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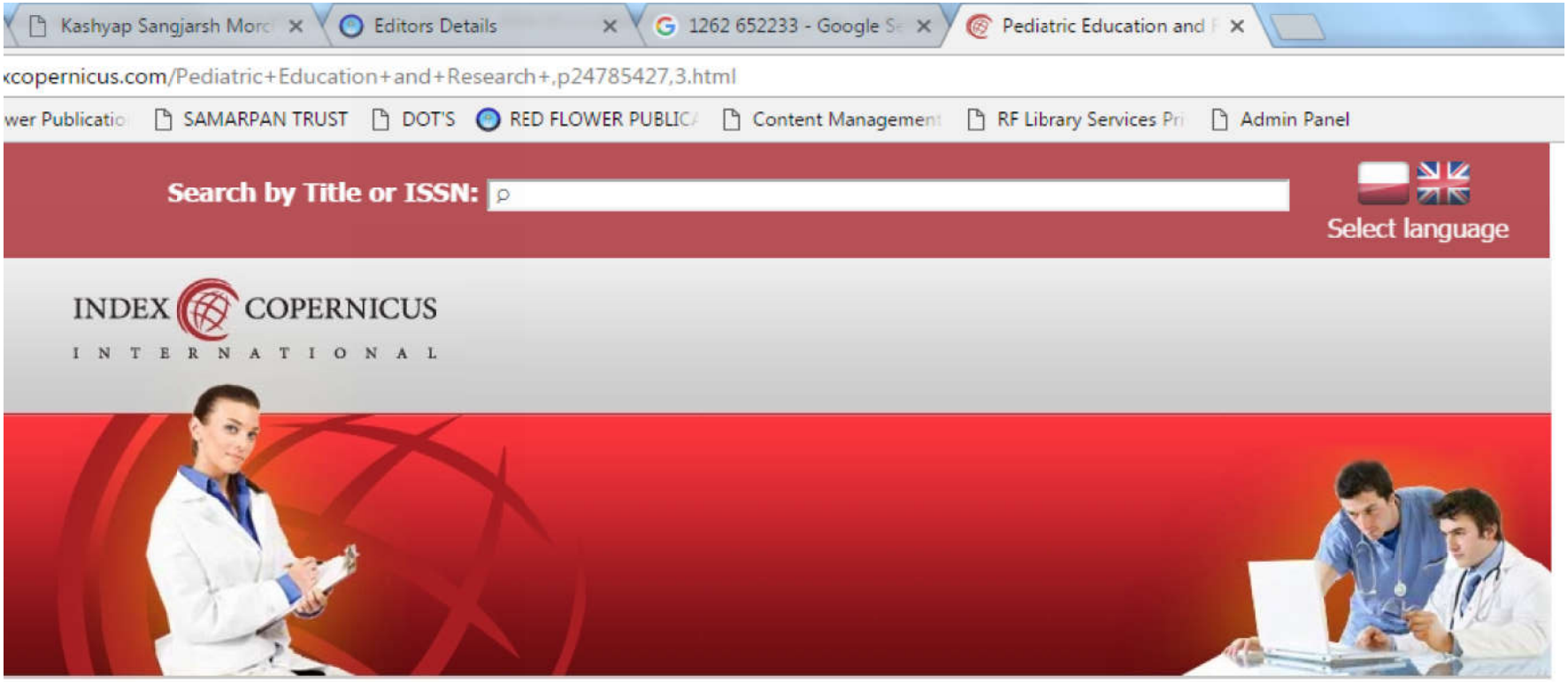
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Sociodemographic and Perinatal Factors Affecting Pneumonia: A Cross Sectional Study

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Abstract

Objective: Objective was to evaluate the sociodemographic and perinatal factors determining severity of acute lower respiratory tract infection (ALRTI) among children below five years. *Materials and Methods:* Cross sectional study conducted at HSK hospital & Research centre, Bagalkot, from March-2014 to August-2015. 200 ALRI cases aged 2 months to 5 years as per WHO criteria for pneumonia were evaluated for sociodemographic and perinatal factors determining severity of pneumonia. *Results:* In our study out of 200 cases admitted with ALRTI 3 had Pneumonia, 155 had Severe Pneumonia and 42 had Very Severe Pneumonia according to WHO ARI programme. There were 116(58%) male in the study. More than half were infants 107(53.5%), 57(28.5%) were of birth order 3 or more and 88(44%) were living in overcrowded houses. 36(18%) were home delivery, 32(16%) were preterms and 43(21.5%) had low birth weight (LBW). In our study 41(20.5%) cases received pre lacteal feeds, 138(69%) cases were exclusively breast fed and 53 (26.5%) were incompletely immunized. Statistical significance was found between EBF, gestation at delivery and pneumonia severity. *Conclusion:* Certain factors like young age, male sex, overcrowding, high birth order, low SES, prematurity, lack of EBF, Incomplete immunization, are associated with increased incidence of pneumonia. Measures like improving the living conditions and educating about need for family planning, birth spacing and adequate antenatal care to prevent preterm, LBW, overcrowding and malnutrition can decrease the ALRTI incidence. Promotion of EBF, immunization coverage and adequate perinatal care can reduce the disease burden.

Keywords: WHO; ALRTI; Pneumonia; EBF.

Introduction

Acute Lower Respiratory tract infection (ALRTI) is the leading cause of under-5 childhood morbidity in the world, with nearly 156 million new episodes each year, of which India accounts for a bulk of 43 million [1]. Of the 7.6 million children who died in the first five years of life in 2010, Pneumonia caused 1.4 million deaths (18.3%) and 4% of that are in the neonatal period. In India an estimated 4 lakh pneumonia deaths occurs annually, which is highest among all the countries in the world [2]. The incidence of pneumonia is more than 10 fold higher and number of childhood related death due to pneumonia around 2000 fold higher in developing than in developed country [3]. This difference is due to high prevalence of malnutrition, low birth weight and indoor pollution in developing countries [4].

Respiratory-tract infections are caused by a mixture of viral and bacterial pathogens and are particularly common in low birth weight children and children exposed to poor nutritional conditions, indoor air pollution, low socioeconomic status, overcrowded living condition, HIV co-infection, vitamin D deficiency, and low immunization coverage [5].

Pneumonia not only has an immediate effect

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during childhood, but can also lead to impaired lung function years later in adulthood.[6]Respiratory tract infections increases demands for medical interventions in terms of both outpatient services and hospital admissions causing significant financial burden on the economy of the country. Also cause missed school days, and caring for a sick child may lead to absenteeism from work.

The utility of simple clinical signs like rapid breathing and chest in drawing to diagnose pneumonia in infants and young children has been well established. The use of these clinical signs in the early detection and treatment of children with pneumonia by primary health care workers forms the basis for the case management strategy formulated by the WHO to control mortality and morbidity [7]. It is important to know risk factors that can be used to identify a child who have a higher risk of dying from ALRTI so that these children can be given more intensive therapy [8].

Since ALRTI is the commonest cause of morbidity and mortality in under five children, the aim is to study these children's sociodemographic and perinatal factors determining severity of ALRTI.

Objectives

To evaluate the sociodemographic and perinatal factors determining severity of ALRTI.

Material and Method

We conducted a Cross Sectional study of ALRTI in children aged 2 months to 5 years as per WHO guidelines [7] over a period of 18 months from March 2014 to August 2015 admitted in tertiary care teaching hospital.

Children with Congenital Heart Diseases, Tuberculosis, Bronchial asthma, Hospital acquired illness and those who were admitted outside for the same illness were excluded from the study as it could alter the severity and outcome of cases.

The data collected was tabulated in Microsoft Excel and analyzed with SPSS software, Chi Square test for proportion and other appropriate statistical tests like percentage and proportions were applied.

Results

In our study out of 200 cases admitted with ALRTI 3 had Pneumonia, 155 had Severe Pneumonia and 42 had Very Severe Pneumonia according to WHO ARI programme.

Table 1: Comparing Socio demographic factors with Pneumonia severity

Variability		Pneumonia	Severe	Very severe	Total	'p' value
Age in months	2-12	0	86	21	107	0.142
	13-60	3	69	21	93	
Sex	Male	3	88	25	116	0.315
	Female	0	67	17	84	
Birth order	1	2	49	6	57	0.21
	2	1	62	23	86	
	3	0	25	9	34	
	≥3	0	14	4	23	
SES	I	0	4	0	4	0.381
	II	2	30	11	43	
	III	1	57	12	69	
	IV	0	60	16	77	
	V	0	4	3	7	
Overcrowding	Yes	1	68	19	88	0.92
	No	2	87	23	112	
Total		3	155	42	200	

'p' <0.05 was considered as statistically significant.

In the present study there were 116 (58%) male and 84 (42%) female cases out of which 107 (53.5%) were infants and remaining 93 (46.5%) were between the age group of 1-5 years. 153 (76.5%) were of lower social status (class III-V) and 47 (23.5%) were belonging to upper status (class I and II). 57 (28.5%) are of birth order 3 or more than 3 and 88 (44%) were living in overcrowded condition

No statistical significance was found with WHO severity classification of Pneumonia and the studied

socio demographic risk factors. Majority of the cases were of birth order 2(86) belonging to class III (69) and class IV (77) living in overcrowding condition (44%).

Table 2: Comparing Perinatal risk factors with Pneumonia severity

Variability		Pneumonia	Severe	Very Severe	Total	'p' value
Place of Delivery	Hospital	2	128	34	164	0.762
	Home	1	27	8	36	
Gestation	Term	2	125	41	168	**0.021**
	Preterm	1	30	1	32	
Birth weight	≥2.5	2	91	34	127	0.065
	≤2.5	0	37	6	43	
	Not known	1	27	2	30	
Immunization	complete	2	116	29	147	0.725
	Incomplete	1	39	39	53	
Prelacteal feeds	Yes	0	33	8	41	0.642
	No	3	122	34	159	
EBF	Yes	2	114	22	138	**0.031**
	No	1	41	20	62	
Total		3	155	42	200	

In our study out of 200 cases 36 (18%) were delivered at home, 32 (16%) were preterm births and 41 (20.5%) cases received pre lacteal feeds. 53 (26.5%) were with missed immunization at birth or with incomplete immunization for age.127 (63.5%) were of normal birth weight, 43 (21.5%) were low birth weight and birth weight was not known in 30 (15%) cases. In our present *study statistical significance was found between pneumonia severity and gestation at delivery, Exclusive breast feeding*.

Discussion

In the present study, 200 ALRI cases were studied for the risk factors affecting pneumonia severity. ALRI among infants was 53.5% which in accordance with previous studies by Savitha et al [9], Broor et al [10] and Sehgal et al [11] where infants with ALRI accounted about 52-62%. Young age (infants <12 months) was found to be risk factor for poor prognosis [12]. This could be due to waning of maternally conferred passive immunity towards the latter half of infancy with other contributing factors like narrow airways.

We observed that male (58%) outweighed females (42%) which were in comparison with studies done by Savitha et al [9] (64.4%), Broor et al [10] (73.1%). This probably can be explained by the importance of cultural factors, such as preference in seeking medical care for boys.

76.5% cases belonged to lower socioeconomic status and overcrowding was seen in 44% cases. 28.5% were of birth order ≥3. All these can predispose the children to acquire ALRTI due to increased prevalence of malnutrition, poor ventilation and spread of viral infections. Savitha et al [9] found parental illiteracy, low socioeconomic status, overcrowding and partial immunization were significant socio demographic risk factors for ALRI. They also found that administration of prelacteal feeds, early weaning, anaemia and malnutrition as significant risk factors.

In our study, 18% cases were home delivery, 16% were preterm births and 21.5% had low birth weight. 20.5% cases received pre lacteal feeds and 26.5% were with partial/incomplete immunization. Only gestation was significantly associated with pneumonia severity. This could be explained by higher incidence of malnutrition, anaemia among preterms.

Tupasi et al [13] in their study found that the risk factors that increase the incidence and severity of lower respiratory infection in developing countries include large family size, higher birth order, overcrowding, low birth weight, malnutrition, lack of breastfeeding, pollution and young age. Zafar F et al [14] observed that delivery at home and respiratory infection in the early neonatal period were found to be associated with increased likelihood of pneumonia compared to birth in hospitals. Jackson et al [15] in their review found consistent significant association between 7 risk factors (low-birth-weight, undernutrition, indoor air pollution, incomplete immunization at one year, HIV, breastfeeding, and crowding) and severe ALRI.

Conclusion

Acute Lower Respiratory Tract infection remains one of the major causes of morbidity and mortality in children and frequent cause of health care seeking both on outpatient and inpatient basis. Infants and male children are affected frequently.

Among the studied risk factors period of exclusive breast feeding and gestational age at birth were significantly associated with pneumonia severity and majority belonged to lower socio economic group living in overcrowding condition. Young age, preterms, Low-birth weight babies, malnutrition, overcrowding, Indoor pollution and poor socioeconomic status continues to be an important predisposing factor for childhood respiratory illness.

Measures like improving the living conditions and educating about need for family planning, birth spacing and adequate antenatal care to prevent preterm, low-birth weight, overcrowding and malnutrition can decrease the ALRTI incidence. Effective utilization of under-five clinics to ensure availability of proper nutrition to combat malnutrition and anaemia, and increasing the immunization coverage and promoting exclusive breast feeding can reduce the disease burden. Training of local health personnel in early recognition, treatment and referral of sick and at-risk children helps to improve the morbidity and mortality profile.

References

1. Rudan I, Boschi-Pinto C, Biloglav Z, Mulholland K, Campbell H. Epidemiology and etiology of childhood pneumonia. Bull World Health Organ. 2008; 86:408-16.
2. The Inter-Agency Group for Child Mortality Estimation (IGME). Estimates of underfive mortality rates by country, the 2011 release. www. childmortality.org (accessed Jan, 2013).
3. Thoma J. Sandora and Theodore CS. Community-acquired Pneumonia. Nelson Textbook of Pediatrics, 19thedi. Elsevier Saunders Vol 2; 1474-9.
4. WHO (1999), Health Situation in the South-East Asia Region 1994- 1997, Regional Office for SEAR, New Delhi.
5. Wonodi CB, Deloria-Knoll M, Feikin DR, et al, for the Pneumonia Methods Working Group and PERCH Site Investigators. Evaluation of risk factors for severe pneumonia in children: the Pneumonia etiology Research for Child Health study. Clin Infect Dis 2012; 54:S124-31.
6. Johnston I, Strachan DP, Anderson HR. Effect of pneumonia and whooping cough in childhood on adult lung function. N Engl J Med 1998; 338:581-7.
7. World Health Organization programme for the control of acute respiratory infections. Technical bases for the WHO recommendations on management of pneumonia in children at first level health facilities. Geneva: WHO; 1995.
8. Kumar A, Saha E, Patra D, Chakraborty S. Outcome of Acute Lower Respiratory Tract Infection in Children. Indian Medical Gazette, 2011 Oct; 394-9.
9. Savitha MR, Nandeeshwara SB, Pradeep MJ, Farhan-ul-haq, Raju CK. Modifiable risk factors for acute lower respiratory tract infections. Indian Journal of Pediatrics 2007; 74:477-82.
10. Broor S, Pandey RM, Ghosh M, Maitreyi RS, Rakesh L, Tanu S et al. Risk factors for severe acute lower respiratory tract infection in under five Children. Indian Pediatrics 2001; 38: 361-9.
11. Sehgal V, Sethi GR, Sachdev HPS, Satyanarayana V. Predictors of mortality on subjets hospitalized with acute lower respiratory tract infections. Indian Pediatr, 1997; 34:213-9.
12. Jroundi I et al. Risk factors for a poor outcome among children admitted with clinically severe pneumonia to a university hospital in Rabat, Morocco. International Journal of Infectious Diseases. 2014; 28:164-70.
13. Tupasi TE, Velmonte MA, Sanvictores ME et al. Determinants of morbidity and mortality due to acute respiratory infections: implications for intervention. J Infect Dis 1988; 157: 615-23.
14. Zafar F, White F. A comparison of 'cough and cold' and pneumonia: risk factors for pneumonia in children under 5 years revisited. Int J Infect Dis 2002; 6:294-301.
15. Jackson et al: Risk factors for severe acute lower respiratory infections in young children- a systematic review and meta-analysis. Croat Med J. 2013; 54:110-21.

