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Original Research Article

Pediatric Education and Research
Volume 6 Number 3, September - December 2018
DOI: https://dx.doi.org/10.21088/per.2321.1644.6318.1

Severe Acute Malnutrition with Anemia and Vitamin Deficiency in Hospitalized Children: Two Centres Cross Sectional Study

Sunil Kumar Rao[1], Dhilip Kumar[2], Rakesh Kumar[3], Abhishek Abhinay[4]

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Abstract

Context: Severe acute malnutrition (SAM) is a major cause of morbidity and mortality in children under 5 years of age, especially in developing countries. Co-morbid conditions such as infections, micronutrient deficiencies and parasitic infestation are mainly responsible for this high mortality in SAM. *Aims:* To describe anemia and vitamin deficiency in children admitted with SAM at two tertiary level hospitals. *Settings and Design:* Observational cross sectional study, conducted at two centers. One centre is the Department of Pediatrics, Institute of Medical Sciences, Banaras Hindu University, and Varanasi and another is Department of Pediatrics, Shyam Shah Medical College Rewa MP. *Methods and Material:* Children between 6-60 months fulfilling the WHO criteria of SAM were enrolled. Cases were divided into edematous and non edematous groups. We collected data on demography, anthropometry, history and clinical examination including sign of vitamin deficiency. Investigations included CBC, general blood picture, serum electrolytes and calcium, serum albumin, blood sugar. Children were managed as per WHO SAM protocol. *Statistical analysis used:* The SPSS version 18 was used for data analysis. A p value <0.05 was considered significant. *Results:* Hundred four patients from MP group and 109 patients from UP group were finally enrolled in the study in which 98 (94.2%) patient out of 104 from MP and 77 (60%) patients out of 109 from UP less than 36 months. In this study male are more affected than female in UP group, male to female ratio is 1.79:1 as (64.2% verse 35.7%). In MP group SAM is more prevalent in lower middle class 51% as compared to upper lower class 31.7%. However in UP groups under nutrition is more common in upper lower 34.8% than lower middle 28.4%. We found out that, Vitamin B deficiency was most common (45.87%) followed by Vitamin A (16.5%) and most common deficiency sign were cheliosis, angular stomatitis and conjunctival xerosis in UP group while in MP group also most common Vitamin B deficiency (18.2%) followed Vitamin A deficiency (8.6%). *Conclusions:* Anemia and dermatitis are significantly coexist with edematous SAM children at both centers and vitamin deficiencies was frequently seen in edematous SAM children in UP.

Keywords: Severe Acute Malnutrition; Anemia; Vitamin Deficiency.

Introduction

Severe acute malnutrition is a major cause of morbidity and mortality in children under 5 years of age, especially in developing countries. It often has an intercurrent illness, and these co morbidities may have direct effects on cardiovascular physiology and carry a high risk of mortality in themselves It is supposed to be responsible for more than one million child death yearly worldwide [1]. Co-morbid conditions such as infections, micronutrient deficiencies and parasitic infestation are mainly responsible for this high mortality in SAM [2]. It results in the various path physiological changes in the body systems including significant changes in haematological parameters. Low red cell counts resulting in anemia has always been a constant feature of protein energy malnutrition and may be normochromic normocytic, microcytic hypochromic or macrocytic [3,4]. White cell changes demonstrate the synergistic relationship which SAM has with infections and thymic atrophy [5]. So, we are comparing the prevalence of anemia and multivitamin deficiency at two centre child admitted with SAM.

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Received on 06.12.2018, **Accepted on** 24.12.2018

Materials and Methods

This is an observational cross sectional study, conducted at two centers. One from September 2016 to May 2018 in the Department of Pediatrics, Institute of Medical Sciences, Banaras Hindu University, and Varanasi and another at Department of Pediatrics, Shyam Shah Medical college Rewa MP. Inclusion criteria were children fulfilling the WHO criteria of SAM. Cases were divided into edematous and non edematous groups. We collected data on demography, anthropometry, history and clinical examination including sign of vitamin deficiency. Investigations included CBC, general blood picture, serum electrolytes and calcium, serum albumin, blood sugar. Children were managed as per WHO SAM protocol. The SPSS version 18 was used for data analysis. A p value <0.05 was considered significant.

Results

Demographical data as shown in table 1 there were 109 children with SAM from UP and 104 from MP. Out of these, 28 (26.9%) children had edema in MP group which is almost one third in comparison to non edematous group although there is equal incidence of edema (50%) in case of UP group children.

There is no any significant difference was found in age group, gender in both the centers although little higher incidence was found in lower middle class than upper lower class in both the groups but data was comparable socioeconomic status incidence.

Table 2 depicts there is high incidence of severe anemia (Hb<7g/dl) was found in UP (p=0.01). In MP out of 28 edematous children 42.8% had anemia and 30.2% in case of non edematous group. In UP out of 55 edematous children 52.7% had anemia in comparison to nonedematous children 31.4%. In both the group edematous children had more anaemia. It was also found that anemia is more common in non edematous children in MP and more in edematous children in UP.

Table 3 show there is 61 (55.9%) children had skin changes in UP out of 109 in comparison to UP 24% (25 children out of 104) that is significant difference in skin changes (p=<0.001) in both the group.

In multivitamin deficiency, both B complex and vitamin A is more deficient in UP group in edematous and nonedematous patient although data is comparable except that there is higher zinc deficiency was found in MP group in both edematous and nonedematous patients.

Table 1: General characteristics of study population

Variable Edematous (n=28)		Shyam Shah Medical College, MP (n=104)			IMS BHU, UP (n=109)		
		Non Edematous (n=76)	Edematous (n=55)		Non Edematous (n=54)		
Age groups (months)	6-12	14	48	P=0.325	12	19	p=0.07
	13-36	13	23		29	17	
	37-60	1	4		14	18	
Gender	Male	10	36	P=0.20	36	34	p=0.94
	Female	18	40		19	20	
Socioeconomic status	Upper lower (IV)	16	17	P=0.59	15	23	p=0.06
	Lower middle (v)	29	22		5	26	

Table 2: Comparative analysis of anemia in study populations

Haemoglobin (Hb) (g/dl)	MP (n=104)		UP (n=109)		p value
	Edematous (n=28)	Non Edematous (n=76)	Edematous (n=55)	Non Edematous (n=54)	
<or= 7	12	23	29	17	0.01
7.1 to 11	15	51	20	13	0.005
>11	1	3	6	14	1

Table 3: Comparative analysis of vitamins and Skin changes in study populations

Clinical signs	MP (n=104)		UP (n=109)		P value
	Edematous (n=28)	Non Edematous (n=76)	Edematous (n=55)	Non Edematous (n=54)	
B complex	6	13	28	22	0.06
Vitamin A	4	5	11	7	0.447
Zinc	5	11	3	7	1
Skin changes	8	17	49	12	<0.001

Discussion

Hundred four patients from MP group and 109 patients from UP group were finally enrolled in the study in which 98 (94.2%) patient out of 104 from MP and 77 (60%) patients out of 109 from UP less than 36 months. Sharma [6] in their study also reported that prevalence of malnutrition is significantly high in children less than 24 months of age. Similarly, Mamidi et al. [7] in their study on hospital based treatment of severe malnutrition reported that 71.1% of children were below 24months of age. It is mainly due to rapid growth pattern and increased requirement of substrates for energy and building of tissue in initial 2-3 year age, thus deficiency of protein, energy and other micronutrients in these year results in malnutrition.

In this study male are more affected than female in UP group, male to female ratio is 1.79:1 as (64.2% verse 35.7%). Similar to this, Ashraf et al. [8] reported that malnutrition is relatively more common in males as compared to that of females (53.7% v/s 46.3%) and also Aneja et al. [9] found similar finding in their study on malnutrition observed that 55.5% of children were males as compared to females (44.5%). However in MP group we found female were more than male (55.7% v/s 44.2%) with a ratio of 1.26:1. Almost similar finding to Joshi et al. [10] observed that incidence of malnutrition was higher in females (78%) as compared to that in males (22%). Singh et al.[11] and Rao et al.[12] reported that extent of malnutrition was significantly higher in girls than boys.

In MP group SAM is more prevalent in lower middle class 51% as compared to upper lower class 31.7% as similar to Ashraf et al. [12] in their studies reported that majority of malnourished children belonged to lower socioeconomic status (IV and V) i.e. 72.8% and 90% respectively. However in UP groups under nutrition is more common in upper lower 34.8% than lower middle 28.4%. This is similar to Wagstaff and Watanabe [13] found inverse relation between underweight and socioeconomic inequality

In present study anemia was found in 97.1% in MP group and 72.4% in case of UP group which is higher than 51% from Columbia as reported by Bernal C et al. 2008 [14]. It was further observed that children with SAM was having 29.1% severe anemia UP group and 23.1% MP group in present study which is contrary to the study from Delhi as reported by Thakur et. al. [15]. This can be contributed to nutritional deficiency as majority of the patients had dietary deficiency.

