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Contents

Original Articles

Review Article

Case Reports


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Clinical Profile of HIV Infected Children 18 months – 15 years of Age

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Abstract

Context: Clinical presentation of HIV infected children is different than adults. Disease progression is rapid. About 30% HIV infected children under one year of age die undiagnosed. Clinical scenario is changing with time and with available treatment. *Aims:* To study the profile of clinical presentation in HIV infected children. *Settings and Design:* Study was conducted in a government medical college of north India. It was hospital based observational study. *Methods and Material:* 140 HIV infected children between the age of 18 months to 15 years diagnosed as per guide lines of NACO were included in study. Clinical and immunological staging was done according to WHO staging criteria. Written consent was taken and counselling was done. *Statistical Analysis Used:* To describe nominal data simple percentages were used. Mean and Standard Deviations were used to describe normally distributed data. *Results:* Common age of presentation was between five to ten years. Perinatal transmission was the commonest mode of transmission (97.8%). Fever (60.7), Recurrent loose stools (45.7%), Chronic cough (27.8%), Itching and rashes over body (25%) were the common complaints. Anaemia (85%), Lymphadenopathy(59.2%), Skin lesions(47%), Chronic Supporative Otitis Media(12%) were the common clinical findings. Central Nervous System involvement was seen in six and Cardio Vascular System involvement in four children was noted. One child presented with joint involvement and one had Non Hodgekin's Lymphoma. *Conclusions:* Vertical transmission was the commonest route of infection in children. Prolong fever, recurrent diarrhoea, frequent respiratory infections associated with under nutrition, anaemia and skin lesions were common clinical presentations. One third children were orphan and they were worst sufferer.

Keywords: Clinical Profile; HIV Infection; Children.

Introduction

Children are infected mostly through vertical transmission. 50–70% vertical transmission occur intrapartum [1] across the mucous membrane in the oropharynx or in stomach [2]. Clinical presentation of HIV infected children are different than in adults and varies widely [3,4]. Most of them may be asymptomatic at birth and in early years of life, physical examination may be normal. Initial signs and symptoms may be subtle and non specific. On the basis of disease progression, children are divided in to three groups; *Rapid progressors*- They undergo rapid downhill course, disease progression is very fast. Most of them (33%) succumbed to opportunistic infections before they are diagnosed, i.e. under 1 year of age [5-7]. *Less rapid progressors*- They become symptomatic usually after infancy, presents with failure to thrive and recurrent infections. *Slow progressors*- They remain asymptomatic for longer period. Their disease progression and pattern behaves like adults. Some children become long term survivor without therapy [8,9]. Some perinataly infected girls have been reported to survive to reproductive age and have given birth to un infected infants [10]. Early diagnosis may be utilised as a window of opportunity for disease modification. Initiation of early therapy

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during acute phase limits establishment of latent reservoir. HAART in acute phase of HIV-I leads to development of strong HIV-I specific CD4T cell proliferation in response to HIV antigen [11]. It helps in lowering of viral set point. Availability of Anti retroviral treatment has changed the picture of clinical presentation.

Most of the work on this subject is focused on adults. Data regarding paediatric HIV status, changed clinical scenario, response to infection, progression of disease and pattern of opportunistic infections are in scanty. To understand these aspects of disease in children, particularly in this region of country, we decided to undertake this study.

Present study is a hospital based observational study, done at S. N. Children Hospital Allahabad and O.P.D. of ART Centre, S.R.N. Hospital Allahabad to study the presenting complaints and physical findings before diagnosis. Both the hospitals are associated hospital of M.L.N. Medical College Allahabad. Study period was April, 2005 to October, 2012.

Methods

140 children of both sexes between age 18 months to 15 years with HIV positive status confirmed as per NACO guidelines of HIV testing, at ICTC MLN Medical College Allahabad, attending the ART Centre SRN Hospital Allahabad and OPD of SN Children Hospital Allahabad during the above mentioned period were included in this study. Using the pre designed proforma these children were enrolled in the study. Care takers were counselled by qualified counsellors and informed consent was taken from guardians for any investigation and treatment. Detailed history and full clinical examination was done in all cases. History of presenting complaints with their duration, severity and recurrence, what were the initial presenting symptoms, age at which complaints started and how they progressed were enquired in detail. History of blood transfusion, operative procedures, confidential interview regarding sexual behaviour, parents and sibling's HIV status and their survival was enquired. Socio-economic status was decided on the basis of modified Prasad's criteria. Children were classified in different grades of malnutrition according to IAP classification. Weight was recorded on electronic weighing machine. Clinical and immunological staging was done according to WHO staging criteria. Every child was investigated for Complete Blood Count and CD4 count. CD4 count was done with Partec CyFlow® counter flow cytometer. CD4% was used in children below 5 years of age.

Analysis of data

To describe nominal data, simple percentages were used. Mean and standard deviations were used to describe normally distributed data.

Observation

Out of 140 children, 91 were male and 49 were female. Maximum number of children (61, 43.38%) were in age group 5-10 years. Mean age of children was 7.67 years (SD ± 3.50). 75.71% children belonged to rural area and 24.29% from urban area. 85% children were from Hindu religion and 15% were Muslim. 37.15% children were in Socioeconomic status IV, followed by 35%, 17.14%, 7.14% and 3.57% in Socio Economic Status III, II,V and I respectively. 33.57% children were orphan as shown in Figure 1. 47.14% children were in grade II, 27.86% in grade-I, 20.71% in grade-III and 4.29% were in grade-IV malnutrition. No child was found nutritionally normal. In our study (137) 97.86% children were infected perinatally and (2)1.43% through blood transfusion. In one case mode of transmission could not be traced as shown in Figure 2. Fever, loose stools

Table 1: Distribution of cases according to presenting complaints

Presenting complaints	Number	Percentage
Fever	85	60.71
Loose stools	64	45.71
Cough	39	27.85
Itching over body	35	25.00
Difficulty in breathing	22	15.71
Abdominal distention	21	15.00
Not gaining weight	18	12.87
Ear discharge	17	12.14
Loss of weight	15	10.71
Joint pain	01	00.71
Oral ulcer	22	15.71

Table 2: Distribution of children according to Clinical signs at the time of presentation

Presenting signs	Number	Percentage
Pallor	119	85.00
Lymphadenopathy	83	59.28
Skin lesions	66	47.14
Respiratory distress	37	26.42
Hepato-splenomegaly	29	20.71
Isolated hepatomegaly	25	17.80
Isolated splenomegaly	10	07.10
Parotid swelling	11	07.85
Vitamin A deficiency	21	15.00
Oropharyngeal candidiasis	10	07.14
Clubbing	09	06.42
Icterus	05	03.57
CNS signs	06	04.28
CVS signs	04	02.85
Cyanosis	03	02.14
Joint involvement	01	00.71

Table 3: Type of skin lesions of children in the present study. (n-66)

Type of skin lesions	Number	Percentage
Scabies	13	09.28
Papular Purpuric Eruptions	13	09.28
Pyoderma	12	08.57
Seborrhoeic dermatitis	07	05.00
Fungal skin infection	05	03.57
Fungal nail infection	03	02.12
Chicken pox	04	02.85
Herpes zoster	02	01.42
Cheloid	02	01.42
Non-healing ulcer	02	01.42
Molluscum contagiosum	03	02.14

Table 4: WHO Clinical staging according age. (n-140)

WHO clinical stage	(Age group (in years) 1.5 - 05	05 - 15	Total
Clinical stage I	04	13	17
II	22	50	72
III	14	29	43
IV	01	07	08

Table 5: WHO Immunological staging according to age. (n-140)

WHO immunological stage	Age group in years 1.5 - 05	05 - 15	Total
Stage I	07	43	50
Stage II	11	16	27
Stage III	14	24	38
Stage IV	09	16	25

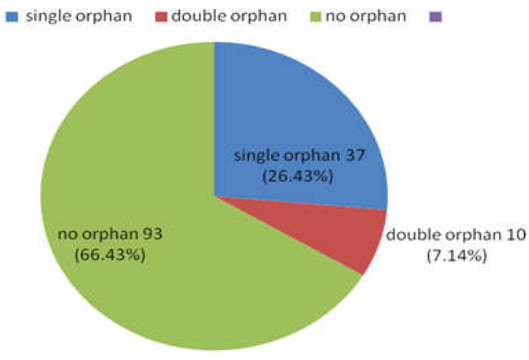

Fig. 1: Number of orphan children

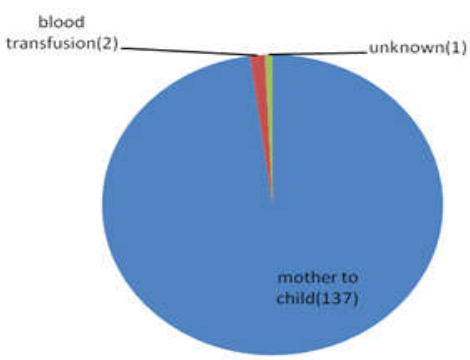

Fig. 2: Mode of Transmission

and cough were among the common presenting complaints (Table 1). Anaemia, Lymphadenopathy and skin lesions were common examination finding (Table 2). Scabies, Papular pruritic eruption and pyoderma were commonest skin lesions (Table 3). Maximum number of children (51.42%) were in clinical stage-II, followed by 30.72% in clinical stage-III (Table 4). 35.72% children were in immunological stage-I, followed by 27.15% in immunological stage-III (Table 5).

Discussion

In present study male to female ratio of children was 1.86:1. This trend in sex distribution may be due to more concern for male children in our society as seen in other diseases also. Mean age of children was 7.67 years (SD ± 3.5) in the present study. Shel A et al[12] also found mean age of children 7 years (SD 3.4years). N Kumarasami et al [13] found that most perinatally infected children become symptomatic by 5 years of age. In our study 33.57% children were orphan. Out of which 7.1% lost their both parents. Before HIV/AIDS epidemic only 2% children in developing world were orphan but according to Foster et al this number has increased to 7 – 11%. In our study significant malnutrition (grade II,III and IV) was present in 72.14% children. No child was nutritionally normal. Rakesh Lodha [15] found 81.3% children had failure to thrive. In our study 97.86% children under 15 years of age were infected through vertical transmission, which is consistent with the findings of Agarwal D et al [16] 94%, Merchant R H et al [17] 86.6%. In our study fever was the most common symptom present in 60.7% children followed by loose stools. Agarwal D et al [16] and Rakesh Lodha et al [15] have reported fever as the most common symptom. Anaemia was the most common (85%) finding in present study. Adetifa I M et al and Claster S [18] also reported anaemia as the commonest finding. In our study lymphadenopathy and skin lesions were 59.2% and 47.14% respectively. Emodi J et al [19] have reported lymphadenopathy in 59% and skin lesions in 37% children. In our study maximum number of children (51.42%) were in WHO clinical stage II while Agarwal D et al [16] have reported maximum number of cases in WHO clinical stage I. In our study on the basis of immunological staging, maximum number of cases were in stage I and minimum number in stage IV, which is consistent with the findings of Shet et al [12] and Agarwal D et al [16].

Conclusion

This study concludes that out of 140 children, majority of children were infected through vertical transmission except 2 children who acquired infection through blood transfusion and in one child mode of acquiring infection was not ascertained. Mean age of presentation was 7.67 years. Majority of children were in WHO clinical stage II and immunological stage I. Recurrent and prolonged episodes of fever and diarrhoea, frequent respiratory tract infections and failure to thrive associated with anaemia were the main findings. Different types of skin lesions were seen in 47% cases. Scabies, popular pruritic eruptions, pyoderma and seborrhoic dermatitis were the common skin conditions. Central nervous system involvement was seen in six children. 4 children had CVS involvement. One male child presented with pain and swelling of both knee and ankle joints. One child presented with Non hodgkins lymphoma. Severe anaemia and severe malnutrition was associated with poor prognosis, which was common in children who were orphan and in lower socioeconomic status. It was observed that HIV infection was transferred to the mothers of these children by their husbands who were working at distant places and staying there for longer time and through vertical transmission children were infected.