Original Article

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Determinants of Nutritional Anaemia in Adolescents

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Abstract

Objectives: To associate the severity of nutritional anaemia with serum levels of ferritin, vitamin B12 and folate; and to determine demographic, socio-economic and nutritional correlates for nutritional anemia in adolescents. *Methods:* Cross-sectional hospital-based study among 200 adolescents (10-18 y) with anemia. Dietary intake (24-h recall), and serum levels of folate, vitamin B12 and ferritin were estimated. *Results:* Iron, folate and vitamin B12 deficiency was present in 30.5% 79.5% and 50% of adolescents, respectively. Statistically significant association was observed between severity of anemia and serum vitamin B12 levels, iron intake, folate intake, Vitamin B12 intake, vegetarian diet, attainment of menarche and history of worm infestation. *Conclusion:* Folate and vitamin B12 deficiencies are more common than iron deficiency in anemic adolescents. Low dietary intake of these nutrients seems to be a significant determinant of their deficiencies.

Keywords: Ferritin; Folic Acid; Iron-Deficiency; Vitamin B12 Deficiency.

Introduction

Adolescence is a vulnerable period in the human life cycle for the development of nutritional anemia. Anemia has a negative effect on cognitive performance in adolescents [1]. Choudhary, et al [2]. reported two-thirds of the anemic adolescents in community were suffering from iron deficiency anemia (IDA). In a study by Patra, et al. [3] on severely anemic adolescents admitted in a tertiary care hospital, megaloblastic anemia was most common type of anemia (42.5%) and iron-deficiency accounted for 15% cases. Compared to the vast amount of work done in pregnant mothers and young children, there are relatively few published studies in India evaluating deficiencies of Iron, Vitamin B12 and Folate in adolescents having nutritional anemia and its association with severity of anemia. This study was planned with an objective of finding association of the levels of micronutrients with the severity of nutritional anemia in adolescents.

Adolescence has been defined by the World Health Organization as the period of life spanning the ages between 10 to 19 years [4]. This is the formative period of life when the maximum amount of physical, psychological and behavioral changes takes place and this is a vulnerable period in the human life cycle for the development of nutritional anaemia, which has been constantly neglected by public health programs [3]. The prevalence is high in developing countries like India & is mainly ascribed to poverty, inadequate diet, pregnancy & lactation, late diagnosis and poor access to health services in developing countries [5].

Nutritional anaemia constitutes the most important cause of anaemia in adolescents. It is mainly due to deficiency of Iron, Vitamin B12 and Folate. Young people are particularly susceptible to develop anaemia because of their rapid growth and associated high iron requirements. Also adolescent's eating behavior is guided by many factors such as personal self-esteem & body image, making them skip meals to

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reduce weight and peer pressure indulging them in unhealthy food habits making them prone to nutritional anaemia .

Aim & Objectives

1. To study the types of nutritional anemia in adolescents (10-18 yrs.) attending the hospital and correlate severity of nutritional anemia with serum levels of ferritin, Vit B12 & folate.
2. To determine demographic, socio-economic & nutritional factors for anemia in adolescents.

Materials and Methods

The study was a cross-sectional observational study. Adolescents coming to outpatient department or admitted in the hospital satisfying the inclusion criteria were included in the study.

Adolescents (age 10-18 years) attending the outpatient department or admitted in the hospital, and having hemoglobin values below the cut-offs (Hb <12g/dL in 10-18 y girls and 10-14 y boys and Hb<13g/dL in 15-18 y boys) were included in the study after written informed consent from parent/guardian. Those who had received blood transfusion or hematinics in past 4 weeks or having apparent infection (fever, diarrhea, cough or burning micturition) or any chronic disease were excluded from the study. A detailed history and physical examination of the study population was carried out. The premorbid dietary intake of the child was assessed by 24-hour recall method. This data was entered in 'Diet soft' software from which the daily intake of iron, folate and vitamin B12 was calculated. Complete blood count with peripheral smear examination was done. Serum Ferritin was estimated.

The severity of anemia was graded as mild (>10 g/dL but below age related cut-off for defining anemia), moderate (7-9.9 g/dL) and severe (<7g/dL). Serum vitamin B12 level of <200 pg/mL, folate level <5 ng/mL and ferritin level < 30ng/mL were considered as deficient.

Inclusion Criteria

Age group: 10-18yr age.

Haemoglobin levels below the following cut-off values:

Girls aged 10-18yrs Hb <12gm/dl.

Boys aged 10-14yrs Hb <12gm/dl.

Boys aged 15-18yrs Hb <13gm/dl.

Exclusion Criteria

Those who received blood transfusion in past 4 weeks. Those with known haematological or any other systemic disorder (thalassemia etc.) and/or evidence of apparent chronic infection (Tuberculosis, malaria etc.)

Table 1:

Severity of anaemia	Males	Females	Total
Mild (11-11.9 gm/dl)	56 (56%)	44(44%)	100 (100%)
Moderate (8-10.9 gm/dl)	47 (47%)	53 (53%)	100 (100%)
Severe (<8 gm/dl)	35 (35%)	65 (65%)	21 (100%)

Discussion

Our study showed that deficiency of folate, vitamin B12 and iron are common in anemic adolescents. Higher proportion of severely anemic individuals (20.5%) in our study can be attributed to hospital-based nature of the study. Prevalence of iron deficiency, vitamin-B12 deficiency and folate deficiency in our study is comparable to earlier reports from India [3-6]. Under the National programs (Iron plus initiative and weekly iron folate supplementation), the beneficiary receives supplemental iron and folic acid. Deficiency of B12 is currently not being addressed through these programs [7,8]. Vegetarianism was significantly associated with severe anemia which was similar to the findings by Verma, et al. [9]. Attainment of menarche was also significantly associated with of anemia which was in agreement with the findings of Heath, et al. [10] who reported that high menstrual blood loss was associated with increased risk of anemia. A significant association between history of worm infestation and severity of anemia in the present study was in agreement to findings of Shield, et al. [11] who demonstrated a statistically significant inverse correlation between hookworm egg count and hemoglobin level.

Our study findings are limited by the hospital-based design of the study. Another limitation is that presence of infections which were not picked up on

detailed history and examination could have erroneously elevated the serum ferritin above the cut-off used for defining iron deficiency anemia.

Conclusion

Folate deficiency was the most common followed by Vitamin B12 deficiency & then irons deficiency. Low intake of all the three nutrients is a significant determinant towards causing nutritional anaemia. Supplementation with not only iron and folic acid but also Vitamin B12 is required through national programmes. History of worm infestation is a significant risk factor which should be taken care off.

References

1. Webb TE, Oski FA. Iron deficiency anemia and scholastic achievement in young adolescents. J Pediatr. 1973; 82:827- 30.
2. Choudhary A, Moses PD, Mony P, Mathai M. Prevalence of anaemia among adolescent girls in the urban slums of Vellore, south India. Trop Doct. 2006; 36:167 9.
3. Patra S, Pemde HK, Singh V, Chandra J, Dutta A. Profile of adolescents with severe anemia admitted in a tertiary care hospital in Northern India. Indian J Pediatr. 2011 Jul; 78(7):863-5.
4. WHO. Iron Deficiency Anaemia Assessment Prevention and Control: A Guide for Programme Managers. 2002; 45:122-126.
5. Ahmed F, Khan MR, Banu CP, Qazi MR, Akhtaruzzaman M. The coexistence of other micronutrient deficiencies in anaemic adolescent schoolgirls in rural Bangladesh. Eur J Clin Nutr. 2008; 62(3):365-72.
6. Abdelrahim, II, Mahgoub HM, Mohamed AA, Ali NI, Elbashir MI, Adam I. Anaemia, folate, zinc and copper deficiencies among adolescent schoolgirls in eastern Sudan. Biol Trace Elem Res. 2009; 132:60-6.
7. Vir SC, Singh N, Nigam AK, Jain R. Weekly iron and folic acid supplementation with counseling reduces anemia in adolescent girls: a large-scale effectiveness study in Uttar Pradesh, India. Food Nutr Bull. 2008; 29:186-94.
8. Kapil U, Bhadoria AS. National Iron-plus initiative guidelines for control of iron deficiency anaemia in India, 2013. Natl Med J India. 2014; 27:27-9.
9. Verma M, Chhatwal J, Kaur G. Prevalence of anemia among urban school children of Punjab. Indian Pediatr. 1998; 35:1181-6.
10. Heath AL, Skeaff CM, Williams S, Gibson RS. The role of blood loss and diet in the aetiology of mild iron deficiency in premenopausal adult in New Zealand women. Public Health Nutr. 2001; 4:197-206.
11. Shield JM, Vaterlaws AL, Kimber RJ, Payne R, Casey GJ, Blunden RW, et al. The relationship between hookworm infection, anemia and iron status in a Papua New Guinea highland population and the response to treatment with iron and mebendazole. PNG Med J. 1981; 24:19-34.

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Health Status of Orphan Children Infected with Human Immunodeficiency Virus

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Abstract

Introduction: Children are infected with Human Immunodeficiency Virus mostly through vertical transmission. In vast majority of cases, their father and mother both are HIV positive and sooner or later these children become orphan. Though, there is significant decrease in new cases of HIV, both in children as well as in adults, but the number of orphan children is increasing. These children are exposed to variety of adversities which affects their health and survival. To study the health status of HIV infected orphan children and how does it differ from their non orphaned counterparts, this study was undertaken. *Methods:* This is an hospital based observational study, in which HIV positive children diagnosed as per NACO HIV testing guidelines, between age 18 months to 15 years were included. Their orphan status was decided by history given by their caregivers/ guardians. Thorough clinical examination and required investigations of both, orphan and non orphan HIV positive children was done and their health status, morbidity and mortality were assessed. *Result:* Out of 140 children, 47 were orphan and 93 non orphan. Mean age of double orphan children was 11.6 0 (SD±1.42) years and single orphan children was 9.13 (SD±2.66) years. Grandparents, aunts and maternal uncle were the care givers of these orphan children. Severe anaemia and severe under nutrition was more common in orphan children than in non orphan. In 27.66% orphan children severe immune suppression was noted as compared to 12.90 % in non orphan children. Recurrent serious opportunistic infections were more common in orphan children than in non orphan. Out of six deaths, five children were orphan. No significant difference was noted in socio economic distribution, treatment adherence and follow up.

Keywords: Health Status; HIV Infection; Orphan Children.

Introduction

Children are mostly infected through vertical transmission, from their mothers, either during pregnancy and intra partum period or during breast feeding [1]. Prevalence of HIV infection is decreasing globally and so in India also. Incidence of new HIV infection in children is also decreasing. 58% reduction in number of HIV infected children (< 15 years of age) has been reported during 2002-2013 worldwide [2]. According to UNICEF India (http://www.unicef.org /india/children) there are 220,000 children infected by HIV in India while NACO (http;//www.nacoonline.org) gives this figure to be 70,000. Number of orphan children due to HIV/AIDS is increasing. Roughly 1,500,000 – 2,500,000 children have been orphaned by AIDS and about 6,000,000 – 10,000,000 have HIV positive parents (http://www.childline india.org.in). Over the period of time these children will lose their one or both parents and will be orphaned. According to an estimate, only 2% children in developing countries were orphan before AIDS era but this number has increased to 7 – 11% with HIV/ AIDS epidemic [3]. An 'orphan' is defined as a child who has 'lost one or both parents'- United Nations. These are the children who are the most vulnerable to become the victim of variety of diseases, environmental

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hazards, neglect and exploitations. To study the health status, routine care and follow up, morbidity and mortality in these children and to compare it with HIV infected non-orphaned children, this study was under taken.

Methods

This is a hospital based observational study, carried out at S.N. Children Hospital, Department of Paediatrics, M.L.N. Medical College Allahabad, UP, during the period April, 2005 – October, 2012. Children between the ages 18 months – 15 years, who were freshly diagnosed HIV positive as per NACO HIV testing guidelines, were included in the study. Children below the age of 18 months and previously diagnosed HIV positive children were not the part of this study. These children were selected from ART Center, S.R.N. Hospital and S.N. Children Hospital Allahabad, UP. Both hospitals are the associated hospital of M.L.N. Medical College Allahabad. Parents and / care givers were counseled by qualified counselors. Informed and written consent was taken to interrogate and examine the children. Detailed history was taken and thorough clinical examination of every child was done. Complete Blood Count (CBC) and CD4 count of every child was done. Investigations for Opportunistic Infections (OI) were carried out where ever required. Every child was classified for WHO clinical and immunological staging. Data was recorded on pre structured proforma. Orphan status of children was decided by history obtained from care givers. The children who lost their both parents were called 'double orphan' and those who lost their one parent were termed as 'single orphan' as per definition given by UNICEF. These children were assessed for their nutritional status, Haemoglobin level, CD4count and CD4%, immunological staging, presence of opportunistic infections, morbidity and mortality. They were also assessed for treatment compliance and follow up visits. Every child was followed up minimum for 18 months. Those who failed to appear in the follow ups for six months they were labeled as 'lost to follow up'.

Statistical analysis- Simple percentages were used to describe nominal data, mean and standard deviations were used to describe normally distributed data. Z proportion test was used to compare two independent groups and p values were calculated.

Result

A total of 140 HIV positive children between ages 18 months to 15 years were the part of this study. 97.86% children were infected through vertical transmission. Out of these, 10 children lost their both parents and 37 lost either father or mother. 93 children had their both parents alive. Age and sex wise distribution of these children has been shown in table 1. Mean age of double orphan children was 11.60 (SD±1.42) years and single orphan was 9.13 (2.66) years. 36.16% orphan children were significantly under nourished (weight for age is < 60% of expected) while in non orphan group 19.34% children were in state of significant under nutrition. Severe anaemia (Hb%≤7gm) was noted in 14.80% orphan children while in non orphan it was 5.37%. In both the groups around 80% children were in lower socio economic status according to B. G. Prasad's modified classification, as shown in Table 2. On the basis of WHO clinical staging 82.98% orphan children were in stage II and III and 81.71% non orphan children. Distribution of children on the basis of WHO immunological staging (Table 3) shows that 27.66% orphan children were in stage IV as compared to non orphan children where it is 12.9% only. Table 4 shows p values for different comparison groups. 25.53% orphan children had recurrent attacks of serious opportunistic infections while 19.35% non orphan children had such OIs. Out of total six deaths, five children were orphan. OIs responsible for death were cryptococcal meningitis, tubercular meningitis, encephalopathy, milliary tuberculosis and severe bronchopneumonia with fungal infection, one in each case. Four (8.51%) orphan and Five (5.37%) non orphan children were lost to follow up. Grand parents, aunts and maternal uncles were the care givers of orphan children. At the end of 18 month, 70.56% orphan children and 72.75% non orphan children were noted to be regular in follow up and treatment adherence.

