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A Hospital-based Study on Demographic Features of Children with Severe Acute Malnutrition

Udaya K

Assistant Professor, Department of Pediatrics, Koppal Institute of Medical Sciences, Koppal, Karnataka 583231, India.

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Abstract

Background: Severe acute malnutrition (SAM) remains a major health hazard to children, as the mortality rates among SAM children are nine times higher than those in well-nourished children. This study was conducted to assess the demographic features of malnourished children. *Methods:* A total of 100 malnourished children were included over a period of six months. A preliminary data of children regarding age, start of complimentary feeding (CF), maternal education, parity were noted. Anthropometric measurements such as height/length, weight, mid-upper arm circumference (MUAC) were recorded in all the patients. *Results:* Majority were in the age group of 6–18 months (64%) and least were in 45–59 months. Females were more with M:F ratio of 1:1.1. 94% of families resided in rural areas. Most of the children hailed from middle (58%), followed by lower (41%) class and one child belonged to upper socioeconomic class. About 70% of children were born to multiparous mothers. Approximately 50% children were of low-birth weight. Only 8% babies were bottle fed and in 76% children, weaning time was inappropriate. *Conclusion:* The short birth interval, low socio-economic status, lower mother's educational level and delay in the initiation of complimentary feeding were the important risk factors of SAM among children. Children with SAM need to be treated with special attention at the primary point of care to reduce mortality.

Keywords: Malnutrition, Anthropometry, Feeding.

Introduction

Severe acute malnutrition (SAM) in children is a serious public health problem with major concerns for child survival, damaging the cognitive and physical development of children, in turn affecting the economic productivity of individuals and society.[1] Globally, it is estimated that nearly half (45%) of deaths in children under 5 years of age is due to undernutrition.[2]

Objective

To assess the demographic patterns of children with severe acute malnutrition.

Materials and Methods

This is a hospital-based, cross-sectional study conducted from July 2019 to December 2019 in the Department of Pediatrics in a tertiary care center. A

Corresponding Author: Udaya K, Assistant Professor, Department of Pediatrics, Koppal Institute of Medical Sciences, Koppal, Karnataka 583231, India.

E-mail: uday.bmc@gmail.com

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total of 100 malnourished children in the age group of 6–59 months were included over a period of six months. A preliminary data of children regarding age, sex, socioeconomic status, residence, start of complimentary feeding (CF), maternal education, parity were noted. Each child was subjected first to measurement of length or height. For children <2 years of age, recumbent length was measured. Subsequently, MUAC and body weight measurement were assessed. A written consent was obtained from the informants of all children and an approval from the ethical committee of the institution was received.

Inclusion and exclusion criteria: Children between age group of 6–59 months and who fulfilled the criteria for SAM were included in this study. Children were excluded if they were in shock, had severe respiratory difficulty, or significant bleeding at the time of admission.

Results

Majority were in the age group of 6–18 months (64%) and least were in 45–59 months. M:F ratio was 1:1.1. 94% of families resided in rural areas. Most of the children hailed from middle (58%), followed by lower (41%) class and one child belonged to upper socioeconomic class. None of the mothers were graduates, 10% of mothers completed pre-university education, 26% higher secondary education, 21% primary education and rest 43% had no formal education (Table 1). About 70% of children were born to multiparous mothers. Approximately 50% children were of low-birth weight. Only 8% babies were bottle fed and in 76% children, weaning time was inappropriate. Interval between pregnancies of index children was less than 2 years in 61% and more than 2 years in 39% babies.

Table 1: Distribution of SAM children based on mothers' education

Level of education	Percentage
Graduation	0
Pre-university college	10
Higher secondary school	26
Primary education	21
No formal education	43

Discussion

Malnutrition is one of the leading causes of morbidity and mortality among children under the age of 5 in low and middle income countries like India. According to National Family Health Survey –3, 7.9% of under-five children in India suffer from SAM.[3] Majority were in the age group of 6–18 months (64%), followed by 19–31 months (18%) and least were in 45–59 months (Fig. 1). Similar results were reported by several published studies.[4–6] This may suggest the risk of development of SAM is higher in first few years of life as initial 2 years of life are the critical period for growth and development of a child.

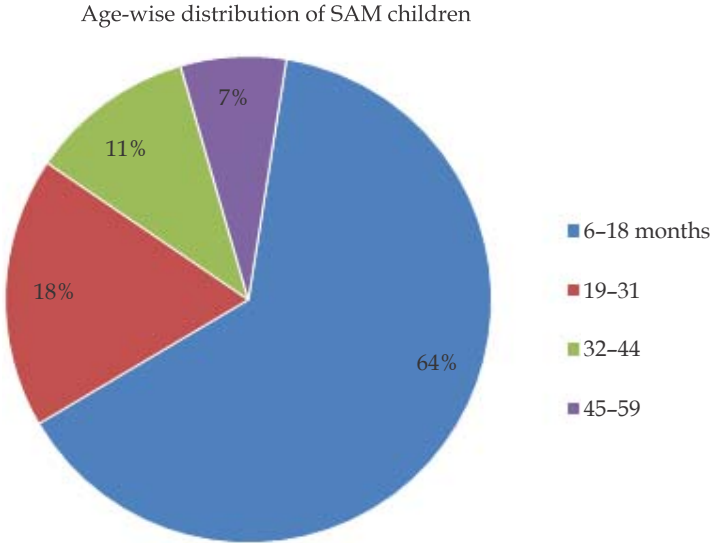

Fig. 1: Distribution of subjects according to age groups.

The proportion of females with SAM was higher than male. This could be due to discrimination of female child regarding amount and nutritious content of diet and more attention given to growth of male child in the community.[5]

Few studies also documented similar results in terms of maternal education, socioeconomic status, birth interval and parity of mother.[7,8] The prevalence of SAM was high in lower and middle socioeconomic classes, which was comparable with other studies.[9,10] This could be explained by the fact that children from families of low socioeconomic status have limited access to food, health services, hygiene and sanitation.

The prevalence of SAM was high in children hailing from rural areas (96%) as compared to urban areas. This could be due to improper road, poor infrastructure, increased family size, poor knowledge of hygiene and sanitation in rural areas.[7,11]

Birth interval <24 months was also an independent determinant for SAM which is similar to studies from Ethiopia, Bangladesh and India.[7,12,13] This may be because of poor knowledge of spacing method.

Conclusion

Younger age, lower maternal education, lower socioeconomic status, lesser birth interval, improper initiation of complimentary feeding predisposes a child to SAM. Improvement in our education system, an effective family planning program, a poverty alleviation program, awareness about spacing method and the timely initiation of complementary feeding might decrease the prevalence of SAM in India. There is a need for further prospective studies to determine the association of above risk factors with malnourishment. Communal education especially of women and young people is essential to improve nutritional levels in the rural regions.

Conflict of interest: Nil

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Birth Defects in Newborns: A Prospective Observational Study from a Rural Tertiary Care Teaching Hospital of Central India

Mohit Kurundwadkar[1], Smita P Jategaonkar[2], Manish Jain[3]

[1]Senior Resident [2]Assistant Professor [3]Professor and Head, Department of Pediatrics, Mahatma Gandhi Institute of Medical Sciences, Sevagram, Wardha, Maharashtra 442102, India.

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Abstract

Introduction: Worldwide, the incidence of congenital anomalies is estimated at 3–7%, but actual numbers vary widely between countries. There is a paucity of Indian literature on birth defects. *Aims and objectives: (*1) To Study the Proportion of birth defects in live births in a tertiary care teaching hospital in rural central India. (2) To follow-up these birth defects for a period of 2 months and to study the outcome. *Materials and methods:* In this prospective observational study every live birth was screened by clinical examination and further relevant investigations. Study subjects were all live births in the calendar year 2015. Telephonic follow-up was undertaken at 2 months of age. *Results:* A total of 4493 neonates were born, out of which 127 babies had birth defects. The total number of individual birth defects was 153 (3.4%). The most common system involved was cardiovascular system. *Conclusion:* Early detection is essential for proper management of these defects and offers the best chance for survival. Follow-up of babies' birth defects is highly recommended. Standard guidelines advocacy and policies at national level are needed. In resource-limited setting the novel way of telephonic follow-up is highly beneficial and economical.

Keywords: Newborns; Birth defects; Babies; India; Neonates.

Introduction

Although the global incidence of congenital anomalies remains 3–7%, it may have geographic variations.[1] In the United States and Canada where congenital anomalies are diagnosed intrauterine and aborted, the incidence ranges from 2–5% of all live births.[2,3] Even in Asia, the magnitude of congenital anomalies varies with reported incidence at birth of 2.5% of infants in India. Early diagnosis and treatment of all birth defects is of clinical and public health importance. It is important to do epidemiologic surveys of congenital anomalies in developing countries like India that host a wide variety of environmental factors, socioeconomic status and ethnic groups with varying marital habits. In this context, our paper aims to **s**tudy the proportion of birth defects in live births in a tertiary care teaching hospital in rural central India and follow them up for a period of 2 months for the outcome.

Materials and Methods

This prospective observational study was conducted wherein every live birth was screened by clinical

Corresponding Author: Smita P Jategaonkar, Assistant Professor, Department of Pediatrics, Mahatma Gandhi Institute of Medical Sciences, Sevagram, Wardha, Maharashtra 442102, India.

E-mail: jategaonkarpa@gmail.com

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examination. Suspected cases underwent relevant investigations (ultrasonography of abdomen and pelvis for newborns with suspected gut or renal anomalies, neuroultrasonography for suspected hydrocephalus. 2D echocardiography for suspected congenital heart diseases; etc.) All live births at the hospital from January 1, 2015 to December 31, 2015 were included the study.