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To Study the Incidence, Etiology, Laboratory Profile and Risk Factors of Febrile Seizures

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Abstract

Background: Febrile convulsion (FC) is the most common type of seizure that occurs in children aged 6-60 months. It occurs in two forms including simple and complex febrile seizure. *Objectives:* The purpose of this study was to determine the clinical, epidemiologic and laboratory features of patients admitted to our hospital with febrile seizure in 2014-2015. *Methods:* In this cross-sectional study, the patients with diagnosis of febrile seizure were the target population. We obtained patient's data based on clinical examination, history and information registered in hospital medical files including demographic, clinical and laboratory findings. *Results:* During the study period, 60 children with febrile seizures and mean age of 26.2 ± 19.5 months were studied. Male to female ratio was 1.2:1. 37 (61.9%) children had simple seizures and 23 (38.1%) children had complex seizures. 12 (19.4%) of the patients had family history of febrile seizures and family history of epilepsy was positive in 6.3% of cases. Ninety one percent of cases were born with normal vaginal delivery. Also, only 2 patients (3.1%) had less than 37 weeks of gestational age at birth. The mean rectal temperature of the patients was 38.5 ± 0.67 ^{o}C. Gastroenteritis was the most common cause of fever in our patients. *Conclusion:* The highest frequency of Febrile Convulsion was seen in younger than 20-month-old children. Except for the lower incidence of positive history of prematurity and higher prevalence of gastroenteritis, results of the present study are relatively similar to other studies..

Keywords: Seizures; Febrile; Children.

Introduction

Febrile convulsion is one of the most common types of seizures in childhood from which 2-5 percent of children suffer and usually occurs between 3 months and 5 years old. According to the definition of International Epilepsy Association, febrile convulsion occurs in infants older than 1 month together with febrile illness, without any evidence of the central nervous system infection, without history of neonatal seizures or a previous unprovoked convulsion and does not meet the features of other symptomatic convulsions. It is divided into two types: simple and complex. Simple convulsion usually takes less than 10-15 minutes, generalized tonic-colonic, tonic, colonic or atonic. Complex Febrile Convulsion has one or more of the following features: a focal onset or showing focal deficit during convulsion attack, a duration longer than 15 minutes, during the first 24 hours, it occurs more than once. Despite its benign nature, the febrile convulsion is one of the most common reasons for admission to pediatric emergency wards worldwide. In these patients, in most cases, fever is the result of upper respiratory system, gastroenteritis and urinary tract infection [1-3]. The incidence of Febrile Convulsion varies in different places of the world, ranging from 5-10 % in India, 8.8% in Japan and 14% in Guam [3]. This illness was distinguished from other types of convulsions in the mid-nineteenth century [4]. Recurrence is very common in this illness, but neural evolution does not

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change in patients. Numerous conducted studies have noted hazardous factors for its recurrence for infants less than 15 months old, including fever background, convulsion history in first degree relatives, complex convulsion and looking after in daily care units [5]. The study by Kong revealed that the convulsion history in first degree relatives was the only substantial risk of convulsion recurrence [6].

Objectives

With respect to the issue that Febrile Convulsion is the most common seizure type in children, the objective of this study was to assess the clinical, epidemiological and laboratory characteristics of Febrile Convulsion in children and its comparison with similar studies in other parts of the world.

Methods

In this cross sectional study, 60 children aged between 6-72 months presenting with Febrile Convulsion who were admitted to the pediatric emergency ward of our hospital between June 2014 and June 2015 were evaluated. Data were collected regarding age, gender, type of convulsion (generalized or focal), duration of convulsion, type of the febrile convulsion (simple or complex), rectal temperature, family history of convulsion, familial background of epilepsy, past history of the febrile convulsion, underlying causes of fever, existence or nonexistence of meningitis signs and symptoms, gestational age at birth, clinical and laboratory data. Patients with a past history of unprovoked convulsion, metabolic disorders, known illnesses of central nervous system and neurological deficits were excluded from this study. Abnormal cerebrospinal fluid analysis included one or more of the following features: positive gram stain, more than 5 white blood cells, and low glucose content of cerebrospinal fluid or increased CSF protein. Anemia is defined as hemoglobin levels less than 11 g/dl for age group 6-72 months. A written informed parental consent was obtained for each patient in this study.

Results

The mean age of patients was 26.2 ± 19.5 months. 33 (54.4%) were boys and 27 patients (45.6%) were girls. The highest frequency of Febrile Convulsion was seen in the six to 20-month age group, which included 32 children (53.9%). In contrast, the lowest frequency belonged to the age group of 34-48 months, which contained only 4 children (6.3 %). There was a history of prematurity in 2 children (3.1%). A family history of Febrile Convulsion and epilepsy was found in 12 cases (19.4 %) and 4 cases (6.3 %), respectively. 54 (90%) children were presented with generalized convulsions and 6 (10%) had focal convulsion. Type of Febrile Convulsion was simple in 37 (61.9%) and complex in 23 cases (38.1%). The majority of patients (78%) had seizure durations less than or equal to 15 minutes. The mean rectal temperature during convulsion attack was 38.3^{o}C ranging from 38 to 40 ^{o}C. According to history and physical examination, it was determined that for 26 patients (43.7%) indications for lumbar puncture were put. Lumbar puncture was performed for 20 patients and for 6 patients; it was not approved by their parents. Among them who were put under lumbar puncture, 3 patients had abnormal findings in cerebrospinal fluid analysis in favor of meningitis. For each patient, an average of 4.6 laboratory diagnostic tests was performed. Cerebral imaging was conducted on 11 patients (18.7 %), however, these tests did not show any abnormality in any of the cases. Among all the patients, convulsion of 20 individuals (37.5%) was controlled with therapeutic measures. Proportion of patients with febrile seizures to all hospitalized patients for seizure disorders was 30%. 23 (38.7%) patients were visited by a physician for current illnesses before seizure attack. 35 (58.5%) had a background of antibiotic consumption during the current illness. (Table 1) shows the abnormal laboratory findings in patients with Febrile Convulsion. Gastroenteritis was the most common cause of febrile illness in our study (Table 2).

Table 1: Frequency of Abnormal Laboratory Findings in Patients with Febrile Convulsion

Lab Abnormality	Cases, n = 60, No. (%)
Leukocytosis	14 (23.8)
Leukopenia	2 (3.7)
Thrombocytosis	7 (12.5)
Thrombocytopenia	4 (6.9)
Anemia	21 (35)
Hypoglycemia	5 (8.1)
Hypernatremia	3 (5)
Hyponatremia	6 (10)

Table 2: Distribution Frequency of Etiology of Fever in Patient with Febrile Convulsion

Etiology of Fever	Cases, n = 60, No. (%)
Urinary Tract Infection	10 (16.2)
Gastroenteritis	22 (38.1)
Meningitis	3 (5)
Respiratory Tract Infection	12 (20)
Otitis Media	1 (1.2)
Unclassified	12 (19.5)
Total	60 (100)

Discussion

Febrile convulsion is the most common type of seizure during childhood which occurs in 2-5% of children. It usually occurs in children between 3 months and 5 years. Fortunately, most febrile seizures are benign and rarely cause brain damage. Although febrile seizures are benign in nature, when seizures occur, they may lead to fear and anxiety of parents and subsequently it potentially affects the family's quality of life. Physical, psychological and behavioral disorders may manifest due to the lack of sufficient information of parents about febrile seizures. In our study, the majority of children were under 2 years old and our findings were similar to other studies in which Febrile Convulsion was in the age range of 6 months to 3 years with peak incidence at the age of 18 months [9]. In the present study, prevalence of Febrile Convulsion was slightly predominant in males than females and this is in agreement with the results of other studies [7,8]. In our study, 12 patients (19.4%) had a positive family history of Febrile Convulsion, while this percentage in the other studies varied from 25% to 40% [10]. 4 (6.3%) in this study had a positive family history of epilepsy, while this frequency varied from 1.6% to 9% in other studies [11]. Ninety percent of children in the present study had generalized convulsion that is similar to the other studies. In our study, 37 children (61.9%) were suffering from simple Febrile Convulsion, while this was between 60 to 90 percent for other studies.

Conclusion

The highest frequency of Febrile Convulsion was seen in younger than 20-month-old children. Except for the lower incidence of positive history of prematurity and higher prevalence of gastroenteritis, results of the present study are relatively similar to other studies.

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Cognitive Dysfunctions in Children with Epilepsy

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Abstract

Background: Epilepsy, a chronic neurological disorder is common in children. The global decline in intellectual functioning may occur in children with seizure disorder. *Objectives:* The present study was undertaken to assess cognitive function in children with epilepsy. *Method:* This was a hospital based observational study on 60 children aged 2-5 years and 68; 6-14 years suffering from idiopathic epilepsy. 74 and 64 normal age and gender matched healthy children were also selected as control. Sixty six children suffering from epilepsy aged 6-14 years were also assessed for their memory functioning with healthy control (n=71). Binet-Kulsrestha intelligence scale and PGI memory scale were used for cognitive assessment. *Result:* Epileptic children had significantly lower scores on cognitive functioning in domains of visual-perception ($p<0.001$), motor-coordination ($p<0.001$), language development ($p<0.001$), and immediate recall ($p<0.001$) as compared to control. In children aged 6-14 years with epilepsy, significantly lower values were observed for analysis, synthesis and reasoning ($p<0.001$) and memory ($p<0.001$) as compared to control. Epileptic children also had significantly lower overall IQ. *Conclusion:* Parent should be counseled about decline in cognitive functions and IQ in epileptic children for early intervention and appropriate measures.

Keywords: Epilepsy; Cognition; Memory; Intelligence Quotient(IQ).

Introduction

Epilepsy is a chronic disorder that affects intellectual abilities and memory functioning in children. They are at risk for developing learning problems due to low intelligence and memory deficit. Difficulties with abstract reasoning and reduced information processing have also been observed in epileptic children [1]. These children show poor motor precision and visual-motor coordination as well as memory impairment as compared with normal healthy children [2]. It has been documented that decreased neuronal excitability and brain damage results into slow motor and psychomotor speed, poorer attention and mild memory impairment in epileptic children [3,4].

Further, various factors are known to disrupt neurocognitive functions in epileptic children such as seizure type and syndrome, age of onset of seizure as well as seizure frequency, intensity and duration. Studies have reported that onset of seizure before 5 years of age is significant risk factor for intellectual function in partial as well as generalized seizure [5, 6]. Mandelbum and Burak (1997) have reported poor intellectual performance in children with generalized and non-convulsive seizures compared to partial and convulsive seizure [7].

Thus, it is important to recognize intellectual and memory deficits in epileptic children as early as possible so that appropriate medical, psychological, and educational interventions can be planned. In view of the above, present study was conceptualized with the objectives to explore specific cognitive deficits and memory functioning in children suffering from seizure disorder.

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Material and Methods

The participants of the study were recruited during the period from 2010 to 2014 from the Epilepsy Clinic and Out Patient Department of Pediatrics, Institute of Medical Sciences, Banaras Hindu University.

Children aged 2-5 to 6-14 years suffering from epilepsy were taken as study groups and age and gender matched children without any disease/mild ailments served as control.

All the children were subjected to assessment for different cognitive functioning. Binet-kulshrestha intelligence scale, which is an Indian adaptation of famous Stanford-Binet test, form L-M (1960) [8] was used for both the age groups for the assessment of intellectual functioning. In this adaptation, the essential shape and features of the original L-M form have been retained. The test was administered in a separate place, in the presence of one of the family member, preferably the mother. The basal age, mental age and IQ were calculated as per the instructions given in the manual. Further, each protocol was analyzed separately for 2-6 years for the visual-perceptual, motor-coordination, language development, immediate recall, concept formation and reasoning. For 7-14 years, analysis was done for synthesis and reasoning, verbal ability, memory, spatial ability and orientation.

