The present forthcoming study was performed at Department of Pediatrics, at a tertiary care hospital, Bhuj. All fit neonates were to be examined for TSB levels at 24 hours and over again at 5 days. Serum bilirubin was expected for all registered cases within 18 to 30 hours of life spectrophotometrically utilizing twin Beam method. Children after that followed up clinically for the manifestation and development of jaundice every 12 hours till discharge from the department. TSB estimation was repeated if the clinical assessment of jaundice was more than 10 mg/dl by any observer using Kramers Rule. **Results:** A total of 199 neonates were originally enrolled. 37 of 199 cases did not follow up. Significant Hyperbilirubinemia was present in 20.9% cases of those babies who developed jaundice. A TSB of <6mg/dl at 24+6 hours was there in 99 infants. In the due 49 cases the TSB at 24+6 hours was >6 mg/dl. 20 cases out of this eventually went on to develop a positive study outcome. Sensitivity of TSB at 24+6 hours >6mg/dl in recognizing those who will have hyperbilirubinemia =90.9%, Specificity was 76.12%, Positive predictive value was 41.6% and Negative predictive value was 97.9%. **Conclusion:** The current research establishes that a TSB at 24+6 hours <6mg/dl has an elevated predictive value in recognizing those infants who are doubtful to build up consequent hyperbilirubinemia and these neonates can be released before time from the hospital.

Keywords: Infants; Hyperbilirubinemia; Jaundice; Sensitivity; Specificity.

Introduction

Jaundice is the frequent irregular physical finding in the first week of life. Jaundice is the noticeable form of hyperbilirubinemia. It emerges in infant skin at Serum Bilirubin >5 mg/dl. Jaundice arises in 60% of term and 80% of preterm infants. However, noteworthy jaundice occurs in 6% of term babies and is the mainly widespread cause for admission after early hospital discharge [1].

Physiologic jaundice frequently emerges on the 2nd to 3rd day of life, generally cresting by 3rd to 4th day to 6-8 mg/dl and after that fall. In early infants, the crest may be 10-12 mg/dl on the fifth day of life, perhaps increasing over 15mg/dl devoid of any exact irregularity [2].

Bilirubin which is a non-polar complex is bound to albumin and accepted to liver, where it is in use up by hepatocytes. Bound to a cytoplasmic protein, ligandin, it is passed to the endoplasmic reticulum for conjugation with glucuronide [3-7].

Unconjugated hyperbilirubinemia may be grounds to numerous issues that enhance the load of bilirubin. Apprehensions concerning jaundice have augmented after reports of Bilirubin induced brain damage happening in healthy term infant. Elevated levels of unconjugated bilirubin are potentially neurotoxic and can guide to extensive brain injury mainly brutally to Basal ganglia.

Early release of strong term newborn after delivery has turned out to be a widespread practice since financial restraints, only some hospital beds and

Corresponding Author: Arun Parikh, Associate Professor, Dept. of Paediatrics, Gujarat Adani Institute of Medical Science, Bhuj, Kutch, Gujarat 370001, India.

E-mail: drpiyushpujara@gmail.com, researchguide86@gmail.com

Received on 04.05.2018, Accepted on 22.05.2018

huge patient load [8-10]. Therefore, the ideas of forecast of Jaundice proffer a gorgeous alternative to choose up babies at risk of neonatal hyperbilirubinemia. It is extremely significant in background of developing country like India wherever expensive investigations and usual follow up is further than the achieve of the huge mainstream.

Material & Methods

The present forthcoming study was performed at Department of Pediatrics, at a tertiary care hospital, Bhuj. All fit neonates were to be examining for TSB levels at 24 hours and over again at 5 days. If clinical jaundice emerged in between TSB examine was done straight away and then every day till 5 days of age and the maximum interpretation was noticed as max out TSB. Hyperbilirubinemia was distinct as TSB level >17 mg/dl. Inclusion criteria were: All infants with Gestational age ≥ 35 weeks and neonatal evaluation by prolonged New Ballard Score and nonexistence of important sickness. Infants of Rh-negative mothers were incorporated simply if the child was also Rh-negative.

Methodology

All children delivered the prior day in the labour room were inspected and a thorough antenatal and postnatal history was recorded. The blood sample of infant was sending for grouping and TSB opinion. The babies were then followed up clinically by 2 observers for the manifestation and sequence of jaundice each 12 hour till discharge from the department TSB assessment was repetitive if the clinical measurement of jaundice was more than 10 mg/dl by several observer using Kramers Rule. Hyperbilirubinemia was defined as TSB level ≥ 12 mg/dl between 24 to 48 hour of life ≥ 15 mg/dl between 48 to 72 hour of life and 17 mg/dl beyond 72 hours of life.

Bilirubin assessment was completed spectrophotometrically by twin beam method and examined by Wako Bilirubin Tester Model SE 101 DII.

Statistical Analysis

The data was coded and entered into Microsoft Excel spreadsheet. Analysis was done using SPSS version 15 (SPSS Inc. Chicago, IL, USA) Windows software program. The variables were assessed for normality using the Kolmogorov-Smirnov test. Descriptive statistics were calculated.

Results

A total of 199 neonates were originally enrolled. 37 of 199 cases did not follow up. Seven infants were admitted to NICU afterward with a analysis of septicemia and were removed from study. All these three cases were excluded from study. Absolute information was accessible for 150 infants. Significant Hyperbilirubinemia was present in 20.9% cases of those babies who developed jaundice. A TSB of <6 mg/dl at 24+6 hours was there in 99 infants. Only two infants after this developed hyperbilirubinemia subsequently.

In the due 49 cases the TSB at 24+6 hours was >6 mg/dl. 20 cases out of this eventually went on to develop a positive study outcome. Sensitivity of TSB at 24+6 hours >6 mg/dl in recognizing those who will have hyperbilirubinemia =90.9%, Specificity was 76.12%, Positive predictive value was 41.6% and Negative predictive value was 97.9%.

Table 1: Showing the incidence of Clinical Jaundice and significant Hyperbilirubinemia

Total enrollments	Clinical Jaundice present	Percentage	Significant Hyperbilirubinemia (>17 mg/dl)	Percentage
150	107	71.03	21	14.01

Discussion

The current research establishes that a TSB level ≤ 6 mg/dl at 24 \pm 6 hour can be utilized to calculate the reduce jeopardy for subsequent hyperbilirubinemia. Bhutani et al. [10] described that infants who develop hyperbilirubinemia have larger serum bilirubin levels, the authors found that 6.1% of neonates had pre-discharge serum bilirubin, 32.1% of these infants demonstrates hyperbilirubinemia afterward [10]. Alpay, et al accounted that TSB levels of [3] 6 mg/dl in the first 24 hour forecast jaundice in all infants consequently [5]. similar finding was reported by Awasthi et al. [11] and Agarwal et al. [12].

Grover et al. [13] described the mean primary day TSB worth in the neonates who consequently developed hyperbilirubinemia was 7.716 mg/dl contrast to a value of 5.154 mg/dl in those who did not, statistically significant difference was observed in above findings. ($p \leq 0.05$)

Lavanya et al. [14] described mean period of commencement of significant jaundice to be 61 ± 32 hours. The mean period of phototherapy was 49 ± 26 hours. In the findings by Chawla et al. [15] 997 neonates were registered, of which 931 completed follow up. Amongst registered neonates 34.5% were low birth weight. Bilirubin nomogram was build utilizing 40th, 75th and 95th percentile values of hour-specific bilirubin. Between 49 neonates with pre-discharge STB in high danger region 34 developed SHB Among 342 neonates with pre-discharge STB in low risk zone, 32 developed PHB. Region under curvature for this risk measurement strategy was 0.73. Hour-specific bilirubin nomogram and STB dimension can be utilized for forecasting consequent require of phototherapy. Additional studies are desirable to authenticate presentation of risk separation zones definite in this hour-specific bilirubin nomogram.

Conclusion

The current research establishes that a TSB at 24+6 hours $<6\text{mg/dl}$ has a elevated predictive value in recognizing those infants who are doubtful to build up consequent hyperbilirubinemia and these neonates can be released before time from the hospital.

Sources of funding: Nil.

Conflict of interest: None declared

References

1. Narang A, Gathwala G, Kumar P. Neonatal Jaundice: An analysis of 551 cases. *Indian Pediatr* 1997;34:429-32.
2. Singhal PK, Singh M, Paul VK, Deorari AK, Ghorpade MG. Spectrum of neonatal hyperbilirubinemia: An analysis of 454 cases. *Indian Pediatr* 1992;29:319-25.
3. Brown AK, Johnson L. Loss of concern about jaundice and the reemergence of kernicterus in the era of managed care. In: Fanroff AA, Klaus MH, Eds. *The Year Book of Neonatal and Perinatal Medicine*. Philadelphia; Mosby, 1996.pp.17-28.
4. Maisels MJ, Newman TB. Kernicterus in otherwise healthy, breast-fed term neonates, *Pediatrics* 1995;96:730-33.
5. Alpay F, Sarici SU, Tosuncuk HD, Serdar MA, Inanc N, Gokcay E. The value of first day bilirubin measurement in predicting the development of significant hyperbilirubinemia in healthy term newborns. *Pediatrics* 2000;106:e16.
6. American Academy of Pediatrics. Practice parameter: Management of hyperbilirubinemia in the healthy team newborn. *Pediatrics* 1994;94:558-67.
7. Davidson LT, Merrit KK, Weech AA. Hyperbilirubinemia in the newborn. *Am J Dis Child* 1941;61:958-80.
8. Knudsen A. Prediction of the development of neonatal jaundice by increased umbilical cord blood bilirubin. *Acta Paediatr Scand* 1989;78:217-21.
9. Maisels JM, Newman TB. Predicting hyperbilirubinemia in newborn: the importance of timing. *Commentary. Pediatrics* 1999;103:493-94.
10. Bhutani VK, Johnson L, Sivieri EM. Predictive ability of a pre discharge hour - specific serum bilirubin for subsequent significant hyperbilirubinemia in healthy term and near term new borns. *Pediatrics* 1999; 103:6-14.
11. Awasthi S, Rehman H. Early prediction of neonatal hyperbilirubinemia. *Indian JPediatr* 1998;65:131-39.
12. Agarwal R, Kaushal M, Agarwal R, Paul VK, Deorari AK. Early neonatal hyperbilirubineamia using first day serum bilirubin level. *Indian Pediatrics* 1997;34:429-32.
13. Shivani Randev and Neelam Grover: Predicting Neonatal Hyperbilirubinemia Using First Day Serum Bilirubin Levels [*Indian J Pediatr* 2010;77(2):147-50.
14. K Radha Lavanya, Ashish Jaiswal, Pramod Reddy and Srinivas Murki: Predictors of Significant Jaundice in Late Preterm Infants: Received: June 27,2011; Initial review: July 12, 2011; Accepted: October 24, 2011.
15. Umesh Pathak, Deepak Chawla, Saranjit Kaur and Suksham Jain: Bilirubin Nomogram for Prediction of Significant Hyperbilirubinemia in North Indian Neonates October 04, 2012.

Spectrum of Acute Kidney Injury and its Clinical Profile and Outcome in Children Admitted to PICU

Basavaraj Patil¹, Savita Patil², Harsha S.³

¹Associate Professor ³Sr. Resident, Department of Pediatrics, Mahadevappa Rampure Medical College, Kalaburagi, Karnataka 585105, India. ²Consultant, Department of Obstetrics and Gynecology, Chinemayagiri Maternity & Children Hospital, Kalaburagi, Karnataka 585103, India.

Abstract

Objective: To determine incidence, risk factors, and outcome of acute kidney injury (AKI) in Pediatric Intensive Care Unit (PICU). **Materials and Methods:** This prospective observational study was conducted over period of 1½ years. All patients within the age group of 1 month to 18 years admitted in the PICU (Pediatric Intensive Care Unit) at Basaveshwar teaching and general hospital and Sangameshwar hospital attached to Mahadevappa Rampure Medical College during a period from December 2015 to August 2017. **Results:** Out of 1007 patient screened 7 patient were excluded, out of which 2 where congenital dysplastic kidney, 2 were known case of nephrotic syndrome, 3 where known case of chronic kidney disease. Out of 1000 patients 69 children had AKI, giving incidence of 6.9%. The median age of boys and girls were 4.56±3.84 and 4.49±4.01 respectively. 58% of the patients were boys. The median admission serum creatinine value in AKI patients was 2.91±2.48 mg/dL. In the present study, pre-renal causes accounted for (54) 78.3% of AKI. AKI Stage 1, 2, 3 was diagnosed in 11 (15.9%), 14 (20.3%) and 44 (63.8%) respectively. **Conclusions:** AKI continues to be associated with adverse outcomes, including high mortality, morbidity and prolonged hospital stay. Mechanical ventilation & Hypovolemia were independent risk factors. Higher the stage of AKI mortality increases. Early diagnosis of AKI using new defined criteria (AKIN, RIFLE, pRIFLE) [4,5,6] along with early and appropriate management of risk factors will prevent the progression of AKI and decrease the mortality and morbidity of AKI patients.

Keywords: Acute Kidney Injury; Critically Ill Children; Pediatric Intensive Care Unit; pRIFLE.

Background

Acute kidney injury (AKI) is an important condition in hospitalized patients, associated with adverse short- and long term outcomes. Mortality rates in critically ill children with AKI are high, ranging between 9% and 67% and increase if complicated by multiorgan failure, organ transplantation and acute respiratory distress syndrome. Most cases of incident AKI represent acute tubular necrosis (ATN) that is secondary to hypovolemia, sepsis or the use of nephrotoxic agents.

AKI is defined as rapid deterioration of renal function resulting in retention of nitrogenous wastes and inability of kidney to regulate fluid and electrolyte homeostasis. In the past, a lack of

objective diagnostic criteria has resulted in wide variability of definitions that have been used for this condition.

Recent reviews emphasize disparities in the definition of AKI have resulted in large variations in reported incidence and outcomes.

The definition and staging of AKI has been recently standardized using the RIFLE classification proposed by the Acute Dialysis Quality Initiative Group, and the one suggested by the Acute Kidney Injury Network (AKIN) [4,5,6].

These classifications have been examined in hospitalized adults and children, and found useful in characterizing AKI.

Most pediatric studies on the incidence of AKI are limited to the developed countries and are based on retrospective analysis of records.

Corresponding Author: Savita Patil, Consultant, Department of Obstetrics and Gynecology, Chinemayagiri Maternity & Children Hospital, Kalaburagi, Karnataka 585103, India.
E-mail: dr.basavarajpatil@gmail.com

Received on 05.08.2018, **Accepted on** 31.08.2018

The Spectrum and burden of AKI in developing countries may be different from that of developed countries. Only a few retrospective studies have been conducted to determine the incidence and profile of AKI in critically ill-children from the developing world in recent years.

Detection of the incidence, etiological profile and outcome of AKI is important for commencement of preventive and therapeutic strategies.

Objective

To determine the clinical profile of acute kidney injury in pediatric ICU.

To determine the outcome of patients with acute kidney injury in pediatric ICU.

Materials and Methods

This prospective observational study was conducted over period of 1½ years. All patients within the age group of 1 month to 18 years admitted in the PICU (Pediatric Intensive Care Unit) at Basaveshwar teaching and general hospital and Sangameshwar hospital attached to Mahadevappa Rampure medical college during a period from December 2015 to August 2017.

Informed parental consent was obtained from the parents prior to the study.

Serum levels of creatinine was estimated at admission and at daily intervals in PICU patients till discharge from PICU. Urine output measured and recorded as ml/kg/hour [4,5,6].

Diagnosis and staging of AKI was based on Acute Kidney Injury Network (AKIN) definition & classification [4,5,6].

Inclusion Criteria

Patients aged 1 month to 18 years, admitted to pediatric intensive care unit (PICU) (Basaveshwar Teaching and General Hospital and Sangameshwar Hospital, Kalaburagi)

Exclusion Criteria

Patients with known kidney disease such as congenital polycystic kidney disease.

Children who were diagnosed with chronic kidney disease on first visit.

Based on the AKI criteria, AKI was defined as abrupt (within 48h) reduction in kidney function with an increase in creatinine level. The illness was categorized as stage 1 (increase of creatinine by 1.5-1.99 times baseline), stage 2 (increase to 2-2.99 times baseline) and stage 3 (increase to ≥ 3 times baseline).

Shock was defined in presence of tachycardia, feeble pulses, cool peripheries, hypotension (blood pressure < 2 SD for age and sex) or capillary filling time > 3 seconds. Sepsis was the presence of systemic inflammatory response syndrome with suspected or proven infection.

Statistical Method

The biochemical and other numerical parameters was compared using t test, Z test, and chi-square or Fischer exact test and other applicable methods.

Data analysis was done using statistical software SPSS.

Results

Out of 1007 patient screened 7 patient were excluded, out of which 2 where congenital dysplastic kidney, 2 were known case of nephrotic syndrome, 3 where known case of chronic kidney disease. Out of 1000 patients 69 children had AKI, giving incidence of 6.9%. The median age of boys and girls were 4.56 ± 3.84 and 4.49 ± 4.01 respectively. 58% of the patients were boys. The median admission serum creatinine value in AKI patients was 2.91 ± 2.48 mg/dL. In the present study, pre-renal causes accounted for (54) 78.3% of AKI. AKI Stage 1, 2, 3 was diagnosed in 11 (15.9%), 14 (20.3%) and 44 (63.8%) respectively.

Table 1: Sex wise distribution of AKI cases and non-AKI cases

Age in years	AKI cases		Non AKI cases		Total	
	No.	%	No.	%	No.	%
Males	40	58.0	544	58.4	584	58.4
Females	29	42.0	387	41.6	416	41.6
Total %	69 (6.9%)	100.0	931 (93.1%)	100.0	1000 (100.0%)	100.0
X ² -test value & P-Value, sig	X ² = 0.0053 P>0.05, Not significant					

NS= not significant, S=significant, HS=highly significant, VHS=very highly significant

There were 40 (58.0%) male AKI cases and 29 (42.0%) female AKI cases in the study.

The sex ratio of total study cases of Male to Female was 1.4:1

The sex ratio of AKI cases of Male to Female was 1.38:1 this is almost same of the total cases

There was no statistical significant difference of sex among AKI and Non-AKI groups ($p>0.05$) (Table 1).

Statistically very highly significant difference of Anuria, Gross hematuria and Encephalopathy among AKI and Non-AKI groups ($p<0.001$) was observed and there were statistically significant differences of Vomiting, Loose motion among AKI and Non-AKI groups ($p<0.05$). The symptoms of Anuria, Gross hematuria, Encephalopathy,

Vomiting and Loose motion were significantly less in the non-AKI cases as compared to AKI cases.

There were no statistical significant difference of Oliguria, Fever, Seizures, Breathlessness and Gl. Hemorrhage among AKI and Non-AKI groups ($P>0.05$).

In the present study, the most common condition associated with AKI was sepsis, encephalitis. Other causes were dengue, pneumonia, DKA, Acute gastro enteritis, acute post-streptococcal glomerulonephritis, hemolytic uremic syndrome and congestive cardiac failure.

AKI was associated with increased mortality. Mortality rate was 34.8% compared to non AKI. In the present study, mortality was 9.1% in Stage 1, 28.5% in Stage 2 and 43.3% in stage 3. Mortality was high in stage 3.

Table 2: Comparison of Staging and outcome in the AKI cases

Staging	No of cases	Improved	Outcome Died	χ^2 -test values P-value & significance
1 st Stage	11	10(90.9%)	1(9.1%)	$\chi^2= 4.12$
2 nd Stage	14	10(71.5%)	4(28.5%)	$P<0.05$
3 rd Stage	44	25(56.8%)	19(43.3%)	S
Total	69	45(65.2%)	24(34.8%)	--

Table 3: Comparison of symptoms among AKI and Non-AKI cases

Symptoms	AKI cases (n= 69)	Non-AKI cases (n=931)	χ^2 -test values P-value & significance
Oliguria	3 (4.3%)	39 (4.2%)	$\chi^2=0.0072$ $P>0.05$, NS
Fever	53 (76.8%)	737 (79.2%)	$\chi^2=0.051$ $P>0.05$, NS
Vomiting	35 (50.7%)	344 (36.9%)	$\chi^2=5.09$ $P<0.05$, S
Loose motion	16 (23.2%)	154 (16.5%)	$\chi^2=5.31$ $P<0.05$, S
Anuria	5 (7.2%)	0 (0.0%)	$\chi^2=37.8$ $P<0.001$, VHS
Seizures	12 (17.4%)	176 (18.9%)	$\chi^2=0.73$ $P>0.05$, S
Breathlessness	28 (40.6%)	298 (32.0%)	$\chi^2=1.87$, $P>0.05$, NS
Gross hematuria	5 (7.2%)	1 (0.1%)	$\chi^2=31.82$, $P<0.001$, VHS
Gl. Hemorrhage	1 (1.4%)	0 (0.0%)	$\chi^2=0.431$, $P>0.05$, NS
Encephalopathy	8 (11.6%)	5 (0.5%)	$\chi^2=33.74$, $P>0.05$, VHS

NS= not significant, S=significant, HS=highly significant, VHS=very highly significant

Table 4: Comparison of outcome among AKI and Non-AKI cases

Groups	No of cases	Outcome		χ^2 -test values P-value & significance
		Improved	Died	
AKI cases	69	45(65.2%)	24(34.8%)	$\chi^2= 230.19$ P<0.000 VHS --
Non-AKI cases	931	922(99.0%)	9(1.0%)	
Total	1000	967(96.7%)	33(3.3%)	

VHS=very highly significant

There was statistically very highly significant difference of outcome in AKI and Non-AKI cases ($p < 0.001$).