We found out that, Vitamin B deficiency was most common (45.87%) followed by Vitamin A (16.5%) and most common deficiency sign were cheliosis, angular stomatitis and conjunctival xerosis in UP group while in MP group also most common Vitamin B deficiency (18.2%) followed Vitamin A deficiency (8.6%). Soni et al. [16] compared different vitamin deficiency between malnourished and normally nourished children and observed prevalence of vitamin A deficiency in (15.7% v/s 1.8%), vitamin B in (7.6% v/s o.4%), vitamin D in (11.9% v/s 2%) and vitamin C in (1.1% v/s o%) children. The different vitamins deficiency seen in SAM patients is because of lack of adequate nutritious food intake and the food which is taken have very low amount of vitamins and minerals making them deficient in the required vitamins. Incidence of zinc deficiency is more in MP (15.3%) than in UP (9.1%). Limitation of present study is we could not estimate the serum level of vitamins and zinc.

Conclusion

Anemia and dermatitis are significantly coexist with edematous SAM children at both centers and vitamin deficiencies was frequently seen in edematous SAM children in UP.

Key Messages

Edematous SAM children have more vulnerable for vitamins deficiencies and skin manifestations. Hence we recommend skin, mucosa and eye care has integral part of management of SAM.

Acknowledgement: Nil

Conflict of Interest: Nil

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Original Research Article

Pediatric Education and Research
Volume 6 Number 3, September - December 2018
DOI: https://dx.doi.org/10.21088/per.2321.1644.6318.2

The Prevalence of Anemia and Severity in Tribal Versus Non-Tribal School Going Children of Mysore District, India

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Abstract

Background: An estimated 30 per cent of the world's population is anaemic, with the global prevalence among 6-12 years children to be 36 percent. Anaemia prevalence is higher in developing countries than developed countries. *Objectives:* Our aim was to estimate haemoglobin levels in tribal and non-tribal school going children [5-10 years] of Mysore and to compare the prevalence and severity of anemia between them. *Study design and settings:* This is a cross sectional community based study. *Methods:* School going children aged 5-10 years belonging to the tribal (Jenukuruba) and non-tribal belt of H.D. Kote Taluk, Mysore, were included. The sample size was estimated to be a minimum of 457 in each group. Hemoglobin estimation, clinical examination and anemia grading was done in all. *Results:* Out of 497 Jenukuruba tribal children, 89.5% of them were diagnosed as anemic, while only 63.6% were anemic among the Non tribal children [p<0.0001]. The mean Hemoglobin of tribal children was 8.62g/dl while that of non-tribal children was 10.94g/dl [p<0.0001]. Prevalence of anaemia was more among the girls than boys in tribal children., whereas anemia was more among boys than in girls in non-tribal children. While 22.54% of tribal-children had severe anemia, only 2.8% were severely anemic among non-tribal children. There is a correlation between the BMI and Hemoglobin. *Conclusions:* The prevalence of anemia in the present study subjects was higher than the national prevalence. There is need to improve the nutritional status of the children. Good training should be provided for the health workers to detect pallor and to take appropriate action.

Keywords: Anemia; Tribal; Non Tribal; Jenukuruba; BMI.

Introduction

Tribal people in Karnataka constitutes 6.95% of the total population of the state [1]. The total number of tribal people recognized by the Government in Karnataka is about 42, 48,978 [1]. The population of *Jenu Kurubas* is 36,076 in Karnataka mostly living in the districts of Mysore, Kodagu, and Chamarajanagar [1]. Tribal populations have been neglected since many generations and along with this, associated poverty has left them in poor state of health. Many practices such as late initiation of breastfeeding, no feeding of colostrum, faulty weaning practices, lack of access to health services, illiteracy, unhygienic personal habits accounts for the poor health of the tribes [1]. In developing countries, as such anemia and malnutrition form major public health problems among the school age children [2]. An estimated 30 per cent of the world's population is anaemic, with the global prevalence of anaemia among 6-12 year old children to be 36 percent [3]. Anaemia is a nutritional problem and its prevalence is higher in developing countries than developed countries [4]. Unfortunately, despite efforts from the government and non-governmental organizations to take primary health care to these marginalized people, there has been a very limited number of studies reported on the health status of the tribal communities of the Karnataka State [1]. Therefore this study was undertaken to compare the prevalence and severity of anemia between tribal and non-tribal school going children.

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Received on 18.10.2018, **Accepted on** 22.10.2018

Material and Methods

This is a cross sectional community based exploratory study.Children aged between 5-10 years going to the primary school and belonging to the tribal (Jenukuruba) and non-tribal belt of H.D. Kote Taluk, Mysore, were included. Inorder to estimate sample size, the prevalence of anemia as reported by Prabhakar and Gangadhar were utilized [5]. Knowing that prevalence of anemia is 78%, with 95% confidence level and 5% allowable error, the required sample size was estimated to be a minimum of 457 in each group. About 500 children, each from the tribal and non-tribal population were selected randomly formed the study group. Consent was taken from the parents and school teachers. Tribal children belonging other than Jenukuruba tribes were excluded. Four Government tribal schools and four private schools were selected. The selection of schools was done by random sampling procedure (Lottery method). JSS university ethical committee clearance was taken. Hemoglobin estimation and relevant clinical examination were done in all students. The blood sample was obtained by finger prick and were analysed for hemoglobin estimation by Cyanmethaemoglobin method of Dacie and Lewis. Anemia grading was done according to Kraemer and Zimmermann as mild (10-11.5 g/dl), moderate (7-9.9 g/dl) and severe anemia (<7.0 g/dl) [6].

Statistics: Summary statistics was done by calculating proportions, Mean, Standard Deviation, Coefficient of Correlation. Inferential statistics was done by using the Chi-Square Test.

The Chi-Square test is used to find the association between two caterogical variables of interest, Independent-t Test. To compare the mean of two independent groups Pearson's correlation was used. The data entry was done in Microsoft excel sheet and all the statistical analyses was carried out using SPSS.13 software.

Results

In the Tribal group, 497 (247 male+250 female) children and in the non-tribal group 500 children (246 male+254 female), were studied. So, a total of 997 cases formed the study subjects [Table 1]. Out of 497 Jenukuruba tribal children, 89.5% of them were diagnosed as anemic, while only 63.6% were anemic among the Non tribal children [Table 2].

Among the tribal children, 22.54% of them had severe anemia (Hb<7g/dl), while only 2.8% were severely anemic among the non-tribal children. Moderate anemia was found in 58.55% of the tribal children as opposed to 26% among the non-tribal children. Mild anemia was detected in 8.45% of the tribal children when compared to 34.8% among the non-tribal children [Table 2].

Table 1: Age and gender wise distribution of tribal and non tribal children

		Tribal children Number (%) 497 (100)	Non tribal children Number (%) 500 (100)	p value
Age group in years	5--5.99	99(19.9)	97(19.4)	0.99
	6--6.99	100(20.1)	100(20.0)	
	7--7.99	98(19.7)	100(20.0)	
	8--8.99	100(20.1)	104(20.8)	
	9--9.99	100(20.1)	99(19.8)	
Gender	Male	247(49.7)	246(49.2)	0.99
	Female	250(50.3)	254(50.8)	

Table 2: Distribution of anemic children among tribal and non tribal children

		Tribal children Number (%)	Non tribal children Number (%)	p value
Anemia status	Mean Hb ±S D	8.62 ±1.97	10.94 ±1.98	<0.0001
	Not anemic	52(10.5)	182(36.4)	
	Anemic	445(89.5)	318(63.6)	<0.0001
According to Classification of anemia	Mild	42(8.45)	174(34.8)	
	Moderate	291(58.55)	130(26)	<0.0001
	Severe	112(22.54)	14(2.8)	
Anemia according to gender	Male	211(85.43)	169(68.70)	
	Female	234(93.60)	149(58.66)	<0.0001

The mean Hemoglobin of Tribal children was 8.62g/dl while that of non-tribal children was 10.94g/dl [p<0.0001]. Prevalence of anaemia was more among the girls than boys in tribal children. Whereas anemia was more among boys than in girls in non-tribal children [Table 2]. The difference in the prevalence of anemia between the tribal and non-tribal children according to gender and age specific groups is shown in the Table 3. The most common sign of anemia was pallor among both the tribals (61.22%) and the non tribals (33.6%). Other common findings among the tribal children were platynychia (55.1%), chelosis (51.22%), Koilonychia (47.55%), bald tongue (44.69%), glossitis (39.39%), and angular stomatitis (25.1%) which were less common among the non tribals [Table 4]. The mean BMI of Tribal children as 13.3 while the mean BMI of non-tribal children were 16.02. [p<0.0001]. There is a correlation between the two independent variables i.e. BMI and Hemoglobin [Graph 1].