A pre-designed, pre-tested questionnaire was used to collect socio-demographic information and clinical examination in all live births was done to screen for birth defects. Initial section of the study tool included socio-demographic details of family. Second section had questions regarding antenatal history, treatment received, weight and height, head circumference of the neonate. Third section had questions related to parental age, gestation, relevant description of the birth defect by the international classification of diseases (ICD-10). At 2 months of age a follow-up call undertaken which included discussion regarding the current health status of the baby, enquiry about the reports of any pending/newly done investigations, and immunization status.

Results

Over the period of one year a total of 4493 neonates were born, out of which 127 presented with birth defects. The total number of individual congenital anomalies found in these neonates was 153. The proportion of birth defects among the inborn live births was 3.4%. Gender distribution showed male preponderance of 1.6:1. Interestingly, majority (39.37%) of anomalies were noted in neonates weighing more than 2.5 kg. About 39.36% of mothers giving birth to children with congenital anomalies were in the age group of 21–25 years. Only 3.94% of the mothers were aged more than 35 years and 14.17% of the mothers were below the age 20. Also, 86.62% of fathers giving birth to children with congenital anomalies were in the age group 24–40 years. Only 2.36% were more than 40 years, while 11.02% were younger than 24 year of age.

Most of the newborns had vaginal delivery (55.12%) as compared to Caesarean section (40.16%) and forceps (4.72%) deliveries. However, 15 babies died before the age of 2 months. The most common system involved was the cardiovascular system (30.7%) followed by the urinary (21%), musculoskeletal (13%), genital, gastrointestinal and central nervous systems (Fig. 1). Overall, congenital heart disease was the most common birth defect observed in our study. The next common birth defects were congenital hydronephrosis, polydactyly and congenital tallipes equinovarus, in that order.

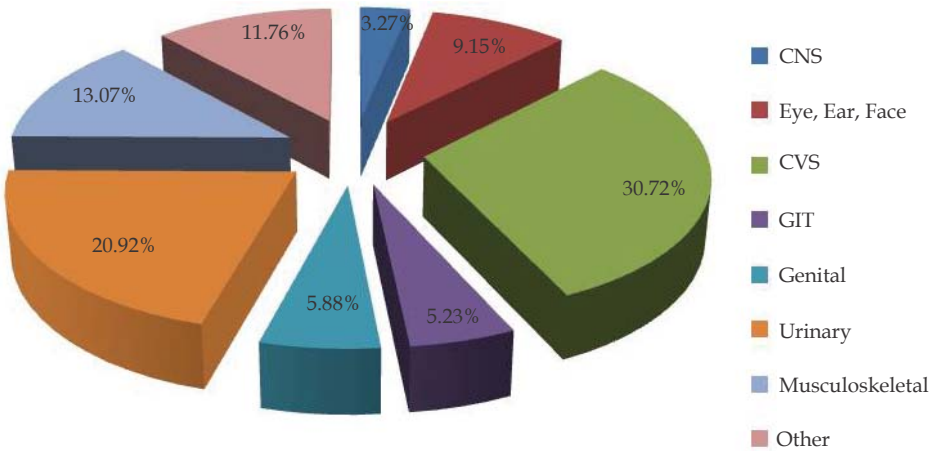

Fig. 1: Systemic distribution of birth defects; CVS- cardiovascular system, CNS- central nervous system.

Discussion

In this hospital-based observational study the fraction of birth defects was found to be 3.4%. In our study male children outnumbered the female (60.63% and 37.80%, respectively), and the remaining 1.57% babies' gender was ambiguous. The male: female gender distribution of 1.5:1 found in our study is comparable to the study conducted by Bakare et al. where the ratio was reported to be 1.55:1.[4] Male preponderance for congenital anomalies has been reported in many studies.[1,5,6] The male preponderance for congenital anomalies is thought to be due to their greater vulnerable to oxidative stress.[6]

Table 1: Follow-up of the babies with birth defects (after 2 months)

Follow-up	Result	No. of babies	Percentage (%)
Current health status	Satisfactory	58	51.8
	Unsatisfactory	4	3.6
	Not applicable	50	
	Lost to follow-up	47	41.6
	Death	3	3.0
	Total	**112**	**100.0**
New report	Yes	27	24.0
	No	85	76.0
	Total	**112**	**100.0**
Immunization status	Up to date	65	58.0
	Not up to date	0	0.0
	Lost to follow-up	47	42.0
	Total	**112**	**100**
Outcome	Death after discharge	3	2.6
	Alive	62	55.4
	Lost to follow-up	47	42.0
	Total	**112**	**100.0**
Lost to follow-up	Yes	47	42.0
	No	65	58.0
	Total	**112**	**100.0**

It is interesting to note that in this study, 44.1% of congenital anomalies were in lowbirth weight babies. Several studies report a significant association between low-birth weight and congenital anomalies.[6–8] Recently, Sarkar et al. reported that the incidence of congenital anomalies was significantly higher in preterm babies as compared to the full-term babies.[9] In our study, however, 78.74% of the newborns were delivered after 36 weeks of gestation and only 21.26% were preterm, less than 36 weeks of gestation. This could be attributed to the fact that our study was exclusively for "in-born" babies, most of whom were planned deliveries.

Furthermore, in our study, the mode of delivery also showed a difference among the neonates with congenital anomalies; they were more common for normal deliveries (55.12%) than for Cesarean section (40.16%) and forceps deliveries (4.72%). However, this is in contrast with another study where mode of delivery had a statistically significant association with congenital anomalies where Caesarean section was more common than normal delivery.[9]

As such, we have found that the frequency of congenital anomalies was highest in mothers aged between 21 and 24 years (39.36%). Suguna et al. also reported rising incidence of congenital anomalies with advancing maternal age.[10] But in our study, advanced age of the mother did not seem to influence the frequency of malformed babies. This, also, is in accordance with a similar study conducted by Taksande et al. in which it was observed that the mothers of babies with congenital anomalies were mostly (90.49%) between 20 and 30 years of age and that women less than 20 years had 1.11% babies with congenital anomalies, and in 8.40% babies with birth defects the mothers were more than 30 year-old.[11] Similarly, as reported by Agrawal et al., 68.75% mothers of newborns with congenital anomalies were in age group 20–35 years and only 12.5% cases were under 20 years.[12] Other researchers also published analogous findings in this regard.[13–15]

The frequency of congenital anomalies was highest in fathers aged 25–29 years. In this study no significant association was found between paternal age and occurrence of congenital anomalies. Interestingly, several studies have found advanced paternal age as well as younger age groups to be at risk of congenital anomalies. With regard to the pattern of congenital anomalies in our study, the most common single system involved was cardiovascular system. And, the commonest birth defect reported was congenital malformations of heart. This was found to be in accordance with the available literature.[11,16]

Follow-up has been the unique part of this study. Two-month time frame was specifically designed

to cover the parameter of immunization at 6 weeks of age in the follow-up; this provided us a useful indicator. However, on literature search we could not find any similar study that included follow-up, results of which could be compared with our study. The following specific parameters were collected for study during follow-up: current health status, new reports, immunization status, and outcome and lost to follow-up.

Overall, we would like to underscore the following:

(1) Despite having high incidence of congenital malformations, there are no well-accepted preventive measures in developing countries like India. It indicates that strong preventive measures, in that regard, are the need of hour.

(2) Increasing awareness about maternal care during pregnancy and awareness programs on congenital malformations need to be highlighted to decrease the incidence of congenital anomalies and the associated co-morbidities.

(3) Regular antenatal visits and prenatal diagnosis for prevention, early intervention and even medical termination of pregnancy should be systematically encouraged.

(4) Conventionally, ultrasound scans are generally done at the 20th week of gestation and 37th week of gestation. However, additional scans at 12th week, that are capable of diagnosing plethora of birth defects, need to be taken into available protocols.

(5) Role of fetal echocardiography in detecting cardiovascular birth defects should be emphasized for early diagnosis, effective intervention and better prognosis.

(6) The study strongly recommends the role of follow-up in studying the status of babies with birth defects and also brings forth the need of standard guidelines advocacy and policies at national level.

Conclusion

The proportion of congenital anomalies was 3.4% among all live births in our hospital in the year 2015. The most commonly affected system is the cardiovascular system, followed by urinary system and musculoskeletal system. Early diagnosis and correction of the malformed babies could offer them the best chance for survival. Also, antenatal awareness of both parents regarding optimal screening may reduce the overall incidence of these malformations. Further multi-centric studies are needed to substantiate these findings.

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Original Research Article

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Neonatal Peripherally Inserted Central Catheters (PICCs): Complication Rates & Average Duration of Stay Related Catheter Tip Location

Triya Malde

Assistant Professor, Department of Pediatrics, Shri MP Shah Government Medical College, Jamnagar, Gujarat 361008, India.

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Abstract

Objectives: Present study was performed with an objective to compare complication rates & average duration of stay between, peripherally located catheter tip, non–centrally (intermediate) located catheter tip and centrally located catheter tip of percutaneously inserted central catheter (PICCs) in neonates. *Material and methods:* Present retrospective cohort study was performed at level III neonatal care in neonates who underwent PICCs placement through saphenous vein. We analyzed the data of the neonates from our NICU from March 2009 to September 2009 who underwent PICCs placement. Patients demographics, catheter duration and catheter complications were analyzed retrospectively. Catheter tip location was determined by X-ray. Difference in the complication rates, premature removal rates and average duration of stay in peripheral, non-central (intermediate) and central groups were analyzed. *Results:* Data of total 49 neonates was analyzed. Of the 49 PICCs, in 16 (32.6%) catheter tip was in peripheral location, in 23 (46.9%) catheter tip was in non-central (intermediate) location and in 10 (20.4%) catheter tip was central location. The peripheral group had complication rate of 81.2% (13/16), while the intermediate group had complication rate of 26% (6/23), and central group had no complications. Average duration of stay of catheter in peripheral group was 7.1 days, intermediate group was 10.7 days, and central group was 28.3 days. *Conclusion:* Placement of the tip of the PICCs beyond the sapheno femoral junction even though not central is acceptable in standard care of newborn.