For memory functioning, PGI memory scale for children [9] was used. This scale was used on children above 7 year of age. It measures 10 aspects of memory such as remote memory, recent memory, mental balance, attention-concentration, delayed recall, immediate recall, similar pairs, dissimilar pairs, visual retention and recognition.

Detailed information about epilepsy such as age of onset, type and frequency of seizure, antiepileptic medications and its compliance and family history were recorded through semistructured questionnaire. The protocol of the study was approved by the Institute Ethical committee and informed consent was taken from the parents or authorized representative of each child.

Statistical Analysis

Data were analyzed using SPSS software version 16.0. Student's t –test and Kruskal-wallis test was used to compare the observations on different study groups.

Result

One hundred and thirty nine children suffering from epilepsy and 157 sex and age matched controls were included in the study. These children were further divided in two age groups i.e. 2-5 (60 epileptic and 74 controls) and 6-14 years (79 epileptic and 83 controls).

Table 1 mentioned median values for different domains of cognitive functioning in 2-5 years age groups. Epileptic children had significantly lower scores for visual-perception ($p<0.001$), motor-coordination ($p<0.001$), language development ($p<0.001$), and immediate recall ($p<0.001$) as compared to their matched controls. In the higher age group, significantly lower values were observed for analysis, synthesis and reasoning ($p<0.001$) and memory ($p<0.001$) in the epileptic children (Table 2). Further, epileptic children showed significantly lower overall IQ as compared to controls and were true for both the age groups (Table 3).

Children having partial seizure were compared with those suffering from generalized seizure. In the lower age groups, poor scores for language development ($p<0.01$) and immediate recall ($p<0.001$) were observed in children suffering from partial seizure. For higher age group, significant differences were observed for memory functioning ($p<0.05$). However, no such differences were observed between the two groups in overall intellectual functioning.

Table 4 showed observations on memory scale. Significant differences were observed for delayed recall ($p<0.001$), immediate recall ($p<0.01$) and similar pairs ($p<0.05$), when children with seizure disorder were compared with control group.

Table 1: Median values for different domains of IQ (2–5 years)

Groups	Visual perception		Motor coordination		Language development		Immediate recall		Concept formation		Reasoning	
	Median	SD	Median	SD	Median	SD	Median	SD	Median	SD	Median	SD
Controls (N=74)	0.6663	0.253	0.6660	0.380	1.000	0.209	0.5000	0.344	0.6330	0.342	0.5000	0.359
Epilepsy (N=60)	0.5000	0.264	0.2500	0.209	0.6660	0.301	0.5000	0.236	0.7500	0.356	0.6660	0.418
						Kruskal Wallis						
KW	20.745*		25.949*		24.490*		12.726*		0.469		0.025	

*p=<0.001

Table 2: Median values for different domains of IQ (6-14 years)

Groups	Analysis, synthesis & reasoning		Verbal ability		Memory		Spatial abilty	
	Median	SD	Median	SD	Median	SD	Median	SD
Controls (N=68)	0.8333	0.214	0.6660	0.278	0.7500	0.265	0.6660	0.342
Epilepsy (N=64)	0.6000	0.301	0.7140	0.285	0.5000	0.308	0.6660	0.298
			Kruskal Wallis					
KW	34.881*		0.392		24.584*		0.657	

*p=<0.001

Table 3: Mean±SD for intellectual functioning in both age groups

Groups	Control (N=68)	Epilepsy (N=68)	t- test
2-5 years	99.10±9.24	95.71±9.57	2.077*
6-14 years	94.77±7.45	87.81±10.15	5.043**

*p=<0.05, **p=<0.001

Table 4: Median values for different components of memory (mean±SD)

Memory components	Control (n=71)		Epilepsy (n=66)		KW
	Median	SD	Median	SD	
Remote memory	4.00	1.441	3.000	1.708	2.140
Recent memory	5.000	0.969	4.000	0.998	0.797
Mental balance	5.000	1.204	5.000	1.041	0.976
Attention & concentration	5.000	0.861	5.000	0.851	0.008
Delayed recall	4.000	1.92	3.000	1.694	13.049***
Immediate recall	6.000	2.76	5.000	1.807	9.467**
Similar pair	2.000	1.383	2.000	1.312	6.084*
Dissimilar pair	0.000	0.976	0.000	0.591	2.223

Discussion

Epilepsy is a chronic neurological condition resulting in brain damage which in turns leads to cognitive and behavioral difficulties which may range from mild attention and concentration problems to difficulty in recent memory and executive functioning [4,10]. Present study was focused to find out cognitive deficits in children suffering from seizure disorder. It was observed that visual-perception, motor coordination, language development, analysis-synthesis and reasoning and memory were poor in these children. Attention problems, memory and language impairment as well as deficits in executive functioning in children suffering from seizure disorder have also been reported by several other researchers [1,11,12,13]. Gulati et al (2014) [11] reported that children with epilepsy showed difficulty in learning, memory, problem solving and concept formation as has been observed in the present study. Chanbers et al (2014) [12] in a case control study found that children with epilepsy have significantly lower scores on memory, language and attention as compared to controls. Language dysfunction have also been observed while lexical knowledge of word finding difficulties and anomia is found to be more common language problems in such children [13].

Intellectual disability and cognitive impairment have been reported by a community based study on 85 children with active epilepsy [14]. Kernan et al (2012) compared complex partial seizure and childhood absence epilepsy with control. They demonstrated that children with complex partial seizure and childhood absence epilepsy showed mild generalized cognitive deficit and impaired intellectual functioning as compared to control [15]. Subnormal global cognitive functioning has been reported in approximately 1 out of 4 individuals in a community based cohort study [16].

In Indian context, very few studies are available on epilepsy and memory functioning. Nehra et al(2013) [2] reported that when 34 children with epilepsy were compared with controls using PGI memory scale significant differences were observed for memory domains such as recent memory, remote memory, attention and concentration, immediate recall, delayed recall, similar pairs, mental balance, visual retention and recognition. While, in the present study significant differences were found for delayed recall, immediate recall and memory. The hypotheses most frequently used to explain the memory deficit in

epileptic patients has been that it involves the consolidation of memory traces or the transfer of the memory trace from short-term memory to long-term storage. A lesion decreases auditory processing capabilities thus inducing a reduction in coding. This poor initial encoding of information further leads to the impairment of delayed recall.

A specific cognitive profile, observed in the present study in children with seizure disorder, showed decline in their cognitive abilities which might be the result of epileptogenic process which can irreversibly damage the brain, especially maturing brain even if seizure is well controlled with anti epileptic medications. It can lead to cognitive changes finally leading towards global intellectual deficits. Thus, early identification of cognitive deficit and its management is necessary for the favorable outcome in children suffering from epilepsy.

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Prognostic Significance of Early Platelet Count Decline in Preterm Newborns

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Abstract

Objectives: Decline of platelets with or without thrombocytopenia is observed in critically ill preterm newborns. Prognostic signifi cance of platelets count in Neonatal Intensive Care Unit focused on outcome after thrombocytopenia. We aimed to estimate the changes in platelets count within the first 7 days of life in preterm newborns and its relation to fi nal outcomes. *Methods:* Retrospectively, the platelets count during the first 7 days of life, and its association with mortality, length of stay among survivors (LOS), and later severe morbidities were determined. Appropriate regression analyses were used to examine possible relations between studied variables. *Results:* Platelets drop that did not reach thrombocytopenia level in the first 7 days of life happened in 61.7%. Platelets count drop in the first 7 days of life was a predictor of mortality, LOS, and major morbidities such as intraventricular hemorrhage and necrotizing enterocolitis. *Conclusions:* Platelets count drop within the first 7 days of life independent of thrombocytopenia can be used to predict increased mortality, LOS, and the development of later severe morbidities in critically ill preterm neonates.

Keywords: Intraventricular Hemorrhage; Thrombocytopenia.

Introduction

Thrombocytopenia in newborns has an overall prevalence that ranges from 1% to 5% [1-4]. It is even much higher in newborns admitted to Neonatal Intensive Care Unit (NICU) reported to be ranging from 18% to 35% [2,5,6]. From 22 weeks' gestation onward, the platelets count reaches and maintains a level above 150×10^9/L and only 2% of term newborns have platelets counts below this level at birth, thereby a platelets count below 150×10^9/L has been used to defined thrombocytopenia [5,6]. Severe thrombocytopenia (platelets $<50 \times 10^9$/L) occursin fewer than three per 1000 term newborns; the most important cause being alloimmune thrombocytopenia [3,4].

In contrast, approximately 70% of newborns born at a weight <1000 g has thrombocytopenia at some point during their NICU stay and up to 20% of sick preterm newborns can develop severe thrombocytopenia [7].

Many neonatal and maternal conditions are associated with thrombocytopenia. However, the most common explanations for severe thrombocytopenia were acquired varieties of consumptive thrombocytopenia, especially in septicemic preterm newborns [2,8]. Thrombocytopenia has been independently related to mortality and major morbidities as intraventricular hemorrhage (IVH), disseminated intravascular coagulopathy, and necrotizing enterocolitis (NEC) [9-11]. On the other hand, the role of thrombocytopenia in some serious morbidities as IVH is difficult to establish, especially when IVH occurs in preterm newborns with normal platelets counts [12]. In adults and pediatric ICU patients, the drop in platelets numbers and not only thrombocytopenia was shown to be a good predictor of clinical outcome. In critically ill adults, ≥ 30% drop in the absolute platelets numbers, without

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thrombocytopenia was associated with ventilator-related pneumonia [13,14]. In PICU, adrop in platelets counts >27% and thrombocytopenia were independently related to mortality [15]. In preterm newborns, despite that the drop in absolute platelets count, without thrombocytopenia, was suggestive of fungal more than bacterial sepsis [16]; the association of morbidity and mortality with this drop of platelets counts, especially without thrombocytopenia, is poorly understood. Platelets drop in preterm newborns can be transient, or it may progress to severe thrombocytopenia.

Most studies investigating platelets count changes in preterm infants, and its prognostic significance focused on outcome in newborns with platelets <50–100 × 10^9 /L [17,18]. Using a cut-off value similar to adult studies (≥ 30% platelets drop), a single study was published in newborns that investigated the signifi cance of platelets drop at 7 and 28 days of life in extremely preterm newborns [19]. The authors demonstrated signifi cant associations of platelets drop with mortality and major morbidities in extremely preterm newborns. Since our local NICU population and setting might be different we aimed in this study to use a similar approach to check the reproducibility of such presumed prognostic value of platelets drop during the first 7 days of life as calculated from the initial platelets count immediately after birth, with or without later thrombocytopenia in our preterm newborns. We hypothesized that in our local setting the rate of early platelets count decline in preterm newborns could predict final NICU outcome and the severity of later thrombocytopenia.

Methods

Study Design

In a retrospective cohort study design, an analysis of the medical records of all preterm newborns admitted to NICU in our Hospital from January 2014 to December 2015 was conducted. Our hospital is a tertiary care health center that serves a large population area.

Inclusion Criteria

The criteria for inclusion of charts reviewed in this study were newborns who: (i) Have gestational age (GA) below 32 weeks; (ii) were admitted on the 1st day of life; and (iii) survived for more than 7 days.