Table 3: Distribution of anemic children according to age groups and gender

Age group in years	Anemia in males			Anemia in females		
	Tribal	Non tribal	p value	Tribal	Non tribal	p value
5-5.99	41(83.6)	32(68.09)	0.07	45(90)	30(60)	0.001
6-6.99	44(88)	28(56)	<0.0001	46(92)	20(40)	<0.0001
7-7.99	42(87.5)	35(70)	0.035	48(96)	22(44)	<0.0001
8-8.99	43(86)	32(64)	0.011	48(96)	35(64.8)	<0.0001
9-9.99	41(82)	42(85.7)	0.6	47(94)	42(84)	0.11
Total	211(85.4)	169(68.7)	<0.0001	234(93.6)	149(58.66)	<0.0001

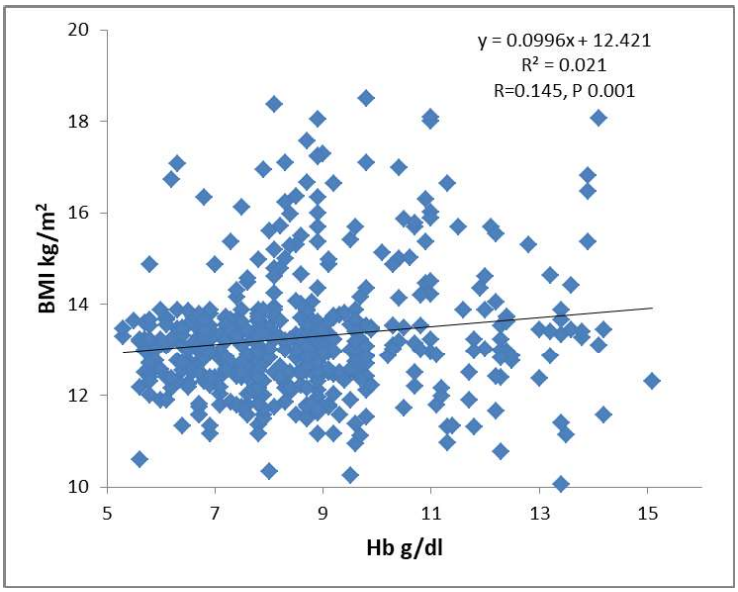
Table 4: Comparison of clinical signs between tribal and Non tribal children.

Clinical signs	Tribal children N=490*		Non tribal children N=500		P Value
	Number	%	Number	%	
Pallor	300	61.22	168	33.6	<0.0001
Glossitis	193	39.39	19	3.8	<0.0001
Gingivitis	32	6.53	0	0	<0.0001
Platynychia	270	55.10	100	20.0	<0.0001
Koilonychia	233	47.55	11	2.2	<0.0001
Bitot's spot	66	13.47	8	1.6	<0.0001
Cheliosis	251	51.22	140	28	<0.0001
Conjunctival Xerosis	124	25.31	110	22.0	0.2
Stomatitis	123	25.10	74	14.8	<0.0001
Bald tongue	219	44.69	74	14.8	<0.0001

*7 children were not co-operative

Table 5: Comparison of the present study with other similar studies in the tribal children

Similar tribal children studies		Anemia (%)	Common Gender	Mild (%)	Moderate (%)	Severe (%)
Vyas and Choudhry, 2005, 6-12 years, Rajasthan Tribes (8)		93.7	----------	0.6	60,	32.9
Rao and Vijay, 2006 6-11 Years, Bihar Tribes (2)		78	----------	----------	--------	----
Sahu et al, 2007, 5-14 years, Orissa Tribes (9)		99	No difference	35.2	59.4	5.4
Prabhakar and Gangadhar 2009, 6-10 years, Karnataka Tribes(5)		77.7	Girls	26.2	36.5	14.8
Kumar et al, 2013, 6-12 years Haryana Schedule caste (10)		85.72	Girls	51.12	33.84	0.75
Bhise et al,2013,8 to 16 years, Maharashtra Tribes(11)		77.10	Girls	42.9	28.1	6.1
Devi and Devi,2013, 6-12 years, Manipur Schedule caste (12)	Boys	64.20	Boys	37.79	16.97	9.59
	Girls	55.02		34.57	14.87	5.58
Prabhakar & Gangadhar,2016 6-10 years, Karnataka Tribes (7).		91.4	Girls	7.2	74.3	9.9
Present, 5-10 years, Karnataka Tribes [Jenukuruba]		89.5	Girls	8.45	58.5	22.5



Graph 1: Scatter plot showing correlation between BMI and Hemoglobin in tribal children.

Discussion

According to WHO, if the prevalence of anaemia is morethan 40% at the community level then the problem is of high magnitude [4]. Anemia is associated with impaired growth, delay in development, behavioral abnormalities and impair cognitive functions in infants and children [7]. In general, tribal populations are considered to be under-privileged in India and anemia is more common in these underprivileged tribal population than the general population [5,7].

An estimated 30 per cent of the world's population is anaemic, with the global prevalence of anaemia among 6-12 year old children to be 36 percent [3]. We observed anemia in 89.5% of Jenukuruba tribal children in the age group of 5-10 years which is comparable to most of other tribal children in the same age group in India [Table 5]. However, the prevalence of anemia in the present study subjects was higher than the national prevalence. Few studies have shown more prevalence than our study in tribal children like the studies from Rajasthan and Orissa [8,9]. In contrast few reported less prevalence like that from Scheduled Caste School Children of Manipur [12].

We detected anemia in 64% of non-tribal children between the age group of 5-10 years, which is similar to study done by Sethi et al. (66.4%) from Delhi [13]. However, Gomber et al. observed anemia in 41.8% of children aged 5-11 years from urban slums [3]. Verma et al. showed that the prevalence of anemia in the 5-15 years age group of urban school children in Punjab as 51.5% [14]. Study from South India observed the prevalence of anemia in 52.88% of children in the age group of 8-16 years [15].

Eventhough we detected anemia in the age group of 5-10 years, similar observations has been made in the preschool and adolescent tribal children also. Haemoglobin estimation showed that 92.40% of scheduled caste preschool children suffereing from anaemia [16]. Philip et al. detected anemia in 95.7% of tribal preschool children in Wayanad district of Kerala [17]. Anemia was detected in 55% scheduled caste preschool children of Amritsar and Faridkot districts of Punjab [18]. Anemia was observed in 70.5% of adolescent girls of Scheduled Caste community of Amritsar [19].

The present study revealed that majority of tribal children were moderately anemic (58.5%) and 22.5% were of severe grade. In most of the studies anemia observed was either moderate or mild variety [Table 5]. Severe anemia was found in 22.5% of our tribal children which is comparable to a study of tribal children from Rajasthan [8]. However study among Jenukuruba tribal children like ours revealed lower prevalence severe anemia(14.86%) in a study by Prabhakar and Gangadhar [5]. We observed anemia in male tribal children more than females which is similar to the observation made in other studies [Table 5]. Whereas the prevalence of anaemia was 64.20% for boys as against 55.02% of girls in tribes of Manipur [12].

We detected anemia by clinical examination (pallor) in 61.2% of tribal children. Clinical examination showed that 95.1 per cent children were anemic in a study of tribal children from Rajasthan [8]. Authors suggest that the clinical examination

can be used to assess anemia as it requires less time, money and energy [8]. Verma et al. found clinical pallor in 44% of total children, while 51.5% were anemic. They opined that clinical assessment of anemia form the tip of iceberg and the true state can be assessed by estimation of Hemoglobin concentration in the blood [14]. In the current study platynychia, koilonychias, cheliosis and glossitis were observed among tribal school children in significant number. A study from Rajasthan also detected flat and plate nails (24.3%), atrophic lingual papillae (12.4%) and koilonychia (44.3%) in their tribal children [8].

There is a correlation between BMI and Hemoglobinin our tribal children. Study among Soliga tribes observed that the prevalence of anemia was higher in low weight children when compared to normal BMI children. They noticed that 94.3% of children of normal BMI had anemia [7]. Study from South India also showed that 51.3% children with normal BMI were found to be anemic [15]. Anemia was not significantly varied with BMI in a study involving Maharashtra tribal children [11].

Prabakar and Gangadhar attributed mainly cereal-based diet and less awareness of nutritional diet as the cause of anemia in soliga children [7]. Study from tribes of Orissa found that 93.7% children were taking food with low bioavailability of iron (5-10%) [9]. Another study from Mysore concluded that the cause for high frequency of anaemia among the Jenukuruba tribal children may be due to dietary inadequacy of iron, lack of safe drinking water and poor knowledge on the part of the mother regarding nutritional requirement of the children [5]. Study from Delhi urban slums revealed that pure or mixed iron deficiency anemia as the commonest type of anaemia and was noted in 68.42 per cent of school children [3].

Anemia can lead to cognitive disabilities in children. Hence an intervention to cure and prevent anaemia should be started for school age tribal children [8]. Sahu et al. suggested that reorientation of primary health care functionaries to cover the children under NNAPP with the help of ICDS workers and school authorities [9]. However a study from Bangalore, South India revealed low anemia prevalence in school-aged children (13.6%) and they attributed possible effect of school health initiative programmes like deworming and vitamin A supplementation [20].

