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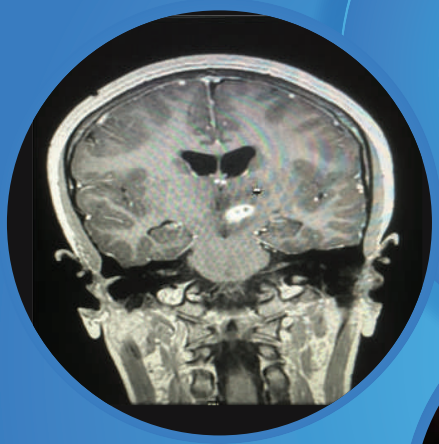

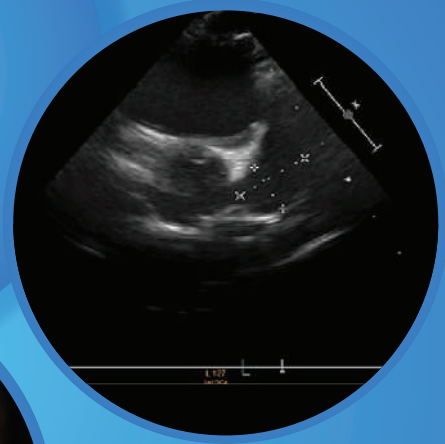

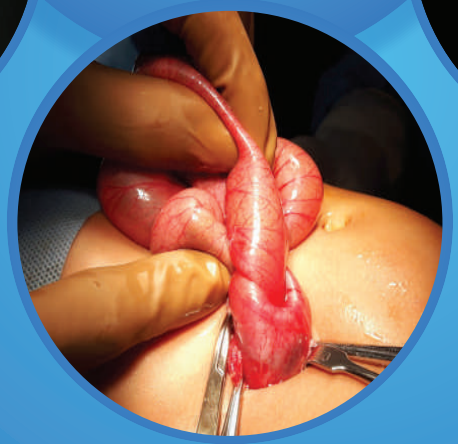

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Contents

Editorial

Warm Greetings from the editorial team of this issue of Chettinad Health City Medical Journal.

With immense pleasure we bring out this issue of Chettinad Health City Medical Journal, which has been uninterruptedly growing with contributions from energetic,enthusiastic,qualified and high profile researchers from all over the world.

Though the major focus of this issue is on Paediatrics, we still have articles from other specialities, like the original article on Assisted Reproduction and an article on the study of deaths due to electrocution. The original articles from Paediatrics will enlighten us on simpler investigation and treatment modalities, as in how a urinary pH test can be used as a predictor for urinary tract infection, and how we can avoid surgery in most of the children with uncomplicated intussusceptions.

In the review article on Approach to chest pain in children and adolescent , authors highlight that in majority of cases detailed history and physical examination would suffice and only few cases need investigations and referral to cardiologist .

We have case reports on Tuberous Sclerosis Masquerading as febrile seizure, Tuberculoma vs Neurocysticercosis, Salbutamol overdose and Kawasaki disease. Interesting case reports from other specialities, on Anaesthetic management for airway laser surgery for Laryngomalacia and on Terlipressin induced ventricular tachycardia have also been included.

The class room article on development of infant and young child is quite informative to all the medical students and practitioners and will help to pave way for early diagnosis and thereby early intervention.

The pages of history takes us on a journey to explore and witness the discovery of Insulin from Banting's ideas, and the article on Dialogue with the stalwart highlights the meeting with one of the senior most practicing pediatrician and a popular teacher in Tamil Nadu : Prof. Dr.Mohammed Thambi.

With wide coverage of various topics of differing medical specialities , this issue of the journal will be interesting and also very informative for all the enthusiastic readers.

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Original Article

Non-Operative Management of Intussusception in Children: A Single Surgeon's Experience

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Chettinad Health City Medical Journal 2016; 5(2): 60 - 63

Abstract

Background: Intussusception is one of the most common causes of acute abdomen in infants and preschool children. With the introduction of non-operative enema reduction into practice, the role of surgical management of intussusception in children is becoming narrower. Advantages of ultrasound guided saline enema reduction (USGSER) over fluoroscopic guided pneumatic reduction made it standard first line approach in majority of centers. Aim of this study is to analyze the results of intussusception cases managed by USGSER by a single surgeon. Methods: Retrospective review of all cases of intussusception from February 2010 to August 2016 was done. Demographics, clinical presentation, investigations, management and outcomes were reviewed. Results: Out of 59 intussusceptions managed during the study period, 5 cases taken for upfront surgery were excluded. All the remaining 54 cases have undergone USGSER and 51 (94%) were successfully reduced. Conclusion: In view of high success rate with very minimal complications, non-operative management should be the first line treatment for all uncomplicated cases of intussusception in children.

Key Words: Intussusception, ultrasound, saline enema.