Exclusion Criteria

Exclusion criteria included newborns who: (i) Were transferred before completing their treatment; (ii) had thrombocytopenia on admission; (iii) did not have platelets count taken on 1st day of admission (iv) had thrombocytopenia with or without decline in plateletscount before day 7 of life; (v) received blood product transfusion in the first 7 days of life

Intervention

All preterm newborns admitted in a 1-year period were identified. Needed data were extracted by examining the hospital patient database, medical files, laboratory system, and electronic records. No direct patient intervention was done. The following data were collected:

(1) Demographic and antenatal data (GA, birth weight, sex, use of antenatal steroids [completed course], and method of deliver); and

(2) Clinical data and outcome (intrauterine growth retardation, diagnosis on admission, mortality, morbidities such as IVH [grades 3 and 4 using head ultrasound by a radiologist], other major hemorrhage such as pulmonary hemorrhage [defi ned as hemorrhage requiring prompt medical intensive action as blood product transfusion and sustained medical care], NEC [presence of pneumatosis intestinalis in abdominal radiograph read by a radiologist], sepsis [blood culture proven], and length of stay [LOS] among survivors). Platelets count recorded on the 1 st day of admission was taken as the base to which subsequent platelets numbers were compared. All subsequent counts in the first 7 days of life were recorded to defi ne the lowest count in the first 7 days. Drop in the platelets counts was defined as a decrease of a ≥ 30% from the fi rst day platelets count. Thrombocytopenia in the study was defined as platelets count <150 × 10^9/L that was proven in two consecutive measurements.

Outcome Measures

Our primary outcome measures were mortality and LOS among survivors. Secondary outcome included IVH (grade 3–4), ROP (grade 3–4), NEC, and culture proven sepsis (Gram-positive and Gram-negative fungus)

Statistical Analysis

Data were analyzed using SPSS software (SPSS for Windows, version 16.0, SPSS Inc., Chicago, IL, USA). To analyze the prognostic value of platelets decline with and without thrombocytopenia included newborns were classified into four groups;

Group 1: No thrombocytopenia with no platelets decline;

Group 2: No thrombocytopenia with platelets decline,

Group 3: Thrombocytopenia with no platelets decline,

Group 4: Thrombocytopenia with platelets decline.

Continuous variable were expressed as mean ± standard deviation (SD) and categorical variables as number (percentage). Normality was checked by 1-sample Kolmogorov-Smirnov test. The assumption of a normal distribution was rejected at an alpha <0.1. Descriptive analysis was performed on demographic and baseline clinical characteristics; as well as the age of platelets count nadir, and magnitude of platelets declines in the first 7 days. Comparisons between the four groups were performed using analysis of variance and Chi-square for categorical variables. Logistic regression was used to examine for the odds of mortality and morbidities in the four groups. Variables that reached a significance level of 0.15 in univariate analysis were included in logistic regression analysis, using the Group 1 (no thrombocytopenia no drop) as reference. P < 0.05 was considered as statistically significant.

Results

Of 300 neonate admitted to the hospital in the study period, 110 preterm newborns (52 males, 58 females) were included in our study. The mean (SD) GA for our sample was 28.3 (1.6) (range, 26–32) weeks; and mean (SD) birth weight was 1025 (385) (range, 610–2400) grams. Clinical characteristics of all preterm newborns involved in the study based on study group are shown in Table 1.

Table 1: Comparison of patients characteristics between study groups

Study groups	Group 1	Group 2	Group 3	Group 4	P
N (%)	19	57	23	11	
Male: female	8:11	29:28	11:12	4:7	>0.05
Gestational age (weeks)	29.1 (1.8)	27.6 (1.5)	28.4 (1.2)	27.1 (1.1)	<0.01
Birth weight (g)	1800(450)	1010(160)	1300(180)	950(300)	<0.01
Antenatal steroids	23(74.2)	77(78.6)	36(87.8)	17(94.4)	>0.05
Cesarean section	17(54.8)	31(31.6)	33(80.5)	15(83.3)	<0.05
Age at platelets drop (d	NA	5(1.2)	NA	3(1.6)	>0.05

Group 1: No thrombocytopenia with no platelets decline; Group 2: No thrombocytopenia with platelets decline; Group 3: Thrombocytopenia with no platelets decline;
Group 4: Thrombocytopenia with platelets decline; NA: Not applicable

Table 2: Probability of death in various study groups in relation to Group 1

	OR	95% CI	P
Group 2	1.29	0.26-6.42	>0.05
Group 3	1.14	0.18-7.30	>0.05
Group 4	7.25	1.28-41.14	<0.05
Gestational age (week)	0.62	0.41-0.83	<0.01
Birth weight (kg)	0.87	0.88-0.88	<0.05

OR: Odds ratio; CI: Confidence interval

Table 3: Drop of platelet count on day 7 and odds of morbidities

	Group 2 (n=57)			Group 3 (n=23)			Group 4 (n=11)		
	OR	95% CI	P	OR	95% CI	P	OR	95% CI	P
IVH	8.18	1.05-63.56	<0.05		No cases		19.09	2.10-173.38	<0.01
Other major hemorrhages	1.28	0.14-11.87	<0.05	0.75	0.05-12.48	>0.05	8.57	0.87-83.91	>0.05
NEC	8.18	1.05-63.55	<0.05		No cases		19.09	2.10-173.38	<0.01
Gram-positive infection	1.17	0.40-3.46	<0.05	3.00	0.95-9.46	>0.05	4.16	1.10-15.8	<0.05
Gram-negative infection	4.96	1.10-22.33	<0.05	1.14	0.18-7.30	>0.05	11.60	2.10-64.0	<0.01
Fungal infection	8.18	1.05-63.56	<0.05		No cases		24.00	2.66-216.31	<0.01

OR: Odds ratio; CI: Confidence interval; IVH: Intraventricular hemorrhage; NEC: Necrotizing enterocolitis

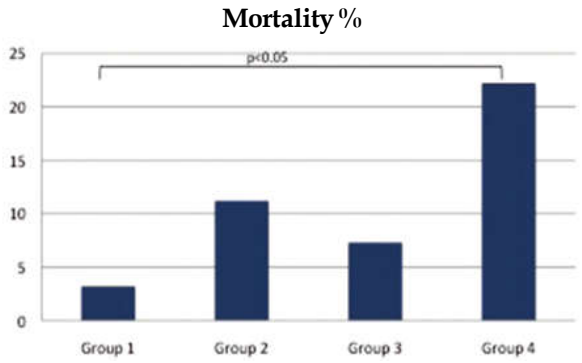

Fig. 1: Mortality rates in study groups

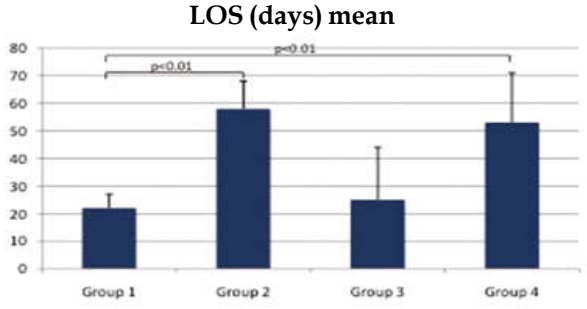

Fig. 2: Length of stay among survivors in study groups

Preterm newborns in Group 4 had significantly lower GA and birth weight than the other groups. Platelets drop occurred at an earlier age in preterm newborns, who developed thrombocytopenia. On the other hand, thrombocytopenia occurred earlier in children who demonstrated platelets drop in the first 7 days of life. The overall incidence of thrombocytopenia was 31.4% in our sample with 61.7% had ≥ 30% drop in platelets counts before the age of 7 days. The relationship between mortality and the study groups are shown in Figure 1. The mortality rates were higher in preterm newborns with ≥ 30% drop in platelets count being highest in Group 4 (both thrombocytopenia and ≥ 30% drop in platelets count). Preterm newbornswithout thrombocytopenia and ≥ 30% drop in platelets count (Group 2) had mortality rate higher than Group 1 (no thrombocytopenia with no platelets decline) and Group 3 (thrombocytopenia without platelets drop). Significance was reached only when Group 4 was compared to Group 1 ($P < 0.05$). Nineteen newborns died before discharge with an overall mortality rate in the study group of 10.1%. Table 2 shows the odds of mortality in various groups of the study. After controlling for other demographic and clinical factors, ≥ 30% drop in platelets counts was associated with significantly increased odds of mortality when it was associated with thrombocytopenia in preterm newborns. When taken together preterm newborns with ≥ 30% drop in platelets showed an increased mortality odds, (1.84 [0.63–5.34], $P > 0.05$) when compared to children with no platelets drop; however, this did not reach statistical significance. In those preterm newborns who died before discharge, mean ± SD ages of death were 33.0 (9.9), 12.7 (7.4), 20.1 (6.3), and 15.6 (7.6) days in Group 1, 2, 3, and 4, respectively. From the 110 studied preterm newborns, 29 (15.4%) showed evidence of grade 3 or 4 IVH, 10 (5.3%) had other major bleedings, 29 (15.4%) showed evidences suggestive of NEC, 46 (24.5%) had Gram-positive bacterial infection, 38 (20.2%) had Gram-negative bacterial infection, and 30 (16.0%) had fungal infection. The odds of having major morbidities in each study group are shown in Table 3. The odds of having IVH and NEC were significant ($P < 0.05$ and <0.01, respectively) in both Group 2 and Group 4 (those preterm newborns who had ≥ 30% drop in platelets either without or with thrombocytopenia). Preterm newborns in Group 3 (thrombocytopenia with no platelets decline) did not have IVH or NEC cases. The odds of other major hemorrhages were increased only in Group 4. While the odds of having Gram-positive infection was significant ($P < 0.05$) only in preterm newborns with ≥ 30% drop of platelets with thrombocytopenia (Group 4), it was not significant in both Group 2 and Group 3. The odds of having Gram-negative or fungal infection were significant in preterm newborns with ≥ 30% drop in platelets count either without thrombocytopenia (Group 2) or with thrombocytopenia (Group 4). The odds of having Gram-negative infection were not signifi cant in preterm newborns with only thrombocytopenia without platelets drop (Group 3) and there were no cases of fungal infection among preterm newborns in this group.The association between LOS and platelets evaluation was shown in Figure 2. There was significant increase in the LOS in preterm newborns with ≥ 30% drop of platelets count either without thrombocytopenia (Group 2; $P < 0.01$) or with thrombocytopenia (Group 4; $P < 0.01$) when compared to the normal preterm newborns (Group 1) while LOS of Group 3 was not signifi cantly different from those of normal preterm newborns (Group 1; $P > 0.05$). m There was no significant difference in LOS between Group 2 and Group 4 ($P > 0.05$).

Discussion

Although drop in the platelets count without thrombocytopenia is a common observation in preterm newborns; usually, it neither trigger action nor used to draw conclusions on expected clinical coarse or prognosis except after reaching the

threshold of thrombocytopenia or even severe thrombocytopenia [8,20]. Our study confi rmed the signifi cant association between drop of platelets with or without thrombocytopenia and poor outcomes. Our findings agree with the results of Rastogi et al., who demonstrated a significant association of mortality and major morbidities in preterm newborns below 28 weeks gestation and platelets drop in the first 7 days of life [19]. Previous studies in critically ill older children and adults have shown similar results [13-15]. The rate of thrombocytopenia in our study (31.4%) was much lower than that reported previously in preterm newborns [9]. This is mostly a refl ection of excluding thrombocytopenia occurring in the first 7 days of life that is mostly caused by maternal causes. Using same criteria, the incidence of thrombocytopenia in extremely preterm neonates below 28 weeks gestation was found to be 48.6% [19]. The authors believed that this incidence of thrombocytopenia in their study was more likely to be refl ective of primary neonatal causes. However, other authors have published thrombocytopenia rates similar to ours when calculated for all babies admitted to NICUs [1,21].

The platelets drop and thrombocytopenia in newborns have traditionally been attributed to a combined process; impaired platelets production and increased platelets consumption and sequestration [22]. Impaired platelet production usually results in low platelet count that is either present at birth or develops within 72 h of life [21-23].