Conclusion

Majority of children in our study (both tribal and non-tribal) were anemic. However, anemia was more common in tribal children when compared to non-tribal children. Mean BMI values were significantly low in tribal children and there was a correlation between BMI and Hemoglobin. Probably many factors like less availability of iron rich foods, good nutritious food, poverty and illiteracy play a role as the cause. There is need to improve the nutritional status of the tribal children. This can be done by periodic screening, regular deworming, health education, iron supplementation and proper implementation of national health programmes. Good training should be provided for the health workers to detect pallor and to take appropriate action.

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Original Research Article

Pediatric Education and Research
Volume 6 Number 3, September - December 2018
DOI: https://dx.doi.org/10.21088/per.2321.1644.6318.3

Thrombocytopenia after Phototherapy for Indirect Hyperbilirubinemia among Breastfed Term and Preterm Neonates

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Abstract

Objective: To compare the incidence of thrombocytopenia in breastfed term and preterm neonates with unconjugated hyperbilirubinemia receiving phototherapy. *Material and Methods:* A prospective study was done over a period of 5 months in NICU of a tertiary care hospital; 64 breastfed term and preterm (≥34 - <37 week) neonates with unconjugated hyperbilirubinemia requiring phototherapy were selected for the study after applying inclusion and exclusion criteria. Serum bilirubin and platelet count were sent on admission, after 24 hours and 48 hours of phototherapy. Phototherapy was administered according to AAP criteria, using LED phototherapy (10 bulbs) positioned within 15 - 20 cm of the patient's body. It was interrupted only for feeding and nursing for around 20 minutes every two to three hours. Platelet count and total serum bilirubin levels, before and after 24 and 48 hours of phototherapy were estimated. *Results:* After 24 to 48 hours of phototherapy, neonates showed statistically significant decrease in the platelet count; term (2.38 to 2.07 x 109/L, p = 0.0245) and preterm (2.27 to 1.96 x 109/L, p=0.0177), along with expected decrease of serum bilirubin levels. There was no association in the incidence of thrombocytopenia between the gestational age (term vs preterm, 6/32 vs 9/32 babies, p= 0.4741). *Conclusion:* Thrombocytopenia does occur following phototherapy in neonates. However it is clinically insignificant and asymptomatic. Gestational age of the neonate is not a factor associated with the incidence of thrombocytopenia after phototherapy in neonates with unconjugated hyperbilirubinemia.

Keywords: Thrombocytopenia; Indirect Hyperbilirubinemia; Phototherapy; Neonates; Platelet Count.

Introduction

Jaundice is a common problem in the first week of life of a newborn. Approximately 85% of all term newborns and most premature infants develop clinical jaundice [1]. In most cases, it is physiological and benign. However, in some neonates severe jaundice may lead to kernicterus with implications for future development. Treatment in the form of phototherapy and/or exchange transfusionis recommended. Human albumin or intravenous immunoglobulin [2] or fenofibrate [3] is also tried. Phototherapy has emerged as the most widely used form for the treatment of unconjugated hyperbilirubinemia.

Though phototherapy is relatively safe, Maurer [4] and Pishva [5] observed that neonates exposed to phototherapy suffer from thrombocytopenia. There is no consensus till now according to AAP (American Academy of Pediatrics) and NNF (National Neonatology Forum of India)guidelines, whether thrombocytopenia should be considered as a definite side effect of phototherapy or not. In 1966, Zieve [6] demonstrated effects of high intensity white light on human platelet *in vitro;* platelets which had been briefly exposed to light following photosensitization by hematoporphyrin lost the ability to aggregate and release potassium, acid phosphatase, serotonin and adenosine triphosphate. Electron photomicrographs of these altered platelets showed depletion of cytoplasmic materials and smooth membrane contours as compared to controls. Maurer [7] observed similar kind of platelet abnormalities within 96 hours of

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Received on 01.12.2018, **Accepted on** 14.12.2018

exposure in rabbits. It caused decrease in blood riboflavin levels and altered the excretory pattern of tryptophan metabolites, both of which are photosensitive. In the same study, phototherapy was also found to cause an increase in platelet production rate possibly secondary to reduction in platelet life span, and when bone marrow compensation was inadequate the platelet count fell [7].

Aims and Objectives

To compare the incidence of thrombocytopenia in breastfed term and pre term neonates after phototherapy.

Materials and Method

A prospective study was done at an NICU of a tertiary care hospital during five months study period (March 2015 – Jul 2015); approved by IRB (Institutional Review Board)/ HEC (Human Ethics Committtee). Unconjugated hyperbilirubinemia was defined as direct bilirubin less than 1.0 mg/dl if total bilirubin is less than 5 mg/dl or less than 20% of the total bilirubin if the total bilirubin is greater than 5 mg/dl. Preterm birth was defined as delivery before 37 completed weeks of gestation and term birth after 37 completed weeks of gestation. Thrombocytopenia was defined as platelet count <150 × 10^9/L. Mild, moderate and severe thrombocytopenia was defined as platelet counts between 100–150, 20–99 and < 20 × 10^9/L, respectively [8]. 64 breastfed neonates; 32 term and 32 preterm, with unconjugated hyperbilirubinemia requiring phototherapy for at least 48 hours were included (Figure 1).

Neonates who had direct hyperbilirubinemia, inborn error of metabolism, neonatal septicemia, congenital anomalies, and anti-platelet drugs given to baby or mother were excluded. Also, neonates who developed features suggestive of sepsis during the course of phototherapy, severe hyperbilirubinemia requiring exchange transfusion or addition of any other modality of treatment besides phototherapy were excluded from the study.

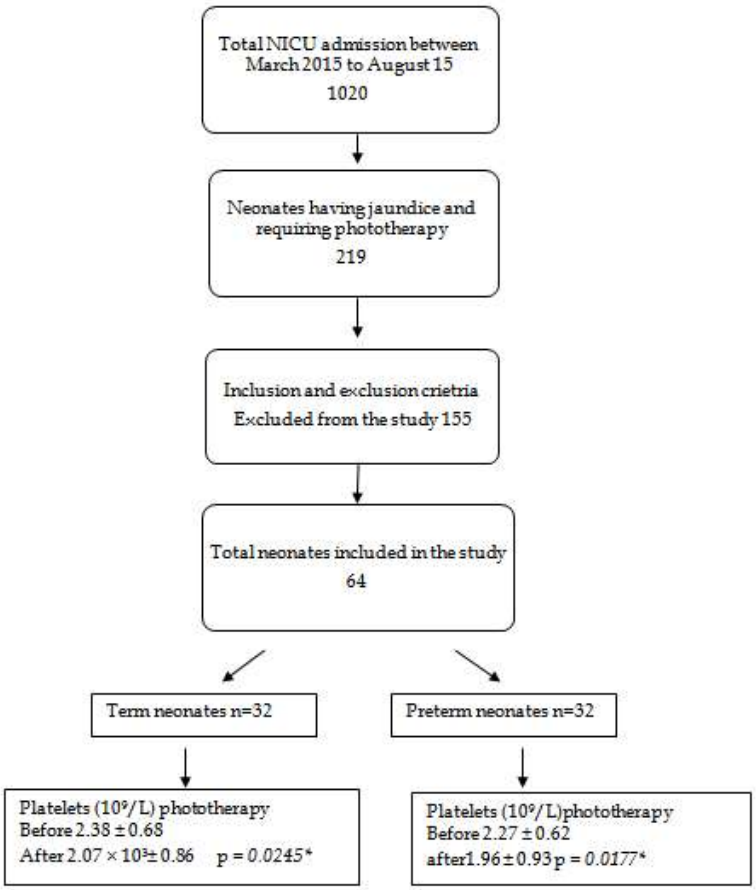

Fig. 1: Selection and outcome flow chart

*Chi square with Yates' correction

Informed written consent was obtained from parents or guardians. After history taking and clinical examination, baseline investigations like CBC, platelet count, serum bilirubin (total, direct, indirect), mother and baby blood grouping, direct and indirect Coomb's test, C- reactive protein, G-6 PD and blood culture were sent on the day of admission. Serum bilirubin and platelet count were sent on admission, after 24 hours and 48 hours of phototherapy. Phototherapy was administered according to AAP criteria for treatment of neonatal hyperbilirubinemia [9]. All neonates received phototherapy using LED phototherapy (10 bulbs) positioned within 15 - 20 cm of the patient's body. It was interrupted only for feeding and nursing for around 20 minutes every two to three hours.

Neonatal factors like sex, gestational age, birth weight, age at onset of jaundice and laboratory parameters like platelet count and level of serum bilirubin before starting phototherapy were compared between the two groups. Parameters like platelet count and incidence of thrombocytopenia after phototherapy were also recorded for both the groups.

Results

Thirty two (32) breastfed term neonates and 32 preterm neonates with unconjugated hyperbilirubinemia requiring phototherapy for at least 48 hours were selected for the study. There was no difference in sex and age at onset of jaundice in studied groups. The gestational age and birth weight were statistically different and comparable. There was significant difference in serum bilirubin level before starting phototherapy (Table 1).