The case fatality rate of Non-AKI was 1.0%. whereas the case fatality rate of AKI was 34.8%.

Overall death rate was 3.3%.

Hypovolemia and Need for Ventilation were significant risk factors for AKI ($p < 0.001$). In the present study, the median duration of PICU and Hospital stay was 9.98 ± 7.27 in AKI group compared to 7.41 ± 5.62 days in Non AKI group ($p < 0.001$)

Discussion

AKI is a clinical condition that commonly occurs in critically ill patients in the pediatric intensive care unit .studies has shown AKI is independently associated with poor outcome. Published data about AKI in Indian children are limited.

In the present study, the incidence of AKI in PICU was 6.9%. Compared to other Indian studies by Krishnamurthy et al. [2] and Mehta et al. [1]; however the incidence rate is lower.

In the present study, median age was 4.56% among boys and girls constituted 4.49%, 58% were boys among AKI patients which is comparable to Krishnamurthy et al study [2].

According to shweta naik et al. [3] presence of infection sepsis were significant predictors of AKI comparable to our study

In the present study hypovolemia and need for mechanical ventilation were significant risk factors similar to Shweta Naik et al. [3].

In the present study, the mean and standard deviation of Serum Creatinine value in AKI patients was 2.29 mg/dL, while in Krishnamurthy et al. study [2], it was 1.1 mg/dL.

In the present study, pre-renal causes accounted for (54) 78.3%of AKI. This is different from other previous studies such as Krishnamurthy et al. [2] and Mehta et al. [1] Garuda rama et al. [7] study prerenal

cases were more followed by renal and post renal .which is comparable to our study. In our study renal cases accounted for (13) 18.8%, post renal (2) 2.9%

The mortality in present study, mortality was 34.8%, which is comparable to Mehta et al study [1].

Conclusion

AKI continues to be associated with adverse outcomes, including high mortality , morbidity and prolonged hospital stay. Mechanical ventilation & Hypovolemia were independent risk factors. Higher the stage of AKI mortality increases. Early diagnosis of AKI using new defined criteria (AKIN, RIFLE, pRIFLE) along with early and appropriate management of risk factors will prevent the progression of AKI and decrease the mortality and morbidity of AKI patients.

Reference

1. Incidence of Acute Kidney Injury in Hospitalized Children: Poonam Mehta, Aditi Sinha, Abdus Sami, Mani Kalaivani, Ashima Gulati, Madhulika Kabra, Sushil K Kabra, Rakesh Lodha and Arvind Bagga, Indian Pediatrics; Volume 49 july 16, 2012.
2. Sriram Krishnamurthy, Parameswaran Narayanan, Sivaprakasam Prabha, Nivedita Mondal, Subramanian Mahadevan, Niranjana Biswal, and Sadagopan Srinivasan. Clinical profile of Acute Kidney Injury in a pediatric intensive care unit from southern India: A prospective observational study. Indian J Crit Care Med. 2013 Jul-Aug; 17(4): 207-213.
3. Shwetha Naik, Juoti Sharma, Rameshor Yengkom, Vijay Kalrao, Atul Mulay. Acute Kidney Injury in critically ill children: Risk factors and outcomes. Indian journal of critical care medicine. 2014 March;18(3): 129-33
4. Sean M. Bagshaw, Carol George, Rinaldo Bellomo. A comparison of the RIFLE and AKIN criteria for acute kidney injury in critically ill patients. Nephrol Dial Transplant 2008;23:1569-74.

5. Sharon Phillips Andreolli. Acute kidney injury in children *Pediatr Nephrol*. 2009 Feb;24(2):253-263.
 6. Schrier RW, Wang W, Poole B, Mitra A. Acute renal failure: definitions. Diagnosis, pathogenesis, and therapy. *J Clin Invest*. 2004 Jul;114(1):5-14.
 7. Garuda Rama. Study of Acute Kidney Injury in children: Its Aetiology, Clinical profile and Outcome. *Journal of Evidence based Medicine and Healthcare*; 2015 March 16;2(11): 1577-85.
-

Special Note!

Please note that our all Customers, Advertisers, Authors, Editorial Board Members and Editor-in-chief are advised to pay any type of charges against Article Processing, Editorial Board Membership Fees, Postage & Handling Charges of author copy, Purchase of Subscription, Single issue Purchase and Advertisement in any Journal directly to Red Flower Publication Pvt. Ltd. Nobody is authorized to collect the payment on behalf of Red Flower Publication Pvt. Ltd. and company is not responsible of respective services ordered for.

Survival and Major Morbidity Pattern of Preterm Infants Admitted in a Level -3NICU: A Retrospective Study

N. Venugopalan¹, Salahudeen², Pavan Kumar D.³, Vidya Singaravelu⁴, Manoj Narayanan K.A.⁵

¹Professor & Head ²Senior Resident ³Assistant Professor ⁴Associate Professor, Dept. of Pediatrics, DMWIMS Medical College, Naseera Nagar, Meppadi (P.O), Wayanad, Kerala 673577, India.

Abstract

Objectives: To assess the morbidity pattern and mortality among preterm infants admitted in Level - 3 NICU of a teaching hospital of a backward district of Kerala and to analyze the quality neonatal survival and cause of death in such preterm neonates. **Methods:** A retrospective study of all preterm infants admitted in the level - 3 NICU of a teaching hospital for a period of one year from 01/01/2017 based on hospital records. This includes both inborn and out born babies. Gestational age at delivery, mode of delivery, birth weight, major congenital anomalies, number of deaths and causes of deaths are recorded. Data are entered in Microsoft excel and data are correlated for any significance. **Results:** A total of 141 infants born below completion of 34 weeks of gestation were included in the study. 8 infants (6%) were of extremely low birth weight out of which only one died in the hospital. Six infants (4.25%) died during NICU stay. Major morbidities were present in 84% of all infants, and multiple morbidities were present in 66% infants. **Conclusions:** In a level 3 NICU of a teaching hospital 6% of babies were of extremely low birth weight and all except one survived. Death and morbidity are directly related to gestational age.

Keywords: Preterm Infants; Gestational Age; Neonatal Asphyxia.

Introduction

Preterm infants by definition are babies born before completion of 37 weeks of gestational age. These infants are at high risk of death and morbidity. Infants born before 28 weeks of gestation if also having multiple congenital anomalies are prone for neonatal asphyxia, respiratory distress, sepsis, hypoglycemia and prolonged neonatal hyper-bilirubinaemia. With advances in perinatal and neonatal care, more infants are surviving at lower gestational ages [1-3]. However, the rates of mortality and severe neonatal morbidity increase with decreasing gestational age [4-6]. Death and major morbidity can be avoided if prompt care is given to these infants in a level 3 neonatal ICU. This study highlights the favorable outcome among preterm

infants treated in a level 3 NICU of Wayanad, a relatively backward district of Kerala. Wayanad is an officially declared backward district of Kerala. It has the largest contingent (%) of tribal population in the state. About 18% of the district's population (about 11 L) are tribes. DMWIMS Medical College Hospital provide Level - 3 neonatal care and is the only institution with a level - 3 NICU in this hill district catering to a total population of about 20 lacks taking in to account the adjacent districts of Tamiladu and Karnataka.

Objectives

To assess the rates of mortality and morbidity among preterm infants admitted in NICU of a tertiary care hospital of a backward district of Kerala and to examine the cause of neonatal survival and death at each gestational age.

Corresponding Author: Manoj Narayanan, Associate Professor, Pediatrics, DMWIMS Medical College, Naseera Nagar, Meppadi (P.O), Wayanad, Kerala 673577, India.
E-mail: saisailesh.kumar@gmail.com

Received on 26.04.2018, **Accepted on** 05.05.2018

Methods

For the purpose of this study those babies born before 34 weeks of gestation alone are included. Those born after 34 weeks are neurologically mature enough to suck by themselves while feeding and are not routinely admitted in NICU for observation and stabilization.

This is a retrospective analysis of hospital data of infants born below 36 weeks of gestation for a period of one year from 1st January 2017 to 31st December 2017. This includes both inborn and out born infants admitted in the NICU of a tertiary care hospital during the study period. Gestational age at birth, maternal morbidity, birth weight of the infants, congenital anomalies, other comorbidities, number of hospital days, type of support provided and outcome are recorded. These data are entered in to Microsoft Excel for data analysis. Individual perinatal characteristics and maternal demographic characteristics are compared in the survivors and non survivors

Definitions

Moderate preterm: Gestational age from 32 to 36 completed weeks

Very preterm: Gestational age between 28 to 31 completed weeks

Extreme preterm: Gestational age less than 28 completed weeks.

Low birth weight: <2500g

Very low birth weight: <1500g

Extremely low birth weight: <1000g

Results

A total of 1036 babies were admitted in the NICU of the institution from 01-01-2017 to 31-12-2017. 947 were in born and 89 were born in other institution and referred in. Out of 1036, 141 (31.61%) were born below 34 weeks of gestation. These 141 babies were included in this retrospective cohort study. Mean birth weight increased with each increasing gestational week, from 354 g at 24 weeks to 2450 g at 34 weeks. 77 infants in the cohort were male (54.6%), and 64 (45.4%) were females (Table 1).

122 infants were born in this hospital and 19 were born in peripheral hospitals and referred for neonatal care (Figure 1).

Among these 15 (10.6%) were between 24-28 weeks of gestation, 21(14.9%) were between 29-31 weeks

Table 1: Sex distribution among preterm infants

Sex	No.	Percentage
Male	77	54.6
Female	64	45.4
Total	141	

of gestation and 105 (74.5%) were between 32-34 weeks of gestation (Figure 2). Out of these 20 (14.2%) were born of cesarean delivery. 8 infants

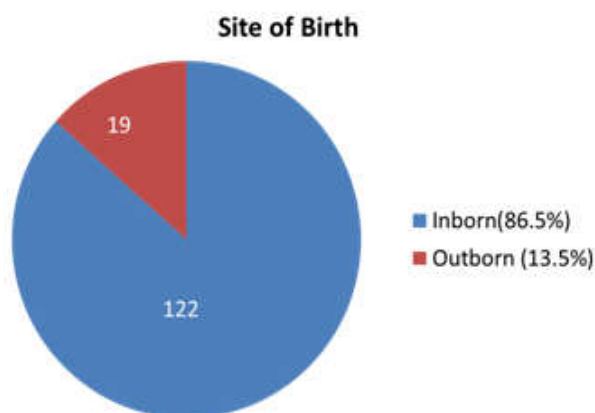


Fig. 1: Number of inborn and out born infants

were of extremely low birth weight (5.6%), 32 (22.7%) were between 1000-1500 gm and 101 (71.7%) were between 1500 to 2500 gm weight category (Figure 3). There were 6 deaths (4.25%) out of which only one was among the extremely low birth weight category (Figure 4). The total number of deaths in the NICU for the year was 12 out of a total admission of 1036. This shows that 6 babies out of 141 die constituting mortality of 4.26% for those below 34 weeks of gestational age. Only 6 out of the remaining 895 (above 34 weeks of gestation) died during the study period constituting a mortality of 0.67%.

Minimum stay in the hospital was 1 day and maximum was 79 days with a mean of 21 days. Out of 6 deaths 5 occurred in the first week and one in the third week. Major congenital heart diseases

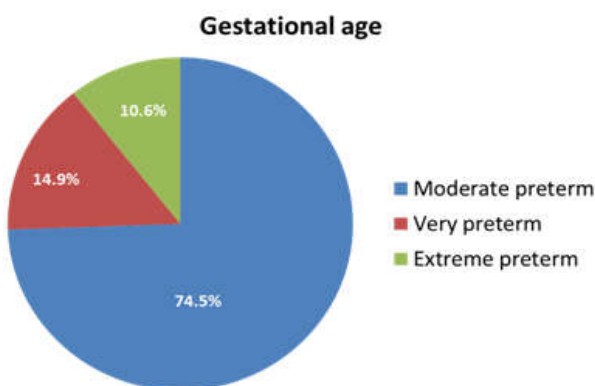


Fig. 2: Number of infants born at different gestational ages

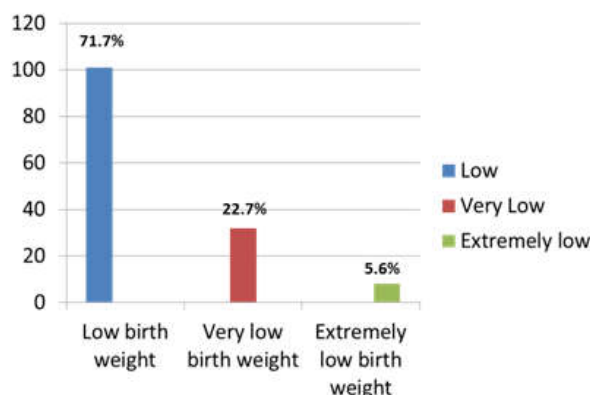


Fig. 3: Number of children in different weight groups

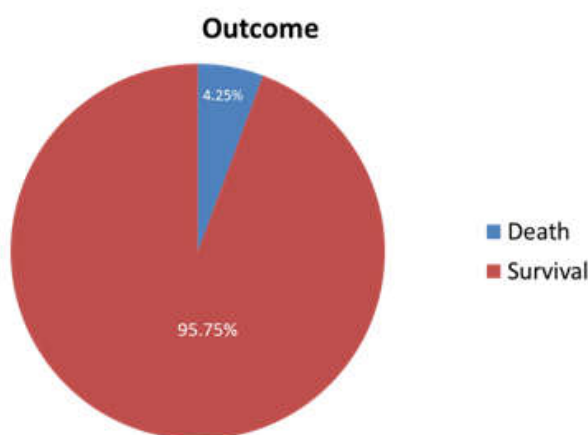


Fig. 4: Death among preterm infants

detected were ASD and PDA. Other problems were Hydrocephalus, Lung immaturity, Retinopathy of prematurity (ROP), other congenital anomalies like limb and chest wall abnormalities, jejunal atresia, undescended testes, tracheo esophageal fistula and hydro-ueronephrosis. Multiple major abnormalities were detected more in infants born below 31 weeks of gestation. Major associated comorbidities noted were perinatal asphyxia, hyaline membrane disease, respiratory distress, neonatal sepsis, hypoglycemia, neonatal hyper-bilirubinaemia, pneumothorax, metabolic acidosis and multiorgan dysfunction syndrome. Respiratory distress syndrome was present in 22 (15.6%) infants, ASD in 4 (2.8%) and Retinopathy of prematurity (ROP) in 6 (4.25%) infants.

All the preterm deliveries were attended by pediatrician. All preterm deliveries were informed in advance to NICU prior to delivery and most of the cases referred from outside are informed before being shifted from those institutions, so that NICU is prepared for receiving the baby. All babies < 34 weeks were shifted to NICU for preterm care. 22 (17%) infants needed resuscitation at birth. All the premature

infants were nursed in thermo-neutral environment (around 36.5°C). Oil is applied to skin to reduce convective heat loss and evaporative water loss. Stable babies are given kangaroo mother care to provide warmth. All preterm infants developed jaundice within the first week. If the baby is found to be jaundiced serum bilirubin is estimated and phototherapy started based on bilirubin level. Jaundice was not controlled by phototherapy in one infant and was subjected to exchange transfusion.

Discussion

In a hospital-based cohort of infants born between 24 and 34 weeks of gestation and taken care of in a level - 3 NICU, survival during the hospital stay was substantially good across all gestational age and birth weight. Although 84% of infants had one or other morbidity, all of them responded well to resuscitation and supportive care in the NICU.

Even though sepsis, respiratory distress and ROP have been reported among this cohort, it is less when compared to other studies [7-11]. Few other studies have shown higher rates of cerebral palsy, cognitive impairment, and behavioral problems among those born at very early gestations [12-15], and causing major neonatal morbidities and high risk for significant impairment in later life. These possible morbidities can be assessed only on subsequent follow up for a sufficiently long period after discharge from NICU.

The rates of attempted resuscitation for infants in our study were similar to several other studies [16]. These infants received interventions such as surfactant therapy, tracheal intubation, ventilator support, parenteral nutrition, or chest compressions. It is possible that the differences in resuscitation practices may influence survival and mortality. Previous studies have proposed individual decision-making in resuscitation of extremely preterm infants [17,18] whereas other studies have examined more major differences in resuscitation practice and subsequent outcomes at the hospital level [19-21]. Population-based studies and analyses are needed to gain a better understanding of the determinants of, and extent to, which regional practice variations influence mortality and quality of survival rates.

Differences in perinatal factors and maternal demographic characteristics have not influenced mortality. But increased birth weight and female sex, demonstrate favorable predictors for survival, as described by Tyson et al. [22]. Majority of extremely

preterm births in this cohort occurred in hospitals and this may be the reason for low mortality. Better survival was seen in infants born at a hospital with a regional-level NICU, which has been reported in other studies [23,24].

Future population-based studies are needed to examine neurodevelopmental and other outcomes in these infants after discharge from the hospital. Large, population-based studies of neurodevelopmental outcomes in survivors of extreme prematurity have been conducted in the United Kingdom [13,25], Sweden [26], Australia [27], and France [12].

Conclusions

This study provides information on mortality of preterm infants treated in a tertiary level-3 NICU in a backward district of Kerala state in India. It also explores the resuscitative and supportive care to the preterm infants and look at major morbidity among survivors. Mortality among this cohort is only 4.25% and only one baby among the 8 extremely low birth weight category was lost. This study highlight that survival can be considerably improved if delivery is conducted in an institution with level - 3 NICU facility and immediate NICU care is provided to all infants who need it. Even though there are multiple anomalies and comorbid conditions in these babies survival is comparable to other advanced NICUs.