In our study, it is reasonable to assume that the decreased production play a little role as we excluded all newborns with early thrombocytopenia. Hence, the most plausible explanation for the platelets drop and thrombocytopenia in our study is increased consumption and sequestration.

It was shown that late onset thrombocytopenia in newborns is almost exclusively caused by sepsis or NEC [24]. Thrombosis and platelet activation/immobilization at sites of infl ammation (as in the gut during NEC) were suggested as the processes behind platelets consumption in such conditions [23]. Such affected neonates were often profoundly sick, required intensive care, and had 10–15% mortality [24]. In our study NEC, Gram-negative bacterial and fungal infections were associated with platelets drop even in the absence of thrombocytopenia. Platelets drop occurred several days before reaching the level of thrombocytopenia and mostly before other signs of illness appeared. As such, platelets drop in the first 7 days of life represents a strong indicator of the later development of these two problems (infections and NEC). Another group found that such prognostic value of platelets drop remained valid in predicting mortality and serious morbidities at 28 days of life [19].

Many authors have expressed doubts regarding whether thrombocytopenia itself directly contributes to adverse outcome or is simply a marker of the severity of precipitating complications, which themselves carry a poor prognosis [21,23,25]. Our results would lend evidence to the latter reasoning and extend this reasoning to platelets drop not only thrombocytopenia. Indeed the early use of platelet concentrates to prevent moderate thrombocytopenia (platelets, 50–150 × 10^9/L) failed to reduce hemorrhage [26], reflecting the diffi culty clinicians face to assess the clinical impact of thrombocytopenia in newborns. As regards type of infection, our results suggest a strong correlation between platelets drop with or without later thrombocytopenia in preterm newborns and both Gram-negative bacterial and fungal infections. Gram-positive bacterial infection odds increased only in newborns that had shown thrombocytopenia after early platelets drop. Thrombocytopenia happening without prior early platelets drop did not show any association with any type of infection. These findings agree in part with the results published by Rastogi et al. [11], who mreported Gram-negative bacterial or fungal infections happening only in newborns with platelets drop with or without thrombocytopenia. In our study, we still see children with Gram-negative bacterial infection in the other two groups (no drop, no thrombocytopenia and thrombocytopenia without a drop). On the contrary to our findings, they reported signifi cant associations between platelets drop with or without thrombocytopenia and Gram-positive bacterial infections [19]. These differences might be attributed to the differences in the local NICU environments as rates of nosocomial infections and predominant environmental pathogens; as well as clinical and demographic backgrounds of newborns included in the study. Actually, our reported rates of various types of infections are higher than those reported in other units [19]. However, although infections have been recognized as a factor that enhances platelets destruction [27], the role of specific type of infection or organism is controversial [17,19].

Intraventricular hemorrhage was associated with platelets drop even if no thrombocytopenia developed. There were no IVH cases in the group that developed thrombocytopenia without prior observed platelets drop. In previous works association between IVH in preterm newborns and thrombocytopenia was questioned. In patients with severe thrombocytopenia and in contrast to cutaneous and gastrointestinal hemorrhage no relationship between the lowest

platelet count recorded, and the presence of IVH or pulmonary hemorrhage were found[28]. Furthermore, it was not clear how to interpret the association seen in some studies between lower platelets counts and higher prevalence of IVH. Two possible explanations have been propagated; thrombocytopenia might have caused the IVH, or it was a result of IVH through a consumptive process [25,28]. Our results illustrate that probably thrombocytopenia is not the main trigger for IVH and other factors contribute to its pathogenesis.

This study is mainly limited by the nature of its design. Being a retrospective study might have influenced several key variables validity resulting in both selection and information biases. The collection of samples in our study was part of routine investigations that were not scheduled on similar timing for all patients, which may have resulted in missing some newborns with a drop of platelets or thrombocytopenia. Furthermore, definitions of morbidities such as IVH, infections, and NEC; as well as investigating newborns for them, were largely dependent on the treating physician. Newborns with thrombocytopenia were subjected to investigations as head ultrasound, blood cultures, and abdominal X-rays more than newborns without thrombocytopenia. This might have falsely increased the association between thrombocytopenia and such morbidities.

Conclusion

Our study highlights, the need to consider not only thrombocytopenia but also platelets drop when predicting the outcome and major morbidities in preterm newborns. The early platelets drop even without the later development of thrombocytopenia is an early indicator of poor outcome and major morbidities, mainly infection. There is a need to investigate these observations in prospective study design.

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Isolated Gastric Outlet Obstruction - Sequelae of Corrosive Ingestion in Paediatric Age-Group: Literature Review

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Abstract

Background: Accidental corrosive ingestion is common in the paediatric age group. Severity may vary from no injury to a fatal outcome [1]. However, isolated gastric outlet obstruction (GOO), though rare, is a well-known complication of corrosive ingestion. We present five such cases. *Cases' Summary:* The mean age of presentation was 5.4 years. There were 4 males and 1 female. The average time duration between ingestion of the corrosive and presentation was 40 days. The common presenting symptoms were abdominal pain and non-bilious vomiting mixed with food particles seen in all five patients. Endoscopy was done in four patients - all four patients had antral and pyloric thickening. The scope could be negotiated across the pylorus only in one patient with difficulty. It could not be negotiated in the other three patients. Retrocolic, isoperistaltic gastrojejunostomy was done in four patients and pyloroplasty was done in one patient. Post-operative recovery was uneventful in all patients. *Conclusion:* Corrosive ingestion is an important cause of significant morbidity, especially in developing countries. Early diagnosis and appropriate endoscopic and/or surgical intervention usually lead to a good outcome.

Keywords: Ingestion; Corrosives; Gastric Outlet Obstruction; Total; Partial; Children.

Introduction

Accidental corrosive ingestion is a common problem in the paediatric age group [1]. In children, alkalies are the ingested agents in about 80% of cases [1, 2, 3]. Severity may vary from no injury to a fatal outcome [1].

Isolated gastric outlet obstruction (GOO), though rare, is a well-known complication of corrosive ingestion [1]. There is a paucity of such reports in literature and most reports are in adults in which various management modalities with varied outcomes are discussed [1]. The management of such injuries is challenging.

We report five cases of gastric outlet obstruction following accidental corrosive ingestion in children.

Cases' Summary

Demographic Details

Five children with corrosive ingestion and clinical features of GOO and their management are described. The mean age of presentation was 5.4 years (range-3 years to 6 years). There were 4 males and 1 female. The time duration between ingestion of the corrosive and presentation was 40 days (range: 20 days to 60 days).

Clinical Features

The common presenting symptoms were abdominal pain and non-bilious vomiting mixed with food particles seen in all five patients. The female child presented with severe dehydration, acidosis

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and electrolyte imbalance along with these symptoms. Visible peristalsis was seen in four patients.

Management and Outcome

All patients were admitted. Fluid and electrolyte balance was corrected. An upper gastrointestinal tract contrast (water soluble) study was done. Endoscopy (oesophago-gastro-duodenoscopy) was done in four patients. All five patients had grossly dilated stomach on contrast study. There was small streak of dye going distally across the pylorus in two patients (Figures 1, 2 and 3). Three patients had complete GOO (Figure 4).

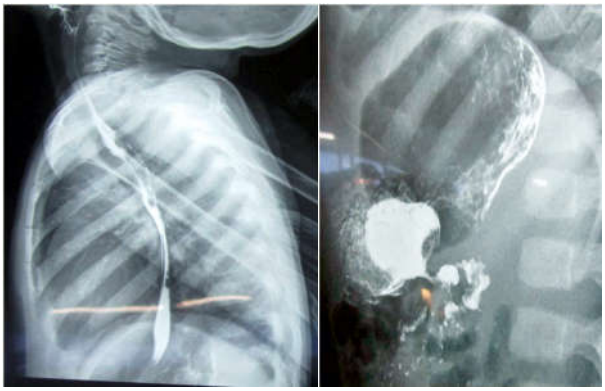

Fig. 1 and 2: Upper gastrointestinal contrast X-rays (Lateral and Antero-posterior) of a patient showing partial Gastric Outlet Obstruction.

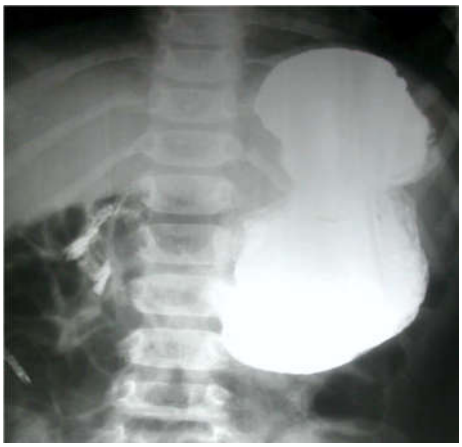

Fig. 3: Upper gastrointestinal contrast X-ray of a patient showing total Gastric Outlet Obstruction

At endoscopy, all four patients had antral and pyloric thickening. The scope could be negotiated across the pylorus only in one patient with difficulty. It could not be negotiated in the other three patients.

Exploratory laparotomy was done in all patients. One patient had only pyloric thickening and pyloroplasty was done. Four patients had grossly dilated stomach with thickened and stenosed pylorus and antrum. A retrocolic, isoperistaltic gastrojejunostomy was done . Post-operative recovery was uneventful in all patients. Orals were gradually started at fifth post-operative and patients were discharged after one week. At follow-up, all patients are aymmptomatic and are gaining weight.

Discussion

Both acids and alkalies act as corrosives after ingestion and produce considerable and progressive damage to the upper gastrointestinal tract [4]. The nature of the agent, the amount and concentration ingested, the amount of food already present in the stomach at the time of ingestion and the intention (suicidal or accidental) of ingestion are the factors affecting the degree of mucosal injury [4, 5]. Presence of a pre-existing comorbid condition compounds the damage [4]. The burden of morbidity caused by the corrosive substances is more in developing countries because of the easy availability of these agents as items of household use which are not under regulatory control [4].

Human exposure to these corrosive agents is usually either accidental or suicidal, the circumstances being different in paediatric and adult populations [4]. Accidental ingestion of corrosive substances is seen most common in young children (80%) [4]. Children under 5 years of age are curious by nature and unaware of the dangers of these toxic agents [1]. Hence, they are more prone to accidental ingestion of these corrosives [4]. This coupled with easy availability of corrosive substances in the form of cheap toilet cleaners which is kept in empty bottles of mineral water is a common cause of such incidences in developing countries like India [4]. Isolated pyloric and antral stenosis without oesophageal injury, though uncommon has been reported. There are only few reports in literature which highlight the management of corrosive stricture of pylorus and antrum of the stomach in children [5, 6].

There is a common belief that alkali ingestion causes severe esophageal damage and limited gastric injury due to the buffering action of acid [7]. However, pyloric stenosis has recently been reported to occur with corrosive alkali ingestion [1,3,7,8,9]. There is a well known tendency of acids "to lick the esophagus and bite the pyloric antrum" [5]. The lower viscosity and specific gravity of corrosive acids when compared to liquid alkalis, lead to their rapid transit through the esophagus and the damage primarily occurs in the antrum and pyloric region of the stomach

[5, 10]. This rapid transit is coupled with antral spasm which causes pooling of the corrosive consequently causing more damage to the antrum [5]. The stomach, with its columnar epithelium is more susceptible to corrosive damage when compared with oesophagus which has a more resilient squamous epithelium [5, 11]. As suggested by Nuutinin et al, acids cause burn injuries more often than alkalies which more often develop into scars as a result of coagulation necrosis of the tissue in contact [1]. With a severe injury to the stomach, gastric outlet obstruction (GOO) may occur as early as three weeks or as late as 10 weeks [7].