Table 1: Neonatal parameters of study subjects at the start

	Term (32)	Preterm (32)	p value
Sex (M:F)	20:12	14:18	0.2104*
Gestational age (weeks)	38.2± 1	35.4± 1	<0.0001†
Birth weight (kg)	2.6±0.4	1.7±0.3	<0.0001†
Age at onset of jaundice	4.2±2.1	4.2±1.6	0.843†
Serum bilirubin (mg/dl)	19± 3.4	13.1±4.1	<0.0001*
Platelet count (x 109/L)	2.38± 0.68	2.26±0.62	0.468*

*Chi square with Yates' correction, † Unpaired t test

After the phototherapy, both term and preterm neonates showed a statistically significant change in the platelet count as compared to pretreatment level (Table 2).

Table 2: Changes in platelet count before and after phototherapy

	Term (32) Platelets (x 109/L)	Preterm (32) Platelets (x 109/L)
Before phototherapy	2.38 ± 0.67	2.26 ±0.62
After phototherapy	2.07± 0.86	1.96±0.92
p value	0.025	0.018
Thrombocytopenia	6 (18.75%)*	3 (9.38%)*

*p= 0.474

There was no association between the gestational age (term/preterm) and the incidence of thrombocytopenia (p=0.4741, Fisher's exact test).

Discussion

We studied 64 subjects, 32 breastfed term and 32 breastfed preterm neonates with unconjugated hyperbilirubinemia who were treated with phototherapy. Both the groups differed in gestational age and birth weight as per the study design. There was a significant difference in serum bilirubin level before starting phototherapy between the two groups as the cut-off value for starting phototherapy is lower in preterm babies. However, there was no significant difference in platelet counts before starting the treatment.

In our study, the incidence of thrombocytopenia after phototherapy was found to be 18.75% and 9.38% in term and preterm neonates respectively (Table 2), which is low as compared to previously reported incidence of 39.02% and 16.66% by Khera [10]. The term group had higher incidence of thrombocytopenia after phototherapy as compared to preterm neonates; however there was no statistical significance when compared for gestational age by Fisher's exact test. Similar observations were seen by Khera [10] and Pishva [5] in their study. None of the neonates with thrombocytopenia, in our (9/64) as well as other studies had clinical manifestations of bleeding or other complications [5,10]. The reason for the same could be that thrombocytopenia was transient and rarely severe in all the three studies.

Limitations of our study were that, maternal parameters like pregnancy induced hypertension, and flux of phototherapy were not recorded.

Conclusion

After phototherapy, both term (p = 0.0245) and preterm (p=0.0177) neonates showed statistically significant decrease in the platelet count as compared to pretreatment levels. However, there was no association between the gestational age (term/

preterm) and the incidence of thrombocytopenia (p=0.4741). Although thrombocytopenia occurs following phototherapy in both term and preterm neonates, it is not life threatening, or causes any clinical signs or symptoms.

What is Already Known?

Transient thrombocytopenia can occur following phototherapy for unconjugated hyperbilirubinemia in breastfed term and preterm neonates and this decreased platelet count is not associated with bleeding manifestations.

What this Study Adds?

Gestational age of the neonate is not a factor associated with the incidence of thrombocytopenia following phototherapy.

Recommendation

When evaluating a healthy term or preterm neonate undergoing phototherapy, for thrombocytopenia (only); treatment modalities like antibiotics (considering sepsis), or platelet transfusion are not recommended- based on decreased platelet counts only; until and unless other indications are present.

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Original Research Article

Pediatric Education and Research
Volume 6 Number 3, September - December 2018
DOI: https://dx.doi.org/10.21088/per.2321.1644.6318.4

Evaluation of Risk Factors and Prevalence of Bronchial Asthma in Children of North Karnataka Population

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Abstract

Ninety eight (98) school going children of both sexes (56 boys and 42 girls) aged between 6 to 14 years. 5(5%) were 6 years, 7 (7.1%) were 7 years. 8 (8.1%) were 8 years, 10 (10.2%) were 9 years, 12 (12.2%) were 10 years, 11 (11.2%) were 11 year 14 (14.1%) were 12 years. 13 (13.3%) were 13 years 18 (18.3%) were 14 years. The clinical manifestation was 11(11.2%) were asthmatic with obesity 13 (13.2%), had onset of asthma more than 2 years, 11 (11.2%) had relief with bronchodilators, 9 (9.1%) had nocturnal exacerbation of asthma, 13 (13.2%) had seasonal exacerbation, 8 (8.1%) had exercise induced asthma, 12 (12.2%) had trigger induced attacks, 11 (11.2%), a febrile episode 10 (10.2%), had family history asthma / atophy. The risk factors of prevalence of bronchial asthma 3 (3.06%), was cat 5 (5.10%), was dog 10 (10.2%) both (cat and dog), 80 (81.6%), had none. 38 (38.7%) had Smoke exposure, 60 (61.2%) had Dust exposure, 19 (19.3%) used electricity for cooking, 47 (47.9%) used LPG gas for cooking, 32 (32.6%) used open? for cooking. 38 (38.7%) were staying in open atmosphere, 60 (61.2%) were staying in crowd area, 20 (20.4%) were upper, 35 (35.7%) middle, 43 (43.8%) had lower socio-economic status. The study of bronchial asthma in children with different age group and different socio-economic status will certainly help the pediatrician to evaluate the different causes, risk factors and treat efficiently to prevent morbidity of mortality

Keywords: Bronchial; Pediatric; Genetic; Environmental; Asthma.

Introduction

Bronchial asthma is a major public health problem worldwide with wide differences in prevalence and severity throughout the world. Significant increases in the prevalence and severity has been noticed globally over the past few decades in certain geographical regions. Changes in the environmental factors, life style, genetic factors play vital role in the prevalence and aggravation of the symptoms [1,2]. As asthma is a chronic inflammatory disease of the airways causing episodes of airway obstruction this chronic inflammation increases airways hyper responsiveness to stimulants [3]. Asthma not only leads to hospitalization but also an important chronic condition causing school absenteeism. Asthma is generally considered a disease of developed countries and affluent societies in developing countries. But there is little information about the epidemiological trends of asthma in urban India especially in the middle and lower middle class society of India. Hence, attempt is made to study the children of different ages of middle and lower class of socio-economic status with respect to various clinical manifestations, aggravating factors, and to evaluate the risk factors of asthma.

Material and method

Ninety eight (98) school going children of both sexes 56 boys and 42 girls aged between 6 to 14 years attending the outpatient pediatric department of KBN Hospital, Kalburagi, Karnataka were selected

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Received on 13.10.2018, **Accepted on** 31.10.2018

for study. Majority of the children belonged to middle class socio - economically. The radiological, bio-chemical, pathology test were carried out to confirm the diagnose of bronchial asthma.

Pulmonary function test (spirometry) was done only in doubtful cases to confirm bronchial asthma. The patients were classified as per their age, sex and as per their risk factors. The duration of the study was about 2 years (2015 to 2017).

Tuberculosis, chronic lung diseases (bronchiectasis, interstitial lung diseases, preterms, congenital heart disease) children were excluded from the study.

Observation and Results

Table 1- Age wise distribution study among children with bronchial asthma. 5 (5%) children were 6 years old, 7 (7%) were 7 years old, 8 (8.1%) were 8 years old 10 (10.2%) were 9 years old, 12 (12.2%) were 10 years old, 11 (11.2%) were 11 years old, 14 (14.2%) were 12 years old, 13.(13.2%) were 13 years old, 18(18.3%) were 14 years old.

Table 2- Gender wise classification of bronchial asthma in children was Boys 56 (57.1%), girls 42 (42.8%)

Table 3- clinical manifestation of bronchial asthma in children was 11 (11.2%) had asthma with obesity, 13 (13.2%) had onset of asthma more than 2 years, 11 (11.2%) had relief with bronchodilators, 9 (9.1%) had nocturnal exacerbations 13 (13.2%) had seasonal exacerbation 8 (8.1%) had exercised induced asthma, 12 (12.2%) had trigger induced attacks. 11 (11.2%) had Afebrile episodes, 10 (10.2%) had family history of asthma/atophy.

Table 1: Age wise distribution of children with bronchial asthma

(Total No of patients- 98)

Sl no	Age	Number	Percentage
1	6	5	5.1
2	7	7	7.1
3	8	8	8.1
4	9	10	10.2
5	10	12	12.2
6	11	11	14.2
7	12	14	14
8	13	13	13.2
9	14	18	18.3

Table 2: Gender-wise distribution of children with bronchial Asthma

(Total No of patients- 98)

Sl no	Particulars	Number	Percentage
1	Boys suffering with bronchial asthma	56	57.1
2	Girls suffering with bronchial asthma	42	42.8

Table 3: Clinical manifestation of bronchial asthma children

(Total No of patients- 98)

Sl no	Presentation	Number	Percentage
1	Asthma with obesity	11	11.2
2	Age of onset >2 years	13	13.2
3	Relief with bronchodilators	11	11.2
4	Nocturnal exacerbations	9	9.1
5	Seasonal exacerbation	13	13.2
6	Exercise induced	8	801
7	Trigger induced attacks	12	12.2
8	Afebrile episode	11	11.2
9	Family history of asthma / atophy	10	10.2

Table 4: Risk factors for prevalence of bronchial asthma among children

(Total No of patients 98)

Risk factors	Particulars	Number	Percentage
1 pet	a-cat	3	3.06
	b-Dog	5	5.06
	c-both	10	10.2
	d-None	80	81.6
2 Exposure	Smoke	38	38.7
	Dust	60	61.2
3 fuel used for cooking	Electricity	19	19.3
	LPG gas	47	47.9
	Open fire	32	32.6
4 location	Open	38	38.7
	Crowd	60	61.2
5 socio economic status	Upper	20	20.4
	Middle	35	35.7
	Lower	43	43.8

Table 4- Risk factors for prevalence of bronchial asthma in 1-3 (3.06%) children were cat, 5 (5.10%) were dogs, 10 (10.2%) were both cats and dogs.