Table 1: Age and Sex wise distribution of orphan and non orphan children

Age in years	Male		Female	
	Orphan (number)	Non orphan (Number)	Orphan (number)	Non orphan (Number)
1.5-05 yrs.	00	29	00	12
05-10 yrs.	19	16	06	20
10-15 yrs.	15	12	07	04
Total	34	57	13	36

Table 2: Socio Economic Class of orphan and non orphan children.

S.E Class	Orphan		Non orphan	
	n - 47	%	N - 93	%
I – Upper class	03	06.38	02	02.15
II – Upper middle	06	12.76	18	19.35
III – Lower middle	17	36.17	32	34.40
IV – Upper lower	17	36.17	35	37.64
V – Lower class	04	08.52	06	06.46

Table 3: Immunological staging of children.

Immunological staging	Orphan		Non orphan	
	n - 47	%	N - 93	%
I	15	31.91	35	37.65
II	05	10.64	22	23.65
III	14	29.78	24	25.80
IV	13	27.67	12	12.90

Table 4: Comparison of different conditions between two groups and their p values

Conditions	Orphan n - 47	Non orphan n - 93	p Value
Significant under nutrition	36.16	19.34	0.0247
Severe anemia	14.80	05.37	0.017
Severe immune suppression	27.60	12.90	0.0479
Severe OIs	25.33	19.35	0.0214
Mortality	10.63	01.07	0.011
Follow up	70.56	72.75	0.072

All p values < 0.05 are significant.

Discussion

Majority of the children acquire the HIV infection from their mothers and mothers get it from their husbands on most of the occasions. Though, significant decrease in morbidity of HIV infection have been noted due to availability and use of anti retroviral treatment and prophylaxis for OIs [4,5], yet over a period of time parents die and children are orphaned. Number of orphan children due to AIDS/ HIV is increasing [3]. In our study, out of 140 HIV infected children, 47 (33.57%) were orphan, 26.42% single orphan and 6.8% double orphan. Ira Shah [6] has reported 40% HIV infected children were orphan, out of which 7.5% were double orphan. We found 100% orphan children were over the age of five years. Unicef report also says that 95% of all orphans are over the age of five years. Mean age of children when single parent died was 9.13 (SD ±2.66) years and when both parents died was 11.60 (SD ±1.42) years in our study. Ira Shah (6) in her study has reported mean age when mother had died was 5.6 ± 2.2 yrs, when father had died 7.08 ± 3.5 years and when both parents had died was 10.9 ± 2.4 years (p = 0.04). In our study 36.16% orphan children were significantly under nourished while this number was 19.34% in non orphan children. Lack of parental care adversely affects the nutritional status of children. This view is also supported by Bicego et al [7] and Deininger et al [8] where they noted that orphans were particularly disadvantaged in terms of nutritional status and growth. Like many other studies [9,10] we also noted that anaemia was a common finding in children of both the groups but severe anaemia was more common (14.80%) in orphan than in non orphan where it was 5.37%. Under nutrition and anaemia is a common association in HIV infected children [11]. Around 80% children of both the groups were from lower socioeconomic status. We found that 57.44% orphan children were in immunological stage III and IV while 38.70% non orphan children were in III and IV immunological stage. It reflects that more of orphan children were immunologically compromised and this explains why these children had recurrent serious OIs as compared to non orphan children. In our study we noted that 25.53% orphan children had recurrent serious opportunistic infections as compared to non orphan children where this figure was 19.35%. Many other studies have also reported that orphan children are more vulnerable to health problems than non orphaned [7,12-14]. Muller and Abbas [15] 1990, also noted that lack of parental protection opens door for child abuse, neglect and illnesses. We observed that mortality was high in orphan children as out of six deaths, five children were orphan. This could be due to poverty, lack of care, neglect and delay in seeking treatment for OIs.

Deininger et al [8] also found that orphans are at increased risk of illnesses and death. Orphans have greater barrier to care due to social and economic constrains leading to lesser survival [4]. In our study we noted that 31.91% of orphan and 30.10% of non orphan children were on ART. In a study of global information and advice on HIV & AIDS 32% of children with HIV were receiving ART [17] At the end of 18 months, 70.56% orphan and 72.75% non orphan children were noted to be regular in follow up and adherent to ART. This difference is not significant. In other studies also [16], over 75% to 80% children reported perfect adherence to ART. Ira Shah [6] in her study has reported that there was no statistical difference in follow up (p=0.48) or initiation of ART (p=0.04) in orphaned and non orphaned children.

Conclusion

There was no significant difference in socioeconomic status, treatment adherence and follow up visits in HIV infected orphan and non orphan children but there was significant difference in their nutritional and immunological status. More orphan children were severely under nourished, severely anaemic and severely immune compromised than non orphan children. Double orphaned children were worst sufferer. Morbidity and mortality was higher in these children. We conclude that along with regular follow up and treatment adherence, nutritional and emotional support is an important factor to maintain good immunological status to avoid recurrent serious opportunistic infections and there should be someone like mother and father to provide affectionate routine care at home.

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References

1. Bertolli J, St.Louis ME, Simonds RJ, Nieburg, et al. Estimating the timing of mother to child transmission of human immunodeficiency virus in a breast feeding population in Kinshasa, Zaire. J Infect Dis 1996; 174:722-26.
2. UNAIDS (2014) 'The Gap Report'
3. Foster G. Supporting community efforts to assist orphans in Africa. N Engl J Med 2002; 346:1907-10.
4. Nyandiko WM, Ayaya S, Nabakwe E, Tenge C, Sidie JE, et al. Outcomes of HIV-Infected Orphaned and Non-Orphaned Children on Antiretroviral Therapy in Western Kenya. JAIDS 2006(Dec); 43(4):418-25.
5. Tudor-William G, Head S, Weigel R, et al. A palatable four drug combo for HIV infected infants. Paper presented at: XIV International AIDS Conference; Barcelona; 2002 Abstract MoOrB 1129.
6. Shah I. Prevalence of Orphans among HIV Infected Children- a Preliminary Study from a Pediatric HIV Centre in Western India. J Trop Pediatr 2008; 54(4): 258-60.
7. Bicego G, Rutstein S, Johnson K. Dimensions of the emerging orphan crisis in sub- Saharan Africa. Social Sciences and Medicine 2003; 56:1235-47.
8. UNICEF(2003), Deininger, Garaic and Subbarao. 'Africas orphaned generations' NewYork: United Nations Childrens Fund Analysis for various countries documented by Subbarao and Coury (2003).
9. Claster S, Biology of Anaemia, differential diagnosis and treatment options in human immunodeficiency virus infection. J Infect Dis 2002(May); 185(suppl 2): s 105-9.
10. Belperio PS, Rhew DC. Prevalence and outcome of anaemia in individuals with human immuno-deficiency virus: a systemicreview of literature. Am J Med 2004(Apr); 116(suppl 7A):27s-43s.
11. Merchant RH, Oswal JS, Bhagwat RV, Karkare J. Clinical profile of HIV infection. Indian Pediatr.2001 Mar; 38(3):239-46.
12. UNICEF/UNAIDS. Children on the Brink 2002: A joint report on Orphan Estimates and Program strategies. Jul, 2002. www.unicef.org/publications/index_4378.html
13. Makame V, Ani C, Granthum-McGregor S. Psychological well-being of orphans in Dar El Salam, Tanzania. Acta Paediatr. 2002; 91:459-65.
14. Leyenaar JK. HIV/AIDS and Africa's orphan crisis. Paediatr Child Health 2005(May-Jun); 10(5):259-60.
15. Muller O, Abbas N. The impact of AIDS mortality on children's education in Kampala, Uganda. AIDS Care. 1990; 2(1):77-80.
16. Aidsmap (2012, 27 July) 'HIV treatment for children: how programmes are improving diagnosis and retention'
17. UNICEF (2015) Annual Report.

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Study of Weaning Practices in Children between 6 Months to 2 Years Attending OPD

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Abstract

Background: During 2006-10, more than 16 per cent of the world's children under the age of 5 years were underweight for their age. The proportion ranged from 1.0 per cent of children in developed countries to 25 per cent in developing countries. In India, the national family health survey (NFHS) 2005-06 included survey of the nutritional status of young children. Both chronic and acute under nutrition were found to be high in all 7 states for which reports have been received, namely, Haryana, Karnataka, Maharashtra, Orissa, Tamil Nadu, Uttar Pradesh and Goa. Poverty, unsanitary conditions, lack of education and poor rearing practices are some of the major contributory factors. Optimal infant and young child feeding mean that mothers should start to breastfeed within half an hour of birth and continue exclusively for the first six months till two years, together with nutritionally adequate, safe and age appropriate complementary feeding. Improving infant and young child feeding practices in children 0-23 months of age is critical to improved nutrition, health and development of children. One of the ways to effectively reduce the growing under-five mortality rate is the timely introduction of complementary feeding. *Objectives:* To determine various weaning practices in Raichur. To determine the effect of weaning on growth. To determine the effect of weaning on various systemic illnesses. To determine effect of weaning on nutritional status of children. *Methodology:* It is a prospective study comparing the various weaning practices and their effect on growth of the children of age 6 months to 2 years visiting pediatric OPD in Navodaya medical college hospital during the period of November 2014 to December 2015. A total of 300 children were included in the study by simple random sampling. The need for the study has been explained to the parents and prior consent has been taken. Ethical committee clearance has been obtained before performing the study. A detailed history taking with a pre-structured questionnaire on feeding habits of children has been taken. Anthrapometric parameters such as height, weight and mid arm circumference have been measured for all the children to determine the growth parameters in children based on the weaning practices. Complete Systemic examination has been performed to rule out any systemic illness. Investigations such as a complete blood examination, urine examination, stool examination and chest x-ray to rule out systemic infections associated with malnutrition. *Results:* It is found that the prevalence of malnutrition and systemic illness were more in 12months to 24 months when compared to younger age group. The growth parameters are hindered more in the elder age group when compared to younger age group. Coming to weaning practices the most common feeds were annaganji and jowahar roti in younger age group and elder age group respectively. Most of the children of the younger age group are still being breast fed frequently compared to the older age group. *Interpretation and Conclusion:* As discussed earlier weaning is not new to India, as the history suggests that there was literature regarding weaning since the times of Charaka; Shrushutha and Kashyapa which advice exclusive breast feeding for first 6 months and complimentary feeding from 6 months with continuation of breast feeds up to 2 years. Our study shows that there is high prevalence of under nutrition and childhood illness in children of age group 12 to 24 months as compared to children of age 12 to 24 months. As age increases energy density of breastfeeds decreases and frequency of breastfeeding decreases and the nutrition

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is mainly from the weaned foods. Hence, in a setting of inadequate weaning the prevalence of malnutrition and childhood illnesses increase with age.

Keywords: Weaning; Malnutrition; Breastfeeding; Nutrition; Complementary Feeding; Anthrapometry; Childhood Illness**.**

Introduction

Infant feeding practices comprises of breast feeding and complimentary feeding which directly affects the nutritional status of children under two years of age and ultimately has an impact on child survival.

During 2006-10, more than 16 per cent of the world's children under the age of 5 years were underweight for their age. The proportion ranged from 1.0 per cent of children in developed countries to 25 per cent in developing countries [1]. In India, the national family health survey (NFHS) 2005-06 included survey of the nutritional status of young children. Both chronic and acute under nutrition were found to be high in all 7 states for which reports have been received, namely, Haryana, Karnataka, Maharashtra, Orissa, Tamil Nadu, Uttar Pradesh and Goa [2]. Poverty, unsanitary conditions, lack ofeducation and poor rearing practices are some of the major contributory factors. Optimal infant and young child feeding mean that mothers should startto breastfeed within half an hour of birth and continue exclusively for the first six months till two years, together with nutritionally adequate, safe and age appropriate complementary feeding. Improving infant and young child feeding practices in children 0-23 months of age is critical to improved nutrition, health and development of children. One of the ways to effectively reduce the growing under-five mortality rate is the timely introduction of complementary feeding [3]

In Gujarat a community based study of feeding and weaning practices in under five children has been done. This study concludes that, false beliefs and myths attached to child's feeding, deeply rooted in all strata of community, need to be replaced by sound and scientific messages [4].

Hence, it is essential to study the various weaning practices and their effect on growth parameters of children to establish scientifically sound and economical feeding practices.

Materials and Methods

Children of age 6 months to 2 years visiting pediatric OPD in Navodaya Medical College Hospital during the period of November 2014 to December 2015.

Inclusion Criteria

1. Age 6 months to 2years
2. Children who are exclusively breast fed for first 6 months
3. Children without an congenital anomalies
4. Children without any peri-natal insult

Exclusion Criteria

1. Children who are not exclusively breast fed for first 6 months
2. Children with congenital anomalies
3. Children with perinatal insult
4. Children who are not staying with mother for any reason
5. Upper and Upper middle class children according to Kuppuswamy classification

Sampling Method

Simple Random Sampling

Size of the sample: 300

Method of Collection of Data

A detailed history taking with a pre-structured questionnaire on feeding habits of children was performed.

Measuring height, weight and mid upper arm circumference was done. Complete Systemic examination a complete blood examination, urine examination, stool examination, chest X-ray for Systemic infections associated with malnutrition. All the data will be collected, compiled and interpreted to establish proper weaning practices that promote growth, development and health of the children aged 6months to 2 years.

Ethical committee clearance has been taken from the ethical committee board.

Results

Using the 'Harvard' standard as the reference measurement based on the percentage of the median, under nutrition was classified into normal (more than 90%), mild (90-75%), moderate (75-60%) and severe (below 60%) Gomez et al. (1956).

The diagnosis of under nutrition was based on the correlation between age and expected values of body weight, body length and arm circumference. Measurements of body weight and length were recorded for all children while the arm circumference was recorded only for children aged at least 12 months. The arm circumference of children below the age of one year was not measured because of the fact that babies have a lot of fat that makes accurate measurement difficult.

Children were categorized into three groups-normal nutrition (Group A), mild (Group B) and moderate to severe under nutrition (Groups C). These categories were examined separately for those aged 6 to 11 months and for those aged 12 or more months.

Table 1: Distribution of children by age, body weight and their nutritional status

Age in Months	Normal (A)	Mild (B)	Moderate to Severe (C)
6to11	58(50.8)	27(23.6)	29(25.4)
12to 24	66(35.4)	50(26.8)	70(37.6)
Total	124(41.4)	77(25.6)	99(33)
P value	>0.003	>0.05	<0.015

The results are summarized in Table 1-2.

There was a significant difference between underweight among children 6-11 months (25.4%) and that of children aged 12-23 months (37.6%).

Body Weight and Body Length

Overall, 40-44% of children were well nourished; 25-30% suffered form mild under nutrition while 27-33% were moderately to severely under nourished. Comparison between the two age groupings showed that while there was no significant difference in the proportions of children with mild under nutrition, the prevalence of normal nutrition was higher and of moderate to severe under nutrition lower in the 6-11 age group as compared to those 12 months or older.

Table 2: Distribution of children by age and body length and their nutritional status

Age in Months	Normal(A)	Mild (B)	Moderate to Severe (C)
6-11	64(56.1)	33(28.9)	17(15)
12-24	67(36)	55(29.5)	64(34.5)
Total	131(43.6)	88(29.3)	81(27)
P value	<0.001	>0.05	**<0.001**

Only 15% of children aged 6 to11 months were stunted (chronic malnutrition) while stunting was two times more i.e. 34.5% among children aged 12 to 24 months.

Weaning Practices

Breastfeeding and the Introduction of Water and Semisolid Food

Overall, 92.8% of children were breastfed. Most of the children aged 6-11 months (99.3%) and 88.8% of those aged 12 months or more were being breastfed. The difference in the proportions in the two groups was significant. In both groups of children water had been introduced in 97-99% and semisolid food in 90-96% by the age of 6 months. There was no significant difference between the groups in these aspects.