References

1. Costeloe KL, Hennessy EM, Haider S, Stacey F, Marlow N, Draper ES. Short term outcomes after extreme preterm birth in England: comparison of two birth cohorts in 1995 and 2006 (the EPICure studies). *BMJ*. 2012;345:e7976pmid:23212881.
2. Hintz SR, Poole WK, Wright LL, et al; NICHD Neonatal Research Network. Changes in mortality and morbidities among infants born at less than 25 weeks during the post-surfactant era. *Arch Dis Child Fetal Neonatal Ed*. 2005;90(2):F128-F133. pmid:15724036.
3. Serenius F, Ewald U, Farooqi A, Holmgren PA, Håkansson S, Sedin G. Short-term outcome after active perinatal management at 23-25 weeks of gestation. A study from two Swedish tertiary care centres. Part 2: infant survival. *Acta Paediatr*. 2004;93(8):1081-89. pmid:15456200.
4. Ancel PY, Goffinet F, Kuhn P, et al. EPIPAGE-2 Writing Group. Survival and morbidity of preterm children born at 22 through 34 weeks' gestation in France in 2011: results of the EPIPAGE-2 cohort study [published correction appears in *JAMA Pediatr*. 2015;169(4):323]. *JAMA Pediatr*. 2015;169(3):230-238pmid:25621457.
5. Fellman V, Hellström-Westas L, Norman M, et al; EXPRESS Group. One-year survival of extremely preterm infants after active perinatal care in Sweden. *JAMA*. 2009;301(21):2225-2233pmid:19491184.
6. Stoll BJ, Hansen NI, Bell EF, et al. Eunice Kennedy Shriver National Institute of Child Health and Human Development Neonatal Research Network. Neonatal outcomes of extremely preterm infants from the NICHD Neonatal Research Network. *Pediatrics*. 2010;126(3):443-56. pmid:20732945.
7. Rees CM, Pierro A, Eaton S. Neurodevelopmental outcomes of neonates with medically and surgically treated necrotizing enterocolitis. *Arch Dis Child Fetal Neonatal Ed*. 2007;92(3):F193-F198. pmid:16984980.
8. Msall ME, Phelps DL, DiGaudio KM, et al. Severity of neonatal retinopathy of prematurity is predictive of neurodevelopmental functional outcome at age 5.5 years. Behalf of the Cryotherapy for Retinopathy of Prematurity Cooperative Group. *Pediatrics*. 2000; 106(5):998-1005.
9. Calisici E, Eras Z, Oncel MY, Oguz SS, Gokce IK, Dilmen U. Neurodevelopmental outcomes of premature infants with severe intraventricular hemorrhage. *J Matern Fetal Neonatal Med*. 2015;28 (17):2115-20. pmid:25354288.
10. Anderson PJ, Doyle LW. Neurodevelopmental outcome of bronchopulmonary dysplasia. *Semin Perinatol*. 2006;30(4):227-32. pmid:16860163.
11. Mitha A, Foix-L'Hélias L, Arnaud C, et al; EPIPAGE Study Group. Neonatal infection and 5-year neurodevelopmental outcome of very preterm infants. *Pediatrics*. 2013;132(2). Available at: www.pediatrics.org/cgi/content/full/132/2/e372.
12. Delobel-Ayoub M, Arnaud C, White-Koning M, et al; EPIPAGE Study Group. Behavioral problems and cognitive performance at 5 years of age after very preterm birth: the EPIPAGE Study. *Pediatrics*. 2009;123(6):1485-92. pmid:19482758.
13. Marlow N, Wolke D, Bracewell MA, Samara M; EPICure Study Group. Neurologic and developmental disability at six years of age after extremely preterm birth. *N Engl J Med*. 2005;352(1):9-19. pmid:15635108.
14. Hack M, Taylor HG, Drotar D, et al. Chronic conditions, functional limitations, and special health care needs of school-aged children born with extremely low-birth-weight in the 1990s. *JAMA*. 2005;294(3): 318-25. pmid:16030276.
15. Hintz SR, Kendrick DE, Wilson-Costello DE, et al. NICHD Neonatal Research Network. Early-childhood neurodevelopmental outcomes are not improving for infants born at <25 weeks' gestational age. *Pediatrics*. 2011;127(1):62-70. pmid:21187312.
16. Rysavy MA, Li L, Bell EF, et al. Eunice Kennedy Shriver National Institute of Child Health and Human

- Development Neonatal Research Network. Between-hospital variation in treatment and outcomes in extremely preterm infants. *N Engl J Med*. 2015;372(19):1801-11. PMID:25946279.
17. Macfarlane PI, Wood S, Bennett J. Non-viable delivery at 20-23 weeks gestation: observations and signs of life after birth. *Arch Dis Child Fetal Neonatal Ed*. 2003;88(3):F199-F202. PMID:12719392.
18. Partridge JC, Freeman H, Weiss E, Martinez AM. Delivery room resuscitation decisions for extremely low birthweight infants in California. *J Perinatol*. 2001;21(1):27-33. PMID:11268864.
19. Bodeau-Livinec F, Marlow N, Ancel PY, Kurinczuk JJ, Costeloe K, Kaminski M. Impact of intensive care practices on short-term and long-term outcomes for extremely preterm infants: comparison between the British Isles and France. *Pediatrics*. 2008;122(5). Available at: www.pediatrics.org/cgi/content/full/122/5/e1014 PMID:18977951.
20. Lorenz JM, Paneth N, Jetton JR, den Ouden L, Tyson JE. Comparison of management strategies for extreme prematurity in New Jersey and the Netherlands: outcomes and resource expenditure. *Pediatrics*. 2001;108(6):1269-74. PMID:11731647.
21. Pignotti MS, Donzelli G. Perinatal care at the threshold of viability: an international comparison of practical guidelines for the treatment of extremely preterm births. *Pediatrics*. 2008;121(1). Available at: www.pediatrics.org/cgi/content/full/121/1/e193 PMID:18166538.
22. Tyson JE, Parikh NA, Langer J, Green C, Higgins RD. National Institute of Child Health and Human Development Neonatal Research Network. Intensive care for extreme prematurity – moving beyond gestational age. *N Engl J Med*. 2008;358(16):1672-81. PMID:18420500.
23. Jensen EA, Lorch SA. Effects of a birth hospital's neonatal intensive care unit level and annual volume of very low-birth-weight infant deliveries on morbidity and mortality. *JAMA Pediatr*. 2015;169(8):e151906. PMID:26237466.
24. Cifuentes J, Bronstein J, Phibbs CS, Phibbs RH, Schmitt SK, Carlo WA. Mortality in low birth weight infants according to level of neonatal care at hospital of birth. *Pediatrics*. 2002;109(5):745-51. PMID:11986431.
25. Moore T, Hennessy EM, Myles J, et al. Neurological and developmental outcome in extremely preterm children born in England in 1995 and 2006: the EPICure studies. *BMJ*. 2012;345:e7961. PMID:23212880.
26. Serenius F, Källén K, Blennow M, et al. Express Group. Neurodevelopmental outcome in extremely preterm infants at 2.5 years after active perinatal care in Sweden. *JAMA*. 2013;309(17):1810-20. PMID:23632725.
27. Doyle LW, Roberts G, Anderson PJ. Victorian Infant Collaborative Study Group. Outcomes at age 2 years of infants < 28 weeks' gestational age born in Victoria in 2005. *J Pediatr*. 2010;156(1):49-53. e1 PMID:1978300.
-

Long Term Outcome of Treated Posterior Urethral Valve Patients at a Tertiary Care Center

Neha Agarwal¹, Neeraj Agarwal², Megha Agarwal³

¹Assistant Professor, Department of Pediatrics ²Associate Professor, Department of Urology ³Assistant Professor, Department of Obstetrics & Gynaecology, SMS Medical College, Jaipur, Rajasthan 302004, India.

Abstract

Background: Posterior urethral valve is one of the most common causes of bladder outlet obstruction. It's congenital defects which most commonly affect male children. **Aim:** The aim of this study is to evaluate the Long term outcome of treated posterior urethral valve patients at a tertiary care center. **Materials and Methods:** The Present study was conducted in SMS Medical College & Hospital, Jaipur, Rajasthan, India. A total of 40 patients with mean age of 2 years were selected for the study. Parameters like, including age at presentation, serum creatinine, initial creatinine clearance, renal ultrasound findings and ascites were studied. Long-term renal outcome was assessed. **Results:** Of the 40 patients 18 were aged below 2 years i.e. 45%, 21 were aged between 2 to 4 years i.e. 52.5% and 9 of the were aged between 5 to 7 years i.e. 22.5%. Primary fulguration was carried in 77.5% cases and fulguration diversion in 22.5%. **Conclusion:** Posterior urethral disease is very common though its rate has declined for past few years. Urethral valve ablation is safe and easy procedure to be carried out.

Keywords: Urethral Valve; Tertiary Care Unit; Creatinine; Renal Dysfunction.

Introduction

Posterior urethral valve are most common caused congenital urethral abnormality. It is known for causing bilateral renal obstruction [1]. Freedman AL et al reported that posterior urethral valve obstruction is very common in male infants and children [2]. Despite of the recent advances in diagnosis many authors reported that 20% to 40% patients suffer renal failure [3,4]. Posterior urethral valve severity totally depends upon the degree of urinary obstruction. If not treated timely it progresses to renal failure [5].

Diagnostic aids like ultrasonography and postnatally on micturating cysto-urethrogram are used to view the dilated urethral valves. Factors such as age at presentation, initial and nadir serum creatinine, renal parenchymal echogenicity on initial renal ultrasound, VUR on initial VCUG, recurrent UTIs, bladder dysfunction and the presence or absence of popoff mechanisms such as

VURD plays a important role in outcome and prognosis of the disease. The main motto of the treatment is to prevent renal failure [3,7,8]. The rate of renal failure according to the literature in about 19-64% of males diagnosed with PUV prenatally and in 25-40% of infants diagnosed postnatally [9]. So, we aimed to evaluate the Long term outcome of treated posterior urethral valve patients at a tertiary care center.

Materials and Methods

A total of 40 patients suffering from posterior urethral valve admitted in SMS Medical College & Hospital, Jaipur, Rajasthan (India) were selected for the study. All the male infants and children above 6 months of age with posterior urethral valves who were treated with endoscopic valve fulguration were included in the study. Infants below 6 months of age, critically ill child, patients with urosepsis or

Corresponding Author: Neeraj Agarwal, Associate Professor, Department of Urology, SMS Medical College, Jaipur, Rajasthan 302004, India.

E-mail: rohingarg99@gmail.com

Received on 24.03.2018, **Accepted on** 10.04.2018

septicemia and patients with renal failure were excluded from the study. Ultrasound abdomen, micturating cysto-urethrogram (MCUG) and cystoscopy were used for the diagnosis of each patient. Routine blood investigation was performed during hospitalization. A written informed consent was obtained from parents/guardians before the treatment. All the surgical procedure was performed under general anesthesia. Antibiotic was given for 3 years. The outcome of the disease was assessed clinically, biochemically and radiographically. After 3 to 5 days patients were discharged and antibiotic was continued for another 5 to 7 days. Patients parents were asked to report for follow up.

Data Analysis

Data so collected was subjected to analysis using Statistical Package for Social Sciences (SPSS) Version 15.0. Non parametric data has been represented as frequencies and percentages.

Results

A total of 40 patients with posterior urethral valve were selected for the study. Participants included were aged above 6 months with mean age of 2 years. Of the 40 patients 18 were aged below 2 years i.e. 45%, 21 were aged between 2 to 4 years i.e. 52.5% and 9 of the were aged between 5 to 7 years i.e. 22.5%. Most common age group in our study was 2 to 4 years (Table 1). Of the 40 patients 31 patients underwent primary fulguration i.e. 77.5% and 9/40 underwent fulguration diversion i.e.

22.5% (Table 2).

Among 40 patients 25 reported us with fever i.e. 62.5%. 52% patients complaint of dribbling of urine, 37.5% were found to be suffering from abdominal swelling, 30% complaint of inconsistency urine whereas only 2.5% were suffering from haematuria (Table 3). Recurrent UTI was observed in 21/40 patients i.e. 52.5%, 12/40 showed up with urine inconsistency i.e. 30%, 4/40 presented with stricture urethra i.e. 10%, 2 suffered from renal failure and enuresis was evident in 2/40 i.e. 5%. Most common complication in present study was recurrent UTI (Graph 1).

Table 1: Age Distribution of Patients

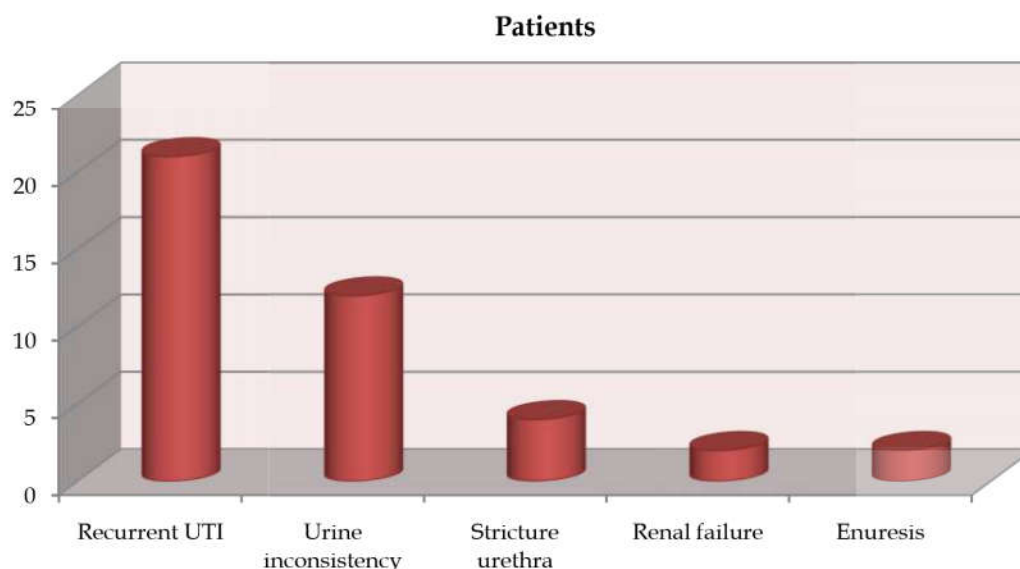
Age	No. of cases	Percentage
<2	18	45%
2-4	21	52.5%
5-7	9	22.5%
Total	40	100%

Table 2: Demographic Details

Procedure	Number of patients
Primary fulguration	77.5%
Fulguration with diversion	22.5%
Total	n = 40

Table 3: Presenting sign and symptom in patients

Sign and Symptom	Patients	Percentage
Fever	25	62.5%
Dribbling	21	52%
Abdominal Swelling	15	37.5%
Inconsistence Urine	12	30%
Hematuria	1	2.5%



Graph 1: Complications after procedure

Discussion

Posterior urethral valve is a common congenital urinary bladder abnormality which is known for its deleterious effect. According to the researchers recently there is an increase in prognosis of the disease. Authors have reported that its mortality rate has decreased from 50% to less than 5% in the last 30 to 40 years [10]. However, there is not much improvement in prenatal diagnosis of posterior urethral valve. Prenatal diagnosis can help to prevent the harmful effect of the congenital disease. Delayed diagnosis leads to poor prognosis and renal damage.

In present study the most common age group was 2 to 4 years i.e. 52.5%. The mean ages of the patients were 2 years of age. Some authors reported that most of the patients reported to them at an age of 1 year [12]. Diamond DA in the year 1992 suggested that PUV can be suspected *in utero* with the help of prenatal ultrasonography the presence of hydronephrosis, dilated proximal urethra, thickened bladder wall, and oligohydramnios can be evident [13]. However, other authors recommended that Fetal intervention for PUV can be a considerable risk to the fetus with a fetal mortality rate of 43% [14]. Fulguration diversion was done in 22.5% cases. Several studies have suggested that urinary diversion is not warranted because it rarely affects renal outcome [15,16]. Most common sign and symptom evident in current was fever (62.5%), Dribbling (52%), abdominal swelling (37.5%) and inconsistent urine (30%). Voiding dysfunction was one of the common causes of presentation in our study. Many other authors have mentioned voiding dysfunction to be the main sign and symptom in posterior urethral valve [17,18].

Posterior urethral valve is often associated with complications. In current study most complication evident was recurrent UTI 52.5%. Our results were similar to those reported by Kifayat Khan et al. [19]. In current study there was no fatality reported. After recurrent UTI, urine inconsistency was most common and only 5% cases of renal failure and enuresis. Urinary inconsistency is considered to be most commonly occurring complication after posterior urethral valve treatment [20]. 20% of patients achieve consistence urine after treatment of PUV. Spontaneous improvement is seen in patients after puberty [10]. 25 to 50% patients suffer from renal failure as late complications after posterior urethral valve treatment. However only 5% was observed in current study [21]. Progression of renal failure is independent of the type of treatment provided.

Conclusion

Endoscopic valve fulguration is an effective method for treatment of posterior urethral valve. Most common age group was 2 to 4 years. If treated at an early stage it can reduce the fatality rate. Complications like recurrent UTI, renal failure, urine inconsistency were evident. The outcome of the disease can be improved early and accurate diagnosis.

References

1. Yohannes P, Hanna M. Current trends in the management of posterior urethral valves in the pediatric population. *Urology* 2002;60:947-53.
2. Freedman AL, Johnson MP, Gonzalez R. Fetal therapy decreased for obstructive uropathy: past, present and future? *PediatrNephrol* 2000;14:167-76.
3. Reinberg Y, de Castano I and Gonzalez R. Prognosis for patients with prenatally diagnosed posterior urethral valves. *J Urol* 1992;148:125.
4. El-Ghoneimi A, Desgrippes A, Luton D et al: Outcome of posterior urethral valves: to what extent is it improved by prenatal diagnosis *J Urol* 1999;162:849.
5. Morri SR, Malin G, Khan K, Kilby M. Antenatal ultrasound to obstruction: systematic review of test accuracy. *BJOG* 2009;116:1290-1299.
6. Martinez JM, Masoller N, Devlieger R, et al. Laser ablation of posterior urethral valves by fetal cystoscopy. *the Fetal Diagnosis and Therapy* January 2015; Doi: 10.1159/000367805.
7. Lopez Pereira P, Espinosa L, Martinez Urrutina MJ et al. Posterior urethral valves: prognostic factors. *BJU* 2003;91:687.
8. Rittenberg MH, Hulbert WC, Snyder HM et al. Protective factors in posterior urethral valves. *J Urol* 1988;140:993.
9. Rainer H, Tanja B, Matthias MW, Ismail T, Felix S, Stephen S. Fetoscopic placement of a transurethral stent for intrauterine obstructive uropathy. *J. Urol.* 2004;171:384-6.
10. Churchill BM, McLorie GA, Khoury AE et al: Emergency treatment and long-term follow-up of posterior urethral valves. *UrolClin North Am* 1990; 17:343.
11. Roth KS, Carter WH Jr, Chan JC. Obstructive nephropathy in children: Long term progression after relief of posterior urethral valve. *Pediatrics* 2001;107:1004-10.
12. Ikuerowo SO, Balogun BO, Akintomide TE, Ikuerowo AO, Akinola RA, Gbelee HO, et al. Clinical and radiological characteristics of Nigerian boys with

- posterior urethral valves. *PediatrSurg Int.* 2008;24: 825-9.
13. Diamond DA, Ford C, authors. Neonatal gross hematuria as a presenting sign of posterior urethral valves. *Urology.* 1992;40:267-9.
 14. Holmes N, Harrison MR, Baskin LS. Fetal surgery for posterior urethral valves: long-term postnatal outcomes. *Pediatrics* 2001;108:E7.
 15. Close CE, Carr MC, Burns MW. Lower urinary tract changes after early valve ablation in neonates and infants: is early diversion warranted? *J. Urol.* 1997; 157:984-8.
 16. Walker RD, Padron M. The management of posterior urethral valves by initial vesicostomy and delayed valve ablation. *J. Urol.* 1990;144:1212-14.
 17. Hodges SJ, Patel B, McLorie G, Atala A. Posterior urethral valves. *ScientificWorldJournal.* 2009;9: 1119-26.
 18. Ikuerowo SO, Balogun BO, Akintomide TE, Ikuerowo AO, Akinola RA, Gbelee HO, et al. Clinical and radiological characteristics of Nigerian boys with posterior urethral valves. *PediatrSurg Int.* 2008;24: 825-9.
 19. Kifayat Khan et al. Management of Infants and Children with Posterior Urethral Valves: Early Outcome of Endoscopic Valve Fulguration in a Tertiary Care Hospital in Peshawar. *Journal of Saidu Medical College* 2017, Vol 7 (1).
 20. Cass AS, Stephens FD. Posterior urethral valves: diagnosis 38 and management. *J Urol* 1974;112:519-25.
 21. De Gennaro M, Mosiello G, Capitanucci ML. Early in children with posterior urethral valves after kidney detection of bladder dysfunction following posterior urethral valves ablation. *Eur J PedSurg* 1996;6:163-5.
-

Clinical Correlates of Working Memory Deficits in School Going Children with and Without ADHD in Ahmednagar

Prajakta Ghatage¹, Sunil Mhaske², Ramesh Kothari³, Ganesh Misal⁴

¹Resident ²Dean ³Professor and Head, Department of Paediatrics, DVVPF's Medical College, Vilad Ghat, Ahmednagar, Maharashtra 414111, India.

Abstract

Both working memory and attention-deficit/hyperactivity disorder (ADHD) have been associated with educational deficits. Since working memory deficits are prevalent in children with ADHD, the main aim of the present study was to examine whether educational deficits are driven by working memory deficits or driven by the effect of ADHD itself. Participants were referred children with (N=100) and without (N=100) ADHD ascertained from pediatric and psychiatric sources. Education deficits were defined as grade retention or placement in special classes, and were assessed using interviews and written rating scales. Working memory was assessed using the WISC-R Freedom from Distractibility (FFD) factor based on digit span, arithmetic and coding. Results were significantly more youth with ADHD had working memory deficits than controls (31.9% vs. 13.7%, $p < 0.05$). In ADHD children, working memory deficits were significantly (p no other differences were noted in other areas of functioning. Although working memory deficits also had some adverse impact on educational and cognitive correlates in non ADHD controls, these differences failed to attain statistical significance. Conclusion was that working memory deficits significantly and selectively increase the risk for academic deficits and cognitive dysfunction in children with ADHD beyond those conferred by ADHD. Screening for working memory deficits may help identify children with ADHD at high risk for academic and cognitive dysfunction.

Keywords: Attention Deficient Hyperkinetic Disorder; Working Memory.

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a prevalent, persistent, and impairing neurobiological disorder estimated to affect up to 7% of children and 5% of adults worldwide [1]. Among the most prominent ADHD-associated adverse outcomes are educational deficits, which include academic under-attainment, increased needs for academic support, and high rates of placement in special classes [2]. ADHD is one of the most prevalent childhood behavioral disorders, and estimates suggest that some where between 4.2% and 6.3% of children meet criteria for this disorder [3] with the boy to girl ratio ranging between 2:1 and 9:1. Children with ADHD often experience significant impairment in major areas of functioning including peer relations, family life, and school. Each of these areas of impairment may affect the media habits of children with ADHD [4].