2- Risk factors of exposure to smoke in 38 (387), 60 (61.2%) were dust.

3- risk factors of bronchial asthma due to fuel used for cooking 19 (19.3%) was electricity 47 (47.9%) was gas (LPG), 32 (32.6%) was open fire.

4- Location of residency – 38 (38.7%) was open, 60(61.2%) was crowded location.

5- socio- economic status - 20 (20.4%) were upper social society 35 (35.7%) were middle, 43 (43.8%) were lower social status children.

Discussion

The present study of risk factors for bronchial asthma in children of north Karnataka population 5 (5.1%) were 6 years age, 7 (7.1%) were 7 years age, 8 (8.1%) 8 years age 10 (10.2%) 9 years age, 12 (12.2%) were 10 years age, 11 (11.2%) were 11 years age 14 (14.2%) were 12 years age 13(13.2%) were 13 years age 18 (18.3%) were 14 years age (Table 1) 56 (57.1%) boys, 42 (42.8%) were girls suffering with bronchial asthma (Table 2) The clinical manifestation of bronchial asthma. 11 (11.2%) children were obese, 13 (13.2%) had on set of asthma, more than 2 years, 11 (11.2%) got relief with bronchodilators 9 (9.1%) nocturnal exacerbation of asthma, 13 (13.2%) had seasonal exacerbation 8 (8.1%) had exercised induced asthma, 12 (12.2%) had trigger induced attacks, 11 (11.2%) had febrile episode, 10 (10.2%) had family history or atophy. These findings were more or less in agreement with previous studies [4,5,6]. Common asthmatic symptoms are wheezing, recurrent coughing, breathlessness [7,8]. It is reported that overcrowding, pollution, poverty, passive (secondhand) smoking, lack of awareness and proper facilities, poor perception of symptoms, social stigmatization, are the factors for prevalence and aggravating factors [9]. Moreover, genetic factors and nutritional status of the children also play a vital role in prevalence of bronchial asthma [10]. Because asthma develops due to interaction between gene and environment hence, parental history of atopy is an index of susceptibility to asthma. A good nutritional status creates immunity which prevents the risk of severity, morbidity and mortality of asthmatic patients.

Summary and Conclusion

The present study of prevalence and assessment of risk factors of bronchial asthma in children of north Karnataka population has encompassed various factors of prevalence and aggravating factors for bronchial asthma in both sexes of different ages in the children. Apart from medication, hygienic atmosphere, nutritious diet is must to overcome the severity and exacerbation of asthma. This study will be quite useful to pediatrician to treat such children with various clinical manifestation but this study demands further genetic, nutritional, patho-physiological, bio-mechanical study to know the mechanism of alveoli and surfactant cells, and broncho-alveolar functions because little is known about the causes of bronchial asthma.

This research paper is approved by ethical committee of KBN institute of medical sciences kalaburgi-585102 (Karnataka).

Conflict of Interest: No

Funding: No.

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Original Research Article

Pediatric Education and Research
Volume 6 Number 3, September - December 2018
DOI: https://dx.doi.org/10.21088/per.2321.1644.6318.5

Outcome of Convulsive Status Epilepticus in Children: A Cross Sectional Study

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Abstract

Background: Convulsive status epilepticus (CSE) is the most common neurological emergency in children and often associated with poor outcome. A prospective cross sectional study was done on 193 north Indian children with CSE, aged one month to 15 years presented in pediatric emergency with primary objective was to record the time taken to control seizure and secondary objective was to find out etiology, laboratory changes, neuroimaging findings. *Methods:* Children (1 month to 180 months) were recruited in study as per protocol. Seizure was managed as per hospital and Indian Academy of paediatrics protocol. Data was entered in predesigned proforma and analyzed for outcomes. *Results:* The mean age of children was 56.7±47.1 months, where as male: female ratio was 2.3:1. Seventy four (38.3%) children belonged to the age group 61-180 months, followed by 13-60 months (34.7%) and 1-12 months (26.9%). Fever; 87 (45.1%) was most common associated symptom followed by vomiting (29.5%), headache (4.7%), and diarrhoea (3.1%). Seizure was clinically controlled (Group-I) in 149 (77.2%), partially controlled (Group-II) in 25 (12.95%), and 19 (9.84%) patients had died (Group-III). The mean duration of CSE at presentation was significantly higher in Group-II and group-III. The mean time taken to control seizure in group-I was 49.7±54.1 minutes, group-II; 135.8±77.7 minutes and in group-III; 118.21±97.5 minutes respectively. The most common CT-scan finding was meningoencephalitis 43 (22.3%), followed by tubercular meningitis 34 (17.6%), bacterial meningitis 33 (17.1%), neurocysticercosis 19 (9.8%) and cerebral infarction (5.2%). CSE was controlled with lorazepam and phenytoin in most of children. *Conclusion:* The duration of CSE at presentation, number of seizure episodes, time taken to control status epilepticus and etiology were the important factors for outcome of status epilepticus.

Keywords: Convulsive Status Epilepticus (CSE); Generalized Tonic Clonic Seizure; Partial Seizure; Prolonged Febrile Seizure; Lorazepam; Phenytoin; Midazolam; Sodium Valproate.

Introduction

Convulsive status epilepticus (CSE) is a common neurological emergency in children, and if continued for long duration may leads to death or long term neurological sequelae [1,2]. CSE requires early expedient management to decrease morbidity and mortality. The incidence of convulsive status epilepticus (CSE) in children range from 10 to 38/100000/year [3,4]. Incidence of status epilepticus in an exclusively pediatric population based study in North London was 18-20/100000/ year. The incidence was highest among children below 1 year of age [5].

In patients younger than 16 years, the most common cause of status epilepticus was fever and/or infection (36%), whereas in adults, this accounted for only 5%. Cerebrovascular disease accounts approximately 3% of status epilepticus in children as against 25% in adults [6]. Shinnar et al. reported more than 80% of status epilepticus in children younger than 2 years and were febrile or acute symptomatic in origin [7]. In contrast, cryptogenic and remote symptomatic causes were more common in older children.

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Received on 28.11.2018, **Accepted on** 14.12.2018

CSE is associated with various biochemical and hematological changes in blood and include hyperglycemia or hypoglycemia and acidosis (both metabolic and respiratory). Acidosis usually resolves with termination of seizure. Aminoff et al., reported metabolic acidosis and mild leukocytosis in blood and cerebrospinal fluid in some patients with status epilepticus [8].

Neuroimaging has significant contribution in the diagnosis and treatment of patients with CSE. It is even more important for those patients who have medically intractable seizures [9]. MRI is the preferred imaging technique but CT scans has a number of advantages such as lower cost, scan speed, ready accessibility, and easy use, which provide a relatively reliable imaging modality for most patients [10]. It can accurately detect hemorrhage, infarctions, gross malformations, ventricular system pathologies, and lesions with underlying calcification. The sensitivity of CT in patients with epilepsy is not higher than 30% in unselected populations [11].

Prognosis of CSE has strong correlation with the underlying disease. It is poor, when CSE had presented with coma and caused by anoxia/hypoxia. Mortality rates related to CSE have decreased over the last 60 years, probably in relation to faster diagnosis and more aggressive treatment. The probability of death is closely correlated with age [12].

The present prospective cross-sectional study was done with primary objectives to record the time taken to control seizure and secondary objective was to find out etiology, laboratory changes, neuroimaging findings.

Methods

This study was carried out in department of Pediatrics, Institute of Medical Sciences, Banaras Hindu University between September 2011 to April 2014 on 193 patients presented and admitted with convulsive status epilepticus (CSE). Ethics committee of the institute approved the study protocol.

Convulsive status epilepticus was defined, for the purpose of the study, as a single clinical seizure lasting more than 30 minutes or repeated seizures over a period of more than 30 minutes without intervening recovery of consciousness, but for practical standpoint, treatment was considered by 5–10 minutes, or when at least two seizures have occurred back-to-back without an intervening return to consciousness.

Inclusion Criteria

The study population included all infants and children aged above 1 month to 15 years admitted in pediatric emergency, ward or pediatric intensive care unit with CSE. The enrolled patients were treated as per recommended protocol of Indian Academy of Paediatrics.