The children usually present with non-bilious vomiting, post-prandial fullness followed by early satiety, decreased oral intake and rapid weight [1, 5]. Patients may present with acute complications like oesophageal perforation (<2% of cases), aspiration pneumonitis and respiratory failure [4]. In patients who present early, after stabilization, an upper gastrointestinal endoscopy is recommended to characterize the nature of injury, if it can be done between 48 and 72 hours of ingestion [4,12]. However, endoscopy is not recommended between 5 and 1 days of ingestion due to a high risk of perforation [4]. The corrosive injuries are graded endoscopically as grade 1, 2a, 2b, 3 and 4 as per the classification system proposed by Zarger et al [4,13]. It has been reported that sequelae like oesophageal and/or gastric stricture and cicatrization is more common with grades 2b (penetration to the submucosa with ulceration or whitish membranes) and 3 (transmural involvement with deep injury and necrotic mucosa) injuries [1,4, 13-16]. A contrast study is recommended to confirm the injuries in such patients and identify the complications [1]. Oesophageal injuries, strictures and cicatrization are more commonly reported as long-term sequelae; however, gastric antral and pyloric injuries causing GOO is also not uncommon in children [4,5,17]. Both alkalies and acids are known to cause GOO.

The management of GOO in such paediatric patients is controversial because of the paucity of literature [1,5,17,18]. However, early surgical intervention has been recommended as the treatment of choice with satisfactory results in various studies [1,5, 7, 20].

The various management options are feeding jejunostomy and endoscopic balloon dilatation of stricture, gastrojejunostomy with or without vagotomy, pyloroplasty, or antrectomy with Billroth I anastomosis [5, 19, 20, 21]. Each of these various procedures has its own advantages and disadvantages [5].

Pyloroplasty and endoscopic procedures like balloon catheter dilatation and intralesional steroid injection are usually indicated in cases with partial obstruction with moderate mucosal injury [1, 5, 22]. However, data of endoscopic management is lacking in children [1]. Erdogan et al has reported successful management of a partial pyloric obstruction by endoscopic balloon dilatation [1, 3]. Pyloroplasty has been reported to be safe, simple and fast procedure for partial GOO in children in many studies [1,4,8,9, 23]. However, there are reports of recurrence requiring surgical intervention [1, 8].

The two procedures commonly performed for complete GOO are gastroenterostomy and gastrectomy [1]. Ozcan et al and Chaudhary et al have reported gastrojejunostomy for complete GOO in children with good long-term results [1, 2, 24]. Gastrojejunostomy is simple, safe and usually has good outcomes in cases with poor nutrition status, extensive perigastric adhesions and unhealthy duodenum [1,5,24,25,26].

Gastric resection is usually reserved for extensive gastric cicatrization [5]. However, it becomes a major surgery in nutritionally depleted patients and has its associated morbidities [5]. Many surgeons have doubts about the risks and benefits of gastric resection in children [1]. Kaushik et al, Ciftci et al, Tekant et al and Erdogan et al have reported Billroth I procedure in children with good results [1,2,8,9,27].

The decision of additional vagotomy and a drainage procedure is usually considered taking into account the diminution of acid and pepsin production due to damage of glandular elements [5,28].

Though rare, paediatric GOO after corrosive ingestion has good outcome with early diagnosis and appropriate management.

Conclusion

Corrosive ingestion is an important cause of significant morbidity, especially in developing countries. Parent education regarding storage of household corrosives away from the children, stringent legislation to curtail unrestricted access of adults to harmful corrosive chemicals and safe packaging of these chemicals in child proof containers with correct labeling are warranted. Early diagnosis and appropriate endoscopic and/or surgical intervention usually lead to a good outcome.

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Review Article

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Significance and Methods for Evaluation of Breast Size among Women

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Abstract

Breastfeeding is one of the important aspects of a woman's life. Asymmetry and/or small size of the breast(s) have important psychological consequences for the mother about her capacity for milk production and ability to breastfeed her baby. Adequate counseling in these cases helps allay anxiety of mothers regarding adequate milk production. Most authors have studied breast size for aesthetic purposes and most studies are on small number of subjects including few adolescents to 125 women and age ranging from 16 to 83 years. 'Aesthetically pleasing breast' have been studied by many authors and defined as size and fullness proportional to the body, have minimal ptosis and no axillary tail, be conical to teardrop in shape, and have the nipple at the anterior most position [12]. Results from aesthetically perfect breasts as well as breast size of the adolescent nulliparous women cannot be extrapolated to the lactating women, hence the requirement of developing nomograms for use during lactation councelling. Breast measurements can be used for counseling mothers for successful breastfeeding and for aesthetic and plastic surgery for re-shaping of the breast. Lactation promotion clinics require such data for adequate counseling of breastfeeding mothers and this decreases the likelihood of discontinuation of lactation. Breast measurements can be used for counseling and aesthetic and plastic surgery for shaping of the breast. Developing nomograms of breast measurements & breast mass/volume estimations can help to counsel lactating mothers regarding adequacy of breast volume.

Keywords: Breast size; Lactation; Lactation counseling.

Introduction

Breastfeeding is fundamental to the growth and development, and survival of the newborn infant as well as wellbeing and health of the mother. Breastfeeding is one of the important aspects of a woman's life. Shape, size, position and asymmetry of the breast(s) have various important psychological consequences on the mother regarding her capacity of milk production and the ability to breastfeed her baby.

The macroanatomy of the female breast comprises of nipple, areola and stroma. The lactating system of the breast consists of alveoli, lactiferous ducts, lactiferous sinuses and lactiferous ductules. The alveoli are made of very small milk secreting cells. Lactiferous ducts carry milk from the alveoli towards the areola. Beneath the areola, many lactiferous ducts coalesce, become wider and form lactiferous sinuses. About 10-20 fine lactiferous ductules transport milk from the lactiferous sinuses to the nipple. The lactating system is surrounded by connective tissue and fat. It is the fat and connective tissue which gives the breast its shape and size [1]. However, the number of milk producing cells in the breasts of all women are almost similar.

The breast(s) attains its hemispherical shape at puberty. Variations in size and position are affected by age of the woman and activity of the gland. During

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pregnancy and lactation, the breasts increase two to three times in size. Following cessation of lactation, breast size decreases and at times become more pendulous.

Shape, size, position, asymmetry, and hypoplasia of the breast(s) has many psychological consequences on the mother making her doubt her capacity of milk production, and the ability to breastfeed the baby, and loosing self confidence due appearance of the breasts [2]. Some women are highly distressed by the shape and size of their breasts and need counseling and adequate treatment. Perceived changes in the appearance of the breast also influence a woman's decision to breastfeed [3]. Breast hypoplasia may be due to imperfect formation of primitive breast tissue during development. It is also seen in ovarian hypofunction and this is amenable to hormonal treatment.

Asymmetry between the sizes of the breasts is often seen during the normal stage of breast development [4] and when breastfeeding is done predominantly from one breast [5]. The breast bud frequently begins to develop on one side before it does on the other. During adolescence one of the breasts may respond better to the circulating hormone and grow bigger than the other breast but eventually both breasts attain same shape and size.

Adequate counseling in these cases helps allay anxiety of mothers regarding adequate milk production. Successful breastfeeding can be done even in deformed breast condition by proper education, counseling and empowerment of the mother and family [6]. Significant difference between the two sides or persistence of difference between the two sides in a growing adolescent, suggestive of asymmetry can be considered for cosmetic correction, after all other underlying pathologies are ruled out.

Most authors have studied breast size for aesthetic purposes and most studies are on small number of subjects including few adolescents [7] to 125 women [8] and age ranging from 16 to 83 years [8,9,10,11]. Aygun et al [7] studied diameter of the nipples and areola of 498 pubertal girls. Kalbhen et al measured breast size on mastectomy samples only [11].

'Aesthetically pleasing breast' have been studied by many authors and defined as size and fullness proportional to the body, have minimal ptosis and no axillary tail, be conical to teardrop in shape, and have the nipple at the anterior most position [12]. Results from aesthetically perfect breasts as well as breast size of the adolescent nulliparous women cannot be extrapolated to the lactating women, hence the requirement of developing nomograms for use during lactation councelling.

Breast measurements can be used for counseling mothers for successful breastfeeding and for aesthetic and plastic surgery for re-shaping of the breast. A simple technique of breast measurement was described by Capraro and Dewhurst in 1975 [2]. It does not require sophisticated equipments and uses only anthropometric measurements to evaluate the breast size and assess the asymmetry between the breasts.

In Capraro and Dewhurst study, the measure 'breast unit' shows an increase in value (indicating an increase in size of breasts) as the adolescent grows. Measurements of breast unit on a normal adolescent girl- Right breast unit at 11,12,14,15 & 16 years was 24.75, 180, 247, 357 & 396 and Left breast unit values at corresponding years were 13.5, 170.5, 252, 340 & 378 respectively, showing a progressive increase with age [2].

Breast size of mothers having even the lowest breast unit- 255 have been found to have measurements similar to those found in the adolescent age group and yet are able to breastfeed the babies well & have sufficient milk output.

Various other methods employed for breast measurements use complex measurements such as mammography [11], torso measurements [12], 3-D imaging [13,23,24,25], casting techniques [14], ultrasound imaging of the breast [11,19,22], water displacement techniques [21], and Grossman disk [20,21]. The wide range of techniques available yield varied results and are sometimes difficult to interpret.

The methods considered most accurate are the direct volume measurement techniques such as plaster casts, paraffin models and water displacement techniques [19]. However, these are time consuming and cumbersome.

Casting methods are conducive to deformation of the breast and may alter the volume measurements. Water displacement techniques are useful for biopsy, pathology, post-surgical and post-mortem samples. Patient stated brassiere cup-sizes have been found to be a poor proxy for the actual measurements of the breast and cannot be used for surgical procedures as it requires accurate measurements. Hence, the constant need to develop accurate and simple measurement techniques.

Breast volumes have been found to be positively correlated to body weight, and chest, lumbar and buttock circumference, and negatively correlated to height [13]. Symmetrical breast has been defined as < 5.6% [9] or < 50 cc difference in the breast volume between the right and left breasts [15].

Breast volume varies during the menstrual cycle and pregnancy. Breast volume is an indicator of whole organ change reflecting responses to the pathologic, physiologic, pharmacologic and environmental factors. It has been reported that breast volume increases by 145±19 ml during pregnancy to 211±16 ml by 1 month of lactation [16,17]. Breast volume also increases in cancer/ tumors due to increase in underlying tissue. Pregnancy and lactation associated breast enlargement is related to hormonal influence on the breast tissue and is independent of maternal age, parity status and frequency of breastfeeding.

The areolar diameter makes up about 25-30% of the breast hemi-circumference. Hauben et al [18] have shown that nipple-areola-breast proportion is 1:3:3 in non-lactating women. The nipple is generally longer in multiparous mothers. Mothers with small nipple projection length are also able to feed the babies well and small nipple does not pose any difficulty in breastfeeding [6].

Lactation and IYCF (Infant and Young Child Feeding) clinics focus on the benefits of breastfeeding, anatomy and physiology of the breast, techniques of breastfeeding, care of the breast(s) and common problems likely to be encountered during lactation and their remedies in order to help resolve maternal apprehensions regarding breast size, and adequacy of breastfeeding. These antenatal/IYCF/lactation promotion clinics can help resolve maternal apprehensions regarding adequacy of breast size during lactation and breastfeeding of the baby.

Lactation promotion clinics require such data for adequate counseling of breastfeeding mothers and this decreases the likelihood of discontinuation of lactation. Breast measurements can be used for counseling and aesthetic and plastic surgery for shaping of the breast. Developing nomograms of breast measurements & breast mass/volume estimations can help to counsel lactating mothers regarding adequacy of breast volume. Studies may be conducted for various ethnic groups/populations for determining their anthropometric parameters and volume nomograms of the breast.