Keywords: Percutaneously inserted central catheter (PICCs); Neonatal.

Introduction

Safe and reliable vascular access is an essential element of modern-day health care. In recent years peripherally inserted central catheter for intermediate and long-term venous access has steadily grown.

Peripherally inserted central catheters (PICCs) have become increasingly popular in the treatment of the neonates in the intensive care units. A PICCs defined as a catheter inserted percutaneously via peripheral vein with the tip residing in central vein.[1] We accept definition of central position of tip if it lies in SVC, high in IVC or RA (based on vessel diameter, blood flow estimates, and physiologic flow dynamics), peripheral position of tip if it lies in saphenous vein, cephalic vein or axillary vein, and intermediate position of tip if it lies in subclavian, femoral, iliac, jugular vein.

Corresponding Author: Triya Malde, Assistant Professor, Department of Pediatrics, Shri MP Shah Government Medical College, Jamnagar, Gujarat 361008, India.

E-mail: drpiyushpujarapcd@gmail.com

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PICCs are commonly used in neonatal practice, where central venous access is often necessary for weeks. PICCs cannot always be advanced to a central venous location and are occasionally left with the catheter tip in noncentral position. There is increasing popularity in placing so called "mid-line catheter" or "long IVs" whose catheter tips are intentionally left in the noncentral position.[2] It has been suggested that centrally placed catheter tips were associated with fewer complications than non-centrally placed catheter tips.[1]

We hypothesized that in case of PICCs inserted via long saphenous vein, if that catheter tip crosses the sapheno-femoral junction (lying in to femoral vein) even if it is not lying high in inferior vena cava would result in decreased in premature removal rates and complication rates, compared with central catheter tip lying in saphenous vein or in tributaries of vein (stuck at the sapheno-femoral junction).

The position of these lines is important because incorrect placement may be associated with complications. Importance must be paid to the correct positioning of the line, preferably with the tip lying within superior vena cava, or inferior vena cava, outside the cardiac chambers. Suboptimal or incorrect positioning can result in variety of complications including perforation, which may lead to extravasation of intravenous fluid, pleural and peritoneal effusion and more seriously cardiac tamponade. The course of the line and positioning of tip is usually assessed using plain film radiography, traditionally using film screen combinations.[3]

PICCs related sepsis is serious complication and its risk is increased by longer duration of catheterization, and is probably influenced by catheter material, frequency of line break for infusion change and drug injection, the presence of multiple lumens and by technique of catheter fixation. The rate of infection can be reduced by staff education.[3]

Delayed effusion into body cavity can occur, probably because of damage to the vascular wall by infused fluid. It may develop after catheter passes into small vein and then cause extravasation.[3]

Materials and Methods

A retrospective study was performed over the period of six months from March 2009 to September 2009. All neonates of the neonatal units at our hospital who required a percutaneous long line through saphenous vein during this period were included in the study. Long lines were inserted by neonatologist using standard procedure. Vygon Premicath 28 G with splitting needle was used in all neonates. After insertion plain radiograph was taken to ascertain the line position. The position of the tip of long line was identified by using plain radiograph. Assessment was made to determine the tip of the PICCs. The reference points by which the position of the tip of the catheter was determined radiologically were the vertebral bodies, medial end of the head of the femur and the midline of the body plane.

Three groups were made according to location of the tip of the catheter radiologically:

Group 1 Central: Tip of the PICCs resides in the superior vena cava (SVC), or high inferior vene cava (IVC) at or above the level of diaphragm.

Group 2 Non-central (intermediate): Tip of the PICCs resides in the femoral vein or iliac vein (crossed the sephanofemoral junction); above the medial end of the head of the femur.[4]

Group 3 Peripheral: Tip of the PICCs resides in saphenous vein (if tip lies anywhere below the head of the femur).

Retrospective data was collected on complications including leaking at the PICCs insertion, phlebitis (erythema, swelling, pain, or palpable cord), infection (positive tip catheter cultures/positive blood culture), and catheter occlusion (inability to infuse or withdraw). All complications necessitated catheter removal. We also looked at premature removal of the catheter rates (removed when needed) in relation to catheter tip location. Average duration of the stay of the catheter was calculated and compared in relation to location of the tip catheter.

Statistical Analysis

All analyses were performed using statistical software, version 13. For the complications rates and premature removal rates. Pair vise comparison of frequency between Group 1 and Group 2 was done using test of proportion. The *p*-values are recorded.

The patient's characteristics were examined univarietely. For simple comparison between central and non central and peripheral PICCs the x^2 test was used for categorical data. For the comparison of duration of the stay of the PICCs in different groups, one-way analysis of variance (ANOVA) was applied. When ANOVA showed a significant difference, Turkey's test for comparison was applied.

Results

From March 2009 to September 2009, data from a total of 49 PICCs were analyzed.

Table 1 shows that 15 neonates (30.6%) were between 28 and 32 weeks, 18 neonates (36.7%) were between 33 and 37 weeks and 17 neonates (34.6%) were >37 weeks of gestation.

Table 1: Distribution of neonates according to gestational age

Gestational age	No. of neonates	%
28–32	15	30.6
33–37	18	36.7
>37	17	34.6
Total	49	100.0

Number of complications were more (81.2%) in the neonates in whom the tip of the PICCs were in peripheral location, and the percentage of complication decreased to 26% if the tip of the PICCs was in intermediate position in femoral vein (crossed the sephano-femoral junction) though not in the central position. There was no complication if the tip of the PICCs were lying in the central position. Pair vise comparison of frequency between Group 1 (Peripheral) and Group 2 (Non-central/ Intermediate) was done using test of proportion. The *p* values are recorded it was significant (<0.001), indicating non-central/intermediate group has reduced rate of complication than peripheral group (Table 2).

Number of premature removal was more frequent in the neonates in whom the PICCs were peripheral in location (81.2%) and in neonates in whom the PICCs were though not in central position but crossed the sephano-femoral junction lying in the femoral vein the no. of premature removal is decreased to (26%). Pair vise comparison of frequency between Group 1 and Group 2 was done using test of proportion. The *p*-values was significant (<0.001), indicating non-central group has reduced rates of premature removal. Average duration of the stay was 6.1 days in peripheral group and it increased to intermediate group to 10.7 days, while it was 27.8 days in central group. (Table 2)

Table 2: Complication rates, premature removal rates & Average duration of stay: Peripheral, Intermediate & Central groups

Complications	Tip located in peripheral position (Group 1 peripheral) *n* = 16	Tip in the femoral vein but not central (Group 2 intermediate) *n* = 23	Tip Crossed the junction and central (Group 3 central) *n* = 10	*p*-value
Swelling	4 (25%)	3 (13%)	0	**<0.001**
Occlusion	6 (37.5%)	3 (13%)	0	
Phlebitis	3 (18.2%)	0	0	
Total no. of complication	13 (81.2%)	6 (26%)	0	
No. of premature removal (Removed when needed)	13 (81.2%)	5 (26%)	0	**<0.001**
Average duration of stay	6.5 days	10.7 days	27.8 days	

Table 3 shows mean duration of the stay of the PICCs in three groups. It shows that mean PICCs duration was greater for the non-central / intermediate group(10.7 ± 4.7 days) than peripheral group (6.5 ± 2.5 days).

Table 3: Duration of stay of the PICCs: between peripheral, non-central, central groups

Duration of stay of the PICCs	Tip located in peripheral position (Group 1) *n* = 16	Tip in the femoral vein but not central (Group 2 non-central/ intermediate) *n* = 23	Tip Crossed the junction and central (Group 3) *n* = 10
Mean	6.56 days	10.78 days	27.8 days
SD	2.5	4.72	8.69

Discussion

In our NICU PICCs were inserted in lower limb in saphenous vein, reasons being:

(1) convenient for nursing staff to maintain the position of the baby.

(2) changes in upper extremity position cause migration of PICCs.

We encountered difficulties in negotiation of the catheter to central location. The reasons could be venous valves, venous tortuosity, veno-spasm and the tip entering into smaller tributaries.

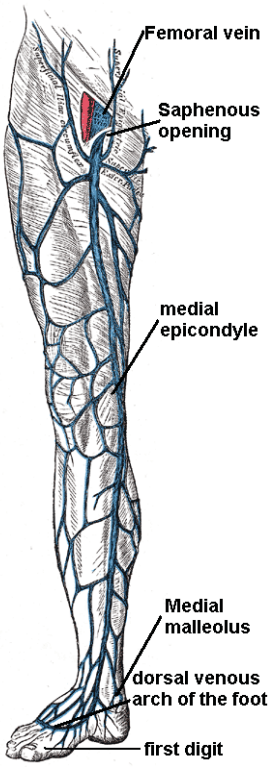

Fig. 1: Course of long saphenous vein.

Figure 1 shows the course of the long saphenous vein and anatomical landmark with venous tortuosity and multiple tributaries which can make the negotiation difficult. Table 4 demonstrates that number of venous valves are more below the saphenofemoral junction, which make the negotiation difficult beyond the saphenofemoral junction.