The academic impairment associated with ADHD is no less insidious. Children with ADHD are more than twice as likely as children without the disorder to be in need of academic tutoring, to repeat a grade, to be placed in special education, and to be diagnosed with a reading disorder [5]. These academic difficulties suggest that, when at home, these children are less likely to engage in reading or homework. Nonetheless, ADHD is frequently associated with executive function deficits (EFDs) in general [6], and working memory (WM) deficits in particular, which are also associated with academic dysfunction (Alloway, Elliott, & Place, 2010; Barkley & Murphy, 2010). This raises questions as to whether academic problems in ADHD are due to ADHD, WM deficits or both [7].

The main aim of the present study was to assess the clinical correlates of working memory deficits in children with and without ADHD. In our study we included children with and without ADHD of

Corresponding Author: Sunil Mhaske, Dean, Department of Paediatrics, DVVPF's Medical College, Vilad Ghat, Ahmednagar, Maharashtra 414111, India.

E-mail: sunilmhaske1970@gmail.com

Received on 30.07.2018, Accepted on 31.08.2018

©Red Flower Publication Pvt.Ltd

both sexes. These children were comprehensively assessed in multiple, non-overlapping domains of functioning. Considering the critical importance of working memory for optimal functioning, we hypothesized that working memory deficits would be associated with impairments in multiple areas of functioning.

Material and Method

Hundred (100) children of both gender were selected for the study. 100 known case of ADHD and 100 control of same age were selected.

Psychiatric assessments relied on the Kiddie Schedule for Affective Disorders and Schizophrenia-Epidemiologic Version (K-SADS-E), (Orvaschel, 1994) conducted directly and individually with the mothers and the children. We used the Freedom from Distractibility (FFD) Factor from the WISC-R to assess working memory. The initial conceptualization of working memory in the revision of the Wechsler Intelligence Scale for Children-Revised was termed the Freedom From Distractibility (FFD) Factor and it was devised from a factor analysis of the Arithmetic, Digit Span, and Coding subtests of the Wechsler scale (Kaufman, 1979). Although the coding subtest was removed in the third edition of the Wechsler Intelligence Scale for Children (Wechsler, 1991), the term FFD Index was kept until the advent of the fourth edition (Wechsler, 2003) where it was replaced with that of "Working Memory (WM) Index" defined the same way (Kranzler, 1997). However, the significant correlation of 0.72 between the Freedom From Distractibility Factor and the Working Memory Index of the WISCIV renders the use of FFD Factor scores an appropriate proxy for WM Index.

We classified participants as having WM deficits using the following rules: 1) participants with a full scale IQ of 120 or less if their Freedom from Distractibility (FFD) score was 1 SD (15 points) lower than their full scale IQ. This method is based on the work of Biederman et al. (2004) indicating that individuals with scores in executive functioning 1 SD below the norm is indicative of poor academic outcomes; or 2) any participant with a FFD of ≤ 85 . Based on the Wechsler Scales, a score of 85 is considered 1 SD below average falling at the 16th percentile; or 3) any participant with full IQ ≥ 120 with a FFD 1.5 SDs (22.5 points) below their full IQ. The predicted score method (vs. simple discrepancy) was employed so that individuals with

high IQs would not be under-identified as not having working memory deficits. This method is often used in the identification of learning disability in high IQ individuals. We chose the FFD subscale because it has been considered by others to be a useful measure of WM (Bowden, Petrauskas, Bardenhagen, Meade, & Simpson, 2013). Statistical Analysis Because significant differences in cognition between children with ADHD and Controls have been well documented (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006), our analytic approach focused on pairwise comparisons within the ADHD and Control groups stratified by the presence or absence of WM deficits. For continuous variables, pairwise ttests were conducted. For categorical variables a chi-squared test was carried out; if the number of participants in any group was below 10 we used Fisher's exact test. All analyses were conducted using the R programming language (R Core Team, 2014). All tests were two tailed. Due to the many comparisons conducted, the alpha level was set at 1%.

Result

There were no significant within group pairwise comparisons for age, sex, SES, or intactness of the family. There was a significant difference between the two ADHD groups in mean number of symptoms of ADHD ($p = 0.008$). However, the observed effect size, though statistically significant, was small with the ADHD+WM group having on average 0.7 more symptoms. There were no meaningful differences between the two groups in the mean duration and age of onset of ADHD. Likewise, there were no meaningful differences between ADHD children with and without WM deficits in number of CBCL Total Problems, social functioning (SAICA) or GAF scores. There was however, a significant difference in the CBCL school competence score between the two ADHD groups with and without WM deficits. The rate of WM deficits was significantly higher in children with ADHD compared to controls (32% vs. 14%, $p < 0.001$). Within group comparisons were made between ADHD children with (ADHD+WM Deficits: $N=88$) and without (ADHD: $N=188$) WM deficits and Controls with (Controls+WM Deficits: $N=33$) and without (Controls: $N=208$) WM Deficits.

Cognitive Outcomes WISC-R subtest scores for Arithmetic, Digit Span, and Digit Symbol were significantly worse in both ADHD and Control participants with WM deficits than in those without WM deficits, as was expected based on the

definition of WM. Likewise, composite scores for both the ADHD and Control groups were significantly worse for those with WM deficits for WISC-R FFD and WRAT Arithmetic, but WRAT Reading was more impaired in ADHD+WM children only. School Outcomes As shown in the rates of grade retention and placement in special classes were significantly

more prevalent in children with ADHD with WM deficits than in those without WM deficits. Although the need for extra help was significantly higher in ADHD children with WM deficits than in those without, this difference did not reach threshold for statistical significance.

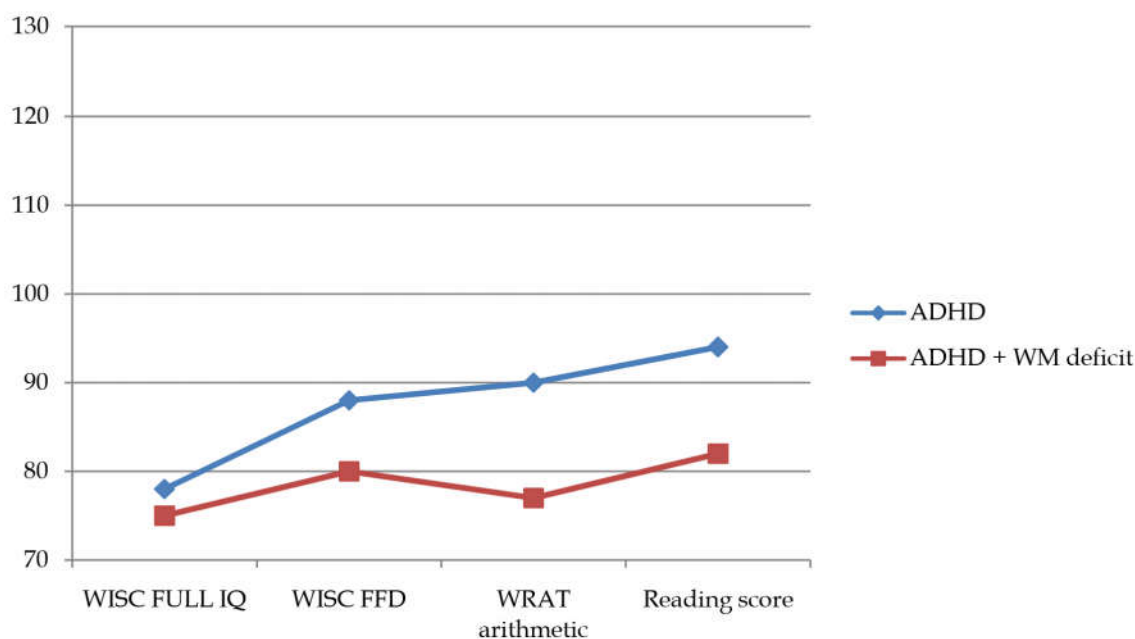


Fig. 1:

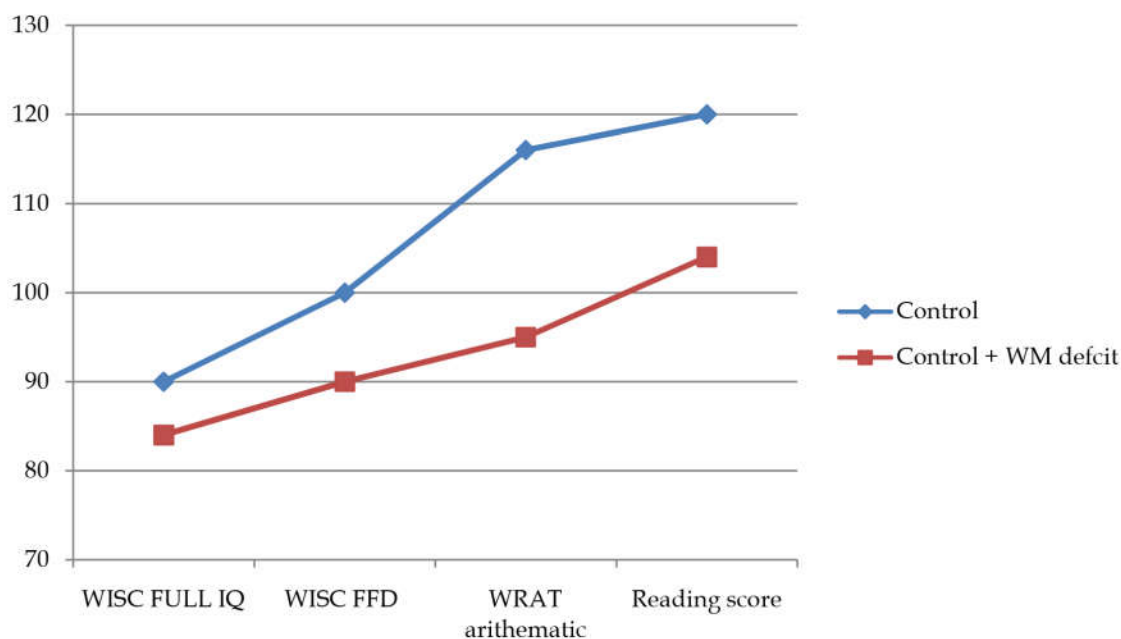


Fig. 2:

Discussion

From our study in children with and without ADHD randomly selected from pediatric and psychiatric sources, we observed that the presence of WM deficits in ADHD children significantly increased the risk for poor scholastic performance, placement in special classes and lower academic achievement in both reading and problems in calculations. Even with stringent statistical controls the results were significantly evident. These deficits could not be accounted for by differences in the clinical features of ADHD [8].

In non ADHD Controls the adverse findings related to education and cognition were found, with the exception of mathematic dysfunction, these findings did not succeed to reach our a priori threshold for statistical significance. These results differed from findings reported by Etchepareborda and Abad-Mas (Etchepareborda & Abad-Mas, 2005) in a non ADHD sample, showing that impaired WM had a negative impact on influenced academic learning processes [9].

The cognitive and academic burdens associated with WM deficits in the have significant clinical conclusions. Clinically, the presence of WM deficits significantly affects the already compromised cognitive an scholastic performance of ADHD children beyond that due to ADHD itself [10]. As the presence of WM deficits can only be documented through cognitive testing, screening for such deficits can help identify a subgroup of ADHD children at very high risk for scholastic performance.

In our study relied on the Freedom From Distractibility Factor from the WISC-R to assess Working Memory, which is not considered a true measure of WM, this index has documented very high correlation with WM index in the WISC-III revision (Wechsler, 1991). Although to a lesser extent, WM deficits also had a adverse effect on educational, scholastic performance and cognitive correlates in non ADHD Controls [11,12,13]. Screening for WM deficits will aid in identifying children at high risk for scholastic performance.

References

1. Achenbach TM., Rescorla LA. Manual for ASEBA School-Age Forms & Profiles. University of Vermont, Research Center for Children, Youth, & Families; Burlington, VT: 2002.
2. Alloway TP, Elliott J, Place M. Investigating the relationship between attention and working memory in clinical and community samples. *Child Neuropsychology*. 2010;16(3):240-54.
3. Alloway TP, Gathercole SE, Elliott J. Examining the link between working memory behaviour and academic attainment in children with ADHD. *Developmental Medicine & Child Neurology*. 2010;52(7):632-38.
4. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders: DSM-IV. 4th ed.. American Psychiatric Association; Washington, DC: 1994.
5. Barbaresi WJ, Katusic SK, Colligan RC, Weaver AL, Jacobsen SJ. Long-term school outcomes for children with attention-deficit/hyperactivity disorder: a population-based perspective. *Journal of Developmental & Behavioral Pediatrics*. 2009;28(4):265-73.
6. Barkley RA, Murphy KR. Impairment in occupational functioning and adult ADHD: the predictive utility of executive function (EF) ratings versus EF tests. *Archives of Clinical Neuropsychology*. 2010;25(3):157-75.
7. Biederman J, Faraone SV, Keenan K, Benjamin J, Krifcher B, Moore C, Tsuang MT. Further evidence for family-genetic risk factors in attention deficit hyperactivity disorder. Patterns of comorbidity in probands and relatives in psychiatrically and pediatrically referred samples. *Archives of General Psychiatry*. 1995;49(9):728-38.
8. Biederman J, Faraone SV, Mick E, Williamson S, Wilens TE, Spencer TJ, Zallen B. Clinical correlates of ADHD in females: findings from a large group of girls ascertained from pediatric and psychiatric referral sources. *Journal of the American Academy of Child & Adolescent Psychiatry*. 1999;38(8):967-76.
9. Biederman J, Monuteaux M, Mick E, Spencer T, Wilens T, Klein K, Faraone SV. Psychopathology in females with attention-deficit/hyperactivity disorder: A controlled, five-year prospective study. *Biological Psychiatry*. 2006;60(10):1098-1105.
10. Biederman J, Monuteaux MC, Doyle AE, Seidman LJ, Wilens TE, Ferrero F, Faraone SV. Impact of executive function deficits and attention-deficit/hyperactivity disorder (ADHD) on academic outcomes in children. *Journal of Consulting and Clinical Psychology*. 2004; 72(5):757-66.
11. Biederman J, Monuteaux MC, Mick E, Spencer T, Wilens TE, Silva JM, Faraone SV. Young adult outcome of attention deficit hyperactivity disorder: a controlled 10-year follow-up study. *Psychological Medicine*. 2005; 36(2):167-79.
12. Bowden SC, Petrauskas VM, Bardenhagen FJ, Meade CE, Simpson LC. Exploring the dimensionality of digit span. *Assessment*. 2013;20(2):188-98.
13. Bunford N, Brandt NE, Golden C, Dykstra JB, Suhr JA, Owens JS. Attention-deficit/hyperactivity disorder symptoms mediate the association between deficits in executive functioning and social impairment in children. *Journal of Abnormal Child Psychology*. 2015; 43(1):133-47.

Craniofacial Anthropometric Measurement of Full Term Newborns in Tertiary Care Hospital

Punam Uke¹, Akash Daswaney², Sam Rodrigues³, Sonal Gore⁴, Amar Taksande⁵

¹Assistant Professor ^{2,3}Intern ⁴Resident ⁵Professor, Department of Pediatrics, Jawaharlal Nehru Medical College, Sawangi Meghe, Wardha, Maharashtra 4420004, India.

Abstract

Introduction: The face is important part of many syndromes of dysmorphogenesis. Main aim of anthropometric studies are to obtain the characteristics of ethnic groups and provide the basis for a comparison among different races. Our study is being conducted to take craniofacial measurements in newborn of rural India and to find correlation between different parameters. It also adds normograms for all the craniofacial anthropometric measurements studied. **Method:** 97 normal neonates comprising 49 male and 48 female were measured 48 hours after birth. The measurement for different cranio-facial dimensions were obtained using vernier calliper and non-stretchable measuring tape. Unpaired 't' test was utilized to compare the parameters. **Result:** The mean value and range for all the parameters was determined. Mean birth weight of studied neonate was 2.52 ± 0.318 and gestational age being 37.95 ± 1.27 . A statistically significant sexual dimorphism was noted to exist in ear length ($p < 0.05$). The mean intercanthal distance was 17.55 ± 3.00 in male and 17.64 ± 2.73 in female, philtrum width were 5.06 ± 1.72 in male and 5.02 ± 1.48 in female and commissural distance were 23.44 ± 4.50 in male and 22.95 ± 3.97 in female. Ear length measurements were 32.46 ± 3.79 in male and 31.25 ± 2.24 in female. **Conclusion:** We here present a reference data set for the newborn population of rural area in India.

Keywords: Newborn; Anthropometric; Craniofacial; Dysmorphogenesis.

Introduction

The literal meaning of anthropometry is the measurement of the human individual for the purpose of understanding physical variation. The face is most important part of many syndromes of dysmorphogenesis [1]. Normative data of facial measurements are very important for knowing degree of deviations from the normal [2]. There are many anthropometric indices of head and face useful in different streams of medical education [3]. Anthropometric measurements are also helpful for other observations in evaluating intrauterine growth, development and in detecting neonatal health problems. The philtrum of the upper lip has a unique dimension and is a characteristic landmark of individual distinction [4]. Since it is frequently involved in disfiguring oro-facial malformations, it is important that a thorough

understanding of its anatomical relationships be established so that functional and aesthetic surgical corrections can be accomplished. Another important anthropometric parameter is commissural distance which also found to have the best single correlation with the philtrum [5]. Ear length is important in the evaluation of congenital anomaly syndrome such as Down's syndrome. There is impact of geographical location, racial and environmental factors on the growth and body composition. Anthropometric studies are mostly conducted with the aim of obtaining the characteristics of ethnic groups in a particular geographical region. It not only assist in understanding the frequency distribution of human morphologies but also in providing the basis for a comparison among different races [6].

Newborn anthropometric data against which deviations from normal could be assessed are

Corresponding Author: Amar Taksande, Professor, Department of Pediatrics, Jawaharlal Nehru Medical College, Sawangi Meghe, Wardha, Maharashtra 4420004, India.
E-mail: amar.taksande@gmail.com

Received on 20.06.2018, Accepted on 14.07.2018

generally lacking in developing countries. Aim of our study is to take craniofacial measurements in newborn of rural India and to find correlation between different parameters. It will also help in development of noemogram of craniofacial antropometric parameters of rural India.

Material and Methods

The study was conducted in Department of Pediatrics of Jawaharlal Nehru Medical College, Sawangi (M), Wardha. It was a cross-sectional study undertaken in 3 month (January 2018 to March 2018) duration. Study was started after approval from ethical committee.

Full term (37-40 weeks of gestation) healthy babies born by any means during the period older than 48 hours. Babies devoid of any gross congenital anomalies were included in study after a written informed consent from one of the parents (Preferably the mother) of the neonates. Sick Full term neonates admitted in NICU, those with birth weight less than 2 kg and unable to get written inform consent were excluded.

Study Procedure

After getting written informed consent following information was collected: Name, Age (days), Birth weight (kg), Sex, Mode of delivery, Gestational Age (weeks). All neonates have undergone craniofacial measurements with the help of vernier caliper and non-stretchable measuring tape.

Facial measurements were taken by pediatric resident and during daytime, when the cases were sleeping to avoid variation due to facial expression. The head circumference was measured using a non stretchable measuring tape in centimeters. The parameters were measured in millimeters using vernier caliper. Measurements of newborn babies were delayed for 48 hours to allow facial swelling and distortions to recede.

Craniofacial measurements will be under following subheadings:

1. Horizontal parameters
2. Vertical parameters

1. Horizontal parameters (Figure 1)

Head Circumference: The lower edge of measuring tape was placed just above the child's eyebrows,

above the ears and around the occipital prominence with the objective of measuring the maximal head circumference [7].

Intercanthal Distance: was measured in mm between the median angles of the palpebral fissures [8].

Philtrum Width: Two points were marked at the base of the philtrum, i.e. at the junction of the vertical ridge of philtrum and vermillion border of upper lip. The width between these points was taken as the philtral width [9].

Commissural distance: was measured between the corners of the mouth [5].