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Case Report

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Jacobsen Syndrome: Are We Informative Enough

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Abstract

Jacobsen syndrome is a rare syndrome caused due to deletion of long arm of chromosome 11. Typical clinical manifestations include physical growth retardation, mental retardation, facial dysmorphism, congenital heart disease, thrombocytopenia. The patient admitted with us at one and a half year had facial anomalies including flat occiput, prominent forehead, trigonocephaly, blue sclera, downslanting palpebral fissure, broad nose, hypertelorism, low set ears, high arched palate with bilateral eversion of foot and thrombocytopenia. Karyotyping confirmed deletion of chromosome 11q.

Keywords: Jacobsen Syndrome; Trigonocephaly; Thrombocytopenia; Heart Defects; 11q Deletion.

Introduction

Jacobsen syndrome (JS) is a rare disorder, yet is a clinically recognisable condition with multiple dysmorphic features. The deletion in most cases involves chromosome 11q.[1]. However fryns *et al.* [2] suggested that the deletion of sub band 11q24.1 is crucial for the clinical presentation. The deletion size ranges from ~7 to 20 Mb, with the proximal breakpoint within or telomeric to sub band 11q23.3 and the deletion extending usually to the telomere. The deletion is de novo in 85% of reported cases, and in 15% of cases it results from an unbalanced segregation of a familial balanced translocation or from other chromosome rearrangements. In a minority of cases the breakpoint is at the FRA11B fragile site [3]. More than 200 cases of JS have been so far reported in the literature[4,5]. The estimated occurrence of JS is about 1/100,000 births [3,4,5]. The female/male ratio is 2:1.

It has a varied spectrum of phenotypic variability, the most consistent being mild to moderate psychomotor retardation, trigonocephaly, facial dysmorphism in the form of skull deformities, hypertelorism, ptosis, coloboma, downslanting palpebral fissures, epicanthal folds, broad nasal bridge, short nose, v-shaped mouth, small ears, low set posteriorly rotated ears & thrombocytopenia. A subset of patients have malformations of the heart, kidney, gastrointestinal tract, genitalia, central nervous system and/or skeleton. Ocular, hearing, immunological and hormonal problems may be also present [3,6].

Case Report

The patient is one and a half years old female born out of non-consanguineous marriage to healthy parents. The pregnancy was uneventful and the child was born at term with a birth weight of 1900 grams. The first presentation of the child was at one and a half years of age with complaints of difficulty in hearing and developmental delay. Examination revealed flat occiput, prominent forehead, trigonocephaly, blue sclera, downslanting palpebral fissure, broad nose, hypertelorism , low set ears, high arched palate with bilateral eversion of foot. Anthropometric measurements suggested < -3 Z score

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for weight, -2 z score for length & microcephaly. Developmental delay in all motor, language & social sectors was present. Investigations revealed thrombocytopenia & BERA suggested mild hearing loss. Chromosomal analysis suggested 11q deletion with karyotype designated as 46, xx, del (11q). Thyroid profile, MRI brain, echocardiography, abdominal imaging and opthalmologicevaluations were normal.

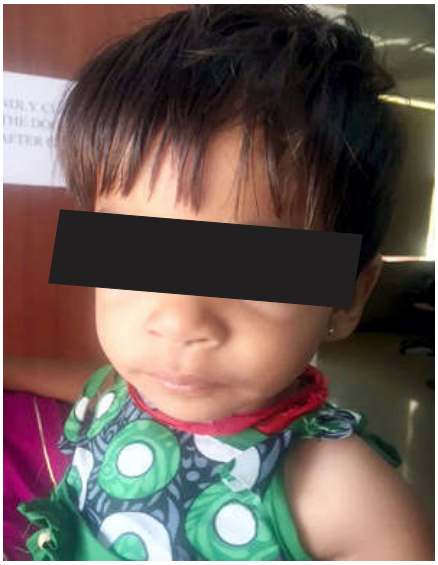

Fig. 1:

Discussion

Jacobsen syndrome is a rare disorder with multiple dysmorphic features. The deletion in most cases involves chromosome 11q. A detailed review of published reports shows that the severity of the observed clinical abnormalities in patients with Jacobsen syndrome is not clearly correlated with the extent of the deletion. The apparent lack of phenotype-karyotype correlation is possibly attributed from undetected mosaicism to redundant gene loci.

There has been an apparent abnormal sex ratio deviating towards the females and ours was also a female patient. Thrombocytopenia observed is usually chronic which was seen in our patient and has to be seen for any association with Paris-Trousseau syndrome in the form of giant platelets and abnormal megakaryocytes in bone marrow. None of these were present in our patient.

On a classical phenotype the diagnosis is suspected on the basis of clinical findings; facial dysmorphism, developmental delay & thrombocytopenia. Cytogenetic analysis is needed for confirmation. Children with JS share some clinical features (short stature, short, wide, sometimes webbed neck, downslanting palpebral fissures, ptosis, aortic or pulmonary stenosis) with Turner and Noonan syndromes. Occasionally, JS children have had a clinical diagnosis of Kabuki syndrome (mental retardation, unusual palpebral fissures, short stature, fingerpads). Thus the differential diagnoses are needed to be kept in mind while assessing.

Dalm *et al.* have recently shown that a subset of JS patients suffer from an impaired adaptive immune response, that is, defects in antibody production. Most patients with JS suffer from combined immunodeficiency in the presence of recurrent infections. Early detection of immunodeficiency may reduce the frequency and severity of infections.[7] There is a wide range of severity of intellectual disability (ID) in JS [8]. Akshoomoff *et al.* had studied 17 JS patients and eight of these patients, including four out of five males and four out of twelve females, fulfilled the diagnostic criteria for Autistic Spectrum Disorder [9].

Management is multi-disciplinary and requires evaluation by a pediatrician, pediatric cardiologist, neurologist and ophthalmologist. Auditory tests, blood tests, endocrine and immunological assessment and follow-up should be offered to all patients. Cardiac malformations can be very severe and require heart surgery in the neonatal period. Newborns with Jacobsen syndrome may have feeding difficulties and tube feeding may be necessary. Special attention should be devoted to hematological problems.About 20% of children die during the first two years of life, most commonly due to complications from congenital heart disease, and less commonly from bleeding. For patients who survive the neonatal period and infancy, the life expectancy remains unknown [10].

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An Unusual Location of Fetus in Fetu: A Rare Case Report

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Abstract

Fetus in Fetu is a rare congenital developmental anomaly, occurs in monozygotic diamniotic twin.It is due to the presence of one of the twin in the body of the other. Most frequently located in the retroperitoneal area, however it has been reported in other locations as well. Hence we report a case of Fetus in Fetu with unusual location. It is the first case in our institution among 323 twin pregnancy during january 2014 to june 2016

Keywords: Fetus in Fetu; Teratoma; Twin Pregnancy.

Introduction

Fetus in fetu is a rare benign congenital anomaly in which the malformed and parasitic fetus grows within the body of its twin. It occurs in the monozygotic diamniotic twin. Incidence is 1 per 500000 births. Most commonly located in the retroperitoneal area, other locations includes head, sacrum and scrotum. Hence, we report a case of Fetus in Fetu presented in the gluteal region, diagnosed and treated in our institution.

Case Report

A 27 year old G2P1L1 mother delivered a female baby through LSCS due to foetal distress. Baby cried immediately after birth, APGAR score was 7/10, weighing about 2.830kg. On examination of the baby there was a swelling in the right gluteal swelling extending upto the thigh,measuring about 15x15cm (Figure 1). On palpation there was loose bones palpable within the swelling(bag of bones) .

X-ray revealed a soft tissue mass with calcified osseous structures (Figure 2 & 3). Ultrasonogram gave an impression as Teratoma with multiple long bones with cartilaginous head with fluid and soft tissue component. CT scan showed a mass with solid,cystic and calcified components. AFP Levels were 1638 IU/ml, beta HCG 0.29m IU/ml. Baby was taken up for surgery with a pre operative diagnosis of polymyelia.Intraoperatively there was a vestigeal lowerlimb and rudimentary parts (Figure 4).

Grossly the specimen showed a malformed vestigeal lower limb, external genitalia and anal orifice leading to a blind pouch. Also received 2 fragments of acetabulum (Figure 5 & 6).

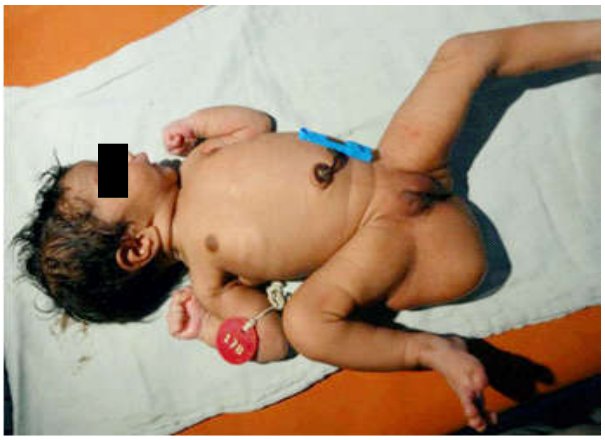

Fig. 1: Image showing swelling in the right gluteal region

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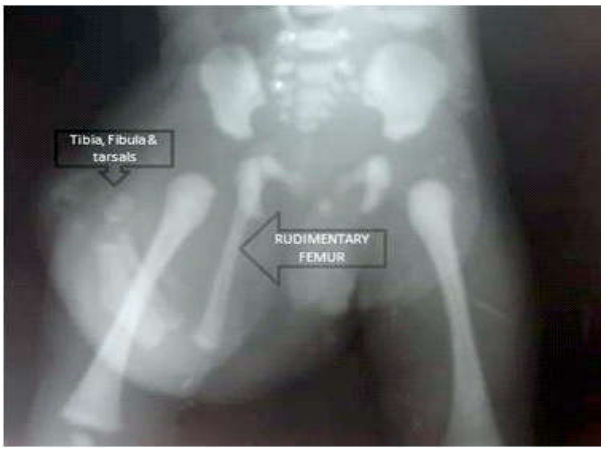

Fig. 2: X-ray images showing soft tissue mass and rudimentary bone

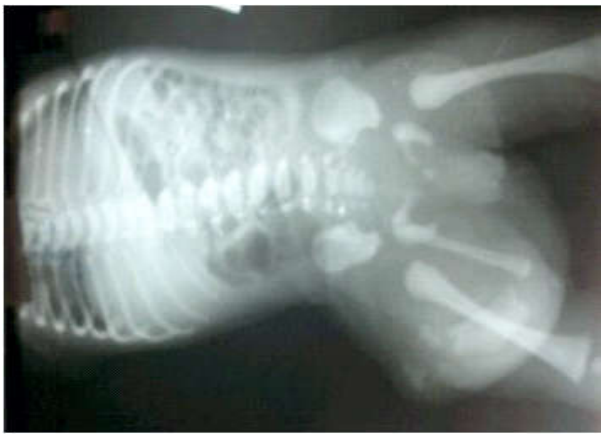

Fig. 3: X-ray images showing soft tissue mass and rudimentary bone

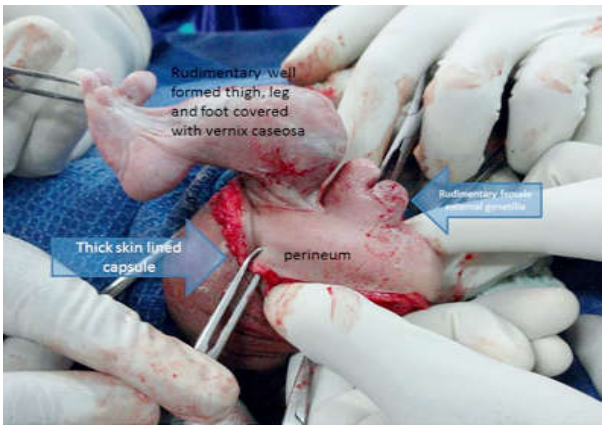

Fig. 4: Intraoperative image showing a vestigeal lower limb and external genitalia

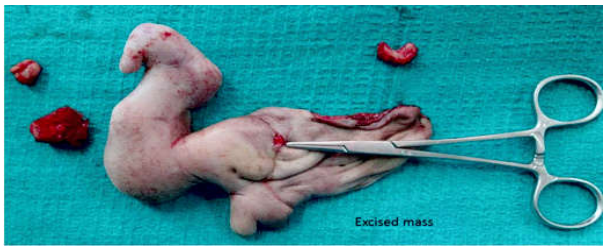

Fig. 5: Image showing external surface of the gross specimen-vestigeal lowerlimb, external genitalia, 2 fragments of acetabulum.