There has not been clear evidence in the literature of an association between catheter tip location and complication rates in neonatal PICCs. Our study demonstrates that central catheter tip location was important factor associated with reduced complication rates, it also demonstrates that though the tip of PICCs is not central but if it is in intermediate vein (out of peripheral vein) the complications rates are decreased and average duration of the stay is increased.

The position of these lines is important because incorrect placement may be associated with complications. Recognized complications of PICCs include catheter occlusion, phlebitis, extravasation, thrombosis and infection.[5,6,7,8]

Importance must be paid to the correct positioning of the line, preferably with the tip lying within superior vena cava, or inferior vena cava, outside the cardiac chambers and beyond the peripheral vein. Suboptimal or incorrect positioning can result in variety of complications including perforation, which may lead to extravasation of intravenous fluid, pleural and peritoneal effusion and more seriously cardiac tamponade. The course of the line and positioning of tip is usually assessed using plain film radiography.[9]

We accept definition of central position of tip if it lies in SVC, high in IVC or RA (based on vessel diameter, blood flow estimates, and physiologic flow dynamics) and noncentral position of tip if it

Table 4: Number of valves in veins of lower limb

	Number of valves
Inferior vena cava	0
Common iliac vein	0
Internal iliac vein	0
External iliac vein	1
Femoral vein	1
Superficial femoral vein	1–4
Profunda femoris vein	0
Popliteal vein	1
Long saphenous vein	
reinforced ostial valve	1
reinforced trunk valves	2–3
fine, transparent valves	6–20

lies in brachycephalic, jugular, subclavian, femoral, iliac vein, and peripheral if tip lies cephalic, axillary or saphenous vein (Fig. 2). The central locations represent the region of the highest blood flow.[10–16]

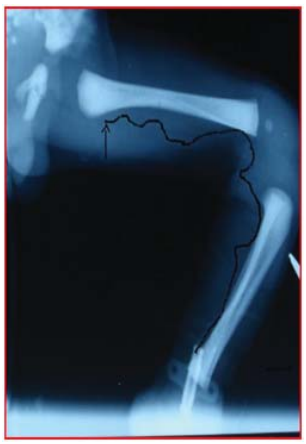

Fig 2: Tip located in saphenous vein (Peripheral group) position.

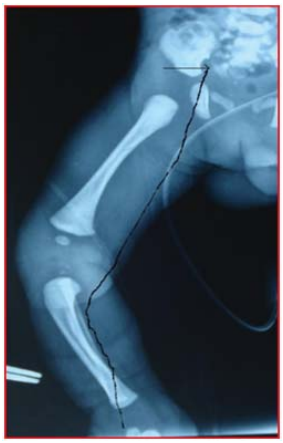

Fig 3: Tip placed in femoral vein crossed the junction but not central (Intermediate group).

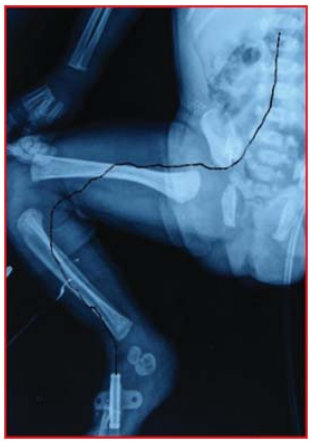

Fig. 4: Tip placed high in IVC (central group).

In our study there was no incidence of pericardial effusion, cardiac tamponade as even in centrally located tip position group as the tips were outside the heart. We report that in neonatal PICCs placement, even if the tip of the catheter is not in central position as it is difficult to negotiate, but in intermediate vein (out of the peripheral vein) it serves the purpose in care of LBW babies without life-threatening complication (e.g. pericardial effusion, cardiac tamponade, sepsis) (Fig. 3). The use of the PCCs should be encouraged by looking at their multiple advantages.

Decreased complication rates, decreased rates of premature removal and increased average duration of stay of PICCs with centrally and noncentrally versus peripherally located PICCs tip is likely related to a combination of factors including vessel size, blood flow rate, turbulent flow, and endothelial injury. Smaller vein diameter result in decreased blood flow, causing turbulence which increases the risk of endothelial injury, thrombophlebitis and thrombosis.[17,18]

Addition of this study to exiting knowledge. Complication rates, premature removal rates are decreased and average duration of stay is increased once the tip of the catheter advances high in intermediate vein (out of the peripheral vein) even though not central (Fig. 4). Non-centrally (in intermediate vein) placed catheter tips are associated with fewer complications than peripherally placed catheter tips.[19]

Conclusion

Complication rates, premature removal rates are decreased and average duration of stay is increased once the tip of the catheter advances high in intermediate vein (out of the peripheral vein) even though not central. Centrally and non-centrally (in intermediate vein) placed catheter tips are associated with fewer complications and prolonged duration of stay than peripherally located catheter tips. Placement of the tip of the PICCs beyond the sapheno femoral junction even though not central is acceptable in standard care of newborn.

Conflict of Interest: none

Source of Support: Nil

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Original Research Article

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Iron Toxicity Measurement by Serum Ferritin Levels Due to Blood Transfusion in Patients of Thalassemia Major

Ajit Inder Kaur[1], Sanjay Chavan[2], SR Agarkhedkar[3], D Upase[4]

[1]Resident, [2]Professor, [3]Professor and Head, Department of Pediatrics, [4]Consultant Hematologist and Oncologist, Dr. D.Y. Patil Vidyapeeth, Pimpri, Pune, Maharashtra 411018, India.

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Abstract

Introduction: Thalassemia is genetic blood disorders inherited from a person's parents that can result in the abnormal formation of hemoglobin. In beta thalassemia major ineffective erythropoiesis, frequent blood transfusions lead to iron overload. Excessive iron can cause irreversible organ damage. Iron overload can be measured by serum ferritin levels. *Aims and objectives:* To study iron toxicity due to blood transfusion in patients of thalassemia major. To study the correlation between iron toxicity and serum ferritin levels. Correlation between iron toxicity and number of blood transfusions. To study growth parameters in thalassemia penitents pertaining to iron toxicity. *Methods:* Fifty cases of thalassemia major were enrolled. Detailed history and clinical examination was done and blood samples collected to test for serum ferritin levels. Ferritin levels were performed by using indirect enzyme-linked immunosorbent assay (ELISA) kit along with normal and abnormal controls. Data were analyzed to determine the association between variables. *Results:* Majority of the patients were in ≤5 years of age (40%) followed by 6–10 years (32%). Sixty-four percent subjects were male and 36% were female. At baseline majority of the children (44%) were having serum ferritin level between 1000 and 2500 ng/ml followed by 2501 and 4000 ng/ml (32%) and 4001 and above (14%). At the last follow-up serum ferritin level was observed to be increased with majority having serum ferritin level between 1000–4000 ng/ml (68%). At the last follow-up it was observed that the total dose of iron chelator was increased as compared to baseline and was statistically significant. *Conclusion:* Thus we conclude that majority of the children suffering from thalassemia major were having iron toxicity due to blood transfusion at the baseline and at the last follow-up also. Increasing age and number of transfusions were significantly associated with serum ferritin levels statistically. Positive correlation between Sr. Ferritin level and chelation was observed at the baseline and at the time of last follow-up also.

Keywords: Thalassemia; Enzyme-linked immunosorbent assay (ELISA); Serum; Hemoglobin.

Introduction

Thalassemia is genetic blood disorders inherited from a person's parents that can result in the abnormal formation of hemoglobin.[1] There are two major types, alpha and beta thalassemia. The severity depends on how many of the four genes for alpha or two genes for beta globin are missing.[1]

Thalassemia occurs when there is decreased or absent production of one of the types of globin

Corresponding Author: Ajit Inder Kaur, Resident, Department of Pediatrics, Dr. D.Y. Patil Vidyapeeth, Pimpri, Pune, Maharashtra 411018, India.

E-mail: ajitinder.kaur@gmail.com

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chains (most commonly either α or β), that cause insufficient amount of normal structure of globin chains.

Children affected with thalassemia have pallor, poor development, and abdominal enlargement. Anemia is due to a combination of ineffective erythropoiesis, excessive peripheral red blood cell hemolysis, and progressive splenomegaly.[2] The red cells are microcytic (mean corpuscular volume <70 fL) with marked anisochromasia. The bone marrow shows marked erythroid hyperplasia, and the serum ferritin level is elevated. Because of chronic anemia and iron overload, endocrinopathies such as hypopituitarism, hypothyroidism, hypoparathyroidism, diabetes mellitus, cardiomyopathy, and testicular or ovarian failure become common as the child with thalassemia grows older.[3,4]

The current management of β-thalassemia major patient is based on regular transfusion of packed red cells and effective chelating therapy.[5–8] The aim of the transfusion therapy is to correct anemia and to maintain sufficient circulating level of hemoglobin (Hb) to suppress endogenous erythropoiesis.[9] Major complication in chronically transfused patients is iron overload.[10] Iron stores in the body exist primarily in the form of ferritin. In the body, small amounts of ferritin are secreted into the plasma. The concentration of this plasma (or serum) ferritin is positively correlated with the size of the total body iron stores in the absence of inflammation. Excess iron is extremely toxic to all cells of the body and can cause serious and irreversible organic damage, such as cirrhosis, diabetes, heart disease, and hypogonadism.[11]

A target ferritin of approximately 1000 mg/L is generally recommended standard practice in thalassaemia major (TIF Guidelines, 2000) and other forms of iron overload resulting from blood transfusion. When the serum ferritin level reaches 1000 ng/L (usually after 10^{th} to 12^{th} transfusion), it is generally taken as the point to initiate iron chelation therapy.