Food Sources and Variety

The child's source of nourishment, apart from breast milk, depends on the food variety readily available in the community, food prepared separately for the child or for the family and those bought from food vendors. In some communities children may be denied certain types of food. The data collected showed that meals were prepared separately for children less than 12 months much more frequently than for older kids (83.1% as against 63.8%; P< 0.001) while vendor food was bought more frequently for older kids than for those less than 12 months (67.9% vs 41.2%). The main food items prepared for kids less than 12 monthswere plain "annaganji"(64.6%), i.e.

rice starch " roti" (14.9%), made from jowar and commercial packed cereals (10.6%).

The main food items prepared for the older kids were plain " annaganji" (19%) bread (13.4%) and (47. 4%) "roti" made of jowar . Bread is usualy taken with tea. The children less than 12 months had relatively more "annaganji" and less bread while the reverse was true of the older children. Jowar roti was consumed more frequently by the children of older age group compared to that of younger age group. Similar comments apply to the main foods bought for the two age groups.

Table 3: Various weaning foods

Food Items	6to11months	12to24 months	P value
Jowar roti	17.2%(57)	36.8%(238)	**<0.001**
Bread	4.2%(14)	14.4%(93)	**<0.001**
Breast	5.1%(17)	2.9%(19)	>0.05
Biscuits	1.5%(5)	2.8%(18)	>0.05
Annaganji or cooked rice	65%(215)	34.2%(221)	**<0.001**
Tea	2.7%(9)	4.2%(27)	>0.05
Commercial cereals	1.2%(4)	0.5%(3)	**<0.03**
Others	3%(10)	4.3%(28)	>0.05
Total	100%(331)	100%(647)	

The various weaning foods given to children of the two age groups had been summarized below (table 3) which was collected by 24 hour recall method.

Note: the number in the bracket indicates the total number of servings of each food time in a day and hence the number of servings for the study group of 300 was found to be 978 of various types of foods.

The results shown in the above table indicate that the staple food of the community were jowar roti and cooked rice. The consumption of roti is more in the older age group compared to increased consumption of rice based diet in the younger age group as the mothers felt that the younger children had difficulty in consumption of roti and were given less frequently were as for older children they were given more frequently because of the easy preparation and longer duration of storage.

Illness	6 to 11 months n=114	12 to 24 months n=186	Total n=300	P value
Diarrhea	58.8% (67)	71.5%(133)	66.7%(200)	**<0.014%**
Fevers	72.8%(83)	87.1%(162)	81.7%(245)	**<0.001%**
Respiratory infections	70.1%(80)	72.5%(135)	71.6%(215)	>0.05%

Childhood Illness

Those examined include diarrhea, frequent fevers and respiratory tract infections. The results are summarized in Table 4.

Among the 300 children 200 presented with diarrhea of which 67 were of younger age group and 133 were of older age group and the difference was statistically significant. Of the 300 children 245 had frequent fevers and the prevalence in younger age group was 72.8% and older age group was 87.1% the difference was again statistically significant. Coming to the respiratory tract infections there wasn't any significant difference in both the age groups.

Diarrhea and frequent fevers occurred in a significantly higher proportion of children aged 12 or more months than in the younger age group. The number of episodes of diarrhea ranged from 1 to 10, with a median of 4. There was however no significant difference in the number of attacks between the two groups. Upper respiratory tract infections also occurred with similar frequency. Fevers were treated mainly with antimalarials and antipyretics.

Antibiotics were given to less than 8% of children. Antibiotics were used more for respiratory tract infections than for diarrhea. In about 10-12% of cases no treatment was given.

Discussion

Prevalence of Under Nutrition

Using the 'Harvard' standard as the reference measurement based on the percentage of the median, under nutrition was classified into normal (more than 90%), mild (90-75%), moderate (75-60%)and severe (below 60%) Gomez et al. (1956) [4].

The number of children who were under nourished in the current study based on weight of the child was found to be 66 i.e. 49.2% of age group 6months to 11 months while that among the older age group were 120 i.e. 64.6%. Among them the prevalence of moderate to severe malnutrition was found to be statistically significant in both the age groups i.e. 25.4% in younger age group, while in older age group it was 37.6% and the p value was found to be <0.015%.

Coming to the length the percentage of children falling withinthe normal range were more in the age group 6 to 11 months when compared to that in 12 to 24 months i.e 56.1% and 36% and the difference was statistically significant.

Coming to stunting only 15% of children aged 6to11 months were stunted (chronic malnutrition) while stunting was two times more i.e. 34.5% among children aged 12 to 24 months. The difference was statistically significant.

The percentage of malnourished children in study area was more than reported by A Mittal et al[11] (26.76%) and Arshad Farooq et al [12] (24.14%). It was observed that majority of the malnourished children were between the age of 12 months to 24 months. The proportion malnutrition was observed to be increasing as age was increasing. Similar trend was also reported by K.D. Bhalani et al.[13] Deterioration of nutritional status with increasing age was may be because of poor weaning practices. During weaning process child is exposed to deleterious synergistic action of malnutrition and infection. Once the child become malnourished due to weakened immune system, child become prone to infection and may fall in vicious cycle of malnutrition and infection, which increases with age.

The first year of an infant's life is the period of most rapid growth and an important nutrition transition, when infants are given various types of complementary foods along with breast milk. Our results suggest that recommended IFPs of the prior periods were important for the gain in weight of the subsequent periods during infancy. In particular, feeding practices during the first quarter and the first half of infancy were significant for the subsequent gain in weight during infancy. A 10% increase in the feeding scale of 1–3 mo and 1–6 mo would increase gain in weight by 90 g and 70 g, respectively. Because weight is more sensitive than length to short-term dietary changes (30) and also to childhood illnesses (31–35), our results of the positive association between IFPs and weight growth during infancy corroborates the importance of following infant feeding recommendations.

Although the associations between IFPs and gain in length during 1–12 mo of life were statistically significant, they were not biologically important. A 10% increase in the feeding scale would cause only a 0.05-cm gain in length during infancy. The small effect of following the infant feeding recommendations on length may result because these infant feeding scales were relatively crude. They did not capture the multidimensional nature of IFPs that might contribute to better linear growth.

Weaning Practices

Breastfeeding and the introduction of water and semisolid food

Overall, 92.8% of children were breastfed. Most of the children aged 6-11 months (99.3%) and 88.8% of those aged 12 months or more were being breastfed. The difference in the proportions in the two groups was significant. In both groups of children water had been introduced in 97-99% and semisolid food in 90-96% by the age of 6 months. There was no significant difference between the groups in these aspects. From the age of 6 months, an infant's need for energy and nutrients starts to exceed what is provided by breast milk, and complementary feeding becomes necessary to fill the energy and nutrient gap (57). If complementary foods are not introduced at this age or if they are given inappropriately, an infant's growth may falter. In many countries, the period of complementary feeding from 6–23 months is the time of peak incidence of growth faltering, micronutrient deficiencies and infectious illnesses (58). (Infant and Young Child Feeding: Model Chapter for Textbooks for Medical Students and Allied Health Professionals).

Even after complementary foods have been introduced, breastfeeding remains a critical source of nutrients for the young infant and child. It provides about one half of an infant's energy needs up to the age of one year, and up to one third during the second year of life. Breast milk continues to supply higher quality nutrients than complementary foods, and also protective factors. It is therefore recommended that breastfeeding on demand continues with adequate complementary feeding up to 2 years or beyond (13).

Complementary foods need to be nutritionally-adequate, safe, and appropriately fed in order to meet the young child's energy and nutrient needs. However, complementary feeding is often fraught with problems, with foods being too dilute, not fed often enough or in too small amounts, or replacing breast milk while being of an inferior quality. Both food and feeding practices influence the quality of

complementary feeding, and mothers and families need support to practice good complementary feeding (13).

Childhood Illness

Diarrhea and frequent fevers occurred in a significantly higher proportion of children aged 12 or more months than in the younger age group. The number of episodes of diarrhea ranged from 1 to 10, with a median of 4. There was however no significant difference in the number of attacks between the two groups. Upper respiratory tract infections also occurred with similar frequency. Fevers were treated mainly with antimalarials and antipyretics.

In the current study it has been observed there is a significant increase in the childhood illness which might be due to the increase in the percentage of malnourished children which age. There was a similar association noted with malnutrition and childhood illness in the studies conducted by Bhatia V et al in chandigarh and Awasthi S et al in Uttarpradesh respectively.

Conclusion

Our study shows that there is high prevalence of under nutrition and childhood illness in children of age group 12 to 24 months as compared to children of age 12 to 24 months.

There was higher intake of annaganji in 6months to 11 months children and jowar roti in 12 to 24 months. The frequency of breast feeding has gradually decreased with increasing age.

As age increases energy density of breastfeeds decreases and frequency of breastfeeding decreases and the nutrition is mainly from the weaned foods. Hence, in a setting of inadequate weaning, the prevalence of malnutrition and childhood illnesses increases with age.

Recommendations

1. Weaning should be initiated at 6 months of age with appropriate food sourcescommonly available.
2. Proper hygiene to be maintained in handling and feeding the child.
3. Simplehand washing before feeding the child can bring down major illness.
4. Breastfeeding to be continued along with the weaning feeds.
5. All children to be adequately immunized to prevent the vaccine preventablediseases.
6. Maternal education has no role in weaning practices, hence it needs to bestressed that all the mothers to be adequately enlightened regardingbreastfeeding, weaning, immunization and hygiene.

References

1. UNICEF-WHO Joint Committee On Health Policy.
2. Parks's textbook of preventive and social medicine, edition 22, p.508.
3. World health organization, the international code of marketing of breast milk. Substitutes: frequently asked questions W.H.O, 2008.
4. Dinesh Bhanderi1, Sushilkumar Choudhary2, A community based study of feeding and weaning practices in under five children in semi urban community of Gujarat; National Journal of Community Medicine. 2011 Jul-Sep; 2.
5. Kuntal K Saha, Edward A Frongillo, Dewan S Alam, Shams E Arifeen, La Rake Persson, And Kathleen M Rasmussen. A appropriate infant feeding practices results in better growth of infants and young children in rural Bangladesh.Am J ClinNutr 2008; 87:1852-9.
6. Anita Khokhar, S Singh, R Talwar, SK Rasania, SR Badhan, M Mehra. A study of malnutrition among children aged 6 months to 2 years from resettlement colony of Delhi. Indian Journal of Medical Science 2003; 57:286-9.
7. Amir Moroof Khan, Priscilla Kayina, Paras Agrawal, Anita Gupta, Anjur Tupil Kannan. A study on infant and young children feeding practices among Axpasr4lfwquh attending an urban health center in east Delhi. Indian Journal of Public Health 2012; 56
8. Anjali Khanna, DeeptiM.Kadeangadi,Shri MD Mullapur. Infant and young child feeding practices in rural Belgaum: A descriptive study 2014; 3(2).

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Anthropometric Profiles of Children with Congenital Heart Disease

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Abstract

Background: Congenital heart disease (CHD) is often associated with malnutrition and failure to thrive in children. Children with congenital heart diseases are frequently undernourished irrespective of cardiac defect and presence or absence of cyanosis. The aim of the study is to assess the effect of CHD on growth and nutrition and to identify the areas of growth affected with reference to different anthropometric measurements. *Materials and Method:* A case control observational study was carried out in children aged 0-14 years old with CHD in our institute. All patients underwent an anthropometric evaluation (weight, height/length, head circumference, mid-arm circumference, triceps and subscapular skin fold thickness) and standard growth charts (NCHS and WHO) were used accordingly. *Results*: We had total of 40 patients, 27 (68%) had acyanotic congenital malformation, while 13 (32%) had cyanotic cardiac malformation. Majority, 18 (82%) out of 22 cases above 5 years with CHD were underweight. Left to right shunt children with acyanotic malformation were tended to have acute malnutrition and stunting was more severe in children with cyanotic defects, with 100% of them affected. Majority of the children (42%) were undernourished with mid arm circumference below 13.5cms. *Conclusion*: A significantly higher, that is, 82% were underweight and 86% were stunted among children with congenital heart disease. In conclusion it can be stated that children with congenital heart disease have highly statistically significant growth retardation ($P<0.001$) by student t- test.

Keywords: Congenital Heart Disease; Underweight; Stunting.

Introduction

Congenital cardiovascular defects, also known as congenital heart defects (CHD), are structural problems that arise from abnormal formation of the heart or major blood vessels present from birth or manifesting any time after birth or may not manifest at all. The overall incidence of congenital malformation in live birth is 0.8% [1]. CHD comprise about 30% of all congenital malformation in the new born [2]. Severe malnutrion may occur in children with congenital heart defects due to an imbalance between energy intake and consumption. Heart failure and pulmonary hypertension are the most important factors for the development of the severe malnutrition. Children with cyanotic heart disease with pulmonary hypertension are the most seriously affected requiring more aggressive nutritional therapy [3].

Cyanotic patients are affected in growth, depending upon the severity of tissue hypoxemia and degree of physiological adaptation. Weight and height are affected equally in cyanotic patients. Acyanotic lesions especially in combination with septal defect, left to right shunt will affect weight only. In short, acyanotic lesions were related to acute malnutrition whereas cyanotic lesions were related to chronic malnutrition [4]. Infants and children with CHD exhibit a range of delay in weight gain and growth.

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In some cases delay can be relatively mild, whereas in other cases, the failure to thrive can result in permanent physical or developmental impairment [5]. In caring for these children, it is important to develop a nutritional strategy and takes into account all of the factors at play, both physical and psychological. Therefore, there must be an effort between parents, physicians, nurses and other health care professionals to develop a plan that will be appropriate on an individual basis [6]. The aims of this study were to determine the anthropometric profiles and prevalence of undernutrition in children with CHD by using anthropometric measurement. Those measurements are useful in early detection of CHD and assessing the prognosis of the basic cardiac defects and their complication.

Materials and Methods

A case control observational study was carried out in children aged 0-14 years old with CHD who had consultation in our outpatient clinic. This study was conducted from August 2015 to July 2016. Children who met the inclusion criteria for age, and who had no definitive or palliative treatment given, were taken up for the study.

Inclusion Criteria

1. 0-14 years both male and female with congenital heart diseases (cyanotic & acyanotic) clinically detected and confirmed by investigations and
2. Patients who have not undergone any surgical intervention.

Exclusion Criteria

1. Major congenital malformation other than CHD.
2. Neurological disability
3. Other obvious causes of malnutrition
4. Chronic infections
5. Endocrinal causes of growth retardation
6. Known chromosomal abnormality syndromes

Diagnostic criteria of congenital heart disease

1. Clinical examination,
2. Chest X-ray
3. ECG
4. Echocardiogram to determine the type of CHD.

The following anthropometric parameters were studied:

1. Weight
2. Height /length
3. Head circumference
4. Mid-arm circumference
5. Triceps and Subscapular skinfold thickness

Anthropometric measurements were performed using same equipment throughout the study. Assessment of growth in these children by anthropometric measurements was done and compared with 50 th centile for age and sex, NCHS and WHO charts [7]. Statistical analysis was done by using software SPSS version 10.