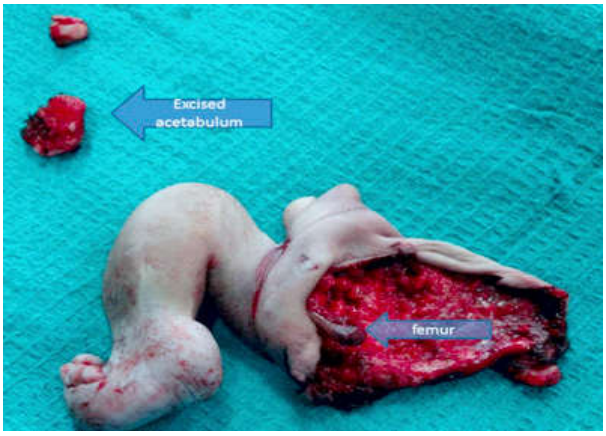

Fig. 6: Image showing external surface of the gross specimen-vestigeal lower limb, external genitalia, 2 fragments of acetabulum.

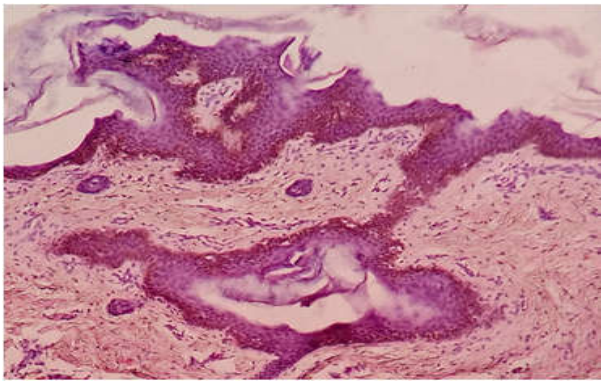

Fig. 7: Showing skin (H&E, 40X)

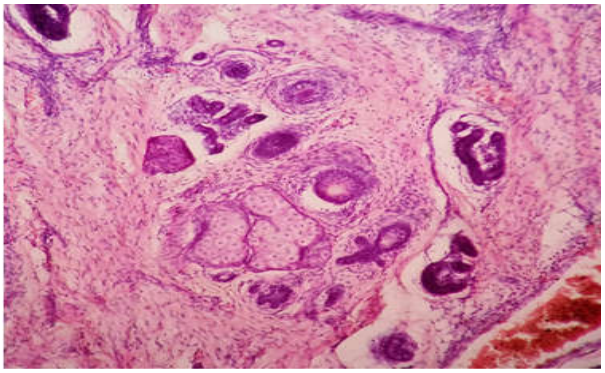

Fig. 8: Showing adnexal structure (arrow) H&E under 40x magnification

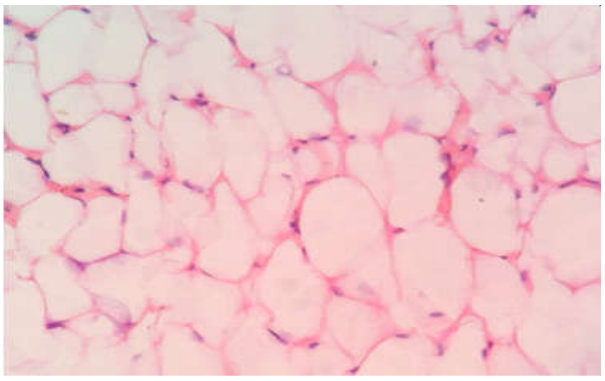

Fig. 9: Showing mature adipocytes(H&E, 40X), Nerve bundle (H&E 40X)

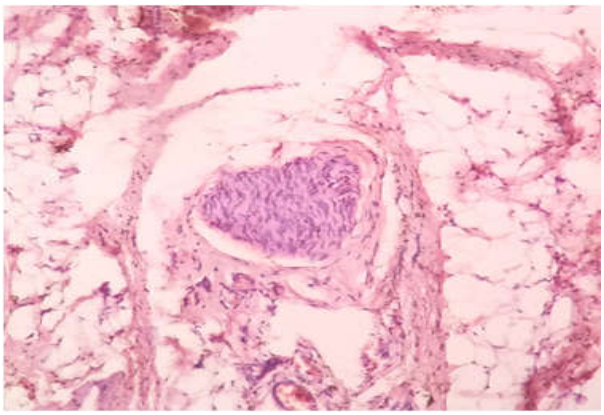

Fig. 10: Showing mature adipocytes(H&E, 40X), Nerve bundle (H&E 40X)

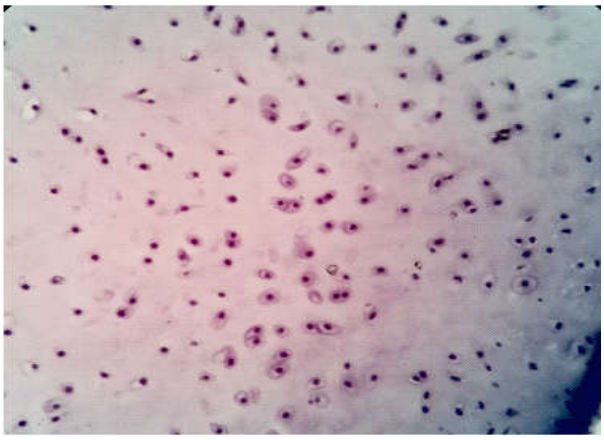

Fig. 11: Showing mature cartilage (H&E, 40X) and acetabulum (H&E,10X)

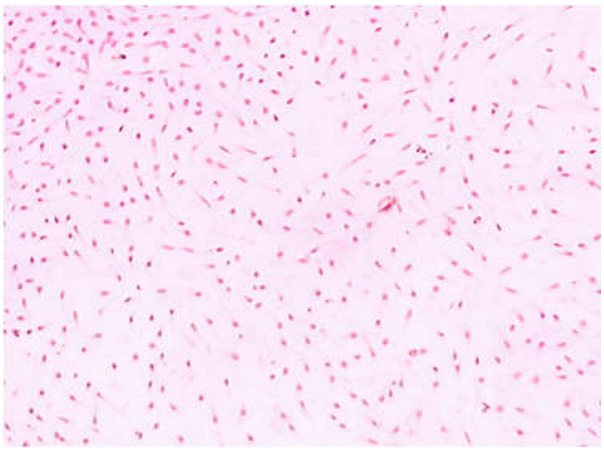

Fig. 12: Showing acetabulum(H&E, 40X& 10X)

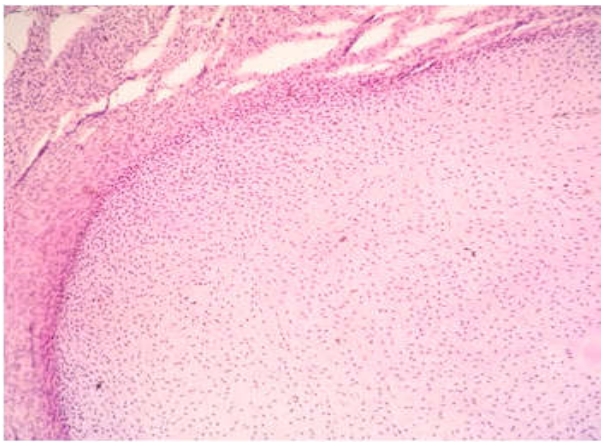

Fig. 13: Showing acetabulum(H&E,40X& 10X)

Multiple sections showed skin (Figure 7) with adnexal structures (Figure 8), mature adipocytes and nerve bundles (Figure 9 &10). Focal areas shows mature cartilages (Figure 11). Sections from the acetabulum shows cartilaginous areas (Figure 12 & 13). Pathological diagnosis was fetus in fetu. Post operative period was uneventful. Baby was discharged without any complications.

Discussion

Fetus in Fetu is a rare congenital anomaly,defined as the existence of parasitic, monozygotic, diamniotic fetus in the body of its twin. It is an unequal division of totipotent inner cell mass of the developing blastocyst leading to the inclusion of a smaller cell mass within a maturing sister embryo [1]. Incidence is 1/500000 births [2]. Most frequently located in retroperitoneal region(80%). It is always detected as an abdominal mass in infancy. Retroperitoneum being the commonest site, however there have been few reported cases of Fetus in Fetu in head, sacrum, scrotum, liver and mediastinum.

There are two theories for Fetus In Fetu. One being Teratoma theory , it may be highly differentiated form of dermoid cyst, itself a highly differentiated form of mature teratoma. Other theory is parasitic twin theory. Fetus in fetu may be a parasitic twin fetus growing within its host twin.Very early in the monozygotic twin pregnancy, in which both fetus share a common placenta. One fetus wraps around and envelope the other. The enveloped twin becomes a parasite.The parasitic twin is anencephalic and lacks some internal organs and as such it is unable to survive on its own [3].

In review of the literature showed that in about 9% of cases of Fetus in Fetu , no vertebral column was found even on the pathological examination. Therefore Gonzalea-Crussi suggested fetus in fetu to be applied to any structure in which the fetal form has a highly developed organogenesis or to the presence of vertebral axis [4].

Despite the requirement of the presence of a vertebral column for diagnosis,there are reports of cases without a vertebral column [5]. Different organs

can be seen in Fetus in Fetu ,including vertebral column 91%, limbs 82.5%, central nervous system 55.8%, gastrointestinal tract-45%, vessels-40% and genitourinary tract-26.5%.

Fetus in fetu may be highly differentiated from mature teratoma. Spencer criteria for diagnosis of fetus in fetu is 1. A distinct sac enclosing fetus. 2. Normal skin coverage, 3. Grossly obvious anatomic parts, 4. Attached to the host through a few relatively large blood vessels, 5. Association with gastrointestinal tract or neural tube.

Teratoma is an accumulation of pluripotent cells in which there is neither organogenesis nor vertebral segmentation. Teratoma is a malignant condition and fetus in fetu never becomes malignant.

A presumptive diagnosis can be made by ultrasonogram, X-ray, CT and MRI. Imaging modalities are useful especially in more developed Fetus in fetu and adult subjected before surgery to evaluate the risk of hemorrhage [6]. CT findings were those of a mass that consists of round or tubular collection of fat that surrounded a central bony structures. Characteristic CT appearance allows the correct prospective diagnosis of this rare entity [7]. With the help of current imaging modalities nowadays,it has been easier to diagnose FIF prior to surgery and even during prenatal periods [8].

Surgery is the curative. Post operative follow up with screening for tumor markers Alpha feto protein and beta HCG was often used and further support on the basis of malignant recurrence of Fetus in fetu [9].

Conclusion

Fetus in Fetu is a pathological condition that occurs from the abnormal embryogenesis in a diamniotic , monochorionic pregnancy. It should be differentiated from the teratoma which has malignant potential. Radiological modalities allow correct prospective diagnosis of this rare entity. Complete excision allows the confirmation of the diagnosis and lowers the risk of recurrence. Final diagnosis of fetus in fetu is not made until histopathological analysis.

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[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.

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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

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Personal author(s)

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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/HSQ 20.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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