Aims and Objectives

Aim

To study iron toxicity due to blood transfusion in patients of thalassemia major.

Objectives

1. To study the correlation between iron toxicity and serum ferritin levels.
2. Correlation between iron toxicity and number of blood transfusions.
3. To study growth parameters in thalassemia penitents pertaining to iron toxicity.

Materials and Methods

Fifty cases of thalassemia major were enrolled from a teritiary hospital in Pune. Detailed history and clinical examination was done and blood samples collected to test for serum ferritin levels. Ferritin levels were performed by using indirect enzyme linked immunosorbent assay (ELISA) kit Orgentec, Germany along with normal and abnormal controls. Data were analyzed to determine the association between variables.

Results

Majority of the patients were in ≤5 years of age (40%) followed by 6–10 years (32%). 64% subjects were male and 36% were female. At the baseline 34% children had received 41–80 transfusions while 24% had received 1–40 transfusions. At the last follow-up 36% had received 41–80 transfusions while 14% had received 1–40 transfusions. At baseline majority of the children (44%) were having serum ferritin level between 1000 and 2500 ng/ml followed by 2501 and 4000 ng/ml (32%) and 4001 and above (14%). At the last follow-up serum ferritin level was observed to be increased and majority of the children were having serum ferritin level between 1000–4000 ng/ml (68%). At baseline maximum mean serum ferritin level was observed among 6–10 years children (3400.76 ± 1564.89) followed by >10 years children (2986 ± 1403.816) and the difference was statistically significant. At the last follow-up also maximum mean serum ferritin level (4036.63 ± 1907.302) was observed among 6–10 years children followed by >10 years children were observed (3656.81 ± 1533.986). Mean dose of chelation drug at baseline was 625 ± 235.135 in >10 years age children while it was 546.88 ± 187.50 in 6–10 years age children with statistically significant difference. At the last follow-up it was observed that the total dose of iron chelator was increased as compared to baseline. The mean serum ferritin level at baseline among 81–120 transfusion was 3190.71 ± 667.032 while 121–160 transfusions was 3107.24 ± 1170.836 and the difference observed was statistically significant. Serum ferritin level was significantly increased after last follow-up, i.e. 13.59%. The mean serum ferritin level among the children with 81–120

transfusion was 4371.88 ± 2487.551 ng/ml while among 121–160 transfusion was 3861.18 ± 1540.155 ng/ml. At baseline maximum requirement of iron chelator dose (750 ± 250.00) was among the patients who had 81–120 transfusions followed by 161–200 transfusions (678.57 ± 237.797) and the difference was statistically significant (Figs 1–3).

At last follow-up the requirement of iron chelators was increasing with number transfusions. And the difference observed was statistically significant. At the baseline there was positive correlation between the number of transfusions and serum ferritin and chelation (0.4 and 0.56 respectively) with significant *p* value. Weak negative correlation between number of transfusion and BMI was observed with non significant *p*-value (p = 0.97). At the follow-up there was positive correlation between number of transfusion and serum ferritin level and chelation (0.36 and 0.57 respectively) with significant *p*-value. Negative correlation between number of transfusion and BMI (–0.33) with significant *p*-value. In the baseline group positive correlation between Sr. Ferritin level and chelation was observed (r = 0.402) while weak negative correlation between Sr. Ferritin level and BMI was observed (r = –0.06). In the follow-up group association between Sr. Ferritin level and chelation and BMI was not significant. At the baseline weak negative correlation between serum ferritin and height, weight and MAC was observed (r = –0.07, –0.02, –0.14 respectively) but the correlation was not significant. At the last follow-up positive correlation between serum ferritin level and height, weight and MAC was observed (r = 0.26, 0.53, 0.28 respectively) but the correlation was not statistically significant (Tables 1 and 2).

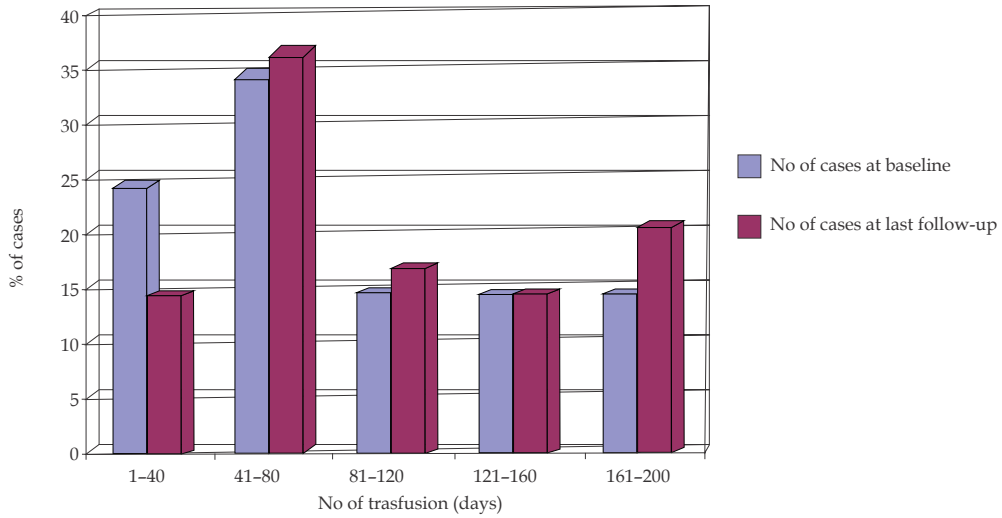

Fig. 1: Bar diagram showing No of blood transfusion wise distribution of cases in study group.

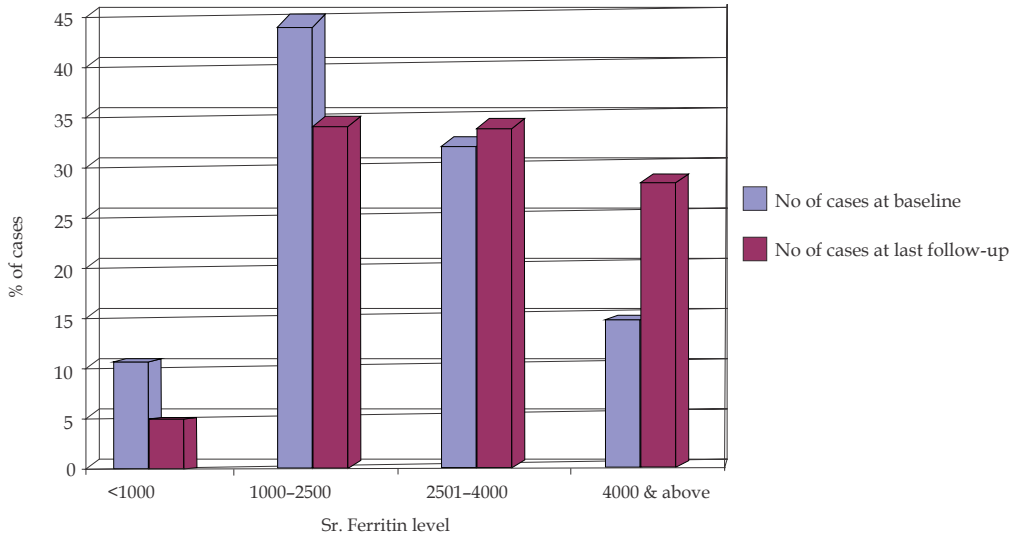

Fig. 2: Bar diagram showing comparison of Sr. Ferritin level at baseline, last follow-up according to number of transfusion in study group.

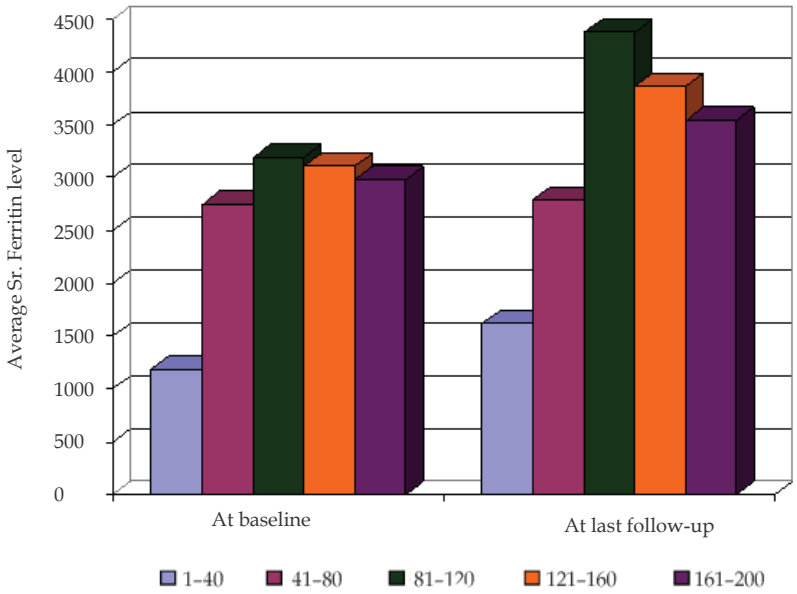

Fig. 3: Bar diagram showing comparison of Sr. Ferritin level at baseline, last follow-up according to no of transfusion in study group.