Results

40 patients with congenital cardiac malformation were registered for this study. 16 (42%) were female and 24(58%) were males. Age wise distribution of children with acyanotic and cyanotic cardiac malformation in the subject group were divided into age groups of <5years, 5-10 years and >10 years. Majority of the children with CHDs, 18(46%) were pre-schoolers (< 5 years), followed by 15(38%) in age group of 5-10 years and 7(16%) above the age of 10 years. 27 (68%) had acyanotic congenital malformation, while 13 (32%) had cyanotic cardiac malformation. Echocardiographic diagnosis of cardiac defects revealed that Ostium Secundum Atrial Septal Defect (ASD) in 11(28%) of the children as the commonest. The second most common cardiac malformation was Patent Ductus Arteriosus (PDA) in 9(22%). Thus ASD and PDA together comprised over half 20 (50%) of cardiac defects. The third commonest cardiac malformation in the present study was Ventricular Septal Defect (VSD) in 8(18%) cases. Tetralogy of Fallot (TOF) in 7(16%) cases ranked fourth and all were male children. There were 2 (6%) cases of D-Transposition of Great Artery (DTGA). There were two cases of Double Outlet Right Ventricle (DORV) in 1(4%) and there were 1 (4%) cases of Ebstein's Anomaly and there was 1 (2%) case of Pulmonary Stenosis. Assessment of the mean observed value for weight in children with congenital cardiac malformation was 14.97 kgs significantly less than 21 kgs expected value for age and sex, NCHS standards (P=0.038) as in Table 1.

According to Indian Academy of Paediatrics (IAP)

Table 1: Comparison of observed mean weight, standard deviation, t-test and P-value.

Group	Mean weight	S.D	t-Value	P-Value
Observed value (CHD)	14.97	8.60	2.0	0.038

Table 2: Assessment of nutritional status by weight for age according to IAP classification for children less than 5 years of age.

CHD	Normal >80%	GR I PEM 71-80%	GR II PEM 61- 70%	GR III PEM 51- 60%	GR IV PEM <50%
Acyanotic - 11	-	3	3	4	1
Cyanotic - 7	0	3	3	1	0
Total-18	-	6(36%)	6(36%)	5(27%)	1(3%)

Table 3: Wellcome Trust Classification – Weight for age for children above 5 years of age.

CHD	Normal >80%	Underweight 80-60%	Marasmic <60%	Total
Acyanotic	3(14%)	15(68%)	0	18
Cyanotic	1(4%)	3(14%)	0	4
Total	4(18%)	18(82%)	0	22

Wellcome Trust Classification – Weight for age for children above 5 years.

Table 4:

Group	Observed value CHD	S.D	t-value	P-value
Mean height(cm)	99.38	107.6	10.9	0.001
Mean head circumference (cm)	44.6	3.3	-1.6	0.05
Mean MAC (cm)	12.70	1.2	-7.0	0.001
Mean triceps skinfold thickness (mm)Observed value CHD	5.5	0.61	-7.0	0.001
Mean subscapular skinfold thickness (mm)	4.51	0.53	-9.9	0.001

There was a significant mean difference between observed value and expected value (p=0.001).

[8] for children less than 5 years, only 3(17%) children with CHD had normal growth in terms of weight for age in the present study. A large majority of children (83%) with cardiac disease failed to gain weight adequately, among whom 6 (36%) had Protein energy malnutrion (PEM) grade I, 6(36%) had grade II PEM and 5(27%) had grade III PEM and 1 (3%) had grade IV PEM. Growth pattern with regard to type – acyanotic and cyanotic malformations revealed that 3(17%) had grade I malnutrition and 3(21%) had grade II 1 (3%) had grade III and 1 (3%) had grade IV malnutrition with acyanotic malformation and; 3(17%) had grade I PEM and 3(14%) had grade II PEM and 4(3%) had grade III PEM in cyanotic malformation as shown in table 2. According to Wellcome Trust classification [9], majority 18 (82%) out of 22 cases above 5 years of age with CHD were underweight (80-60%) but no one was marasmic as in Table 3.

According to Waterlow's classification of stunting [10], height for age (H/A), height was similarly affected in children with cardiac defects. There was statistically significant difference of 8.22 cms between heights of the children with CHD 99.38 cms, compared to expected 107.6 cms, NCHS standards for age and sex (P<0.001) by student t-test as in Table 4. According to Waterlow's classification [8] only 6(14%) of these children had adequate height for age and sex, a large majority 34 (86%) were stunted, among whom half 20 (50%) suffered from moderate to severe stunting and only 14 (36%) suffered from marginal stunting. Stunting was more severe in children with cyanotic defects, with 100% of them affected, 5(14%) were severely stunted while 2(6%) were moderately stunted and 3(8%) had marginal stunting. Majority of children in acyanotic had marginal to moderate stunting.

Head circumference was estimated in children with congenital cardiac malformation and mean was 44.6 cms which was not significant when compared to normal expected mean 46 cms value for age and sex according to WHO standards. There was no significant mean difference between observed head circumference and expected head circumference (P>0.05) .

Mid-arm circumference was estimated in 18 children between the age of 1 to 5 years with congenital heart disease with mean MAC value 12.68 cms and which was highly significant (p<0.001) when compared to normal expected mean 15.28 cms value for age and sex according to WHO standards. There was significant mean difference between observed mid- arm circumference and expected mid-arm circumference (P<0.001) .

The mean observed value for triceps skin fold thickness recorded in children with congenital cardiac malformation was 5.5 mm, significantly less than 8.12 mm (P<0.001) expected value for age and sex, WHO standards. The mean observed value for subscapular skin fold thickness recorded in children with congenital cardiac malformation was 4.51mm, significantly less than 6.17 mm (P<0.001) expected value for age and sex, WHO standards. There was significant mean difference between observed and expected subscapular skin fold thickness (P<0.001).

Discussion

The present study revealed that a majority, 27(68%) children had acyanotic malformation and 13 (32%) cyanotic malformation. Diagnosis of type of leading cardiac defect in the present study was ASD 11 (28%), followed by PDA 9 (22%), VSD 8 (18%) and TOF 7 (16%). While study from Mumbai reported VSD in 29% as the leading defect followed by ASD 24% and TOF 17.6% [10]. A study from Delhi reported VSD (34%) as the commonest diagnosis followed by PDA (18.6%) [10].

Age was found to be an inverse factor, the older one had less chance for malnutrition and was reported by Villasis Keever MA, et al in Mexico [13]. 100% children with cyanotic malformations were stunted, compared to those with acyanotic malformation. The study also showed significant growth retardation for children with cyanotic malformations, more for height, nearly 60% compared to 45% for weight. In contrast the study by Varan B reported that cyanotic children were more malnourished for weight for age and height for age [3]. A study by Tambic-Bukovac L [10], showed statistically significant growth retardation in 222 children with cardiac disease as compared to 50 in the control group, by values of body weight and height (p<0.001). Weight retardation was more marked than retardation in body height (p<0.001). Growth retardation was more significant in the cyanotic children than in those with acyanotic heart disease (p<0.001).

Among the children with left to right intracardiac shunt, growth retardation was found to increase proportionally with size of the shunt and was most significant in patients with large left to right shunt (QP/QS>1.80) (p>0.01) [9]. In this study only 20 (40%) had income above Rupees 1000 per person. Commonest symptoms were recurrent upper respiratory infections with breathlessness in 58% of patient followed by cyanosis in 24% of patients, cyanotic spells in 10% and congestive cardiac failure in 8%. Thus growth is affected in children with congenital heart disease, being highly statistically significant when compared to expected values, in terms of weight height, mid-arm circumference and skin fold thickness for age and sex, 50 th centile, NCHS and WHO standards (p<0.001).

Around 90% of children were malnourished, indicating that children with CHD significantly suffered from growth failure. Failure to gain weight was seen in those with acyanotic malformation while stunting was seen with cyanotic CHD. Mid-arm circumference and skinfold thickness were also reduced significantly (p<0.001) in present study. Head circumference between children with acyanotic and cyanotic defect showed no difference. Severity of the cardiac lesions and malnutrition put children with CHD at risk for increased morbidity and mortality. Hence, strategies for intervening in the monitoring of growth, a more intensive nutritional rehabilitation, and early corrective surgery should be done to optimise the outcome. Some limitations in our study should be considered. Firstly, this study was conducted in hospitalized patients and hence the results in this study are not a truerepresentative of the general population. Secondly, possible risk factors such as number of family members, mid-parental height, were not analysed in this study. Thirdly, our exclusion criteria may have caused selection bias, leading to underestimation of the true prevalence of malnutrition as some excluded cases may have had more severe malnutrition.

Conclusion

Congenital heart disease in children constitutes an organic cause for failure to thrive. The child needs increase calorie intake for increased cardiac work, sympathetic over activity, recurrent respiratory infections, and hypoxia, in addition, congestive cardiac failure causes difficulty in feeding with poor intake and malabsorption. Hence growth failure is inevitable in symptomatic children with congenital heart disease, which was noted among nearly 90% of the cases with regard to weight, height, mid-arm circumference and skin fold thickness, compared to 50 th centile of NCHS and WHO standards being highly statistically significant (P<0.001) by student t-test.

The two main groups of structural heart defects, acyanotic and cyanotic heart disease show a distinct pattern of growth failure. Children with acyanotic malformations such as ASD, VSD, PDA, PS etc.

manifested with characteristic symptoms of poor feeding, fatigue, dyspnoea, tended to gain less weight and to be leaner than those with cyanotic defects such as TOF, DORV, D-TGA, who have decreased oxygen carrying capacity which affects the growing ends of the epiphyseal plates of long bones affecting growth and hence severely stunted in height. Mid arm circumference and triceps and subscapular skin fold thickness were also affected significantly in present study. PEM and growth retardation are probably the most wide spread, health and nutritional problems in developing countries. In India, 43% under-fives are underweight and 48% stunted in comparison a significantly higher 82% underweight and 86% stunted among children with congenital heart disease. The difference of 40% contributed by cardiac defect as cause for growth failure is significant. In conclusion it can be stated that children with congenital heart disease have highly statistically significant growth retardation ($P<0.001$) by student t- test.

Children with CHD are at risk for increased morbidity and mortality. Strategies for growth monitoring, nutritional rehabilitation, and early surgery should be done in these children.

References

1. Daniel Bernstein. Congenital heart disease. Kliegman, Behrman, Bonita F.S, Joseph W, Nina.F.S (eds) Nelson Textbook of Paediatrics, 19 th edition vol II, Elsevier; 2012.p.1549.
2. Hoffman JKaplan S. The incidence of congenital heart disease. Journal of the American College of Cardiology. 2002; 39(12):1890-1900.
3. Julien I.E Hoffman, Samuel Kaplan. The Incidence of Congenital Heart Disease J Am Coll Cardio. 2002; 39(12):1890-900.
4. Varan B, Tokel K, Yilmaz G. Malnutrition and growth failure in cyanotic and acyanotic congenital heart disease with and without pulmonary hypertension. Arch Dis Chil. 1999; 81(1):49-52.
5. De Staebel O et al. Malnutrition in Belgian children with congenital heart disease on admission to hospital. J Clin Nurs. 2000; 9(5):784-91.
6. Mehrizi ADrash A. Growth disturbance in congenital heart disease. J Pediatr. 1962; 61(3):418-29.
7. Menon GPoskitt E. Why does congenital heart disease cause failure to thrive?. Arch Dis Child 1985; 60(12):1134-39.
8. Growth Charts - WHO Child Growth Standards. Cdc.gov. 2016 [cited 8 August 2016]. WHO_ charts. htm#The WHO Growth Charts
9. Achars Textbook Of Pediatrics (4Th Edn) . Google Books. 2016 [cited 8 August 2016].
10. Villasís-Keever MA e. [Frequency and risk factors associated with malnutrition in children with congenital cardiopathy].

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Assessment of Cardiac Function in Malnourished Children of Central India

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Abstract

Background: Malnutrition is a widespread public health problem and the child is the chief victim of interplay of nutritional, socioeconomic and health factors that cause malnutrition. It affects the potential for growth of the child and causes high mortality in the later years of life. There is increased risk of diseases and deaths in the adults who have been undernourished in their childhood. *Aim*: Assessment of cardiac function in malnourished children of Central India. *Materials and Methods*: The present study was conducted to compare cardiac dysfunction between a group of malnourished children and a control group of the same age and sex and to investigate the variables like xray,ECG,2D ECHO and Cardiac troponin among study group. *Results:* Mean among the cases for QT interval was 332.2 $\pm$44.59 msec and controls were 293.4 $\pm$28.54 msec. (P<0.001). Mean among the cases for QTc interval was 426.7 $\pm$27.5 msec and controls was 379.5$\pm$24 msec. (P<0.0001).Mean LVM among cases was 31.58$\pm$13.6 and control was 47.14$\pm$15.68. There was significant difference in LVM among cases and control (P<0.0001). *Conclusion*: Malnutrition has a definite effect on cardiac volume, muscle mass, as well as the electrical properties of the myocardium. The electrocardiographic findings showed increased QT and QTc intervals. The echocardiographic examination shows decreased interventricular septal thickness, decreased left ventricular dimensions, decreased left ventricular posterior wall thickness, decreased fractional shortening and left ventricular mass suggesting cardiac atrophy and impaired left ventricular systolic functions.

Keywords: Malnutrition; ECG; 2D Echo; Xray; Cardiac TroponinI.

Introduction

Malnutrition is a widespread public health problem and the child is the chief victim of interplay of nutritional, socioeconomic and health factors that cause malnutrition [1]. The quality of nation's health is mainly determined by the investment made for development of child population. The three of eight millennium development goals emphasise on child health, reduction of child mortality and improved maternal health, indirectly leading towards a healthy nation [2]. Malnutrition has adverse effects on the child's health. It decreases the life expectancy of the child. It affects the potential for growth of the child and causes high mortality in the later years of life. There is increased risk of diseases and deaths in the adults who have been undernourished in their childhood [3,4]. Nutritional status of the child depends indirectly on the various factors such as level of employment, food availability, food consumption pattern, purchasing power of people, distribution of income, food distribution in the household amongst the family members, level of knowledge, literacy and government schemes. Improved economic development contributes to improved nutrition and vice versa [5].

In India, there is prevalence of wasting and stunting, causes being lack of food, poor hygiene, poor

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sanitary practice in the household, poverty, illiteracy amongst the mothers and lack of health care facilities. These problems prevail more commonly in rural India. Since independence, agricultural production has increased by many folds, however the infant mortality rate and death rate have come down by one–third, and malnutrition has come down by one–fifth only. This means that despite of adequate food production there is prevalence of malnutrition

It has been found out that heart failure and cardiac arrhythmias are amongst the few causes of death in malnutrition. The risk of death increases with mild undernutrition and as the severity of undernutrition increases, the risk increases exponentially.In most children with third-degree malnutrition, cardiac mass is decreased. Cardiac troponins are regulatory proteins of the thin actin filaments of the cardiac muscle. Myocardial cell injury results in the release of cardiac troponin, which differs from troponin isoforms of the skeletal muscle, and thus is a highly sensitive and specific biomarker of myocardial damage [6]. It is a common and serious problem in the developing countries mainly in the rural areas. The present study was conducted to compare cardiac dysfunction between a group of malnourished children and a control group of the same age and sex and to investigate the variables like X-ray, ECG, 2D ECHO and Cardiac troponin among study group.

Materials and Method

Study Design

The present study was a case control study and was done at Tertiary care hospital, Jawaharlal Nehru Medical College & Acharya Vinoba Bhave Rural Hospital (AVBRH) Sawangi,Wardha. The AVBRH, Sawangi is the rural medical college located in Maharashtra. This study was conducted in the Pediatric Department at AVBRH. The sample Size was 100 (50 cases & 50 controls) and study period was from September 2014 to September 2016.