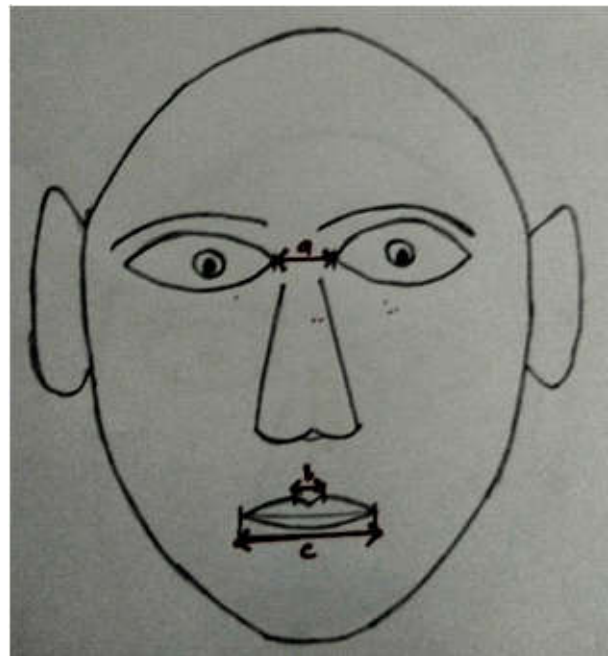


Fig. 1: Measurement of horizontal facial parameters

a = Intercanthal distance
b = Philtrum width
c = Commissural distance

2. Vertical Parameters (Figure 2)

- A. Ear Length: from superior to inferior aspects of the ear [10].
- B. Philtrum length: from base of columella to midline depression of the vermillion border [11].
- C. Lower Lip to Chin: between junction of skin and mucous membrane of lower lip and the lowest point of the chin with mouth closed [11].
- D. Nose Length: Nasion to a point at the tip of the nose in line with the upper edge of both nostril [11].

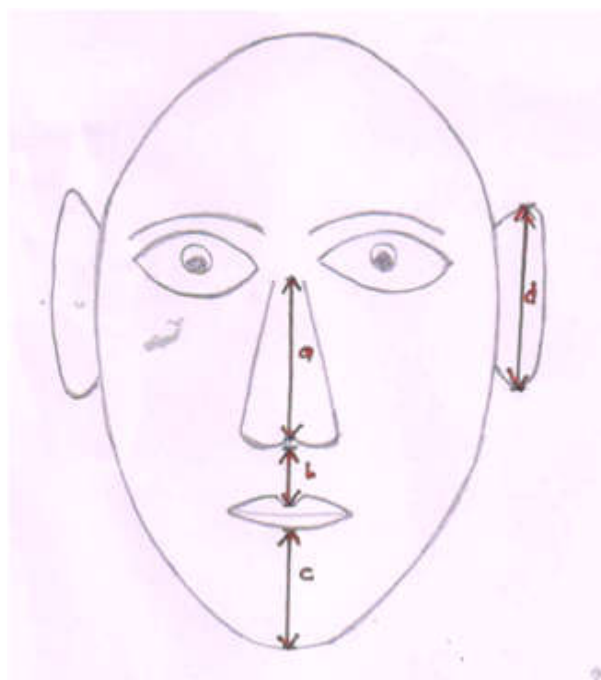


Fig. 2: Measurement of vertical facial parameters

a = Nasal Length
b = Philtrum length
c = Lower lip to chin length
d = Ear length

Statistical Analysis

STATA software was used for statistical analysis. Unpaired 't' test was utilized to compare the parameters as measured for males and females and the 't' distribution table was consulted.

Results

Total 97 neonates were studied. Mean birth weight of studied neonate was 2.52 ± 0.318 and gestational age being 37.95 ± 1.27 . Mean head circumference of neonates were 33.44 ± 1.54 with mean head circumference of male was 33.6 ± 1.54 and female 33.26 ± 1.53 .

Total 49 male and 48 female were studied. All measurements were taken by vernier caliper except head circumference which was taken by non-stretchable measuring tape. The mean intercanthal distance was 17.55 ± 3.00 in male and 17.64 ± 2.73 in female. Measurements of philtrum width were 5.06 ± 1.72 in male and 5.02 ± 1.48 in female. Readings of commissural distance were 23.44 ± 4.50 in male and 22.95 ± 3.97 in female.

Ear length measurements were 32.46 ± 3.79 in male and 31.25 ± 2.24 in female. Philtrum length were 6.81 ± 1.86 in male and 6.25 ± 2.12 in female. Lower lip to chin measurements were 13.37 ± 3.52 in male and 12.77 ± 3.30 in female. Nose length were 16.31 ± 2.59 in male and 15.95 ± 2.43 in female (Table 1).

Discussion

The present study was conducted to obtain a baseline craniofacial dataset for the newborn population of rural India where it is still lacking. Most important implication of our study is to get normal values of these measurements in healthy subjects which are useful for dysmorphologists in the early identification of some craniofacial syndromes and in planning intervention. There is influence of genetic, cultural, environmental and racial factors on craniofacial landmarks. This implies that local values derived from well-defined populations should be used as reference in the evaluation of a case with dysmorphogenesis. Similar studies were done in different part of India with variation in different geographical population.

In a study by Ghosh A et al. [12] done shows, philtrum width was 5.2 ± 0.07 , 5 ± 0.08 in male and female respectively where as in our study it was 5.02 ± 1.71 and 5.02 ± 1.48 values being almost close. Another study by Soni P et al. [13] conducted in Himachal Pradesh get values 6.75 ± 0.98 in male and 6.65 ± 0.87 in female. Also variation was found in nose length in these studies as it was 15.3 ± 0.11 and 15 ± 0.14 in a study by Ghosh A et al¹² with values

Table 1: Mean values (standard deviation) and comparison in sexes for neonates

	Total Parameters	Male	Female	p- value
Horizontal parameters (mm)	Intercanthal distance	17.52(3.02)	17.64(2.73)	0.42
	Philtrum Width	5.02(1.71)	5.02(1.48)	0.50
	Commissural distance	23.35(4.50)	22.95(3.97)	0.32
Vertical parameters (mm)	Ear length	32.31(3.66)	31.25(2.24)	0.026
	Philtrum Length	6.72(1.78)	6.25(2.12)	0.09
	Lower lip to chin	13.37(3.52)	12.77(3.30)	0.21
	Nose length	16.31(2.59)	15.95(2.43)	0.24

16.31±2.59 in male and 15.95±2.43 in female in our study whereas it was 21.04±2.31 in male and 20.82±2.00 in female in a study by Soni P. et al. [13].

Agnihotri G et al. [1] reported intercanthal distance was 20.05±1.43, 20.10±1.56 in male and female respectively whereas in our study values these values are 17.52±3.02 and 17.64±2.73 [1]. Soni P. et al. [13] reported that 19.93±1.43 and 19.47±1.93 with values higher than our study. Ear length 32.31±3.66 in male and 31.25±2.24 in female whereas in study by Agnihotri G et al. [1] these are 37.55±2.24 and 35.21±2.61 respectively which was highly significant.

Conclusion

This study is a small initiative for preparing a reference data set for the newborn population of rural area in India and to provide a ready database to Paediatrician. We here present a set of reference value for the newborn population of rural India.

References

1. Agnihotri G, Singh D. Craniofacial Anthropometry in Newborns and Infants. *Iran J Pediatr* 2007;17:332-8.
2. Farkas LG, Katic MJ, Forrest CR, et al. International anthropometric study of facial morphology in various ethnic groups/races. *J Craniofac Surg* 2005;16:615-46.
3. Williams, P. L.; Warwick, R.; Dyson, M. & Bannister, L. H. In: *Cranium sexual and racial differences*. Gray's Anatomy. 37 th Ed. Churchill Livingstone, United Kingdom, 1989.pp.396-7.
4. Lee ST. A histological study of the philtrum. *Ann Acad Med Singapore*. 1988;17(3):328-34.
5. Franz ML, Sokol AB. The proportioned philtrum: a helpful measurement and ratio. *Cleft Palate J*. 1972;43(3):143-6.
6. Heidari Z., Mahmoudzadeh Sagheb H.R. & Noori Mugahi M.H. Morphological evaluation of head and face in 18-25 years old women in southeast of Iran. *Med. Sci.*, 2006;6:400-4.
7. Alderman J. Gaining and growing. In: *Nutritional assessment*. 33rd ed. London; G Bell and Sons Ltd. 2007.p.71.
8. Alwazzan KA. Variation in mesiodistal crown width and amount of tooth exposure between right and left maxillary anterior teeth. *Egypt Dent J* 1995;41(3): 1283-6.
9. Singh S, Bhalla LR, Khanna VK. Relationship between width of maxillary central incisors and width of philtrum. *J Indian Dent Assoc*. 1971;43(12):264-9.
10. Sivan Y, Merlob P, Reisner SH. Assessment of ear length and low set ears in newborn infants. *J Med Genet*. 1983;20(3):213-5.
11. Shah M, Verma IC, Mahadevan S, et al. Facial anthropometry in newborns in Pondicherry. *Indian J Pediatr*. 1991;58(2):259-63.
12. Ghosh A, Manjari C and Mahapatra S. The craniofacial anthropometric measurement in a population of normal newborns of Kolkata. *Nepal Journal of Medical sciences*. 2013;2(2):125-29.
13. Soni P, Prabhakaran K, Kapoor K, et al. Craniofacial Anthropometric Measurement of Normal Full Term Newborns in Lower Hills of Himachal Pradesh. *IJCRR*. 2017;9(7):23-27.

Paediatric Museum-An innovative Teaching-Learning Method

Sunil Mhaske¹, Prajakta Ghatage², Ramesh Kothari³

¹Dean ²Resident ³Professor and Head, Department of Paediatrics, DVVPF's Medical College, Vilad Ghat, Ahmednagar, Maharashtra 414111, India.

Abstract

Introduction: A lecture is the most widely used teaching method. Learning by lecture is a passive practice. Many pioneering techniques have been used to motivate students to have active attitude toward learning. In our method we are using museum as an innovative teaching-learning method. **Material and Method:** 96 undergraduate students were evaluated by dividing into 2 groups. Group 1 (48 students) was taught through the traditional blackboard-chalk and Power Point presentation method and Group 2 (48 students) was sent to the Paediatric Museum for the same period of time. The knowledge and understanding of the subject was then assessed through a test at the end of the 1 month posting. After both the groups were taught by both methods of teaching, feedback was taken. **Result:** Most of the students accepted that the newer, innovative method of teaching was more interesting and appealing to them. Group 1 scored 68% average and in that group 54% students got above 50%. While the average of Group 2 was 84% and 68 % scored a over 50% marks. The feedback showed that 99% of the students agreed or strongly agreed that learning and clearing their concepts was easier after visiting museum. Students also strongly agreed that this method was time saving and helped them in memory retention. **Conclusion:** Students who had attended the museum had a better understanding of the Paediatric subject and could reproduce the knowledge gained by them in a better way as compared to the students taught through the traditional method.

Keywords: Museum; Teaching; Medical Students.

Introduction

In this era, where medical education plays the most important role in shaping up of a doctor's knowledge and ability to treat patients, the teacher's duties are being more and more scrutinized. Teaching health science subjects in India is largely in the form of instructive lectures which is more teachercentered. The efforts are being made to make teaching more student-centered [1]. During recent decades, newer technologies have been implemented and visual aids such as slides and PowerPoint presentations have been used to boost education. Since lectures have a low effect on the development, the employment of newer techniques is inevitable and actively engage the students in the education process. Effective medical education should be viewed as a continuum, integration of basic science and clinical medicine should occur throughout the curriculum and self-

directed, life long learning should be emphasized [2].

Irby [3] gave importance in his research article about not only teaching but also learning. Creating an environment in which the students can learn effectively and efficiently becomes the new prerequisite, demanding not only that teachers are experts in their fields but also-and more importantly-that they understand how students learn. According to Samarakoon et al. Teaching is considered as 'ever-evolving' processes especially in medical school. He further states that it needs to modernise continuously. James et al. [4], defined learning style as 'the manner in which and the conditions under which learners most efficiently and effectively perceive, process, store, and recall what they are attempting to learn'. According to Kharb et al 'learning style' means as 'an individual's preferred method of gathering, processing, interpreting, organizing and analyzing

Corresponding Author: Prajakta Ghatage, Resident, Dean, Department of Paediatrics, DVVPF's Medical College, Vilad Ghat, Ahmednagar, Maharashtra 414111, India.
E-mail: sunilmhaske1970@gmail.com

Received on 30.07.2018, Accepted on 31.08.2018

information' [5].

Paediatric museum has been designed to teach the students in a way which would enable their hearing and visual impressions to gain access to knowledge according to their own, individual understanding and grasping power.

Material and Method

This study was conducted in the Department of Paediatrics at a tertiary care hospital and Medical College. The Paediatric Museum used for study is one of its kind museum in Asia. 98 undergraduate students of final MBBS from our Medical College were evaluated by dividing them into 2 groups of 48 students each. Permission was taken from the ethical committee of College. Group 1 was taught through the traditional blackboard-chalk and Power Point presentation method for an hour. Group 1 was sent to the Paediatric Museum. The knowledge and understanding of the subject was then assessed through a written test and viva at the end of the 15 lecture series which was covered over 1 month. In the next semester, the batches were exchanged and the group 1 was sent by Paediatric museum. After both the groups were taught by both the methods, a feedback was taken from all the students.

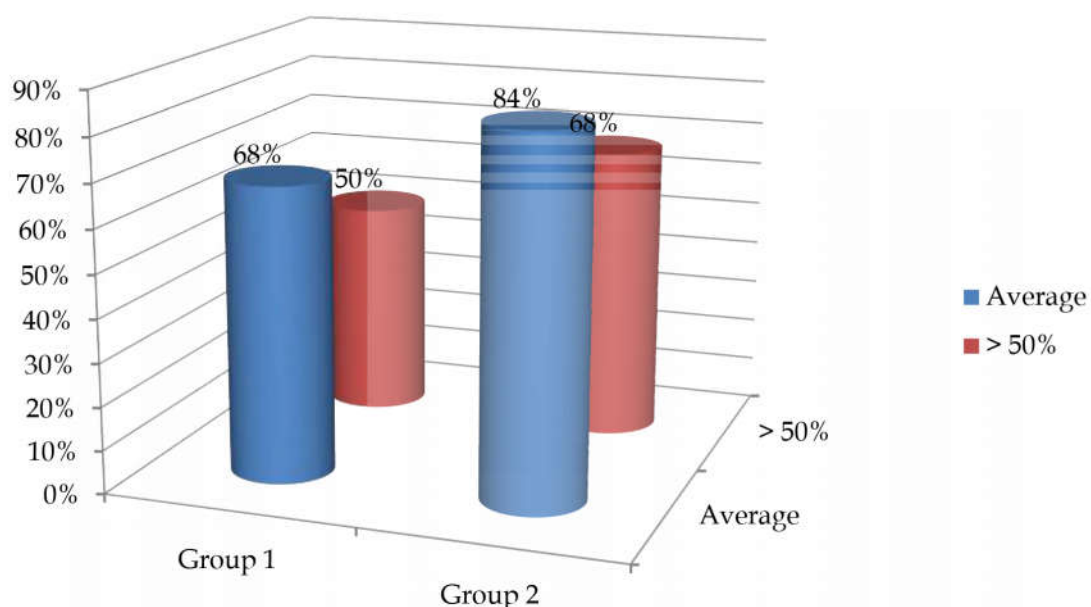


Fig. 1: Paediatric Muesum used in study

Result

Group 1 scored 68% average marks and in that group 54% students got above 50%. While the average of Group 2 which was taught in Paediatric museum had an average of 84% and 68 % scored over 50% marks.

To test the effectiveness of the method, we conducted a quasi-experimental study and then the students were asked to write their learning experiences in feedback form by a questionnaire method. A feedback form was prepared to take the input from the students regarding their experience during the interactive method the results of which are as follows:



Graph 1:

Questions	Totally disagree	Partially disagree	Agree	Strongly agree
Do you think this teaching method has increased your performance?	00	00	36 (37.5%)	60 (62.5%)
Do you think this method has Increased your interest in Peadiatrics?	00	01 (0.68%)	39 (40%)	56 (58.3%)
Helped in memory retention?	00	01 (0.68%)	44 (48.9%)	52 (54%)
Do you think it is better than traditional Teaching method?	00	01 (0.68%)	40 (41.2%)	55 (57.2%)
Helped in increasing confidence and knowledge?	00	01 (0.68%)	33 (34.3%)	63 (65.6%).

Discussion

Medical education is an important factor in the progress of any country. Across the world, increasing attention is being given to the quality of teaching and learning in the medical colleges. Teaching is the noblest profession of all. A doctor is treated as equivalent to god who serves and saves the lives of the people. Our study showed that Group 1 scored 68% average marks and in that group 54% students got above 50%. While the average of Group 2 which was taught in Paediatric museum had an average of 84% and 68% scored over 50% marks. The feedback showed that 99% of the students agreed or strongly agreed that learning and clearing their concepts was easier after visiting museum.

Students also strongly agreed that this method was time saving and helped them in memory retention. Teacher's explanations enable students to understand the content and forging connections between what is known and what is new. Good teaching methods are open to change for effective teaching in the light of evidence collected [6].

The outcomes of the research revealed so the student based education leads to an improvement in student participation, better long-term memory, motivating students, stability of knowledge, higher self-confidence level, recognizing the educational differences, better student master interactions, and creating a sense of team-work. Also, learners got greater in this problem solving method topics in the final exam. These results were compatible with these findings of Nikfar et al. [7], Kermaniyan et al. [8], Momeni Danaei et al. [9], Jafari et al. [10], Hekmatpour et al. [11].

Although traditional medical education methods had produced thousands of well-known, efficient and successful doctors in both developed and developing countries there were increasing calls for fundamental

changes in medical education to meet the needs of the community [12].

In our study as both the groups were taught by both the methods, they found the museum learning as more innovative, interesting way which enabled them to learn at their own pace and helped in better understanding.

Conclusion

Students who had attended the museum had a better understanding of the Paediatric subject and could reproduce the knowledge gained by them in a better way as compared to the students taught through the traditional method.

To decrease the burden on teachers, the biases that can arise because of intrinsic individuality of the teachers and the learning capabilities of the students, the establishment of the innovative "Paediatric Museum as a learning tool" has proved to be worthy.

References

1. Ramani S. Twelve tips to promote excellence in medical teaching. *Med Teach* 2006 Feb;28(1):19-23.
2. Bardes CL, Gillers D, Herman AE. Learning to look: Developing clinical observational skills at an art museum. *Med Educ* 2001;35(12):1157-61.
3. Irby DM. What clinical teachers in medicine need to know. *Acad Med*. 1994;69:333-342.
4. Samarakoon L, Fernando T, Rodrigo C, Rajapakse S. Learning styles and approaches to learning among medical undergraduates and postgraduates. *BMC Medical Education*, 2013;13:42.
5. James WB, Gardner DL. Learning styles: Implications for distance learning. *New Directions for Adult and Continuing Education*, 1995;67:19-31.

6. Koh GCH, Khoo HE, Wong ML, Koh D. The effects of problem-based learning during medical school on physician competency: a systematic review. *Canadian Medical Association Journal*. 2008;178(1):34-41.
 7. Wojtczak, A. Glossary of Medical Education Terms. In *MedEdWorld Glossary*. 2003.
 8. Brown G., & Edmunds S. Lectures. In, J. A. Dent & R. M. Harden (Eds.), *A Practical Guide for Medical Teachers* (4th ed., pp. 61-68). London: Churchill Livingstone Elsevier. 2013.
 9. Hafeez K., Khan M.L.Z., Jawaid M., & Haroon S. Low attendance in lectures at medical colleges of Karachi – A cross sectional survey. *Journal of Postgraduate Medical Institute*, 2014;28(2):161- 64.
 10. Sumera, A. Large group teaching, an effective and efficient teaching methodology. *Journal of Asian Scientific Research*, 2014;4(1):1-5.
 11. Held, S., &McKimm, J. Improve your lecturing. *British Journal of Hospital Medicine*, 2009;70(8):466-69.
 12. Momeni Danaei S, Zarshenas L, Oshagh M, Khoda O, Maryam S. Which method of teaching would be better; cooperative or lecture? *Iranian Journal of Medical Education*. 2011;11(1):24-31.
-

Screening of School Children for Attention Deficit Hyperactivity Disorder using Vanderbilt ADHD Diagnostic Teacher Rating Scale

Jayendra R. Gohil¹, Mrigank N. Gupta², Shruti U. Patel³, Shreya A. Patel⁴

¹Professor HOD ²⁻⁴Resident, Department of Pediatrics, Government Medical College and Sir T G Hospital, Bhavnagar, Gujarat 364002, India.