Table 1: Correlation between number of transfusion and Sr. Ferritin level, chelation, BMI at baseline in study group

Correlation between no of transfusion and	*r*-value	*p*-value
Sr. Ferritin level (*n* = 50)	0.40	0.004
Chelation (*n* = 47*)	0.56	<0.0001
BMI (*n* = 39**)	–0.01	0.97

*3 cases had age <2 years **11 cases had age <5 years

Table 2: Correlation between number of transfusion and Sr. Ferritin level, chelation, BMI at last follow-up in study group

Correlation between no of transfusion and	*r*-value	*p*-value
Sr. Ferritin level (*n* = 50)	0.36	0.011
Chelation (*n* = 47*)	0.57	<0.0001
BMI (*n* = 39**)	–0.33	0.044

*3 cases had age <2 years **11 cases had age <5 years

Conclusion

Thus we conclude that majority of the children suffering from thalassemia major were having iron toxicity due to blood transfusion at the baseline and at the last follow-up also. Increasing age and number of transfusions were significantly associated with serum ferritin levels statistically. Positive correlation between Sr. Ferritin level and chelation was observed at the baseline and at the time of last follow-up also.

Negative correlation of Sr. Ferritin level with height, weight, BMI and MAC in study group was observed. Thus there was growth retardation along with iron overload but the difference was not statistically significant.

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An Unusual Case Masquerading Tuberculous Meningitis: A Case Report

Anuradha Garg[1], Bhawna Dubey [2]

[1]Specialist, Department of Pediatrics, Hedgewar Arogya Sansthan, Vishwas Nagar, Shahdara, Delhi 110032, India. [2]Professor, Department of Neonatology, Atal Bihari Vajpayee Institute of Medical Sciences & Dr RML Hospital, New Delhi 110001, India.

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Abstract

Background: Scrub typhus is known to cause meningitis in children. Literature on neuroimaging findings in scrub typhus is very scarce. Case characteristics: A case of acute meningoencephalitis with raised intracranial tension demonstrated basal meningeal enhancement and ventricular prominence on CECT brain. In view of clinical non-responsive and CECT findings, antitubercular therapy was started. Subsequently rickettsial serology came out to be positive for Orientia tsutsugamushi; good response was observed to doxycycline. *Outcome:* Dramatic clinical response to doxycycline was observed favoring a diagnosis of scrub typhus meningitis.

Keywords: Scrub typhus; Tuberculous meningitis; Basal meningeal enhancement.

Introduction

Scrub typhus has been known to cause CNS involvement in children and meningitis/ meningoencephalitis occurs in 6 to 14% of affected patients.[1,2] However there is paucity of data on this topic in pediatric age group.

Tuberculous meningitis (TBM) which is highly prevalent in India closely mimics scrub typhus meningitis (STM) in terms of clinical features and CSF analysis.[3] However due to scarcity of literature on neuroimaging findings in rickettsial meningoencephalitis it becomes difficult to differentiate between the two entities. Basal meningeal enhancement and early hydrocephalus on CECT (contrast enhanced computed tomography) brain are are a characteristic finding seen in TBM.

We report an unusual case of aseptic meningitis with basal meningeal enhancement and hydrocephalus on CECT brain which proved to be due to scrub typhus in etiology.

Case Report

A 7-year-old developmentally normal boy presented with complaints of fever with chills for three days and one episode of generalized tonic clonic seizure leading to altered sensorium for last few hours. At admission temperature was 38.9°C. Rest of the vitals were within normal limits. His weight was 20 kg. The child was in altered

Corresponding Author: Anuradha Garg, Specialist, Department of Pediatrics, Hedgewar Arogya Sansthan, Vishwas Nagar, Shahdara, Delhi 110032, India.

E-mail: manglaanu@yahoo.com

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sensorium and neck stiffness was present. Tone was increased in all limbs and deep tendon reflexes were brisk. Cranial nerves were normal on examination. Pupils were normal in size, normally reacting. Conjunctivae were congested. Examination of abdomen revealed a mild hepatosplenomegaly. Investigations revealed a TLC of 24 × $10^3/\mu$L with a predominance of neutrophils (70%) hemoglobin of 111 g/L and platelet count of 90 × 10^9/L. RBS and Serum electrolytes were reported to be in normal range. Hepatic transaminases were raised (SGOT-70 U/L, SGPT-68 U/L). Serum albumin was 35 g/L diagnosis of acute meningoencephalitis was kept and broad spectrum antibiotics; ceftriaxone, vancomycin and antiepileptic drugs were administered. Papilledema was present on fundus examination therefore mannitol and dexamethasone were administered. Investigations like typhidot IgM, malaria card test came out to be negative. On day 2 of admission mild distention of abdomen with billious gastric aspirates was noted. Bowel sounds were present and abdomen was nontender. X-ray abdomen and serum electrolytes were normal. The patient did not pass stool for three days. On day 3 due to persistance of fever, altered sensorium and GIT manifestations meropenem was started in place of ceftriaxone. On day 5, nasogastric aspirates and abdominal distention subsided. Due to persistence of papilledema lumbar puncture was withheld. Blood culture and urine culture showed no growth. The history was reviewed. The child belonged to agricultural background and a single eschar was present in inguinal region. There was no rash. Hence serology for rickettsial diseases was sent. CECT brain was done which revealed a mildly prominent ventricular system with periventricular ooze in bilateral occipital lobes with irregular leptomeningeal enhancement along bilateral tentorium cerebelli and basal cisterns (Fig. 1).

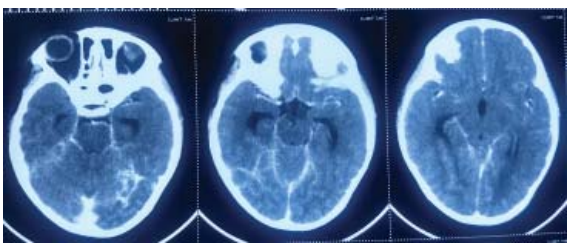

Fig. 1: CECT brain shows a mildly prominent ventricular system (third ventricle) with periventricular ooze in bilateral occipital lobes. There is irregular leptomeningeal enhancement along bilateral tentorium cerebelli and basal cisterns.

Dengue serology and Japanese encephalitis serology came out to be negative. Acetazolamide was started and keeping a possibility of TBM,work up for Koch's was done which came out to be negative. On day 7, the fundus examination showed a resolving papilledema, lumbar puncture was done. Mild pleocytosis (20–25 lymphocytes/mm^3) with raised protein (65 mg/dl), CSF: blood glucose ratio of 0.8, and negative gram and Ziehl-Neelson staining were observed on CSF examination. CSF CBNAAT was also negative. In view of persistent fever and altered sensorium; and CECT findings four drug antitubercular therapy (ATT) was started. After three days IgM ELISA tested positive for Orientia tsutsugamushi, hence oral doxycycline was initiated. Over next 48 to 72, hours fever subsided and sensorium improved dramatically. The papilledema subsided after a couple of days. ATT was continued and doxycycline was given for 10 days. Repeat CECT done after 3 weeks was normal. ATT had been given for a total of 20 days and was omitted. Post-ATT withdrawal the child continued to improve and neurological examination at discharge was normal. His 3 months, 9 months and one year follow-up post-discharge was also uneventful.

Discussion

Rickettsiae are increasingly being recognized as important pathogens causing multisystemic involvement in cases of acute febrile illnesses. Due to low index of suspicion, absence of eschar in a large number and non availability of diagnostic tests many cases are missed. GIT manifestations ranging from vomiting, diarrhea, pain abdomen to acute surgical abdomen can occur in children especially in early part of clinical course.[5] GIT manifestations were present in our case during early course. Normal to low TLC counts in early stages with leukocytosis later on, thrombocytopenia, elevated hepatic transaminases, hypoalbuminemia, hyponatremia are suggestive lab features which point towards rickettsial etiology.[6] Our case also demonstrated thrombocytopenia and elevated hepatic transaminases whereas serum albumin and sodium were in normal range.

CSF analysis in scrub typhus meningitis shows mild to moderate pleocytosis, mildly raised protein, normal CSF: blood glucose ratio.[2,7] We also obtained similar results. Due to our resource limited setting CSF investigations, i.e. PCR for HSV and *M. Tuberculosis* and MRI brain could not be done. We could find very few case reports/studies of STM in children in whom neuroimaging was done. In these CT brain was either normal or non specific showing brain edema.[7,8] Neuroimaging

in adults in such cases shows microhemorrhages, periventricular ooze, infarct in lenticular nucleus and transverse myelitis.[9]

Basal meningeal enhancement and early hydrocephalus on CT favors TBM.[4] Subsequent to ATT initiation IgM ELISA for scrub typhus came out to be positive. Rickettsial and TBM presenting as dual infection causing acute encephalitis syndrome has been reported in adults.[10]

Facing this diagnostic dilemma both doxycycline and ATT were administered initially. Short history, clinical, epidemiological scenario and lab findings pointed to diagnosis of STM. Our patient had thrombocytopenia and splenomegaly which are known to be more commonly associated with STM than TBM.[3]

Dramatic clinical response to doxycycline and normalization of neurological status and CT within such a short period favored diagnosis of STM and hence ATT was omitted. Post-ATT withdrawal he continued to improve which further consolidated the diagnosis of STM.

Hence rickettsial meningitis should be kept as a differential diagnosis in cases of aseptic meningitis with basal meningeal enhancement and ventricular prominence on CT. Clinical differentiation between STM and TBM based on thrombocytopenia and splenomegaly could be highly useful. Facilities like rickettsial serology should be available even in peripheral hospitals in endemic areas to enable timely diagnosis. This case report lays down the foundation on which further research on neuroimaging findings in rickettsial meningitis can be conducted.

Conclusion

Scrub typhus meningitis can cause basal meningeal enhancement and early hydrocephalus on CECT brain.