Method of Collection of Data

Parents of all the subjects (cases as well controls) were interviewed using study questionnaire. The content of the questionnaire included:

A: History

Following details were interviewed

- Demographic characteristics: child's name, sex, address and religion.
- Subject's conditions at birth: gestational age, birth weight
- A detailed dietary history was taken by interviewing the child's mother using a 24-hour recall method.

B:Physical Examination

- General physical examination: Temperature, pulse, respiratory rate, cyanosis, clubbing, lymph nodes, pallor etc.
- Systemic examination: cardiovascular system, respiratory system, abdomen etc was done to exclude the presence of any chronic illness.

C. Anthropometry

- All subjects undergone an anthropometric evaluation at presentation. Anthropometric indices were recorded as recommended of weight to the nearest 0.1 kg by weighing scale machine and height to the nearest 0.5 cm using measuring tape.
- Body mass index (BMI) was calculated by the formula:

  $BMI = weight\ (kg) / height\ (m)^2$.

Inclusion Criteria

- Cases: Children with Malnourished
- Controls: Consisted of age and sex matched subjects without malnourished who were visiting Pediatric OPD and admitted in the Pediatrics ward during the study period.

Exclusion Criteria

- Not willing to participate in the study
- Pre-term infants or intrauterine growth retardation at birth.
- Any documented cardiothoracic event (congenital heart disease, pericarditis, cardiomyopathy, acute severe lower respiratory tract infection, etc.).
- Severe anemia (Hemoglobin level ≤6 g/dl).

Operational Definition

- The WHO recommends the use of standard definitions and classifications for malnutrition (undernutrition) based on calculated Z scores for anthropometric indices [28-29].

Z scores for weight for age (WAZ), weight for height (WHZ) and height for age (HAZ) were recorded.

The WHO global database on child growth and malnutrition (undernutrition) recommends a cut-off z score of ≤-2 to classify low WAZ (underweight), low HAZ (stunting) and low WHZ (wasting) as moderate malnutrition, and a z score of ≤-3 SD to define severe malnutrition.

A z score of ≤-2 indicates that a child's WAZ, WHZ or HAZ is 2 SD below the age- and gender-specific median for the normal population, and 3 SD below the median cut-off if the z score is ≤-3. Normal nutrition is indicated by a WAZ z score of between >-2 and ≤2.

Following Investigations were done in the Subjects

Assessment of cardiac troponin I (**cTnI)** by electrochemiluminescence immunoassay (ECLIM)

Chest X-ray (CXR)

- Cardiothoracic ratio was assessed by the standard way by measuring the cardiac horizontal diameter divided by the internal thoracic diameter with the line passed over the dome of the right diaphragm multiplied by 100.

Electrocardiogram (ECG)

- The 12-lead surface electrocardiograms of all the subjects were obtained using a single channel electrocardiography machine.
- The heart rate, RR interval, QT interval was measured, and corrected QT (QTc) was calculated from all the electrocardiograms.
- QTc was calculated by applying Bazett's equation and the QTc was considered prolonged when it was greater than 440 ms.

$$(QTc = QT/\sqrt{RR})$$

Echocardiography (ECHO)

Transthoracic echocardiography examination was done by using Phillip Echocardigraphy machine with phased array transducers with a frequency of 8 MHZ. M-mode echo is a standard method for assessment of LV function in the absence of segmental wall motion abnormalities.

- *LV Dimensions*
- *Fractional Shortening (FS):* FS was calculated using the following formula:
- *FS=EDD - ESD/EDD X 100*

EDD is the end diastolic diameter of the left ventricle and ESD is the end systolic diameter of the left ventricle

- *Ejection fraction (EF):* EF was measured from the "cubed equation":
- *EF=(EDD)-(ESD)/(EDD) X 100*
- *LV diastolic function:* Using E/A ratio of the mitral flow by pulsed wave Doppler across the mitral valve.
- *Left ventricular mass (LVM) and Left ventricular mass index (LVMI) [7]* to body surface area estimated by LV cavity dimension and wall thickness and end diastole.
- $LVM\ (g) = 0.8\{1.04[([LVEDD+IVSd+PWd]^3 - LVEDD^3)]\}*0.6$

Statistical Analysis

The data was entered, validated and analyzed using STATA 10 software. The numerical data was represented as mean±SD. Comparisons was made between the two main study groups (controls and cases). Continuous variables were expressed as means and SDs if they were normally distributed and as median and range if skewed. Two sample (unpaired) Student t tests, and one way analysis of variance logistic regression analysis was used to compare means and test the relationships between covariates. Each of the variables associated with malnutrition was studied and analyzed. A p value of <0.05 was considered to be statistically significant and p value of <0.001 as highly significant.

Results

The mean age for cases and control was 43.94 ± 39.9 months and majority of the patients were in the age group of 1 to 24 months and there were 64% males and 36% female.The mean weight (kg) of cases was 8.96 ± 4.98 kg and mean weight of controls was 15.37 ±8.16 kg (P < 0.005) and mean WFA among cases was -4.13 ±1.01 and controls were 0.02 ± 0.66 (P value <0.00001). Mean height in cm of cases was 82.08 ± 23.04 cm and mean height in cm of controls was 92.56 ±26.4 cm. (P < 0.03) and mean HFA among cases was -3.21 ± 1.38 and mean HFA among controls was -0.46 ± 0.80 (P <0.00001). Mean BMI for cases was 12.33± 1.98 and controls were 16.83±1.58 (P value <0.0001) and Mean BMI for age among cases was -

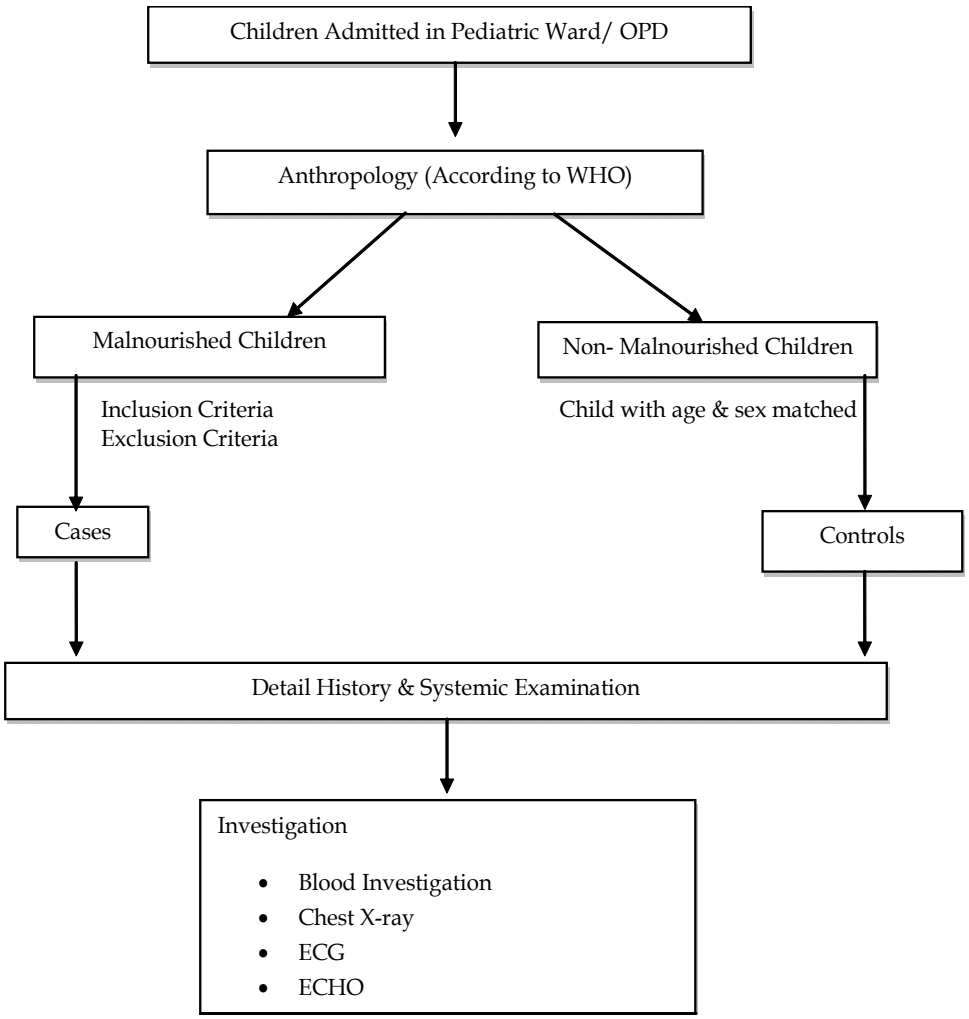

Fig. 12: Flowchart of study

3.09 ± 1.78 and controls were 0.4 ± 0.97.(P value was <0.00001). WHO classification among the cases (n=50), where majority 29(58%) had stunted growth, followed by 14 (28%) had stunted and wasted growth and only 7 (14%) had wasted growth. IAP classification among the cases (n=50), where majority (40%) had IAP grade 2 and 3 respectively and only 20% had IAP grade 4.

Mean CT ratio for cases was 0.47± 0.03 months and controls was 0.49 ± 0.02months (P<0.005) and according to WHO classification, CT ratio among Stunted was 0.48±0.03, wasted was 0.47±0.35 and stunted and wasted was 0.46±0.02 (P>0.10) which was not stastically significant among cases.

Mean troponin I for cases was 0.015 ±0.02mcg/lit and for controls was 0.01 ±0.02mcg/lit(P>0.2) and according to WHO classification,Cardiac troponin I among Stunted was 0.01±0.007 mcg/lit,wasted was 0.01±0 mcg/lit and stunted and wasted was 0.01±0 mcg/lit (P=0.89) which was not stastically significant among cases and control and also among cases.

In ECG, mean HR among cases was 102.1± 17.8 per min and control was 101.5±17.2.(P>0.8) per min.Mean RR interval among cases was 0.6±0.094 secs and control was 0.6±0.099 secs (P>0.7). Mean among the cases for QT interval was 332.2 ±44.59 msec and controls were 293.4 ±28.54 msec. (P<0.001). Mean among the cases for QTc interval was 426.7 ±27.5 msec and controls was 379.5±24 msec. (P<0.0001) There was significant difference in QT interval and QTc interval among cases and controls but according to WHO classification among cases mean QTc among Stunted was 422.4±20.5 msec, wasted was 445.5±52.5 msec and stunted and wasted was 426.0±20.88 msec (P=0.13) which was not stastically significant.

Table 1 shows mean LVIDs among cases was 16.28 ±3.23 and control was 18.28±3.38(p<0.03). Similarly, there was significant difference found in LVIDd, LVPWs, LVPWd, LVIVSs, LVIVSd and FS among cases and controls (P <0.05). Mean LVM among cases was 31.58±13.6 and control was 47.14±15.68. There

was significant difference in LVM among cases and control (P<0.0001). But no significant difference in LVMI among cases and control.

In Table 2 we compared the echocardiographic finding among the cases according to WHO, we found the LVIDd, LVPWs and LVIVSd were significant difference among different types of WHO (p<0.005). Whereas the LVIDs, LVIDd, LVIVSs, LVIVSd, EF and FS were not having significant difference among the type of WHO. There was statistically significant difference was found in LVM among the different type of malnutrion according to WHO (p<0.005) whereas LVMI was not significant among the different type of malnutrion.

Diastolic function shows mean E among cases 0.88±0.12 and mean E among controls 0.93±0.10.(p<0.02). Mean A among cases 0.52±0.11 and controls were 0.58±0.11. (p<0.005). Mean E/A among cases 1.75±0.41and mean E/A among controls 1.63±0.21(p>0.05). There was significant difference in E and A but no difference in E/A ratio among cases and controls and according to WHO classification in cases E/A ratio among Stunted was1.7±0.4 ,wasted was 1.8±0.5 and stunted and wasted was 1.7±0.3 (P=0.70) which was not stastically significant.

Table 1: Echocardiography Findings

Variables	Cases	Controls	P value
LVIDs (mm)	16.28 ± 3.23	18.28±3.38	P= 0.03
LVIDd(mm)	25.52±4.97	29.92±5.49	P <0.001
LVPWs(mm)	8.57±1.85	9.56±1.65	P=0.05
LVPWd(mm)	5.78±1.04	6.76±0.62	P<0.0001
LVIVSs(mm)	7.64±1.49	9.08±1.67	P<0.0001
LVIVSd (mm)	5.96±1.28	6.6±0.8	P= 0.03
FS (%)	35.61±6.28	38.75±4.8	P= 0.005
EF (%)	67.5± 7.09	68.05± 5.11	P=0.6
LVM (gm)	31.58±13.6	47.14±15.68	P<0.0001
LVMI (gm/m^2)	73.64± 24.2	79.26± 14.63	P=0.1

Table 2: Echocardiography finding among the study cases according to WHO classification

Variables	Stunted	Wasted	Stunted and wasted	P value
LVIDs (mm)	16.89±3.00	15.4±2.9	15.4±3.7	0.29
LVIDd(mm)	26.7±4.7	24.14±5.6	23.57±4.5	0.09
LVPWs(mm)	9.34±1.8	7.14±1.77	7.69±0.99	0.001
LVPWd(mm)	6.03±1.05	5.2±0.95	5.51±0.99	0.12
LVIVSs(mm)	7.96±1.5	7.42±1.1	7.07±1.38	0.17
LVIVSd (mm)	6.33±1.3	5.28±1.1	5.52±1.0	0.04
FS (%)	35.9±6.2	35.5±4.3	34.9±7.3	0.88
EF (%)	68.5±7.33	66.54±5.0	65.8±7.4	0.46
LVM (gm)	36.34±14.3	24.8±12.2	25.07±8.18	0.01
LVMI (gm/m^2)	75.13±24.5	72.1±12.4	71.2±28.7	0.87

Discussion

Malnutrition is a complex phenomenon resulting in body composition alterationscomplicated by electrolytic disorders and mineral or vitamin deficiencies. Protein- energy malnutrition (PEM) is frequently located in less-developed countries due to inadequate food intake, socioeconomic or political factors or, at times, due to natural disasters. Malnourished children suffer cardiac abnormalities as hypotension, cardiac arrhythmias, myocardiopathy, cardiac failure and sudden death. It is difficult to find out whether these abnormalities are primary phenomena of malnutrition or secondary to other alterations associated with malnutrition.

In present study mean age for cases was 43.94±39.9. Study by Nagla Hassan Abu F et al [8] showed that forty-five malnourished infants and young children (mean±SD of age was 11.24 ±7.88 months) were matched with 25 apparently healthy controls (mean±SD of age was 10.78±6.29 months). Jose LO et al [10] in their study showed that mean age for malnourished group was 2.40 ±1.82 and control group was 2.52 ±1.75 (P> 0.8). In the present study in both groups majority were males with M:F ratio was 1.8:1. Study by Munde A et al [9] showed that M: F ratio was 1.5:1. Study by AL-Samerraee AS et al [17] shows M:F ratio among cases was 0.4:1 and among control was 0.5:1(P value=0.46).Present study shows mean weight of cases was 8.96 ±.98 kg and in controls was 15.37 ±8.16 kg (P< 0.005) Study by Divya S et al [11] showed that mean body weight for malnourished group was 15.47±4.45 and among control group was

30.60 ±9.70 (P<0.003).

Study by Divya S et al [11] showed that mean height for malnourished group was 107.65±14.27 and among control group was 129.5±14.49 (P=0.003). In present study, we found that mean height in cm of cases was 82.08 ±23.04 cm and of controls was 92.56 ±26.4 cm (P value<0.03).