Abstract

Objective: To screen the school children aged 10-14 years for Attention Deficit Hyperactivity Disorder using Vanderbilt ADHD Diagnostic Teacher Rating Scale. **Method:** This was a cross-sectional observational study. Total 2114 students of three schools between the age group of 10-14 yrs studying in 5th-9th standard were surveyed. Pre-study, the class teachers were trained in a half day workshop about ADHD disease and how to fill up forms. From each class suspected students of ADHD were selected by the teachers and then ADHD symptom scale self report form was filled by the teachers for these students. The filled up forms were analyzed by us. **Result:** Prevalence of ADHD in present study was 65/2114 (3.07%), out of which 27.27% and 14.54% were of hyperactive and inattentive ADHD type respectively, while 58.16% were suffering from mixed ADHD type. Most affected age was 12 years, and male to female ratio was 5.5. Most common symptom of ADHD was, excessive talking (96.62%); most common behavioral problem was, not following rules (70.7%); and most common affected area was, mathematics. **Conclusion:** The self report tool filled up by teachers after training them is able to detect cases of suspected ADHD in 10 to 14 year studying in 5 to 9 standard. There is a 3% predicted prevalence of ADHD by this survey tool in upper-middle class schools.

Keywords: ADHD; Vanderbilt Scale; School Children; Screen; Teacher Rating Scale; Prevalence.

Introduction

Attention Deficit Hyperactivity Disorder (ADHD) is one of the most common childhood brain disorders and can continue through adolescence and adulthood. Symptoms include difficulty staying focused and paying attention, difficulty controlling behavior, and hyperactivity (over-activity). To be diagnosed with the disorder, a child must have symptoms for six or more months and more than or equal to six symptoms of each domain. Parents and teachers can miss the fact that children with symptoms of inattention may have ADHD because they are often quiet and less likely to act out. They may sit quietly, seeming to work, but they are often not paying attention to what they are doing. They may get along well with other children, whereas children who have more symptoms of hyperactivity or impulsivity tend to have social problems. However, children with the inattentive kind of ADHD are not

the only ones whose disorders can be missed. For example, adults may think that children with the hyperactive and impulsive symptoms just have disciplinary problems.

ADHD impacts each child's brain differently, so each child may look quite different in the classroom. Children with ADHD exhibit a range of symptoms: some seem to bounce off the walls, some daydream constantly, and others just can't seem to follow the rules. The American Academy of Pediatrics (AAP) guidelines for ADHD assessment advise: DSM- IV-TR (Diagnostic and Statistical Manual of Mental Disorders Text Revision) criteria, evaluating for comorbid conditions, and a neurological examination; subjectivity arises in recognition of symptoms and degree of functional impairment. In the West, studies have shown that ADHD can be reliably diagnosed by various clinicians. However, this may not be true in India and other similar settings due to low levels of awareness and expertise about ADHD

Corresponding Author: Mrigank N. Gupta, Resident, Department of Pediatrics, Government medical college, and Sir T G Hospital, Bhavnagar, Gujarat 364002, India.
E-mail: mrigan.gupta.012@gmail.com

Received on 29.05.2018, **Accepted on** 22.06.2018

in community clinicians. Clinically, International Classification of Disease-10 (ICD-10) and the DSM-IV-TR criteria are used for diagnosis.

Teachers are often the first ones to recognize or suspect ADHD in children; as ADHD symptoms can affect school performance- and in some cases, disrupt the rest of the class- and because teachers are with children day in and day out.

However, teachers cannot diagnose ADHD hence professional intervention is necessary to consider all cases of the noticed abnormal behavior.

The purpose of the ADHD screening is to separate those students who are suspected of having ADHD from children who do not have any disorder or who are suspected of having an alternative educationally related disability.

The teacher interview should be completed as an early element of the screening.

ADHD-specific rating scale allows teachers to share their global perceptions of the child by completing items about school performance and behavior that map to DSM-IV diagnostic criteria for ADHD. We used Vanderbilt ADHD Diagnostic Teacher Rating Scale [1].

Through a review of the student's school records, the teacher should look for any observations from past teachers that the child has had, like trouble completing class work, remaining focused, or suppressing inappropriate behaviors. Such teacher comments may help to establish the duration (six months) necessary for the diagnosis of the disorder.

Vanderbilt scale is generally used for 6-12 years age, but we have used this scale in 10-14 years age to assess whether it can be used for older children also. Usually, the incidence of ADHD is higher in preschool children and the incidence decreases with age. This early high incidence may be due to extremes of normal behavior which may be diagnosed as ADHD.

Methods

This was a cross-sectional observational study.

Development of Appropriateness Criteria and Instrument-

The tool was translated forwards and backwards from *English to Hindi* and *Gujarati* by bilingual translators maintaining conceptual, content, semantic, operational and functional equivalence of the items, and was validated.

A group of two pediatricians first trained the teachers in half a day workshop on how to recognize the symptoms and gave basic knowledge regarding the disease.

Diagnostic modalities of ADHD in children were reviewed, and clinical expertise regarding personal practice was shared. The former included, the Vanderbilt ADHD Teacher Rating Scale.

Section-A of Vanderbilt ADHD Teacher Rating Scale consists of 9 items related to 'inattention' and 'hyperactivity/ impulsiveness' symptoms (9 items each).

Scoring - Frequency Code: 0 = Never; 1 = Occasionally; 2 = Often; 3 = Very Often. Behaviors are counted if they are scored 2 (often) or 3 (very often).

A score of six or more of the 9 items related to 'only inattention', 'only hyperactivity/ impulsiveness' and 'both' indicate 'predominantly inattentive', 'predominantly hyperactive/ impulsive', and 'combined subtypes', respectively.

Inattention: Requires six or more counted behaviors from questions 1-9 for indication of the predominantly inattentive subtype.

Hyperactivity/ Impulsivity: Requires six or more counted behaviors from questions 10-18 for indication of the predominantly hyperactive/ impulsive subtype.

Combined Subtype: Requires six or more counted behaviors each on both the inattention and hyperactivity/ impulsivity dimensions.

The performance section is scored as indicating some impairment if a child scores 1 or 2 on at least one item.

Apart from these, Vanderbilt scale also has symptoms of oppositional defiant disorders, anxiety and depression ranging from 19-35. We have not done the scoring of these symptoms.

Venue: A cross-sectional observational study was conducted at three different schools that catered to higher middle class in Bhavnagar. Total 2114 students of these schools with age of 10 to 14 years studying in 5th- 9th standard were included.

Training: The teachers were trained in administration of Vanderbilt ADHD Diagnostic Teacher Rating Scale using a standardized operational manual in a half day structured workshop and were taught how to recognize the symptoms and fill up the forms. Two pediatricians were the trainers.

After training from each class suspected students of ADHD were selected by the teacher and then

ADHD symptom scale self report form (Vanderbilt scale) was filled by teachers for these students. Filled up forms were analyzed by pediatricians later on.

Results

Total number of schools covered was three and total number of children included was 2114. Out of this, 135 students were suspected by the teachers and for whom the forms were filled. 65 turned out to be positive.

Prevalence of ADHD in present study was 65/2114 (3.07%), out of whom 58.16% were suffering from mixed ADHD type, while 27.27% and 14.54% were of hyperactive and inattentive types respectively. Proportion of score positive students, age wise and school standard wise, is given in Table 1. Most affected age was 12 and 13 years ($p = 0.0023$, highly significant).

Table 1: Performance in ADHD Vanderbilt teacher rating scale by age group, school standard

Age group (years)	School standard	suspected 135, score positive (65), %	% score positive 65
10	5	21 (3) 14.2	4.6
11	6	9 (5) 55.5	7.7
12	7	32 (25) 78.1	38.46
13	8	37 (23) 62.1	35.38
14	9	36 (9) 25.0	13.86

$p = 0.0023$ highly significant between age group [10,11,14 vs 12,13].

Male to female ratio was 5.5. Out of 65 positive cases, 55 were boys (84.61%) and 10 were girls (15.38%). In both boys and girls mixed type of ADHD was more common followed by hyperactive and inattentive type.

Most common symptom of ADHD was 'excessive talking' 63/65 (96.62%); most common behavioral problem was 'not following rules' (70.7%). Least common was 'loses the thing necessary for work' 30/65 (46.52)

The most common affected area was *mathematics*. 39/65 were poor in mathematics (60%). When further divided, in mixed type of ADHD, 24/38 were poor in mathematics (63.15%), similarly in hyperactive type this ratio was 10/17 (58.82%), and in inattentive type, it was 5/10 (50%).

The confidence interval within which NO ADHD/ Borderline ADHD (few if any, symptoms

beyond those required to make the diagnosis and no more than minor impairment in functioning) should fall is (0.42- 0.64); in our study such students were at 0.53 CI. Cases of Severe ADHD (Severe is reserved for cases with many symptoms in excess of those required for the diagnosis, or several symptoms that are especially severe, or marked impairment resulting from symptoms.) should fall within (0.69- 0.93 CI); in our study such students were at 0.81 CI.

Discussion

This was the first study done in Bhavnagar, Gujarat to assess the prevalence of ADHD. Prevalence of ADHD in present study was 3.07%, which is lower than worldwide (5.3-7.2%) [2]. Vanderbilt ADHD Diagnostic Teacher Rating scale was used in our study, which is one of the most widely used measures of ADHD in the world today [3].

In our study, male to female ratio was 5.5. In the study done by Al zaben et al, prevalence of ADHD in girls and boys was found to be similar [4]. This higher incidence in girls could have been due to cultural differences in different societies in the perception of expected behavior from girls and so (where) even a slightly more than 'expected normal' is counted as non-confirmity. However, generally [5] and from other studies worldwide [6] boy-girl ratio has varied from 2:1 to 9:1.

We found that the most affected age was 12 and 13 years as compared to lesser age groups such as 10 to 11 years; this is in contrast to what has been reported earlier that ADHD prevalence decreases as the age advances [7]. We are not able to explain the reason for this anomaly. There is a fine line between ADHD and natural adolescent boys defiance behavior.

In other studies prevalence of ADHD is more among pre-school children [8,9], whereas our study was done in 10-14 year age group.

The prevalence of ADHD in present study was 65/2114 (3.07%), out of which 58.16% were suffering from mixed ADHD type while 27.27% and 14.54% were of hyperactive and inattentive ADHD type respectively, which is similar to study by Osman AM et al., in which the prevalence of children with ADHD/ inattentive sub type, ADHD/ hyperactive-impulsive sub type, ADHD/ combined subtype were 3.5%, 6.9 % and 1.0 %, respectively [10,11].

What this study adds

This was the first study done in Bhavnagar, Gujarat to predict the prevalence of ADHD in school going children. We did this study in the 10-14 year age group in contrast to most other studies where prevalence in *preschool* children was studied. Prevalence was highest in 12 and 13 years.

References

1. Vanderbilt ADHD Teacher Rating Scale. [https://www.childrenshospital.vanderbilt.org/uploads/documents/med-ped_VADTRS_Quest\(2\).pdf](https://www.childrenshospital.vanderbilt.org/uploads/documents/med-ped_VADTRS_Quest(2).pdf)
2. Homidi M, Obaidat Y, Hamaidi D. Prevalence of attention deficit and hyperactivity disorder among primary school students in Jeddah city, KSA. *Life Sci J*. 2013;10:280-85.
3. Wolraich ML, Bard DE, Neas B, Doffing M, Beck L. The psychometric properties of the Vanderbilt attention-deficit hyperactivity disorder diagnostic teacher rating scale in a community population. *J Dev Behav Pediatr*. 2013;34:83-93.
4. Al Zaben FN, Sehlo MG, Alghamdi WA, et al. Prevalence of attention deficit hyperactivity disorder and comorbid psychiatric and behavioral problems among primary school students in western Saudi Arabia. *Saudi Medical Journal*. 2018;39(1):52-8. doi:10.15537/smj.2018.1.21288.
5. Alhraiwi NJ, Ali A, Househ MS, Al-Shehri AM, El-Metwally AA. Systematic review of the epidemiology of attention deficit hyperactivity disorder in Arab countries. *Neurosciences*. 2015;20:137-44.
6. Rucklidge JJ. Gender differences in ADHD: implications for psychosocial treatments. *Expert Rev Neurother*. 2008;8:643-55.
7. Faraone SV, Biederman J, Mick E. The age-dependent decline of attention deficit hyperactivity disorder: a meta-analysis of follow-up studies. *Psychol Med*. 2006; 36:159-65.
8. Venkata JA, Panicker AS. Prevalence of attention deficit hyperactivity disorder in primary school children. *Indian J Psychiatry*. 2013;55:338-42.
9. Alqahtani MM. Attention-deficit hyperactivity disorder in school-age children in Saudi Arabia. *European J Pediatr*. 2010;169:1113-7.
10. Osman AM, Omer IM, Mohammed AA, Abdalla SE. The prevalence and factors affecting attention deficit hyperactivity disorder among school children in Khartoum State. *Sudanese J Pediatr*. 2015;15(2):29-36.
11. Dalwai S, Unni J, Kalra V, Singhi P, Shrivastava L, Nair MKC. Consensus Statement of the IAP on Evaluation and Management of ADHD. *Indian Pediatr*. 2017 Jun 15;54(6):481-8.

Rebound Hyperbilirubinaemia Following Intensive Phototherapy

Basavaraj M. Patil¹, Harsha A. Sangolgikar²

^{1,2}Associate Professor, Department of Pediatrics, Mahadevappa Rampure Medical College, Kalaburagi, Karnataka 585105, India.

Abstract

This prospective study was conducted in tertiary care hospital attached to Department of pediatrics, Mahadevappa Rampure Medical college Kalburgi. *Aims:* To determine the incidence of post phototherapy rebound hyperbilirubinemia in neonates subjected to phototherapy. *Materials and Methods:* All babies with hyperbilirubinemia admitted to nicu were included in the study. A total of 200 neonates among which 122 were males and 78 were females. Repeat serum bilirubin was measured 24 hours after stopping phototherapy. *Statistical analysis:* Data entry analysis were done using SPSS. *Results:* The general incidence of significant rebound hyperbilirubinemia requiring reinitiation of phototherapy was 9.5%. Birth weight and gestation age has significant impact on rebound hyperbilirubinemia. *Conclusion:* it is necessary to measure rebound bilirubin levels after 24 hrs of stoppage of phototherapy in high risk neonates like premature babies, birth weight <2kg.

Keywords: Bilirubin; Phototherapy; Significant Rebound.

Introduction

Neonatal jaundice is a common problem in newborn, 60% of term newborn and 80% premature infant develop clinical jaundice [1]. Severe jaundice can cause encephalopathy resulting in handicap or death. Intensive Phototherapy is used worldwide for the treatment of neonatal jaundice, resulting in reduced need of exchange transfusion.

Due to alteration in bilirubin production, excretion may persist and cause rebound hyperbilirubinemia after stopping phototherapy. This issue has been addressed previously by some studies, which were retrospective review charts than prospective [2].

This is a Prospective Study

Aims & Objectives

1. To determine the incidence of post phototherapy rebound hyperbilirubinemia in neonates subjected to phototherapy

2. To determine the incidence of significant hyperbilirubinemia
3. To determine the risk factors associated with significant hyperbilirubinemia

Materials and Methods

This study was conducted in tertiary care hospital attached to Department of Pediatrics, Mahadevappa Rampure Medical College kalaburgi.

Study Period

August 2015 to December 2016.

Sample

All babies with hyperbilirubinemia admitted in neonatal intensive care unit at tertiary care hospital attached to department of Paediatrics, Mahadevappa Rampure Medical college Kalaburagi, were included in the study.

Corresponding Author: Basavaraj M. Patil, Associate Professor, Department of Pediatrics, Mahadevappa Rampure Medical College, Kalaburagi, Karnataka 585105, India.
E-mail: dr.basavarajpatil@gmail.com

Received on 05.07.2018, **Accepted on** 30.07.2018

A total of 200 neonates among which 122 were males and 78 were females.

The criteria for starting and of phototherapy were based on American Academy of Pediatrics clinical practice guidelines and subcommittee on Hyperbilirubinemia. Management of hyperbilirubinemia in the newborn >35 weeks of Gestation [3].

For premature infants – infants less than 1 kg phototherapy was started with in 24 hrs, between 1 to 1.5kg – phototherapy at bilirubin levels of 7 to 9mg/dl. Between 1.5 to 2kg -10 to 12mg/dl, 2 to 2.5kg -13 to 15mg/dl [4] Significant bilirubin rebound was defined as postphototherapy bilirubin level needing reinstitution of phototherapy [2].

Repeat Serum bilirubin was measured 24 hours after stopping phototherapy

Inclusion Criteria

1. All term babies
2. All preterm babies
3. Birth asphyxia
4. Sepsis
5. Exchange transfusion

Exclusion Criteria

1. Major congenital anomalies

Method of Collection of Data

An informed consent was obtained from the parents of the newborn before enrolling them in the study. Serum bilirubin estimation was done by Diazo method.

Blood samples for bilirubin were collected from the baby at discontinuation and after 24 hours of stopping of phototherapy. A 'p' value <0.05 was considered statistically significant.

Data Analysis

Data was analysed, continuous variables were compared using student's t test. categorical data using chi square or fisher exact test. Data entry analysis were done using SPSS.

Results

Studies reveal that out of 200 babies subjected to phototherapy, 42 babies developed rebound bilirubinemia, of which 19 cases required reinitiation of phototherapy. This accounts to 9.5% (Table 1).

Table 1: Mean Sr.bilirubin count

Phototherapy	Sr.bilirubin Mean \pm SD	Paired t-test value	P-value & significant
Sr.bilirubin Initiation of phototherapy N=42	17.52 \pm 3.85	t= 12.25	P<0.001, Highly significant
Sr.bilirubin stopping phototherapy N=42	10.67 \pm 2.87	t= 9.12	P<0.001, Highly significant
Sr.bilirubin after 24 hours of phototherapy N=42	13.8 \pm 2.87	t= 3.75	P<0.01, Significant
Sr.bilirubin Reinitiation of phototherapy who required N=19	15.12 \pm 3.30		

Table 2: Association between risk factors required reinitiation of phototherapy

Risk factors	Initiation of phototherapy cases N= 42(%)	Required Reinitiation of phototherapy cases N=19	χ^2 -test & t-test values P-value & significance
Mode of delivery			
LSCS	08(19.0%)	03(15.8%)	$\chi^2=0.09$, P>0.05
NVD	34(81.0%)	16(84.2%)	Not Significant
Obstetric H/o			
Primi	26(62.0%)	12(63.2%)	$\chi^2=0.02$, P>0.05
Multigravida	16(38.0%)	07(36.8%)	Not Significant
Hb			t= 1.74, P>0.05
Mean \pm SD	16.6 \pm 2.8	15.1 \pm 3.3	Not Significant
HCT in %			t= 1.01, P>0.05
Mean \pm SD	46.4 \pm 18.72	40.74 \pm 21.9	Not Significant
Birth weight in kgs.			
< 2.0	14(33.3%)	11(58.0%)	$\chi^2=6.57$, P<0.05
2.0 - 2.5	09(21.4%)	04(21.0%)	Significant
≥ 2.5	19(45.3%)	04(21.0%)	P>0.05 Not significant
Gestational age			
Preterm	15(35.7%)	12(63.2%)	$\chi^2=4.03$, P<0.05
Term	27(64.3%)	07(36.8%)	Significant

Mode of delivery, gravidity, maternal, haemoglobin, didn't have impact on rebound hyperbilirubinemia. Birth weight and gestation age has significant impact on rebound hyperbilirubinemia (Table 2).

Discussion

Even though hyperbilirubinemia is common problem among neonates precise data regarding post phototherapy rebound hyperbilirubinemia, significant hyperbilirubinemia and its risk factors is inadequate.

Many studies report have been flawed by retrospective chart reviews [3].

Previous reports in literature have indicated that rebound hyperbilirubinemia is rare and therefore it is unnecessary to keep an infant in the hospital after phototherapy has been discontinued [2,5].

Factors reported to influence incidence of rebound hyperbilirubinemia include proportion of premature neonates and hemolytic jaundice, severity and onset of hyperbilirubinemia, mode of feeding and presence of other risk factors like G6PD deficiency [2,6].

The AAP Subcommittee on Hyperbilirubinemia now recommends a follow up bilirubin measurement within 24 hours of discharge for those cases in which phototherapy was used for neonates with haemolytic diseases, initiated early, or discontinued before the infant is 3-4 days old.

Specific recommendations on the initiation of phototherapy are well described. On the contrary, criteria for discontinuation of phototherapy are less well described [3].

In the study by Anuradha Bansal et al a total of 17 neonates out of 232 (7.3%) developed significant rebound hyperbilirubinemia. Our study is comparable with this study with incidence of 9.5%

In the study by Lee et al total of 7 babies out of 154 babies (4.5%) had a rebound jaundice which shows much lower incidence in their study compared to our study [7].

M Kaplan et al showed incidence of 13.3% 236 neonates out of which 30 developed significant rebound, which has higher incidence than our study

In our study group, risk factors associated with significant rebound were preterm babies and birth weight less than 2kg which is comparable with Anuradha Bansal et al.