Exclusion Criteria

Patients with hepatic or uremic encephalopathy with convulsive status epilepticus were excluded from study.

Complete blood count, blood glucose, serum calcium, arterial blood gas analysis, cerebrospinal fluid study, renal function tests and imaging (CT-scan/MRI-scan or both) of cranium were done in all patients. Tandom mass screening (TMS) for metabolic diseases was done as needed to find out etiology. Findings of enrolled patients were recorded in a pretested performa and collected data was analyzed by SPSS version [16].

Results

One hundred ninety three (193) children with CSE, presented to pediatric emergency and admitted in paediatrics ward were taken for this prospective hospital-based observational study. Children were divided in three groups based on control of seizure activity: Group-I: Seizure controlled (149), Group–II: Seizure uncontrolled (25) and Group-III: death (19) patients.

The basic characteristics of enrolled children in the study are mentioned in Table 1. The mean age of the children was 56.71±47.1 months (1 month to 15 years). The maximum number of children belonged to the age group 61-180 months i.e. 74 children (38.3%) followed by 13-60 months (34.7%) and 1-12 months (26.9%). In the present study 69.9% were male, and male to female ratio was 2.3:1.

They were further categorized into three groups: Status of generalized tonic clonic seizure (GTCS), status of partial seizure, and status of febrile seizure. 164 (85%) had status of GTCS, 24 (12.4%) had status of partial seizure, and 5 (2.6%) had status of febrile seizure. Eighty nine (46.1%) children had presented with history of more than 4th seizure episode, 14 (7.3%) had 4th episode, 24 (12.4%); third episode, 29 (15%); second episode, and 37 (19.2%); first episode. Fever (45.1%) was most common associated symptom, followed by fever with vomiting (29.5%), fever with headache (4.7%) and

fever with diarrhoea (3.1%). The family history of seizure was present in 11 (5.7%) children.

The hematological and biochemical changes in children with status epilepticus are mentioned in Table 2. Of 193 children, 6 children had died before their CT-scan, 72 (37.3) had normal study. The most common CT-scan finding was meningoencephalitis 35 (18.1%), followed by tubercular meningitis with hydrocephalus; 27 (13.9%), neurocysticercosis 19 (9.8%), cerebral infraction 10 (5.2%), and hypoxic ischemic 6 (3.1%). Other less common findings are mentioned in Table 3.

Table 1: Basic characteristics of children with Status epilepticus

	Group-I (n=149) (Mean±S.D.)	**group-II (n=25) (Mean±S.D.)**	**group-III (n=19) (Mean±S.D.)**	**Total (%) (Mean±S.D.)**
Age (Mean±S.D.)	56.7±47.2	60.6±49.1	51.4±45.1	56.7±47.1
1month-12monthS	41(78.8)	7(13.5)	4(7.7)	52(26.9)
13months-60months	51(34.2)	7(10.4)	9(13.4)	67(34.7)
>60 months	57(77)	11(14.9)	6(8.1)	74(38.3)
Sex: Male	104(77)	18(13.3)	13(9.6)	135(69.9)
Types of S.E.				
S.E of GTCS	131(79.9)	16(9.8)	17(10.4)	164(85)
Partial S.E	13(54.2)	9(37.5)	2(8.3)	24(12.4)
Febrile SE	5(3.4)	0(0.0)	0(0.0)	5(2.6)
Episode of S.E				
First episode	34(22.8)	2(8)	1(5.3)	37(19.2)
Second episode	24(16.1)	2(8)	3(15.8)	29(15)
3rd episode	22(14.8)	1(4)	1(5.3)	24(12.4)
4th episode	10(6.7)	2(8)	2(10.5)	14(7.3)
>4th episode	59(39.6)	18(72)	12(63.2)	89(46.1)
Associated symptoms				
Fever	72(48.3)	6(24)	9(47.4)	87(45.1)
Fever+vomiting	41(27.5)	8(32)	8(42.1)	57(29.5)
Fever+headache	6(4)	2(8)	1(5.3)	9(4.7)
Fever+vomiting+headache	7(4.7)	0(0.0)	1(5.3)	8(4.1)
Fever+vomiting+diarrhea	3(2)	0(0.0)	0(0.0)	3(1.6)
None	17(11.4)	5(20)	0(0.0)	22(11.4)
Family history of seizure	8(5.6)	2(8)	1(5.3)	11(5.7)

Table 2: Laboratory findings in children with status epilepticus (n=193).

	Group-I (n=149) (mean±S.D.)	group-II (n=25) (mean±S.D.)	group-III (n=18) (mean±S.D.)	p-value		
				I vs. II	I vs. III	II vs. III
Hemoglobin(gm/dL)	10.5±2.03	10.7±2.2	10.5±2.5	0.708	0.988	0.824
TLC(cells/mm3)	13884.6± 8194.1	15126.2± 13113.5	18405.6±6996.4	0.526	0.026	0.340
Neutrophil(%)	65.7±14.1	61.9±13.1	71.1±13.5	0.218	0.121	0.031
Lymphocyte(%)	28.4±13.3	30.2±10.6	21.7±11.5	0.537	0.041	0.017
Platelet count (cells/mm3)	279597.9± 161496.3	257640.2± 139651.9	327116.7± 186581.2	0.523	0.248	0.170
Random blood glucose(mg/dL)	110.6±7.6	93.6±16.5	125.8±54.4	0.184	0.322	0.009
Total calcium (mg/dL)	10.6±7.6	10.4±0.8	9.5±1.7	0.868	0.545	0.036
Ionized Calcium (mmol/L)	1.06±0.19	1.08±0.12	0.96±0.23	0.556	0.050	0.034
pH	7.39±0.08	7.36±0.09	7.37±0.13	0.048	0.342	0.671
Hco3(mmol/L)	22.3±5.4	20.9±5.4	19.7±7.1	0.334	0.109	0.556
BE	-2.01±5.3	-2.9±5.9	-4.2±6.9	0.439	0.114	0.520
Na+(mmol/L)	132.4±7.4	135.6±11.5	130.2±8.1	0.064	0.258	0.095
K+(mmol/L)	4.2±0.8	4.4±0.9	4.1±0.9	0.097	0.822	0.249
Urea(mg/dL)	40.4±36.2	50.8±64.4	53.8±49.5	0.245	0.157	0.870
Creatinine(mg/dL)	0.84±0.5	0.92±0.8	1.2±1.7	0.500	0.034	0.448

Table 4 showed the time period of CSE, time taken to control seizure and duration of hospital stay. Mean time of CSE at presentation to our emergency in Group-I, Group-II and Group-III were 67.18±55.97, 108.8±84.17, and 136.05±96.93 minutes respectively. The mean time taken to control CSE in group-I was 49.7±54.1 minutes, whereas in group-II; 135.8±77.7 and group-III; 118.2±97.5 minutes. The time taken to control CSE is more in group-II, and group-III than group-I and were statistically significant. The mean time of hospital stays in group-I was 10.2±4.1 days, group-II: 13.9±8.1 and group-III: 3.57±5.6 days.

Table 5 showed the number of antiepileptic drug used in CSE. Three children with febrile status got controlled with lorazepam and others required additional antiepileptic. CSE got controlled in majority with lorazepam and phenytoin in Goup-I, whereas others needed either midazolam or valproic acid or both and in combination with propofol infusion. Most of patients in Group-II required multiple antiepileptics including propofol infusion but not achieved complete control of seizure. The duration and number of episodes of CSE, time taken to control and underlying etiology were important contributing factors for outcome of status epilepticus.

Table 3: Findings of CT-scan of brain in children with convulsive status epilepticus (n=193)

	Group-I (n=149)	group-II (n=25)	group-III (n=19)	Total (%) (n=193)
Normal study	61(40.9)	8(32)	3(15.8)	72(37.3)
Meningoencephalitis	29(19.5)	2(8)	4(21.1)	35(18.1)
Tubercular meningitis with hydrocephalus	18(12.1)	6(24)	3(15.8)	27(13.9)
Neurocysticercosis	14(9.4)	4(16)	1(5.3)	19(9.8)
Cerebral infarction	9(6.1)	1(4)	0(0.0)	10(5.2)
Cerebral atrophy	4(2.7)	2(8)	0(0.0)	6(3.1)
Periventricular Leukomalacia	4(2.7)	2(8)	0(0.0)	6(3.1)
Subdural effusion	3(2.01)	0(0.0)	0(0.0)	3(1.6)
Brain abscess	3(2.01)	0(0.0)	1(5.3)	4(2.1)
Intra-cranial hemorrhage	1(0.7)	0(0.0)	1(5.3)	2(1.03)
Hydrocephalus	2(1.3)	0(0.0)	0(0.0)	2(1.03)
Corpus callosum agenesis	1(0.7)	0(0.0)	0(0.0)	1(0.52)
Not done	0(0.0)	0(0.0)	6(31.6)	6(3.1)

Group-I: Seizure controlled, Group-II: Seizure uncontrolled, Group-III: Death.

Table 4: Time period of status epilepticus, time take to control seizure and duration in hospital stay (n=193).