Present study shows mean BMI for cases was 12.33±1.98 and for controls was 16.83±1.58 (P <0.005). Jose LO et al [10] in their study showed that mean BMI for malnourished group was 13.58 ±1.09 and control group was 16.22 ±0.82 (P < 0.00)

Study done by Abu Fadan et al [8] reported that cardiac troponin among cases was 0.01±0.01 and controls was 0.009±0.001 which is statistically significant difference in cardiac troponin among cases and controls. In present study, mean cardiac troponin among cases was 0.015±0.02 and controls was 0.01±0.02 which is not significant difference among cases and controls. Troponin I is a contractile protein found almost solely in the myocardium. Based on the previous data it is clear that there is no place for cardiac protein to be released massively in a detectable way in circulation except in severe acute cases or in the presence of complication as severe hemodynamic disturbances or sepsis [85].

Olowonyo et al. [18] found the mean CT ratio in cases was 49.39+ 4.0 and in controls was 55.4+ 3.5. There was a statistical significant difference (p<0.005) found among the cases and controls. Similar finding was present in our study also.

Abdel J F et al [13] study QT parameter showed significant difference(p=0.03), in Hl El Sayed et al [12] study QT among cases was 0.27±0.03 secs and among control was 0.21± 0.04 and QTc among cases was 0.46±0.03 secs and control was 0.41±0.07 secs and both parameters showed significant differences (p=<0.01), in Divya S et al [11] study QTc among cases was 0.455± 0.021 sec and control was 0.419± 0.020 sec which was significant(p=0.0007), in Neeraj Kumar et al [14] study also QTc was significant (p=0.0001) and in present study also QT and QTc showed significant differences.

In study by Abu Fadan et al [6] reported that LVSD, PWD, FS and EF had significant difference among cases and controls(p<0.05). AL-Samerraee AS et al [17] found that LVIDd, LVPWs, LVPWd, LVIVSs and LVIVSd had significant difference among cases and control(p=0.00). In present study LVIDs, LVIDd, LVPWs, LVPWd, LVIVSs, LVIVSd and FS had significant difference but EF did not have significant difference among cases and control

In study by Kothari SS et al [16] LVM among cases was 25.75 + 8.09 gm and control was 32.44 + 11.64 gm had significant difference(p<0.05), Ocal B et al [80] in their also showed significant difference in LVM(p<0.05), study by Amna A et al [15] showed LVM among cases 11.38 + 7.74 gm and among cases 22.35 +12.03 gm(p=0.002) as well as LVMI among cases was 32.34+20.38 and control was 47.58+ 23.45(p= 0.024) similarly in present study also LVM and LVMI had significant difference.

In our study, we found E had significant differences and E/A ratio had no significant difference among the cases and controls, this was similar to those observed by Amna A et al [15] were E among cases was 0.80± 0.21m/s and among control was 1.00 ±0.25m/s(p=0.006) and E/A among cases was 1.79 ±0.99 and control was2.03± 1.13. (p=0.443). In our study, we found E/A had no significant differences found among the cases and controls.

Refrences

1. Pathak et al. Trends in malnutrition among children in India: Growing inequalities across different economic groups Social Science & Medicine. 2011 Aug; 73(4):576–585.
2. Chhabra. R and C. Roks. The Nutrition MDG indicator- Interpreting Progress, World Bank Discussion Paper, Washington D.C, World Bank. (2004).
3. Haq, M.N. Age at menarche and the related issue: A pilot study on urban school girls. Journal of Youth and Adoloscence. 1984; 13(6):559-67.
4. Karim , A, A. K. M. A Chowdhary and M. Kabir. Nutritional status and age at secondary sterility in rural Bangladesh. Journal of biosocial science. 1985; 17(4):497-502.
5. Black, R.E., Singhal, A., Uaur, R. International Nutrition: Achieving Millennium Goals. Nestlé Nutrition Institute Workshop, Muscat. March 2013.
6. Torum, B. and Chew, F. Protein Energy Malnutrition in Modern Nutrition in health and disease. United States Of America: Lea and Febiger. 1994; 8th ed. Vol. 2:950-975.
7. Lang RM, et al. Recommendations for chamber qualification. J Am Soc Echocardiogr 2005;18: 1454-7.
8. Nagla Hassan Abu Faddan, Khalid Ibrahim El Sayh, Hamdy Shams,and Hosni Badrawy. Myocardial dysfunction in malnourished children. Ann Pediatr Cardiol. 2010; 3(2):113–118.
9. A Munde, N Kumar, Rs Beri And Jm Puliyel. Lactate Clearance As A Marker Of Mortality In Pediatric Intensive Care Unit. Indian Pediatrics.

July 15, 2014; Volume.

10. Jose ´ L. Olivares, Margarita Va´Zquez, Gerardo Rodríguez, Pilar Samper, And Jesu´S Fleta. Electrocardiographic And Echocardiographic Findings In Malnourished Children. Journal Of The American College Of Nutrition. 2005; 24(1):38–43.
11. Divya Srivastava, Rajesh Misra, Ashish Prakash, Prachi Gautam And Shyamali Dutta. Correlation Of Anthropometric Parameters With Qtc Interval In Malnourished Children. Indian J Physiol Pharmacol. 2011; 55(1):37–43.
12. HL El-Sayed, MF Nassar, NM Habib, OA Elmasry and SM Gomaa. Structural and functional affection of the heart in protein energy malnutrition patients on admission and After Nutritional Recovery. European Journal Of Clinical Nutrition. 2006; 60: 502–510.
13. Abdel J. Fuenmayor, Rosae. Mora, Akaarc. F~Ienmayor.Qt -Interval Dispersion In Malnourished Children. C'lin. Cardiol. 1998; 21:201-205.
14. Neeraj Kumar, Aakash Pandita, Deepak Sharma, Anita Kumari, Smita Pawar and Kishour Kumar Digra. To Identify Myocardial Changes in Severely Malnourished Children: A Prospective Observational Study. Front Pediatr. 2015; 3.
15. Amna Abdelbagi Mohammed Ahmed. Clinical And Echocardiographic Findings In Children With Malnutrition In Khartoum State. University of Khartoum Graduate College Medical & Health Studies Board. May 2007.
16. Kothari SS, Patel TM, Shetalwad AN, Patel TK. Left ventricular mass and function in children with severe protein energy malnutrition. Int J Cardiol. 1992 Apr; 35(1):19-25.
17. Amal S. AL-Samerraee, Jassim M., Impact of PEM on heart structure. International Journal of Pharmacy and Pharmaceutical Sciences. 2014; 6(6).ISSN- 0975-1491.
18. Olowonyo MT, Ogunkunle OO, Akinbami FO, Jaiyesimi F. The echocardiographic findings in kwashiorkor. J Trop Pediatr. 1995; 41:74–76.

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Crouzons Syndrome: A Review of Literature and Case Report

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Abstract

Crouzon syndrome is an autosomal dominant disorder with complete penetrance and variable expressivity [1]. In 1912 a French neurologist, Octave Crouzon (1874-1938) first described a hereditary syndrome of craniofacial dysostosis in a mother and her daughter which included a triad-cranial deformities, facial anomalies and exophthalmos [2]. The genetic defect appears to emanate from the mutation of fibroblast growth factor receptor 2 (FGFR2) on chromosome locus 10q25-q26, resulting in early fusion of skull bones during fetal development. Crouzon syndrome, also called craniofacial dysostosis characterized midfacial hypoplasia and orbital defects. It accounts for 4.8% of all cases of craniosynostosis [3]. Normally, the sutures in the human skull fuse after the complete growth of the brain, but if any of these sutures close early then it may interfere with the growth of the brain. . Mental retardation is not a hallmark feature unless premature closure of the cranial suture lines impairs brain development [4]. The disease is characterized by premature synostosis of coronal and sagittal sutures which begins in the first year of life. Case report of a 7 year old boy is presented with characteristic features of Crouzon's syndrome with mental retardation.

Keywords: Crouzon's Syndrome; Fibroblast Growth Factor; Premature Synostosis; Hypertelorism; Exophthalmos; Midfacial Hypoplasia.

Introduction

Crouzon syndrome is a rare genetic disorder characterized by premature closure of cranial sutures, exophthalmos and mid facial hypoplasia. Crouzon syndrome occurs in approximately 1 in 25,000 births world wide and 16.5 per 1,000,000 live births in World [5]. It may be transmitted as an autosomal dominant genetic condition or appear as a mutation. No known race or sex predilection exists The majority of the patients with Crouzon syndrome have mutations in the extracellular immunoglobulin III domain of the Fibroblast Growth Receptors 2(FGFR2) gene [6]. The differential diagnosis of Crouzon syndrome includes Apert syndrome, Pfeiffer, Jackson-Weiss, Carpenter and Saethre-Chotzen syndrome. Crouzon syndrome is distinguishable from other craniosynostosis syndromes by lack of hand and/or foot abnormalities [7,8]. Multiple staged surgeries are the general treatment plan for patients with Crouzon syndrome. In this article, we present a case of Crouzon syndrome in a boy aged 7 years.

Case Report

A 7-year-old boy along with his parents reported to our department for treatment. The chief complaint being as presented by the mother was mental retardation and overcrowding of teeth. Since the child's appearance and head size was not normal, the family and medical history were taken in detail. It was diagnose as Crouzon's syndrome associated with mild to moderate mental retardation. Review of

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medical history was unremarkable, specifically, the mother reported normal labor and delivery. There were no anomalies in any siblings or near relatives reported. The child was not on any medications and denied any medical allergies. Further medical history revealed that the enlarged size of the head was noted by the mother ever since he was 6 months and the severity has gradually increased.

On Examination

On examination, enlarged cranial vault with frontal bossing, maxillary hypoplasia and a relative, mandibular prognathism was found. Ocular manifestations such as shallow orbits, hypertelorism, bilateral proptosis, exophthalmos and strabismus were present. Other facial features included short and incompetent upper lip, depressed nasal bridge and low-set ears but without any hearing loss. His hands and feet found to be normal. Past medical history from the parents revealed that the features started developing slowly after birth and was diagnosed as CS at the age of 9 months and the patient had undergone cranial surgery to relieve closed sutures of the skull at the same age.

In Figure 1 Frontal and lateral view face showing the frontal bossing, midface hypoplasia and a relatively large mandible, shallow orbits, hypertelorism, exophthalmos, short and incompetent upper lip, depressed nasal bridge, (Figure 2) lateral view of the face.

The prenatal, delivery and postnatal history was found to be insignificant. Family history revealed no abnormality. He is the first child of clinically healthy parents of non-consanguineous marriage. His developmental milestones were found to be normal. The patient was presented with normal intelligence and little speech difficulty. At the time of his birth, his father was 36 years old and his mother was 22-year-old.

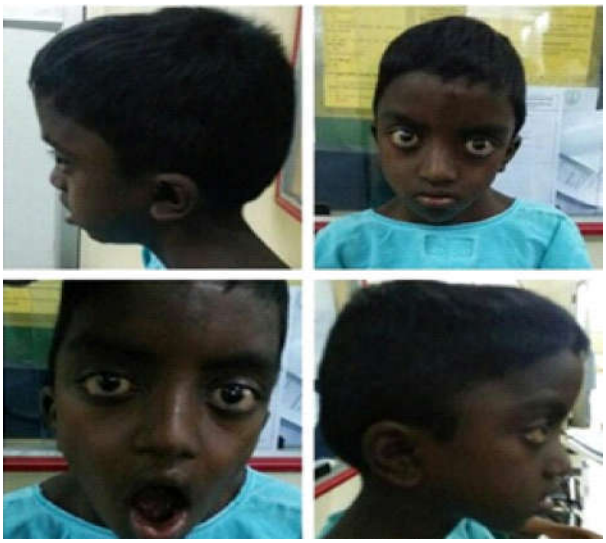

Fig. 1 & 2: Frontal and lateral view face showing the frontal bossing, midface hypoplasia and a relatively large mandible, shallow orbits, hypertelorism, exophthalmos, short and incompetent upper lip, depressed nasal bridge, (Fig 2) lateral view of the face.

On intraoral examination, V-shaped maxillary arch with high-arched palate and bilateral palatal swellings which was mimicking a pseudocleft, retruded maxilla with a relatively large mandible were found. His oral hygiene was poor with crowding of upper and lower teeth, reverse over-jet with posterior cross-bite and anterior open bite, tongue tie and decayed teeth were present.

The skull radiographs revealed the 'scaphocephalic' skull shape, hypoplastic maxilla and zygoma with shallow orbits (Figure 3 & 4) Prominent cranial markings of the inner surface of the cranial vault seen as multiple radiolucencies appearing as depressions resulting in the 'hammered silver' (beaten metal/copper beaten) appearance indicating internal remodeling of the calvaria due to an increase in intracranial pressure as a result of premature cranial suture fusion.

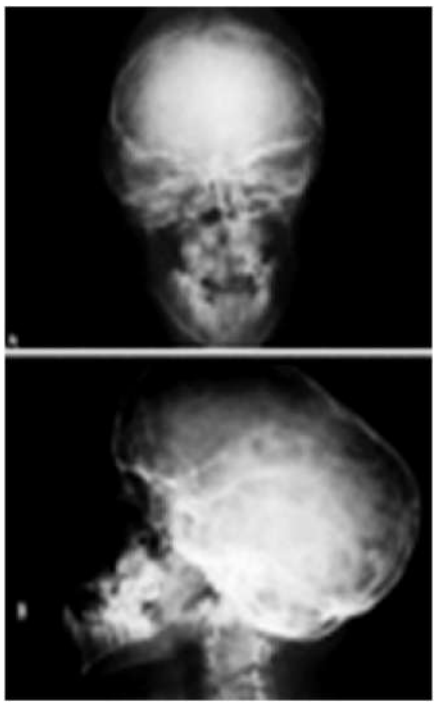

Fig. 3 & 4: AP and lateral view of the skull

AP and lateral view of the skull–demonstrating maxillary retrusion, relative mandibular prognathism and 'hammered silver' (beaten metal/copper beaten) appearance

Three-dimensional computed tomographic (CT)

scans of the skull showed fused sagittal and lambdoid sutures and surgically opened coronal suture, moderate degree of hydrocephalus with diffuse indentation of inner table of skull Other systemic examination was found to be normal. Routine hematological and biochemical tests were within normal limits.

Discussion

CS is inherited as an autosomal dominant fashion but there is an equal incidence of sporadic cases which probably represent new mutations. The sporadic cases are postulated to be associated with advanced paternal age and some investigators have found that this mutation is more common in the sperm of older men. However, the fact that the same mutation can produce a wide range of phenotypic expression makes the mechanism of anomalous development more complex [9]. Penetrance is high although severity is variable. Within the family, members tend to have similar facial deformities but variable calvarial deformities and this phenotypic heterogeneity makes genetic counseling difficult. The phenotypic features of CS may be absent at birth and evolve gradually during the first few years of life [10]. The variability in both cranial and facial malformations depends on the order and rate of progression of sutural synostosis. Premature synostosis commonly involves the sagittal and coronal suture. Lambdoidal sutures are occasionally involved. The craniosynostosis of the sagittal is predominant in boys, while the coronal is more common in girls. The type of obliterated sutures determines the shape of the cranial vault. The skull shape can vary from brachycephaly (most commonly observed) to scaphocephaly (boat-shaped head), oxycephaly, plagiocephaly, trigonocephaly (triangle-shaped head) or in severe disease cloverleaf skull (kleeblattschädel) like deformity-In the present case premature closure of the sutures had caused restricted skull growth and lack of space for the growing brain resulted in 'compensatory' change in the growth of skull and brain toward frontal region, where the coronal sutures were opened surgically causing frontal bossing creating an elongated narrow scaphocephalic skull [11].