Maisels and Kring et al. determined the incidence for rebound hyperbilirubinemia after stopping

phototherapy; And they do recommend repeated serum bilirubin checks 24 hours after discharge only if phototherapy was stopped at higher levels adopted in their study [8].

AL- Saedi et al measurement of serum bilirubin level is not required after termination of phototherapy and adds unnecessary expense, prolongs hospitalization. Which is contrary to our study [9].

Neurotoxic levels of bilirubin may vary with postnatal age, maturity of blood brain barrier, serum albumin concentration, according to Stets, Chung et al. [10].

Conclusion & Recommendations

1. Considering the neurotoxic effects of hyperbilirubinemia on the neonate. We recommend that it is mandatory to measure rebound bilirubin levels after 24 hours of stoppage of phototherapy in high risk neonates like premature babies, birth weight <2 kg.
2. Serum Bilirubin measurement after 24 hours reduces readmission in turn saving the difficulties of caretakers.

References

1. Namsivayam Ambalavanan and Waldemar A. Carlo. Jaundice and Hyperbilirubinemia in New Born. Nelson text book of paediatrics volume 1. chapter 102, page no 871. 20th edition.
2. Anuradha Bansal, Suksham Jain, Veena R Parmar, Deepak Chawla. Bilirubin rebound after intensive phototherapy for neonatal jaundice; Indian Pediatr: 2010;147:607-609.
3. American Academy of Pediatrics clinical practice guideline and subcommittee on hyperbilirubinemia. Management of hyperbilirubinemia in the newborn Infant 35 or more weeks of Gestation. Pediatrics. 2004 Jul;114(1):297-316.
4. Mary Lucia P, Greory, camilia R. Martin, and John P. Cloherty. Neonatal Hyperbilirubinemia. page no 325 seventh edition.
5. Erdevi O, Tiras U, Dallar Y. Rebound bilirubin measurement is not required for hyperbilirubinemia regardless of the background attributes of the newborns. JTrop Pediatr 2004;50:5.
6. Kaplan M, Kaplan E, Hammerman C, N Algur, R Bromiker, M S Schimmel, and A I Eidelman. Post phototherapy neonatal bilirubin rebound: a potential cause of significant hyperbilirubinaemia. Arch Dis Child 2006;91:31-34.

7. ACW Lee, WT Lung, R Li, H Ng, K Tse, NS Kwong; The Management of Neonatal Jaundice After Discontinuation of Phototherapy ; HK J Paediatr (newseries) 1999;4:92-95.
 8. Maisels MJ, Kring E. Rebound in serum bilirubin level following intensive phototherapy. Arch Pediatr Adolesc Med 2002;156:669-672.
 9. Saad A. Al-Saedi: Rebound hyperbilirubinaemia in term infants after phototherapy. Saudi Med J 2002;23 (11):1394-97.
 10. Ip S, Chung M, Kulig J, O'Brien R, Sege R, Glick S, Maisels MJ, Lau J. An evidence based review of important issues concerning neonatal hyperbilirubinemia. Pediatrics 2004;114:e130-153.
-

Rotavirus Vaccine

Sunil Mhaske¹, Vineetranjan Gupta²

¹Professor & Head ²Resident, Department of Paediatrics, P.D.V.V.P.F'S Medical College, Ahmednagar, Maharashtra 414111, India.

Abstract

Rotavirus is the most common cause of diarrhoeal disease among infants and young children. It is a genus of double-stranded RNA viruses in the family Reoviridae. Nearly every child in the world is infected with rotavirus at least once by the age of five. Immunity develops with each infection, so subsequent infections are less severe; adults are rarely affected. There are eight species of this virus, referred to as A, B, C, D, E, F, G and H. *Rotavirus A*, the most common species, causes more than 90% of rotavirus infections in humans. Rotavirus vaccine is a vaccine used to protect against rotavirus infections.

Keywords: Rotavirus; Vaccine.

Introduction

Rotavirus disease is highly contagious. The germ is present in the stool of an infected person and can remain viable for a long time on contaminated surfaces, including people's hands. Children catch it by touching something that's contaminated and then putting their hands in their mouth. The spread of rotavirus infection is a particular problem in hospitals and in day care settings, where it can be easily spread from child to child. It's also easily spread by day care workers, especially when they change diapers without washing their hands afterward. Rotavirus is a double-stranded RNA virus of the reovirus family. Viewed under an electron microscope, the virus is shaped like a wheel, hence its name (*rota* being Latin for "wheel"). Several species and subtypes of rotavirus cause disease in humans [1].

Under an electron microscope, the virus on several electron micrographs. Computer-aided reconstruction of a rotavirus based on several electron micrographs.

History

Rotaviruses were discovered in the 1960s in animals. The virus was first described in humans when it was found by electron microscopy in duodenal biopsies from children with acute gastroenteritis.

Rotavirus vaccine is a vaccine used to protect against rotavirus infections. These viruses are the leading cause of severe diarrhoea among young children. The vaccines prevent 15 to 34% of severe diarrhea in the developing world and 37 to 96% of severe diarrhea in the developed world. The vaccines appear to decrease the risk of death among young children due to diarrhea. Immunizing babies appears to decrease rates of disease among older people and those who have not been immunized [2].

The World Health Organization (WHO) recommends that rotavirus vaccine be included in national routine vaccinations programs, especially in areas where the disease is common. This should be done along with promoting breastfeeding, handwashing, clean water and good sanitation.

Corresponding Author: Vineetranjan Gupta, Resident, Department of Paediatrics, P.D.V.V.P.F'S Medical College, Ahmednagar, Maharashtra 414111, India.

E-mail: myvineet1689@gmail.com

Received on 13.07.2018, **Accepted on** 17.08.2018

The first vaccine for rotavirus, Human-rhesus RRV (Rota Shield). The first multivalent live oral reassortant vaccine developed was Rota Shield (a rhesus rotavirus tetravalent [RRV-TV] vaccine). This tetravalent vaccine contained a mixture of four virus strains representing the most commonly seen G types, G1 to G4: three rhesus-human reassortant strains containing the VP7 genes of human serotypes G1, G2, and G4 strains were substituted for the VP7 gene of the parent RRV, and the fourth strain comprised serotype G3 of rhesus RRV. RRV-TV was extensively evaluated in field trials in the United States, Finland, and Venezuela and proved highly effective (80 to 100%) in preventing severe diarrhea due to rotavirus in each of these settings. Due to the proven efficacy, the RRV-TV vaccine was licensed in August 1998 for routine use in children in the United States at 2, 4, and 6 months of age. Several cases of vaccine-associated intussusception were reported. The period of greatest risk of intussusception was shown to be 3 to 10 days after the first of three oral doses. As a consequence of this rare but potentially dangerous adverse effect, Wyeth, the manufacturer, withdrew Rota Shield from the market in the United States 14 months after its introduction. Unfortunately, the vaccine was not evaluated in terms of risk-benefit for children in resource-poor countries, as the ongoing trials in Asia (Bangladesh and India) and Africa (Ghana and South Africa) were stopped at that time [3].

There are currently three globally available vaccines against rotavirus: Merck's RotaTeq®, GlaxoSmithKline's Rotarix®, Rotavac®. Studies of these vaccines have demonstrated their safety and efficacy among children in every region of the world.

Clinical efficacy trials in Africa and Asia found that the vaccines dramatically reduced severe disease among infants in developing countries, where the majority of rotavirus deaths occur.

Rotarix is a monovalent, human, live attenuated rotavirus vaccine containing one rotavirus strain of G1P[8] specificity. ROTARIX is indicated for the prevention of rotavirus gastroenteritis caused by G1 and non-G1 types (G3, G4, and G9) when administered as a 2-dose series in infants and children [4].

RotaTeq® is an immunisation given to infants by mouth (orally) to protect them from severe rotavirus infection. RotaTeq® is free on the National Immunisation Schedule at 6 weeks, 3 months and 5 months of age.

Rotavac® should be administered as a 3-dose regimen, 4 weeks apart, beginning at 6 weeks of age

and should not be administered to children older than 8 months of age. Rotavac® has a shelf life of 5 years when stored at -20°C till the expiry date and can be stored for up to six months between 5°C ±3°C. Rotavac was licensed for use in India in 2014 and is manufactured by Bharat Biotech International Limited. It is a live attenuated, monovalent vaccine containing a G9P human strain isolated from an Indian child. It is given by mouth in a three-dose series, 4 weeks apart, beginning at 6 weeks of age up until 8 months of age [5].

Doses

RotaTeq® (RV5) is given in 3 doses at ages 2 months, 4 months, and 6 months

Rotarix® (RV1) is given in 2 doses at ages 2 months and 4 months.

Rotavac® (RV 116E) is given as a 3-dose regimen, 4 weeks apart, and should not be administered to children older than 8 months of age.

Adverse Effect

Common side effects of the rotavirus vaccines that may affect 1 to 10 in every 100 children includes: Irritability, loss of appetite, vomiting and diarrhoea (for up to 1 week after vaccination). A rare side effect of vaccination is an intestine (bowel) blockage (intussusception) within 7 days after the first vaccination [6].

Other Vaccine Approaches

Other approaches to the development of rotavirus vaccines are also being pursued. Rotavirus antigens for parenteral delivery have received some attention as virus-like particles prepared in baculovirus, expressed antigens, DNA vaccines, and killed virus. These novel approaches are being pursued using animal model [7].

Conclusion

Rotaviral gastroenteritis is associated with a substantial clinical and economic burden in both developed and developing countries. The disease burden is particularly considerable in infants and young children, producing infections that range from mild diarrhea to severe diarrhea, vomiting, and fever that result in hospitalization and death. The prevalence of the disease may be under-reported

because laboratory confirmation is not typically performed. Because there are currently no specific treatments for rotaviral infection, vaccination is the primary public health intervention for rotavirus infection. At present, approved vaccines (RotaTeq, Rotarix and Rotavaq) produce effective protection against disease (particularly severe disease), and decrease emergency room visits and hospitalizations [8].

References

1. International Drug Price Indicator Guide. Retrieved 6 December 2015.
 2. World Health Organization. April 2015. Retrieved 8 December 2016.
 3. Chanran A, Fitzwater S, Zhen A , et al. Prevention of rotavirus gastroenteritis in infants and children rotavirus vaccine safety, efficacy, and potential impact of vaccines. *Biologics*. 2010;4:213-29.
 4. World Health Organization. Rotavirus vaccines. WHO position paper January 2013. *Wkly Epidemiol Rec*. 2013;88:49-64.
 5. Hwang KP, Huang YC, Bányai K, et al. Severe gastroenteritis associated with G3P [9] rotavirus in Taiwan. *Infection*. 2011;39:271-5.
 6. Christie CD, Duncan ND, Thame KA, et al. Pentavalent rotavirus vaccine in developing countries safety and health care resource utilization. *Pediatrics*. 2010;126:e1499-506.
 7. The American Society of Health-System Pharmacists. Retrieved Dec 14, 2015.
 8. Releve epidemiologique hebdomadaire/Section d'hygiene du Secretariat de la Societe des Nations = Weekly epidemiological record/Health Section of the Secretariat of the League of Nations (*Wkly Epidemiol Rec*). 2013 Feb 1;88(5):49-64.
-

Neonatal Head Injury as a Potential Etiology for Autism Spectrum Disorders and Attention Deficit Hyperactivity Disorder (ADHD)

Santosh Kondekar¹, Amit Rudrakar², Sharan Subramanian³, Surbhi Rathi⁴

¹Associate Professor ^{2,3}Resident ⁴Professor, Department of Pediatrics, Topiwala National Medical College & BYL Nair Charitable Hospital, Mumbai, Maharashtra 400008, India.

Abstract

Autism and ADHD are wide spectrum neurodevelopmental disorders of children with uncertain aetiology. Early neonatal or fetal acute brain insult is one of the suspected aetiologies. In this case of Autism and ADHD history of neonatal head injury is suspected as an aetiological possibility. No such case is reported yet.

Keywords: Autism; ADHD; Head Injury; Neonatal; Subependymal Hemorrhage.

Introduction

There are infrequent case reports of known aetiologies for Autism and Attention deficit hyperactivity disorder. The literature regarding the outcome of non-accidental head injury (NAHI) is scarce and lacks specific detail even though it is generally considered to be poor. Few studies report a wide range of neurological sequelae in children who suffered inflicted traumatic brain injury in infancy [1]. These include motor deficits, visual deficits, epilepsy, speech and language abnormalities, and behavioural problems. There is limited information regarding the long-term outcome of inflicted traumatic brain injury (TBI), including shaken infant syndrome [2]. The purpose was to describe the long-term neurologic, behavioural, and cognitive sequelae seen in this population [3].

Case Report

We hereby report a case of a 7 year old girl, born out of non-consanguineous marriage by LSCS in view of non-progress of labour. On the first day of life, the child suffered non-accidental trauma,

allegedly being thrown by the father for a distance of over three feet in the hospital ward. There is also history of two episodes of fall from the father's hands in the neonatal period.

No immediate consequences were observed by the parents, and the child was not investigated further for the same. At one year of age, child was noticed to have speech delay in the setting of normal gross motor milestones. Over the next three years, parents noted child running amok, hyperactive, with significant paucity of eye contact, and gross deficit in social interaction and communication.

The child presented to us with these symptoms and was certified with a diagnosis of autism, attention deficit hyperactivity disorder with mental retardation. Child also developed three episodes of Grand tonic-clonic seizures and was started on Phenytoin and Risperidone.

A CT scan of the brain revealed subependymal calcification in the frontal horns and body of both lateral ventricles. This raised a dual possibility of tuberous sclerosis versus sequelae of remote trauma, and hence was further evaluated with an MRI of the brain. MRI showed evidence of subependymal micro-infarcts noted in bilateral temporal horns, which could represent sequelae of neonatal trauma suffered by the girl.

Corresponding Author: Santosh Kondekar, Associate Professor, Department of Pediatrics, Topiwala National Medical College & BYL Nair Charitable Hospital, Mumbai, Maharashtra 400008, India.
E-mail: drkondekar@gmail.com

Received on 27.02.2018, Accepted on 14.07.2018

Post imaging, the child was started on therapy cocktail for ADD/Autism, and consequently the child showed an improvement in activity and speech.

We therefore propose that the subependymal infarcts were probably related to concussion related injury on day of life one and probably resulted from the shear stress injury sustained by the brain. With the above correlation between clinical features and imaging findings, an etiological association between neonatal head injury and subsequent Autism/ADHD symptomatology cannot be ruled out.

Conclusion

Autism and ADHD may have a history of non specific early neonatal insult. A prudent , detailed neonatal resuscitation or trauma history may be fruitful in confirming the etiology in each case. Also modified therapy plan may help the child gain speech milestones. Therapy should be actively instituted to demonstrate early improvement in such cases.

Acknowledgement

We acknowledge Dr R Bharmal, Dean TNMC Mumbai for permission of the publication, and Dr Trupti Nikharge, Occupational therapist for her contribution for the case

References

1. Barlow K, Thompson E, Johnson D, Minns RA. The neurological outcome of non-accidental head injury. *Pediatr Rehabil.* 2004;7:195-203.
2. Keenan HT, Runyan DK, Marshall SW, Nocera MA, Merten DF, Sinal SH. A population-based study of inflicted traumatic brain injury in young children. *JAMA.* 2003;290:621-26.
3. Barlow KM. Late Neurologic and Cognitive Sequelae of Inflicted Traumatic Brain Injury in Infancy. *Pediatrics.* 2005;116(2):e174-85.

Manuscripts must be prepared in accordance with "Uniform requirements for Manuscripts submitted to Biomedical Journal" developed by international committee of medical Journal Editors.

Types of Manuscripts and Limits

Original articles: Up to 3000 words excluding references and abstract and up to 10 references.

Review articles: Up to 2500 words excluding references and abstract and up to 10 references.

Case reports: Up to 1000 words excluding references and abstract and up to 10 references.

Online Submission of the Manuscripts

Articles can also be submitted online from http://rfppl.co.in/customer_index.php.

1) First Page File: Prepare the title page, covering letter, acknowledgement, etc. using a word processor program. All information which can reveal your identity should be here. use text/rtf/doc/PDF files. Do not zip the files.

2) Article file: The main text of the article, beginning from Abstract till References (including tables) should be in this file. Do not include any information (such as acknowledgement, your name in page headers, etc.) in this file. Use text/rtf/doc/PDF files. Do not zip the files. Limit the file size to 400 Kb. Do not incorporate images in the file. If file size is large, graphs can be submitted as images separately without incorporating them in the article file to reduce the size of the file.

3) Images: Submit good quality color images. Each image should be less than 100 Kb in size. Size of the image can be reduced by decreasing the actual height and width of the images (keep up to 400 pixels or 3 inches). All image formats (jpeg, tiff, gif, bmp, png, eps etc.) are acceptable; jpeg is most suitable.

Legends: Legends for the figures/images should be included at the end of the article file.

If the manuscript is submitted online, the contributors' form and copyright transfer form has to be submitted in original with the signatures of all the contributors within two weeks from submission. Hard copies of the images (3 sets), for articles submitted online, should be sent to the journal office at the time of submission of a revised manuscript. Editorial office: Red Flower Publication Pvt. Ltd., 48/41-42, DSIDC, Pocket-II, Mayur Vihar Phase-I, Delhi - 110 091, India, Phone: 91-11-22754205,

45796900, 22756995. E-mail: author@rfppl.co.in. Submission page: http://rfppl.co.in/article_submission_system.php?mid=5.

Preparation of the Manuscript

The text of observational and experimental articles should be divided into sections with the headings: Introduction, Methods, Results, Discussion, References, Tables, Figures, Figure legends, and Acknowledgment. Do not make subheadings in these sections.

Title Page

The title page should carry

- 1) Type of manuscript (e.g. Original article, Review article, Case Report)
- 2) The title of the article, should be concise and informative;
- 3) Running title or short title not more than 50 characters;
- 4) The name by which each contributor is known (Last name, First name and initials of middle name), with his or her highest academic degree(s) and institutional affiliation;
- 5) The name of the department(s) and institution(s) to which the work should be attributed;
- 6) The name, address, phone numbers, facsimile numbers and e-mail address of the contributor responsible for correspondence about the manuscript; should be mentioned.
- 7) The total number of pages, total number of photographs and word counts separately for abstract and for the text (excluding the references and abstract);
- 8) Source(s) of support in the form of grants, equipment, drugs, or all of these;
- 9) Acknowledgement, if any; and
- 10) If the manuscript was presented as part at a meeting, the organization, place, and exact date on which it was read.

Abstract Page

The second page should carry the full title of the manuscript and an abstract (of no more than 150 words for case reports, brief reports and 250 words for original articles). The abstract should be structured and state the Context (Background), Aims, Settings and Design, Methods and Materials, Statistical analysis used, Results and Conclusions. Below the abstract should provide 3 to 10 keywords.

Introduction

State the background of the study and purpose of the study and summarize the rationale for the study or observation.

Methods

The methods section should include only information that was available at the time the plan or protocol for the study was written such as study approach, design, type of sample, sample size, sampling technique, setting of the study, description of data collection tools and methods; all information obtained during the conduct of the study belongs in the Results section.

Reports of randomized clinical trials should be based on the CONSORT Statement (<http://www.consort-statement.org>). When reporting experiments on human subjects, indicate whether the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional or regional) and with the Helsinki Declaration of 1975, as revised in 2000 (available at http://www.wma.net/e/policy/17-c_e.html).

Results

Present your results in logical sequence in the text, tables, and illustrations, giving the main or most important findings first. Do not repeat in the text all the data in the tables or illustrations; emphasize or summarize only important observations. Extra or supplementary materials and technical details can be placed in an appendix where it will be accessible but will not interrupt the flow of the text; alternatively, it can be published only in the electronic version of the journal.

Discussion

Include summary of key findings (primary outcome measures, secondary outcome measures, results as they relate to a prior hypothesis); Strengths and limitations of the study (study question, study design, data collection, analysis and interpretation); Interpretation and implications in the context of the totality of evidence (is there a systematic review to refer to, if not, could one be reasonably done here and now?, What this study adds to the available evidence, effects on patient care and health policy, possible mechanisms)? Controversies raised by this study; and Future research directions (for this particular research

collaboration, underlying mechanisms, clinical research). Do not repeat in detail data or other material given in the Introduction or the Results section.

References

List references in alphabetical order. Each listed reference should be cited in text (not in alphabetic order), and each text citation should be listed in the References section. Identify references in text, tables, and legends by Arabic numerals in square bracket (e.g. [10]). Please refer to ICMJE Guidelines (http://www.nlm.nih.gov/bsd/uniform_requirements.html) for more examples.

Standard journal article

[1] Flink H, Tegelberg Å, Thörn M, Lagerlöf F. Effect of oral iron supplementation on unstimulated salivary flow rate: A randomized, double-blind, placebo-controlled trial. *J Oral Pathol Med* 2006; 35: 540-7.