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Case Report

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Dengue Myocarditis, Bicuspid Aortic Valve, Acute Chest Pain in a 12-year Boy Treated with Streptokinase

Jayendra R Gohil[1], Atul M Sheladiya[2], Chintu C Chaudhary[3], Tushar S Agarwal[4]

[1]Professor, [2,3,4]Resident, Department of Pediatrics, Sir TG Hospital and Govt. Medical College, Jail Road, Bhavnagar, Gujarat 364001, India.

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Abstract

Myocardial ischemia and myocardial infarction is rare in children, with Kawasaki disease in children and substance abuse in adolescents, being the main cause. We present a case of a 12-year child suffering from Dengue, with bicuspid aortic valve, complicated by aortic valve insufficiency, who presented with chest pain and ECG changes of myocarditis and acute coronary syndrome; a rare occurrence. He was referred to us with complains of fever, cough, black colored stool, burning abdominal pain, and vomiting after 4 days of hospitalization at a district hospital as dengue illness. On day 9 of illness, he developed large hematemesis, with severe precordial chest pain, and was diagnosed initially as a case of acute myocardial ischemia due to myocarditis following dengue fever. ECG showed ST depression and troponin-I was markedly raised. Oxygen, ventilator, inotropes, streptokinase in divided doses (after bleeding had stopped), and steroids, zinc and vitamins C, E were administered. ECG changes reversed in a few hours and *q* waves started to appear. Later, 2D ECHO showed bicuspid aortic valve with aortic valve insufficiency. Myocardial enzymes, troponin-I and ECG returned to normal after 14 days. He was discharged on cardio supportive medications and advised valve replacement. Bicuspid aortic valve may remain undiagnosed till adolescence and can present rarely as acute myocardial ischemia following Dengue that was treated with streptokinase.

Keywords: Bicuspid aortic valve; Adolescent; Electrocardiogram; Acute myocardial infarction; Streptokinase; Dengue.

Introduction

Though rare, Myocardial infarction and ischemia (MI) in adolescents is associated with substance and alcohol abuse. We report a young patient (12-year) with MI with bicuspid aortic valve, an association that has been reported only once.[1,2]

Case History

A 12-year-old boy was referred from District hospital (120 km) as clinical dengue fever, after 4 days of hospitalization with: fever since 4 days, cough since 3 days, black stool since 2 days, burning abdominal pain since one day, and vomiting two

Corresponding Author: Tushar S Agarwal, Resident, Department of Pediatrics, Sir TG Hospital and Govt. Medical College, Jail Road, Bhavnagar, Gujarat 364001, India.

E-mail: jayukids@yahoo.com

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episodes. His vitals were Temp- 37.3 C, Pulse: 82/min, Respiratory rate: 24/min, BP: 96/62 mm Hg. IV fluid was started (4 ml/kg/h). No Antibiotics were given. Patient was improving except for dry cough. On D3 hemoglobin level reduced from 11 to 9 g/dl and platelet from 60,000 to 35,000/mL. On D8, hemoglobin further reduced to *5* g/dl, while there was no bleeding from any site. On D9, patient developed two episodes of hematemesis and severe burning retro-sternal pain. Red cell concentrate was given and stomach wash with cold saline was done, along with iv pantoprazole, ethamsylate, haemo-coagulase, and tranexamic acid. There was no improvement and retro-sternal pain did not respond to analgesics. 400 ml of blood was collected in seven hours via Ryle's tube.

Vitals: temp-N, pulse-102/min, BP-86/66 mm Hg, resp. rate-30/min. Auscultation revealed a murmur thought to be haemic.

Investigation: On ninth day, as per Table 1, and cardiac markers were: Creatinine kinase (CKMB)-35 U/L, Lactate dehydrogenase (LDH)- 500 U/L.

Table 1: Investigations on ninth day

Investigations	Values
Hb/Hematocrit	4.2 g/dL / 12%
TLC K/uL	6
DLC P/L/E/M %	50/45/3/2
Platlets K/uL	120
ALT/AST U/L	74/110
S creatinine	0.06 mg/dL
Urea	15.3 mg/dL
Sodium	131 mmol/L
Potassium	4.3 mmol/L
Prothrombin time	14 sec/INR 1

TLC— Total leucocyte count, DLC— Differential LC %, ALT— Alanine amino transferase, AST— Aspartate amino transferase, K— Thousand

On *tenth* day were ALT - 55 U/L, AST - 143 U/L, Cardiac Marker: CKMB 124 U/ L (18-51), LDH-754 U/L (125–220), S. Amylaze-71 U/L, S. Troponin-I - 4.5 µg/L (0.0–0.01). ECG: ST depression in all leads with sinus tachycardia 150/min (Fig. 1).

He was provisionally diagnosed as Myocardial Ischemia due to myocarditis caused by dengue

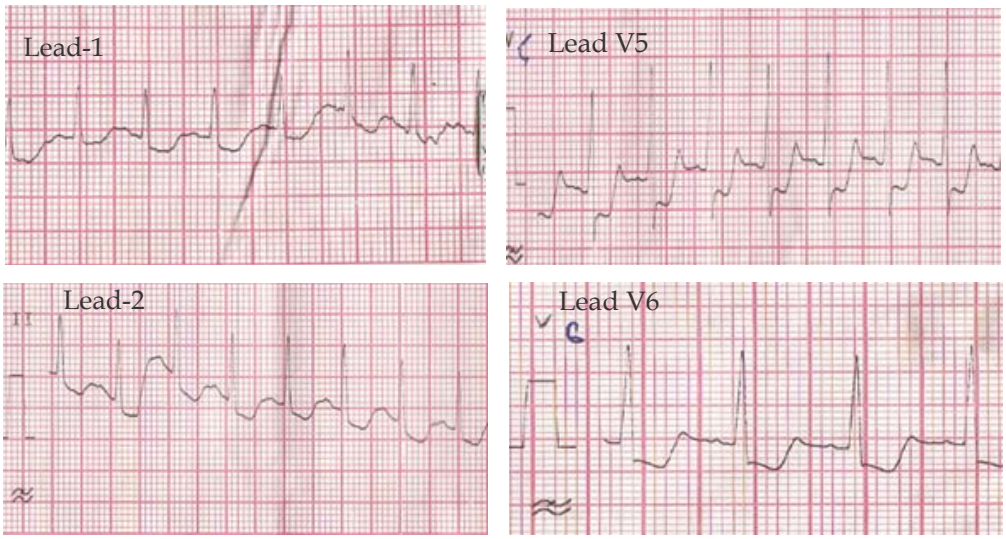

Fig. 1: ECG showing ST depression in all leads with sinus tachycardia.

fever. Chest pain was not relieved by analgesics so inj tramadol was given at 00:30 am. Inj dexamethasone was given as 3 mg/kg loading, followed by 1 mg/kg 8 hourly for myocarditis. Following tramadol chest pain was relieved but he developed respiratory depression, so he was intubated, ventilated and inotropes were started. He was unconscious, on ventilator and with low BP. Within 12 h *Troponin-I* level increased from 4.5 to *17.7* µg/L, however, bleeding had stopped. Further repeat ECG was not obtained. As the triad of severe chest pain, raised biomarker and ECG changes suggested *Acute Coronary syndrome* (ACS); inj. streptokinase was administered as bleeding had stopped and, in ¼ dose of 2000 IU/kg slowly over 15 min.[3] Remaining three doses were given at an interval of 1 hour each. Divided doses were given as bleeding is its side effect. At 9 am, Patient

started improving, with *q* wave in V5 starting to appear, and ST-T changes disappearing (Fig. 2). He recovered from shock and was weaned from ventilator.

Next morning patient was referred to Cardiology center, Ahmedabad (180 km). There he received mechanical ventilator care for 3 days and inj. Methyl Prednisolone (40 mg/kg) for 3 days. 2D ECHO

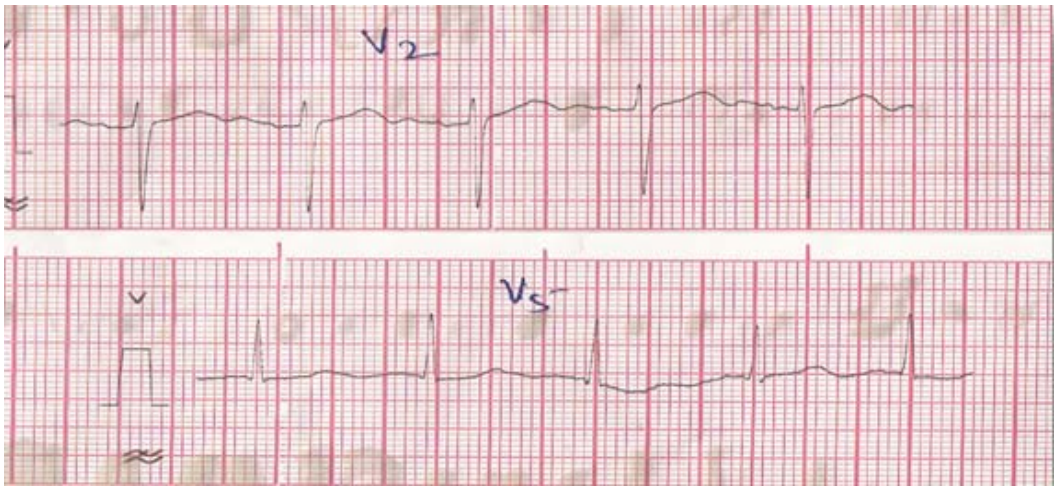

Fig. 2: ECG following 4 divided doses of streptokinase showing disappearance of ST depression.

done for the first time showed *bicuspid aortic valve with valvular insufficiency*, hitherto undiagnosed. He was discharged after five days with advice for valve replacement on follow-up.