The facial and oral malformations consist of hypoplastic maxilla and zygoma, pointed nose (psittichorhina/parrot beak-like nose) due to the short and narrow maxilla, narrow high-arched palate, bilateral palatal swellings (pseudocleft) or cleft palate in some patients and crowding of teeth as well as posterior crossbite and reverse overjet with anterior open bite and relative mandibular prognathism [12].

Approximately one-third of patients with CS suffer from hearing loss due to middle ear deformities and upper airway obstruction occurs due to midfacial hypoplasia and narrow epipharynx. Optic atrophy is frequently seen and has been reported in 30 to 80% of patients [12]. Affected individuals exhibit ocular malformations including hypertelorism, proptosis due to the shallow orbits. Mental ability and psychomotor development is generally within normal limits. However, when the premature closure of the cranial suture lines impairs brain development due to increased intracranial pressure it can lead to mental retardation [13]. The gene for CS could be localized to the FGFR2 at the chromosomal locus 10q 25.3-q26 in more than 50% of cases [14]. Mutation of the FGFR gene is also responsible for other craniosynostosis, such as Apert's, Pfeiffer's, Jackson-Weiss' and Saethe-Chotzen's syndromes [15]. Rarely, acanthosis nigricans may coexist with CS in childhood and is caused by mutation in the FGFR3 gene (locus 4p16.3).

Thorough clinical, radiological and genetic analysis is required for early diagnosis of CS. Prenatal diagnostic testing for FGFR gene mutation is an option for couples at risk for having a child with CS. Ultrasonic prenatal diagnosis of exophthalmos might give a clue regarding the developing problems.

The management requires a multidisciplinary approach and the surgical treatment usually begins in the child's first year with cranial decompression. In the presented case early craniectomy of coronal sutures was done at the age of 9 months to relieve increased intracranial pressure caused by premature multiple suture synostosis [6]. An increased intracranial pressure impairs brain development and can lead to mental retardation. Because of the early diagnosis and intervention in this case, no complications were found in our case except for dysmorphic features and the patient presented with normal intelligence. Skull reshaping may need to be repeated when the child grows and subsequent development of midfacial hypoplasia also needs correction. Procedures for this purpose will include Le Fort III osteotomy or its segmental variants, monobloc frontofacial advancement, or bipartition osteotomy which helps in the cosmetic reconstruction of facial dysmorphisms. The goal is to stage reconstruction to coincide with facial growth patterns and psychosocial development. The prognosis in the case of CS depends on severity of malformation and the patients usually have a normal lifes pan [16].

In our case, complex treatment plan involving prophylactic and therapeutic approach was

formulated for the patient which includes regular mechanical and chemical professional plaque control, with fluoride and chlorhexidine applications to control the intense carioactivity and gingival inflammation.

Conclusion

Clinical diagnosis of Crouzon syndrome is based on clinical findings, molecular genetic testing of FGFR2 gene mutation. Being an autosomal dominant disorder, there is a 50% chance of passing the disease to each child. Prenatal testing of pregnancies with high risk is necessary. Treatment is palliative and surgical management is tailored to individual needs. Prevention of secondary complications such as hydrocephalus, cognitive impairment depends on early treatment of craniofacial anomalies. Exposure keratitis in these children can be prevented by adequate ophthalmologic lubrication in those with severe proptosis.

References

1. Crouzon O. Dysostose cranio-faciale hereditaire. Bull Mem Soc Med Hop Paris. 1921; 33:545–555.
2. Reardon W, Winter RM, Rutland P, Pulleyn LJ, Jones BM, Malcolm S. Mutations in the fibroblast growth factor receptor 2 gene cause Crouzon syndrome. Nat Genet. 1994; 8(1):98–103.
3. Al-Qattan MM, Phillips JH. Clinical features of Crouzon's syndrome patients with and without a positive family history of Crouzon's syndrome. J Craniofac Surg. 1997; 8(1):11–13
4. Bowling EL, Burstein FD. Crouzon syndrome. Optometry. 2006; 77(5):217–222.
5. Kaur H, Waraich HS, Sharma CM. Crouzon syndrome: A case report and review of literature. Indian J Otolaryngology Head Neck Surg. 2006; 58(4):381–382.
6. Regezi JA, Sciubba JJ, Jordan RCK. Oral pathology: Clinical pathologic correlations. London: WB Saunders Company; 1999.p.436–437.
7. Da Silva DL, Palheta NFX, Carneiro SG, Palheta ACP, Monteiro M, Cunha SC, Nunes CTA. Crouzon's syndrome: Literature review. Intl Arch Otorhinolaryngol. 2008; 12(3):436–441.
8. Glaser RL, Jiang W, Boyadjiev SA, Tran AK, Zachary AA, Van Maldergem L, Johnson D, Oldridge M, Wall SA. et al. Paternal origin of FGFR2 mutations in sporadic cases of Crouzon syndrome and Pfeiffer syndrome. Am J Hum Genet. 2000; 66(3):768–777.
9. Cohen MM Jr., editor. Craniosynostosis: Diagnosis, evaluation, and management Crouzon syndrome. In: Cohen MM Jr (Ed). Craniosynostosis: Diagnosis, evaluation, and management. (2nd ed) New York: Oxford University Press; 2000.p.361.
10. Golabi M. Radiographic abnormalities of Crouzon Syndrome. A survey of 23 cases. Proc Greenwood Genet Ctr. 1984; 3:102.
11. Neville BW, Damm DD, Allen CM, Bouquet JE. Oral and maxillofacial pathology . Developmental defects of the oral and maxillofacial region. Elsevier Publishers; 2004.p.40.
12. Abdallah AM. A severe form of Crouzon's syndrome: Clinical and radiological correlation. Saudi J Ophthalmol. 2003; 17:183–185.
13. Jeftha A, Stephen L, Morkel JA, Beighton P. Crouzonodermo- skeletal syndrome. J. Clin. Pediatr Dent. 2004; 28(2):173–176.
14. Preising MN, Schindler S, Friedrich M, Wagener H, Golan I, Lorenz B. On the effect of mutations of the fibroblast growth factor receptors as exemplified by three cases of craniosynos- toses. Klin Monbl Augenheilkd. 2003; 220(10):669–681.
15. Meyers GA, Orlow SJ, Munro IR, Przylepa KA, Jabs EW. Fibroblast growth factor receptor 3 (FGFR3) transmembrane mutation in Crouzon syndrome with acanthosisnigricans. Nat Genet. 1995; 11(4): 462–464.
16. Atkinson FR. Hereditary Craniofacial dysostosis or Crouzon's disease. Med Press Circular.1937; 195: 118–124.

Case Report

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Cornelia De Lange Syndrome: A Case Report of a 5 Year Old Boy

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Abstract

Cornelia de Lange syndrome (CdLS) is rarely seen syndrome with incidence between 1/10,000 and 1/60,000 neonates. It is characterized by typical facial features, besides involving multiple systems. We present here a case of 5 year old male child with typical facial features of CdLS like bushy eyebrows that meet in midline, long eye lashes, long philtrum, thin upper lip, depressed nasal bridge and anteverted nostrils. The patient was diagnosed as having Cornelia de Lange syndrome as he has distinctive facial features in addition to the pre- and postnatal growth retardation, hirsutism and speech delay. He has been followed up by interdisciplinary care team.

Keywords: Cornelia De Lange Syndrome; Incidence; Typical Facial Features; Brachman De Lange Syndrome; Classical CdLS; Mild CdLS; Phenocopy CdLS.

Introduction

Cornelia de Lange syndrome (CdLS) is a rare genetic disorder that affects multiple systems. Typically it is characterised by distinctive facial appearance, intellectual disability, growth failure and hirsutism as prominent features [1].

This syndrome affects both the physical and intellectual development of a child. This syndrome is otherwise called as Brachman de Lange syndrome as this was identified first by a Dutch paediatrician Brachman in 1933. Incidence is rare and affects between 1/10,000 and 1/60,000 neonates [2].

Congenital anomalies of CdLS include malformations of the upper limbs, gastrointestinal malformation/rotation, pyloric stenosis, diaphragmatic hernia, heart defects and genitourinary malformations. Gastroesophageal reflux disease is present in almost all patients [3].

The distinct facial characteristics of CdLS include highly arched eyebrows, synophrys, long eyelashes, short nose with anteverted nares, small widely spaced teeth, and microcephaly. Other frequent findings include loss of hearing, ophthalmic abnormalities, cardiac septal defects, gastrointestinal dysfunction, and cryptorchidism or hypoplastic genitalia. Individuals with a milder phenotype have less severe growth, cognitive, and limb involvement, but often have facial features consistent with CdLS [4].

Sporadic cases of CdLS are most common, but familial transmission with an autosomal dominant hereditary pattern has also been reported [5].

Case Report

A 5 year old male child was presented to Pediatric department, Navodaya medical college, Karnataka with the complaints of not gaining weight. He was born to a consanguineous couple 3rd in order of birth. He was a full term baby born through normal vaginal delivery. Child did not cried for about five minutes after birth.

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Physical examination revealed weight, height and head circumference as 10 kg, 89 cms and 45 cms respectively, all below 3rd percentile as per WHO charts.

General examination reveals arched confluent and bushy eyebrows, well-defined long curly eyelashes, anteverted nares with long philtrum, microcephaly (Figure 1), and excessive body hair (Figure 2) without any gross limb deformities. X-ray of wrist joint showed delayed and hypoplastic appearance of epiphyseal centre in right ulna (Figure 3).

No abnormality has been detected in systemic examination. Ophthalmologic and ear examination also revealed normal findings.

The child had speech difficulties and was using gestures and incomplete words to communicate. He is more aggressive and anxious.

The complete blood count, biochemical parameters and urine analysis were normal. Echocardiography was normal. Genetic analysis was not done due to financial constraints.

The child has been followed up by an interdisciplinary care team (pediatrician, gastroenterologist, neurologist, physiotherapist, dentist, speech therapist, and psychologist). After a series of follow-ups over a period of one year, child was found to be less aggressive, playful and there was a marked improvement in speech suggesting the importance of interdisciplinary approach in managing child with CdLS.

Fig. 1: Typical facial features

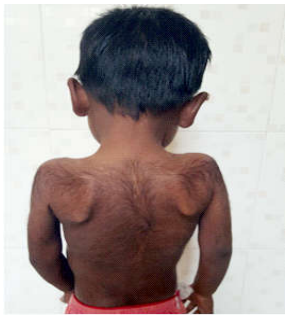

Fig. 2: Excessive body hair

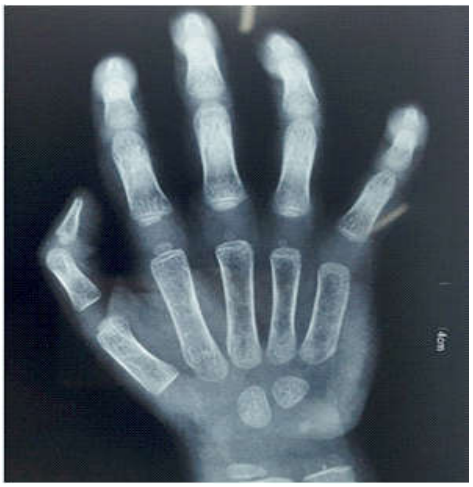

Fig. 3: Hand and wrist X-ray hypoplastic epiphyseal centre

Discussion

CdLS is a rare multisystem disorder with an overall incidence of between 1/10,000 and 1/60,000 [2]. Majority of the CdLS cases are sporadic [6], however familial inheritance with autosomal dominance pattern has also been reported [7].

CdLS is mainly classified into three types based on clinical manifestations. Type I also called as classical CdLS is characterized by typical facial features, severe growth deficiency prenatally and psychomotor retardation.

Type II also called as mild CdLS, has almost similar facial features but with minor skeletal and systemic malformations, which develop with time or are only partially expressed. They may present with border-line psychomotor retardation. Type III also called as "phenocopy CdLS" will have similar phenotypic manifestations of CdLS that may be related to chromosomal aneuploidies or teratogenic exposures [8].

CdLS can be diagnosed with the help of typical clinical manifestations. Genetic analysis of chromosomal mutations is not needed to confirm the syndrome [9].

The major causes of death in CdLS were in the following order: aspiration pneumonia (31%) followed by gastrointestinal disorders like obstruction or volvulus (19%) and then by congenital anomalies like diaphragmatic hernia and congenital heart defects (15%) [10]. Milder form of CdLS has good prognosis when compared with that of classical form [11]. Multidisciplinary approach towards patients with CdLS has a good potential for improving the overall health of patients [12].

Conclusion

Despite its rare occurrence, CdLS poses serious limitation on the quality of life of the affected patient which is evident from the clinical manifestations. As CdLS involves multiple systems, multidisciplinary approach to caring for patients with CdLS is essential. A better understanding of etiology, pathogenesis and clinical manifestations can be established with more number of case studies that will help for establishing strategies for improving the quality of life of affected individuals.

References

1. Boyle MI. Cornelia de Lange syndrome. Clin Genet. 2015 Jul; 88(1):1-12.
2. Hosseininejad S, Bazrafshan B and Alaee E. A Case Report of Cornelia De Lange Syndrome in Northern Iran; A Clinical and Diagnostic Study. J Clin Diagn Res. 2016 Feb; 10(2):SD03–SD05.
3. Tayebi N. Cornelia de lange syndrome. Indian J. Hum. Genet. 2008; 14(1):23-26.
4. Brachmann-De Lange Syndrome. In: Jones KL, Jones MC, Campo DC, editors. Smith's Recognizable Patterns of Human Malformation. 7th edition. Philadelphia: Elsevier, 2013.
5. Schoumans J, Wincent J, Barbaro M et al. Comprehensive mutational analysis of a cohort of Swedish Cornelia de Lange syndrome patients. European Journal of Human Genetics. 2007; 15: 143–149.
6. Brock G, Redon R, Sanlaville D et al. NIPBL mutations and genetic heterogeneity in Cornelia de Lange syndrome. J Med Genet. 2004; 41(128):1-6.
7. McConnell V, Brown T, Morrison PJ. An Irish three-generation family of Cornelia de Lange syndrome displaying autosomal dominant inheritance. Clin Dysmorphol. 2003 Oct; 12(4):241-4.
8. Rohatgi S, Clark D, Kline Ad et al. Facial Diagnosis of Mild and Variant CdLS: Insights From a Dysmorphologist Survey. Am J Med Genet A. 2010 Jul; 0(7):1641–1653.
9. Park K, Lee S, Ki C et al. Cornelia de Lange Syndrome with NIPBL Gene Mutation: A Case Report. J Korean Med Sci. 2010 Dec; 25(12):1821–1823.
10. Schrier S, Sherer I, Deardorff MA et al. Causes of Death and Autopsy Findings in a Large Study Cohort of Individuals with Cornelia de Lange Syndrome and Review of the Literature. Am J Med Genet A. 2011 Dec; 155(12):3007–3024.
11. Verma L, Passi S, Gauba K. Brachman de Lange syndrome. Contemp Clin Dent. 2010 Oct-Dec; 1(4): 268–270.
12. January K, Conway LJ, Deardorff M et al. Benefits and limitations of a multidisciplinary approach to individualized management of Cornelia de Lange syndrome and related diagnoses. Am J Med Genet C Semin Med Genet. 2016 Jun; 172 (2):237-45.

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[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 antisepsis. State of the art. Dermatology 1997; 195 Suppl 2: 3-9.

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

Unpublished article

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

Chapter in book

[7] Nauntofte B, Tenovuo 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.

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

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