	Group-I (n=149) (mean±S.D.)	group-II (n=25) (mean±S.D.)	group-III (n=18) (mean±S.D.)	p-value I vs II	I vs III	II vs III
Time period of SE(min)	67.2±55.9	108.8±84.2	136.1±96.9	0.002	<0.001	0.325
Time take to control seizure(min)	49.7±54.1	135.8±77.7	118.2±97.5	<0.001	<0.001	0.509
Duration of hospital stay(day)	10.2±4.1	13.9±8.1	3.57±5.6	<0.001	<0.001	<0.001

*min-minutes,*SE-status epilepticus. *Group-I Seizure controlled,* Group-II Seizure uncontrolled,* Group-III Death.

Table 5: Anti-epileptic drug used in study subjects

Anti-epileptic drug	Group-I (n=139)	Group-II (n=25)	Group-III (n=19)	Total (%)
LZP alone	3	0	0	
LZP+ PHT	92	2	4	98
LZP+PHT+MDZ	25	4	3	32
LZP+PHT+MDZ+VPA	23	9	7	39
LZP+PHT+MDZ+VPA+LEV	0	5	1	6
Multiple doses of LZP+PHT+VPA+LEV+propofol	6	5	4	15
I.V calcium gluconate	15	0	4	19
Pyridoxime	1	1	0	2

Group-I: Seizure controlled, Group-II: Seizure uncontrolled, Group-III:Death

LZP-Lorazepam, PHT-Phenytoin sodium, MDZ+Midazolam infusion (2-15 mcg/kg/minute), VPA- Sodium valproate, LEV-Levetiracetam

Discussion

Convulsive status epilepticus is pediatric emergency with good outcome at most of centres. Maximum children belonged to 5-15 years age group. GCTS was the most common presentation. Seizure recurrence of more than four episode was seen in 89 children. This can be attributed to poor drug compliance, choice of antiepileptic, poor follow up. Fever triggered seizure was the most common presentation. Acute symptomatic seizure like menigoencephalitis, tubercular meningitis, bacterial meningitis, necurocysticercosis was the most common etiology in study population. The same finding has been reported in previous studies from developing countries [13-17].

The time taken to control seizure was 49.7±54.1 minutes in group I. whereas this duration was high in group II and Group III, leading to poor outcome and even death. So, aggressive treatment with best supportive care is the need of hour for managing such cases in emergency. Kumar et al. also recorded the similar finding in his group of patients [17].

Intravenous lorazepam and phenytoin were common antiepileptics used for control of seizure in most of patients. Second line atiepiletics used were: valproate, midazolam infusion, propofol infusion. These groups of patients (group II and group III) required supportive care and management in PICU.

Mortality rate was 9.8%, which is comparable to other studies where it ranged from 10.8 to 28% [18,19]. Mortality was found higher in some other studies as high as 30% [17,20]. Low morality rate is attributed to better health facility, proper referral, protocol based management at tertiary care hospital like ours.

Limitations

Our study could further delineate causes for metabolic cases. We could not do neuroimaging with MRI. This might have caused missing of some diagnosis like neurodegenerative disorders, metabolic disorders. Further follow up was not done.

Conclusion

Study highlights important burden of an important medical emergency at a tertiary centre hospital. 5-15 years is the most vulnerable group with GCTS as most common presentation of CSE. Acute symptomatic seizure was the most common group. Seizure were well controlled in most of children with protocol based management and utilization of best supportive care. Intravenous lorazepam and phenytoin were found be most effective drugs in controlling seizure. Had the MRI facility were available easily, we could have diagnosed more metabolic and neurodegenerative cases. Even though, this data had brought important point: etiological diagnosis, duration of status epilepticus, seizure episode, time take to control status epilepticus, anti-epileptics used, and other supportive treatment are the important contributing factors for outcome of status epilepticus.

Funding: None

Conflict of Interest: None

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Case Report

Pediatric Education and Research
Volume 6 Number 3, September - December 2018
DOI: https://dx.doi.org/10.21088/per.2321.1644.6318.6

A Rare Entity in a Common Condition - Congenital Tyrosinemia Type 1

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Abstract

Tyrosenemia type 1 is a rare inherited metabolic disorder attributable to a deficiency of enzyme fumarylacetoacetate hydrolase. It has an autosomal recessive pattern of inheritance. The accumulation of tyrosine and its toxic metabolites succinyl acetone and succinyl acetoacetate in various tissues leads to the charecteristic hepatic failure, renal dysfunction and neurological crisis. Here we report 21 months old child presented with signs and symptoms of hepatic failure. This case report highlights that clinical examination has not lost its significance even in this new era of advanced extensive investigations.

Keywords: Congenital Tyrosinemia; Neurological Crisis; Tyrosin.

Introduction

Hereditary Tyrosinemia type 1 is the most severe disorder of tyrosine metabolism. It is characterized by severe progressive liver disease and renal tubular dysfunction. HT1 is caused by mutations in the fumarylacetoacetate hydrolase gene (FAH). Progression of the liver disease may present as chronic or acute form, with rapid deterioration and early death. Acute form of the disease presents within first month of life with hepatic failure and its complications. However, Chronic form present up to 2 years of life with hepatomegaly and developing cirrhosis. Here we report a case with chronic hereditary tyrosinemia type 1 presented as acute hepatic failure precipitated by drug induced hepatitis.

Case Report

A 21 month old girl referred from outside hospital with history of jaundice for 20 days, generalised anasarca with ascites for 10 days, Vomiting and Altered sensorium for 5 days. She got admitted in private hospital 10 days before with above complaints diagnosed as hepatic encephalopathy stage 3, received symptomatic treatment. After stabilization transferred to our hospital for further management. 5 months back child was started on 3 drugs AKT from outside in view of mantoux positive. On examination there was icterus and bilateral pedal edema with features of vitamin D deficiency. Liver was firm, smooth surface, 3 cm below right costal margin with span of 11 cm. our initial impression was 21 month old female child on AKT with acute hepatic failure with recovering Hepatic encephalopathy along with features of rickets most likely AKT induced Hepatitis or Autoimmune Hepatitis or Wilson's disease or other Metabolic cause. lab investigations showed SGOT/SGPT- 312/216, Total bili/Direct bili- 22/9.2, PT/INR-20/1.7, serum ammonia- 330, vitamin Dlevel 12IU, Ca/PO_4 level- 8.7/4.4, ALP 334. All viral markers were negative. USG abdomen was suggestive of mild hepatomegaly with heterogeneous echo texture of liver. ABG showed Normal Anion gap metabolic acidosis. Urine PH was 6 with high phosphorus level. Meanwhile child started on vitamin A, D, E, K supplements with oral ursodeoxycholic acid. Keeping in mind about hepato-renal involvement in form of firm

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Received on 06.03.2018, **Accepted on** 14.11.2018

liver with heterogeneous echo texture, rickets and normal anion gap metabolic acidosis; we went ahead with serum alpha protein level which came as high as 11,860. CT abdomen showed micro-nodular cirrhosis. Liver biopsy done also confirmed micronodular cirrhosis with activity and rulled out Wilson's disease as dry copper weight was normal (77 microgm/gm). Urine succinyl acetone level sent, suggested four fold raise in primary tyrosine metabolites indicating congenital tyrosinemia type 1. Child started on tyrosine free diet. FAH gene study sent for confirmation; report awaited.

Discussion

Type1 tyrosinosis is an inborn error of metabolism caused by deficiency of enzyme fumeryl acetoacetate hydrolase. Autosomal recessive disease associated with FAH gene mutation in chromosome 15q 25.1. Patients have peculiar cabbage like odour, with severe liver involvement within 2-6 months of life or later in the first year with liver dysfunction and renal tubular dysfunction (fanconi syndrome) associated with growth failure and rickets. Children may have repeated neurologic crises include change in mental status, abdominal pain, peripheral neuropathy, and/or respiratory failure requiring mechanical ventilation. Death in the untreated child usually occurs before age ten years, typically from liver failure, neurologic crisis, or hepatocellular carcinoma. Typical biochemical findings include: increased succinylacetone concentration in the blood and urine; elevated plasma concentrations of tyrosine, methionine, and phenylalanine; and elevated urinary concentration of tyrosine metabolites and the compound δ-ALA. Genetic testing for FAH gene use for confirmation. Combined treatment with nitisinone and a low-tyrosine diet has resulted in a greater than 90% survival.

Conclusion

This case reports highlights that clinical examination has not lost its significance even in this new era of advanced extensive investigations. All siblings of a child with tyrosinemia type I should screened with urine and blood succinylacetone analyzed as soon as possible to enable the earliest possible diagnosis and initiation of therapy. Early recognition of tyrosinemia type I and adequate treatment should always be followed by intensive follow up for the risk of development of HCC both at the short and the long term.

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

Discussion

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

References

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

Standard journal article

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

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

Article in supplement or special issue

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

Corporate (collective) author

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

Unpublished article

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

Personal author(s)

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

Chapter in book

[7] Nauntofte B, 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.

No author given

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

Reference from electronic media

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

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