[2] Twetman S, Axelsson S, Dahlgren H, Holm AK, Källestål C, Lagerlöf F, et al. Caries-preventive effect of fluoride toothpaste: A systematic review. *Acta Odontol Scand* 2003; 61: 347-55.

Article in supplement or special issue

[3] Fleischer W, Reimer K. Povidone iodine antiseptics. *State of the art. Dermatology* 1997; 195 Suppl 2: 3-9.

Corporate (collective) author

[4] American Academy of Periodontology. Sonic and ultrasonic scalers in periodontics. *J Periodontol* 2000; 71: 1792-801.

Unpublished article

[5] Garoushi S, Lassila LV, Tezvergil A, Vallittu PK. Static and fatigue compression test for particulate filler composite resin with fiber-reinforced composite substructure. *Dent Mater* 2006.

Personal author(s)

[6] Hosmer D, Lemeshow S. *Applied logistic regression*, 2nd edn. New York: Wiley-Interscience; 2000.

Chapter in book

[7] Nauntofte B, Tenovou J, Lagerlöf F. Secretion and composition of saliva. In: Fejerskov O, Kidd EAM,

editors. Dental caries: The disease and its clinical management. Oxford: Blackwell Munksgaard; 2003. p. 7-27.

No author given

[8] World Health Organization. Oral health surveys - basic methods, 4th edn. Geneva: World Health Organization; 1997.

Reference from electronic media

[9] National Statistics Online – Trends in suicide by method in England and Wales, 1979-2001. www.statistics.gov.uk/downloads/theme_health/HSQ20.pdf (accessed Jan 24, 2005): 7-18. Only verified references against the original documents should be cited. Authors are responsible for the accuracy and completeness of their references and for correct text citation. The number of reference should be kept limited to 20 in case of major communications and 10 for short communications.

More information about other reference types is available at www.nlm.nih.gov/bsd/uniform_requirements.html, but observes some minor deviations (no full stop after journal title, no issue or date after volume, etc).

Tables

Tables should be self-explanatory and should not duplicate textual material.

Tables with more than 10 columns and 25 rows are not acceptable.

Table numbers should be in Arabic numerals, consecutively in the order of their first citation in the text and supply a brief title for each.

Explain in footnotes all non-standard abbreviations that are used in each table.

For footnotes use the following symbols, in this sequence: *, ¶, †, ‡.

Illustrations (Figures)

Graphics files are welcome if supplied as Tiff, EPS, or PowerPoint files of minimum 1200x1600 pixel size. The minimum line weight for line art is 0.5 point for optimal printing.

When possible, please place symbol legends below the figure instead of to the side.

Original color figures can be printed in color at

the editor's and publisher's discretion provided the author agrees to pay.

Type or print out legends (maximum 40 words, excluding the credit line) for illustrations using double spacing, with Arabic numerals corresponding to the illustrations.

Sending a revised manuscript

While submitting a revised manuscript, contributors are requested to include, along with single copy of the final revised manuscript, a photocopy of the revised manuscript with the changes underlined in red and copy of the comments with the point to point clarification to each comment. The manuscript number should be written on each of these documents. If the manuscript is submitted online, the contributors' form and copyright transfer form has to be submitted in original with the signatures of all the contributors within two weeks of submission. Hard copies of images should be sent to the office of the journal. There is no need to send printed manuscript for articles submitted online.

Reprints

Journal provides no free printed reprints, however a author copy is sent to the main author and additional copies are available on payment (ask to the journal office).

Copyrights

The whole of the literary matter in the journal is copyright and cannot be reproduced without the written permission.

Declaration

A declaration should be submitted stating that the manuscript represents valid work and that neither this manuscript nor one with substantially similar content under the present authorship has been published or is being considered for publication elsewhere and the authorship of this article will not be contested by any one whose name (s) is/are not listed here, and that the order of authorship as placed in the manuscript is final and accepted by the co-authors. Declarations should be signed by all the authors in the order in which they are mentioned in the original manuscript. Matters appearing in the Journal are covered by copyright but no objection will be made to their reproduction provided permission is

obtained from the Editor prior to publication and due acknowledgment of the source is made.

Approval of Ethics Committee

We need the Ethics committee approval letter from an Institutional ethical committee (IEC) or an institutional review board (IRB) to publish your Research article or author should submit a statement that the study does not require ethics approval along with evidence. The evidence could either be consent from patients is available and there are no ethics issues in the paper or a letter from an IRB stating that the study in question does not require ethics approval.

Abbreviations

Standard abbreviations should be used and be spelt out when first used in the text. Abbreviations should not be used in the title or abstract.

Checklist

- Manuscript Title
- Covering letter: Signed by all contributors
- Previous publication/ presentations mentioned, Source of funding mentioned
- Conflicts of interest disclosed

Authors

- Middle name initials provided.
- Author for correspondence, with e-mail address provided.
- Number of contributors restricted as per the instructions.
- Identity not revealed in paper except title page (e.g. name of the institute in Methods, citing previous study as 'our study')

Presentation and Format

- Double spacing
- Margins 2.5 cm from all four sides
- Title page contains all the desired information. Running title provided (not more than 50 characters)
- Abstract page contains the full title of the manuscript
- Abstract provided: Structured abstract provided for an original article.
- Key words provided (three or more)

- Introduction of 75-100 words
- Headings in title case (not ALL CAPITALS). References cited in square brackets
- References according to the journal's instructions

Language and grammar

- Uniformly American English
- Abbreviations spelt out in full for the first time. Numerals from 1 to 10 spelt out
- Numerals at the beginning of the sentence spelt out

Tables and figures

- No repetition of data in tables and graphs and in text.
- Actual numbers from which graphs drawn, provided.
- Figures necessary and of good quality (color)
- Table and figure numbers in Arabic letters (not Roman).
- Labels pasted on back of the photographs (no names written)
- Figure legends provided (not more than 40 words)
- Patients' privacy maintained, (if not permission taken)
- Credit note for borrowed figures/tables provided
- Manuscript provided on a CDROM (with double spacing)

Submitting the Manuscript

- Is the journal editor's contact information current?
- Is the cover letter included with the manuscript? Does the letter:
 1. Include the author's postal address, e-mail address, telephone number, and fax number for future correspondence?
 2. State that the manuscript is original, not previously published, and not under concurrent consideration elsewhere?
 3. Inform the journal editor of the existence of any similar published manuscripts written by the author?
 4. Mention any supplemental material you are submitting for the online version of your article. Contributors' Form (to be modified as applicable and one signed copy attached with the manuscript)

Revised Rates for 2018 (Institutional)

Title of the Journal	Frequency	India(INR)		Outside India(USD)	
		Print Only	Online Only	Print Only	Online Only
Community and Public Health Nursing	Triannual	5500	5000	430	391
Dermatology International	Semiannual	5500	5000	430	391
Gastroenterology International	Semiannual	6000	5500	469	430
Indian Journal of Agriculture Business	Semiannual	5500	5000	413	375
Indian Journal of Anatomy	Bi-monthly	8500	8000	664	625
Indian Journal of Ancient Medicine and Yoga	Quarterly	8000	7500	625	586
Indian Journal of Anesthesia and Analgesia	Monthly	7500	7000	586	547
Indian Journal of Biology	Semiannual	5500	5000	430	391
Indian Journal of Cancer Education and Research	Semiannual	9000	8500	703	664
Indian Journal of Communicable Diseases	Semiannual	8500	8000	664	625
Indian Journal of Dental Education	Quarterly	5500	5000	430	391
Indian Journal of Diabetes and Endocrinology	Semiannual	8000	7500	597	560
Indian Journal of Emergency Medicine	Quarterly	12500	12000	977	938
Indian Journal of Forensic Medicine and Pathology	Quarterly	16000	15500	1250	1211
Indian Journal of Forensic Odontology	Semiannual	5500	5000	430	391
Indian Journal of Genetics and Molecular Research	Semiannual	7000	6500	547	508
Indian Journal of Hospital Administration	Semiannual	7000	6500	547	508
Indian Journal of Hospital Infection	Semiannual	12500	12000	938	901
Indian Journal of Law and Human Behavior	Semiannual	6000	5500	469	430
Indian Journal of Legal Medicine	Semiannual	8500	8000	607	550
Indian Journal of Library and Information Science	Triannual	9500	9000	742	703
Indian Journal of Maternal-Fetal & Neonatal Medicine	Semiannual	9500	9000	742	703
Indian Journal of Medical & Health Sciences	Semiannual	7000	6500	547	508
Indian Journal of Obstetrics and Gynecology	Bi-monthly	9500	9000	742	703
Indian Journal of Pathology: Research and Practice	Monthly	12000	11500	938	898
Indian Journal of Plant and Soil	Semiannual	6000	6000	508	469
Indian Journal of Preventive Medicine	Semiannual	7000	6500	547	508
Indian Journal of Research in Anthropology	Semiannual	12500	12000	977	938
Indian Journal of Surgical Nursing	Triannual	5500	5000	430	391
Indian Journal of Trauma and Emergency Pediatrics	Quarterly	9500	9000	742	703
Indian Journal of Waste Management	Semiannual	9500	8500	742	664
International Journal of Food, Nutrition & Dietetics	Triannual	5500	5000	430	391
International Journal of Neurology and Neurosurgery	Quarterly	10500	10000	820	781
International Journal of Pediatric Nursing	Triannual	5500	5000	430	391
International Journal of Political Science	Semiannual	6000	5500	450	413
International Journal of Practical Nursing	Triannual	5500	5000	430	391
International Physiology	Triannual	7500	7000	586	547
Journal of Animal Feed Science and Technology	Semiannual	7800	7300	609	570
Journal of Cardiovascular Medicine and Surgery	Quarterly	10000	9500	781	742
Journal of Forensic Chemistry and Toxicology	Semiannual	9500	9000	742	703
Journal of Global Medical Education and Research	Semiannual	5900	5500	440	410
Journal of Global Public Health	Semiannual	12000	11500	896	858
Journal of Microbiology and Related Research	Semiannual	8500	8000	664	625
Journal of Nurse Midwifery and Maternal Health	Triannual	5500	5000	430	391
Journal of Orthopedic Education	Triannual	5500	5000	430	391
Journal of Pharmaceutical and Medicinal Chemistry	Semiannual	16500	16000	1289	1250
Journal of Plastic Surgery and Transplantation	Semiannual	26400	25900	2063	2023
Journal of Practical Biochemistry and Biophysics	Semiannual	7000	6500	547	508
Journal of Psychiatric Nursing	Triannual	5500	5000	430	391
Journal of Social Welfare and Management	Triannual	7500	7000	586	547
Medical Drugs and Devices Research	Semiannual	2000	1800	156.25	140.63
New Indian Journal of Surgery	Bi-monthly	8000	7500	625	586
Ophthalmology and Allied Sciences	Triannual	6000	5500	469	430
Otolaryngology International	Semiannual	5500	5000	430	391
Pediatric Education and Research	Triannual	7500	7000	586	547
Physiotherapy and Occupational Therapy Journal	Quarterly	9000	8500	703	664
RFP Indian Journal of Medical Psychiatry	Semiannual	8000	7500	625	586
RFP Journal of Gerontology and Geriatric Nursing	Semiannual	5500	5000	430	391
Urology, Nephrology and Andrology International	Semiannual	7500	7000	586	547

Terms of Supply:

1. Agency discount 10%. Issues will be sent directly to the end user, otherwise foreign rates will be charged.
2. All back volumes of all journals are available at current rates.
3. All Journals are available free online with print order within the subscription period.
4. All legal disputes subject to Delhi jurisdiction.
5. Cancellations are not accepted orders once processed.
6. Demand draft / cheque should be issued in favour of "Red Flower Publication Pvt. Ltd." payable at Delhi
7. Full pre-payment is required. It can be done through online (<http://rfppl.co.in/subscribe.php?mid=7>).
8. No claims will be entertained if not reported within 6 months of the publishing date.
9. Orders and payments are to be sent to our office address as given above.
10. Postage & Handling is included in the subscription rates.
11. Subscription period is accepted on calendar year basis (i.e. Jan to Dec). However orders may be placed any time throughout the year.

Order from

Red Flower Publication Pvt. Ltd., 48/41-42, DSIDC, Pocket-II, Mayur Vihar Phase-I, Delhi - 110 091 (India),
 Mobile: 8130750089, Phone: 91-11-45796900, 22754205, 22756995 E-mail: sales@rfppl.co.in, Website: www.rfppl.co.in

Red Flower Publication (P) Ltd.

Presents its Book Publications for sale

- | | |
|--|----------------------|
| 1. Shipping Economics (New for 2018) <i>by D. Amutha, Ph.D.</i> | INR345/USD27 |
| 2. Breast Cancer: Biology, Prevention and Treatment (2015)
<i>by Rana P. Singh, Ph.D. & A. Ramesh Rao, Ph.D. (JNU)</i> | INR395/USD100 |
| 3. Child Intelligence (2005) <i>by Rajesh Shukla, MD.</i> | INR150/USD50 |
| 4. Pediatric Companion (2001) <i>by Rajesh Shukla, MD.</i> | INR250/USD50 |

Order from

Red Flower Publication Pvt. Ltd.

48/41-42, DSIDC, Pocket-II

Mayur Vihar Phase-I

Delhi - 110 091(India)

Mobile: 8130750089, Phone: 91-11-45796900, 22754205, 22756995

E-mail: sales@rfppl.co.in

SUBSCRIPTION FORM

I want to renew/subscribe international class journal **"Pediatric Education and Research"** of Red Flower Publication Pvt. Ltd.

Subscription Rates:

- Institutional: INR7500/USD586

Name and complete address (in capitals): _____

Payment detail:

Online payment link: <http://rfppl.co.in/payment.php?mid=15>

Cheque/DD: Please send the US dollar check from outside India and INR check from India made payable to 'Red Flower Publication Private Limited'. Drawn on Delhi branch.

Wire transfer/NEFT/RTGS:

Complete Bank Account No. 604320110000467

Beneficiary Name: Red Flower Publication Pvt. Ltd.

Bank & Branch Name: Bank of India; Mayur Vihar

MICR Code: 110013045

Branch Code: 6043

IFSC Code: BKID0006043 (used for RTGS and NEFT transactions)

Swift Code: BKIDINBBDOS

Term and condition for supply of journals

1. Advance payment required by Demand Draft payable to Red Flower Publication Pvt. Ltd. payable at Delhi.
2. Cancellation not allowed except for duplicate payment.
3. Agents allowed 10% discount.
4. Claim must be made within six months from issue date.

Mail all orders to

Subscription and Marketing Manager

Red Flower Publication Pvt. Ltd.

48/41-42, DSIDC, Pocket-II

Mayur Vihar Phase-I

Delhi - 110 091(India)

Mobile: 8130750089, Phone: 91-11-45796900, 22754205, 22756995

E-mail: sales@rfppl.co.in, Website: <http://www.rfppl.co.in>

Revised Rates for 2019 (Institutional)

Title of the Journal	Frequency	India(INR)	India(INR)	Outside India(USD)	Outside India(USD)
		Print Only	Online Only	Print Only	Online Only
Community and Public Health Nursing	Triannual	5500	5000	430	391
Dermatology International	Semiannual	5500	5000	430	391
Gastroenterology International	Semiannual	6000	5500	469	430
Indian Journal of Agriculture Business	Semiannual	5500	5000	413	375
Indian Journal of Anatomy	Bi-monthly	8500	8000	664	625
Indian Journal of Ancient Medicine and Yoga	Quarterly	8000	7500	625	586
Indian Journal of Anesthesia and Analgesia	Monthly	7500	7000	586	547
Indian Journal of Biology	Semiannual	5500	5000	430	391
Indian Journal of Cancer Education and Research	Semiannual	9000	8500	703	664
Indian Journal of Communicable Diseases	Semiannual	8500	8000	664	625
Indian Journal of Dental Education	Quarterly	5500	5000	430	391
Indian Journal of Diabetes and Endocrinology	Semiannual	8000	7500	597	560
Indian Journal of Emergency Medicine	Bi-Monthly	12500	12000	977	938
Indian Journal of Forensic Medicine and Pathology	Quarterly	16000	15500	1250	1211
Indian Journal of Forensic Odontology	Semiannual	5500	5000	430	391
Indian Journal of Genetics and Molecular Research	Semiannual	7000	6500	547	508
Indian Journal of Hospital Administration	Semiannual	7000	6500	547	508
Indian Journal of Hospital Infection	Semiannual	12500	12000	938	901
Indian Journal of Law and Human Behavior	Tri-Annual	6000	5500	469	430
Indian Journal of Legal Medicine	Semiannual	8500	8000	607	550
Indian Journal of Library and Information Science	Triannual	9500	9000	742	703
Indian Journal of Maternal-Fetal & Neonatal Medicine	Tri-Annual	9500	9000	742	703
Indian Journal of Medical & Health Sciences	Semiannual	7000	6500	547	508
Indian Journal of Obstetrics and Gynecology	Bi-monthly	9500	9000	742	703
Indian Journal of Pathology: Research and Practice	Monthly	12000	11500	938	898
Indian Journal of Plant and Soil	Semiannual	6500	6000	508	469
Indian Journal of Preventive Medicine	Semiannual	7000	6500	547	508
Indian Journal of Research in Anthropology	Semiannual	12500	12000	977	938
Indian Journal of Surgical Nursing	Triannual	5500	5000	430	391
Indian Journal of Trauma and Emergency Pediatrics	Quarterly	9500	9000	742	703
Indian Journal of Waste Management	Semiannual	9500	8500	742	664
International Journal of Food, Nutrition & Dietetics	Triannual	5500	5000	430	391
International Journal of Neurology and Neurosurgery	Quarterly	10500	10000	820	781
International Journal of Pediatric Nursing	Triannual	5500	5000	430	391
International Journal of Political Science	Semiannual	6000	5500	450	413
International Journal of Practical Nursing	Triannual	5500	5000	430	391
International Physiology	Triannual	7500	7000	586	547
Journal of Animal Feed Science and Technology	Semiannual	7800	7300	609	570
Journal of Cardiovascular Medicine and Surgery	Quarterly	10000	9500	781	742
Journal of Forensic Chemistry and Toxicology	Semiannual	9500	9000	742	703
Journal of Global Medical Education and Research	Semiannual	5900	5500	440	410
Journal of Global Public Health	Semiannual	12000	11500	896	858
Journal of Microbiology and Related Research	Semiannual	8500	8000	664	625
Journal of Nurse Midwifery and Maternal Health	Triannual	5500	5000	430	391
Journal of Orthopedic Education	Triannual	5500	5000	430	391
Journal of Pharmaceutical and Medicinal Chemistry	Semiannual	16500	16000	1289	1250
Journal of Plastic Surgery and Transplantation	Semiannual	26400	25900	2063	2023
Journal of Practical Biochemistry and Biophysics	Semiannual	7000	6500	547	508
Journal of Psychiatric Nursing	Triannual	5500	5000	430	391
Journal of Social Welfare and Management	Triannual	7500	7000	586	547
New Indian Journal of Surgery	Monthly	8000	7500	625	586
Ophthalmology and Allied Sciences	Quarterly	6000	5500	469	430
Otolaryngology International	Semiannual	5500	5000	430	391
Pediatric Education and Research	Triannual	7500	7000	586	547
Physiotherapy and Occupational Therapy Journal	Quarterly	9000	8500	703	664
RFP Indian Journal of Medical Psychiatry	Semiannual	8000	7500	625	586
RFP Journal of Gerontology and Geriatric Nursing	Semiannual	5500	5000	430	391
Urology, Nephrology and Andrology International	Semiannual	7500	7000	586	547

Terms of Supply:

1. Agency discount 12.5%. Issues will be sent directly to the end user, otherwise foreign rates will be charged.
2. All back volumes of all journals are available at current rates.
3. All Journals are available free online with print order within the subscription period.
4. All legal disputes subject to Delhi jurisdiction.
5. Cancellations are not accepted orders once processed.
6. Demand draft / cheque should be issued in favour of "Red Flower Publication Pvt. Ltd." payable at Delhi
7. Full pre-payment is required. It can be done through online (<http://rfppl.co.in/subscribe.php?mid=7>).
8. No claims will be entertained if not reported within 6 months of the publishing date.
9. Orders and payments are to be sent to our office address as given above.
10. Postage & Handling is included in the subscription rates.
11. Subscription period is accepted on calendar year basis (i.e. Jan to Dec). However orders may be placed any time throughout the year.

Order from

Red Flower Publication Pvt. Ltd., 48/41-42, DSIDC, Pocket-II, Mayur Vihar Phase-I, Delhi - 110 091 (India),
Mobile: 8130750089, Phone: 91-11-45796900, 22754205, 22756995 E-mail: sales@rfppl.co.in, Website: www.rfppl.co.in