Discussion

Myocardial infarction is defined as: Elevated blood levels of cardiac enzymes (CKMB or Troponin T) in typical pattern and one of the following criteria are met: ischemic symptoms, pathological Q waves, ST elevation or depression or coronary intervention (stent).[4]

ACS or acute myocardial ischemia/infarction (MI) is extremely rare in adolescents. Patient characteristics associated with ACS include substance abuse, tobacco use, and male sex. The hospital survival for ACS in adolescents is excellent, and the need for catheter or surgical coronary artery intervention is uncommon.[1]

Bicuspid aortic valve is a congenital cardiac anomaly, having an incidence 0.9 to 2.0% and a frequency of 54% in valvular aortic stenosis after 15 years.[5] Association of bicuspid aortic valve is with aortic stenosis, regurgitation, dissection; and infective endocarditis. Left coronary artery is dominant (29–57%) and in 90% it is <5 mm in length, leading to insufficiency.[6,7]

Towbin reported incidence of MI in children from retrospective data from 1954 to 1986. Over 1,00,000 ECGs on 54,605 patients were obtained. Pediatric admissions were 1,013,210 and 31,305 were referred to cardiology. 72 had ECG consistent with MI. These included: anomalous left coronary artery (20%), Kawasaki disease (14%), myocarditis (13%), neonatal critical aortic stenosis (11%), ventricular tumor (5.5%), dilated cardiomyopathy (4%), pulmonary atresia-intact ventricular septum (4%), birth asphyxia (4%), aortic thrombosis (4%), muscular dystrophy (2.5%), coarctation of aorta (2.5%), rheumatic carditis (2.5%), total anomalous pulmonary venous return TAPVR (2.5%), and chest trauma (2.5%).

The positive ECGs represented 0.13% of ECGs, 0.23% of cardiology referrals and 0.007% of pediatric admissions. There was no case of bicuspid aortic valve in this series.[8]

The initial ECG indicated ST segment depression in all the leads and Q wave appeared after the disappearance of the ST segment curve after treatment with streptokinase. The major initial triaging decision in acute coronary syndrome (ACS) is whether or not percutaneous coronary intervention (PCI) is the primary treatment; current guidelines recommend primary PCI in ST-elevation ACS (STEACS)[9] and initial antithrombotic therapy in non-ST-elevation ACS (NSTEACS).[10] However, electrocardiographic STE and NSTE patterns are not uniquely related to distinctly

different pathophysiological mechanisms.[10] Cardiac troponin-I (appears after 4–6 hours and increases and remains high up to 7–10 days) is a sensitive and specific marker of ACS or MI. In this boy, the initial cardiac troponin-I and CK-MB increased by 400 times and 1700 times, respectively. The congenitally bicuspid valve may function normally and go undetected throughout life, may develop calcification and stenosis or may develop regurgitation or infection. Calcification is the most common cause of isolated aortic stenosis.[11] Aortic regurgitation was reported to be the cause in 1.5 to 3%.[12] It may occur in isolation, usually as a result of prolapse of the larger of the cusps, but also in association with aortic root dilatation, coarctation of the aorta, or infective endocarditis.[13] Along with bicuspid aortic valve, this patient had aortic valve insufficiency. Kabra reported that 17% (7/54) children had decreased Ejection Fraction.[14] Bicuspid aortic valve may be a rare (only one case reported so far) cause of ACS in children especially when it is complicated by aortic valve insufficiency. Sometimes ECG changes can be non-supportive, in such cases typical precordial chest pain with raised myocardial enzymes can help in diagnosis of ACS or MI. As guidelines for ACS are for adult patients, we had difficulty in adopting them for a child with congenital heart disease (CHD).

Limitation: Dengue tests were not done and, portable Echo facility was not available at our building.

Conclusion

- Undiagnosed *bicuspid aortic valve* can present as Acute myocardial ischemia (or infarction) or *Acute coronary syndrome* in an adolescent with Dengue fever.
- Administration of inj. *streptokinase* in divided doses over a period of 4 hours in patient having ACS with history of gastrointestinal bleeding resulted in improvement.
- Portable or POC (point of care) Echocardiography is a necessity in intensive care.

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Participation involved informed consent received from the family. All authors declare no conflict of interest and no financial relationship or assistance or interest.

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Growing Pains in Children: Over Diagnosis?

Mitali Borgaonkar[1], Santosh Kondekar[2], Prachi Gandhi[3], Surbhi Rathi[4]

[1]Third Year Resident [2]Associate Professor [3]Lecturer [4]Professor and Head of Unit, Department of Pediatrics, Topiwala National Medical College & B.Y.L. Nair Charitable Hospital, Mumbai, Maharashtra 400008, India.

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Abstract

Background: Growing pains being episodic benign pains are often over diagnosed and under evaluated. *Case Characteristics:* A 4-year-old girl presented with complaints of frequent intermittent knee joint pain especially nocturnally. Serum profile studies were within normal limits. Ultrasound of both knee and hip joint was normal. MRI of right knee showed patellar osteitis with retropatellar serositis. *Outcome:* A short course of oral steroids resulted in symptomatic relief within 3 days where a treatment for 4 months with NSAIDs could not provide major relief.

Keywords: Growing pains; Retropatellar serositis; Kohler's disease; Naish and Apley diagnostic criteria.

Introduction

Growing pains are typically intermittent, nocturnal and poorly localized, usually experienced in the lower extremities. Children suffering from 'growing pains' are characteristically well without any physical problems, despite severe pain experienced in the night. The usual age group is 4–14 years with equal gender preponderance.[1] Here we discuss a case suspected to be one of growing pains but was later diagnosed as Kohler's disease.

Case Report

A 4 year-old girl presented with the complaint of intermittent pain in the right knee especially in the night between 2 and 4 am. These episodes occurred once a week in the beginning; but over the month she had daily episodes of knee pain along with tenderness and restriction of movement with inability to walk and squat mainly during the painful bout. She was symptomatically better during daytime with no reason to skip school or day-to-day activities, yet she refused to run or climb stairs without support. Within a month a similar complaint of pain was also noted in the left knee with intermittent episodes and worsening late at night requiring analgesics for relief.

During this course, she was evaluated by the pediatricians and orthopedists as a case of growing pains. Counseling for benign nature of the disease and weekly follow-up was done. As the pain lasted for more than 3 months, the child was investigated for calcium, vitamin D, rheumatic profiles and MRI of both the knees. The investigations revealed

Corresponding Author: Santosh Kondekar, Associate Professor, Department of Pediatrics, Topiwala National Medical College & B.Y.L. Nair Charitable Hospital, Mumbai, Maharashtra 400008, India.

E-mail: drkondekar@gmail.com

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the serum profiles to be within the normal limits. However, the MRI displayed retropatellar serositis. On further evaluation by a Rheumatologist the diagnosis of Kohler's disease was made.

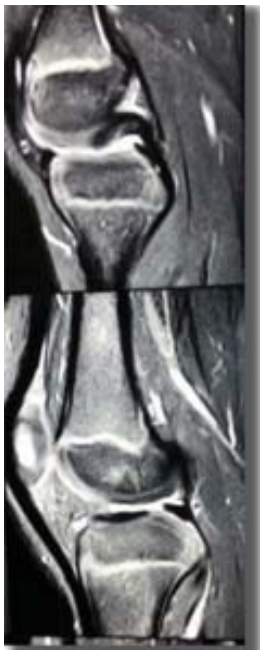

Fig. 1: MRI of knee joint of the same girl s/o of retropatellar serositis.

The child had symptoms lasting for 4 months during which paracetamol and naproxen medication were given along with calcium and vitamin D supplements; which however could not provide major relief. At the end of 4 months, considering the diagnosis as reactive osteitis, a short course of oral steroids was started giving the patient symptomatic relief within 3 days.

Discussion

Kohler's disease is a self-limiting disease characterized by avascular necrosis (AVN) of primary or secondary centers of ossification; classically involving navicular bone but cases involving other bones such as patella have also been reported.[2–4] It is known to have intermittent painful episodes. While growing pain is accepted as a clinical entity, it is a diagnosis of exclusion. The unawareness of a diagnostic criteria often hides the underlying pathology. In our case the term 'growing pains' has been applied mistakenly which misdirected from evaluating in detail a case that happens to be of Kohler's disease.

Kohler's disease shows the following classical features.[5]

- Osteochondrosis due to avascular necrosis of the navicular bone.
- Presents with pain and swelling in the middle part of the foot and usually limps as a result. Patients that walk with a limp tend to walk with increased weight on the lateral side of the foot.
- Tenderness over the navicular and often complain of pain over the apex.

In this case, it involves the patella instead of navicular bone indicating transient patellar osetitis.

Even though growing pains is one of the most common cause of musculoskeletal pains, there is a danger of over diagnosis. The inclusion and exclusion criteria for growing pains should be considered before making a definitive diagnosis in favor of it.

Conclusion

A high degree of suspicion for diagnosis of other entities should be considered when the pain involves joints and bones. The Naish and Apley diagnostic criteria[6,7] specifically points towards growing pains by outlining basic prerequisites such as intermittent lower limb pains for a period of at least 3 months, not specifically located in the joints, and of sufficient severity to interrupt sleep (Practitioners' dilemma). Peterson provided with a definition for better clinical practice, it consists of inclusion and exclusion criteria (Practitioners' dilemma).[8]

Growing pains is in reality a clinical diagnosis without the need for laboratory inestigations or X rays and scans; yet not simply muscular pain but pain in the joint should always be investigated for other diagnoses.

Key message: A high index of suspicion for other diagnoses should be considered when the pain involves joints and bones.

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