Introduction

"Intussusception" is derived from the Latin words "intus" (within) and "suscipere" (to receive)[1] and it means invagination of proximal segment of bowel (intussusceptum) into the lumen of distal bowel (intussuscipiens)[2] . It is one of the commonest causes of intestinal obstruction in infants and preschool children with a reported incidence of 1.5–4.3 per 1,000 live births[3]. Intussusception has been reported in prenatal period[4] to very elderly age group[5], but 3/4th of cases occur in less than 2 years of age with peak incidence between 4 and 10 months of life[6]. It has been classified into several types according to the area of involvement such as Ileo-colic, Ileo-ileo-colic, colo-colic, Ileo-ileal and jejuno-jejunal with Ileo-colic intussusception as the most common type (85%)[7].

Even though Hirschsprung published his experience on hydrostatic reduction in 1876[8], non-operative management of intussusception was not widely accepted till the later half of 20th century. In 1948, Ravitch reintroduced hydrostatic barium enema under radiologic guidance[9], but pneumatic reduction under fluoroscopy became popular only from 1992[10]. Since fluoroscopic facilities are not widely available especially in developing countries, ultrasound guided saline enema reduction (USGSER) gained wide acceptance and became standard first line management for all uncomplicated cases of intussusception.

This study analyses the efficiency of USGSER done by a single surgeon for the management of intussusceptions in children.

Materials & Methods

With the objective to analyze the efficacy of USGSER as the primary management for intussusception in children, retrospective analysis of all cases of intussusception managed by a single surgeon from February 2010 to August 2016 was done. Inclusion criteria: All patients presenting with clinical diagnosis of intussusception and confirmed by ultrasound examination during the study were included.

Exclusion criteria: Patients taken for upfront surgical management for various indications were excluded. All patients were admitted once the diagnosis was confirmed by ultrasound. They were kept nil per mouth, adequately resuscitated with IV fluids and started on IV antibiotics. The parents were explained in detail about the etiopathogenesis, options of operative and non-operative management, advantages of non-operative treatment and the need for emergency surgery in cases of complications or failure and written informed consent was taken. Duty radiologist was informed and emergency operating room was kept ready as standby.

Injection midazolam (0.1 – 0.2 mg/kg) was given slowly through intravenous route at the start of the procedure; this makes the babies quiet and co-operative. A large Foley's catheter (22F or 24F) was inserted per rectally after adequate lubrication and balloon was inflated with 30 ml of normal saline. A saline bottle kept about 100 cm height was connected to the Foley's catheter through an IV drip set and free flow of saline into the rectum was monitored with

ultrasound. Saline gradually fills up the colon and pushes intussusceptum back towards cecum. Usually it takes some time for the proximal part of intussusceptum to get reduced through ileo-cecal valve and babies wake up and strain at that period. Due care to avoid excessive pressure while baby is straining and maintaining constant height of saline bottle on reduction are the keys to avoid perforation. Complete disappearance of mass and free flow of saline through ileo-cecal valve into the ileal loops were the markers of successful reduction which makes the babies become immediately comfortable. Filled saline was allowed to drain under gravity and Foley's catheter was removed. Cases that could not be reduced in the first attempt underwent the same procedure again at 5 minute interval. After successful reduction, babies were kept under monitoring and allowed orally once they passed greenish/yellowish stools. A check ultrasound after 24 hours of reduction to rule out any recurrence was done in all cases and they usually get discharged within 48 hours of procedure. Patients were taken up for emergency surgery if intussusception could not be reduced even after second attempt and in cases of complications.

Results

A total of 59 intussusceptions in 57 patients were managed during the study period. Of the 59, five cases taken up for upfront surgical management for various indications were excluded. Fifty four intussusceptions in 52 patients including one child with 2 episodes of recurrence were managed primarily by USGSER. There were 38 males and 14 females (Male: Female =2.7:1) and age ranged from 4 months to 5 years with 45 (86%) less than 2 years of age. Sudden onset of intermittent colicky abdominal pain was the commonest presenting symptom (96%) and abdominal mass on clinical examination was present only in 22 (40%) patients (Table 1). The diagnosis of intussusception was confirmed by ultrasound in all patients with classical "target sign" on axial view and "sandwich sign" on longitudinal view. The distal level of intussusception was at transverse colon in 18 (33%), at hepatic flexure in 8 (15%), at ascending colon in 26 (48%) and at cecum in 2 (4%) patients.

Clinical Feature	Number	Percentage
Pain abdomen	52	96%
Vomiting	37	68%
Bleeding per rectum	32	59%
Abdominal mass	22	40%

Table 1 - Clinical Features

Successful reduction of intussusception was achieved in 51 out of 54 cases (94%) and none of them had immediate recurrence within 24 hours of procedure. The amount of saline instilled for reduction varied from 300 to 800 ml and average duration of the procedure was about 15 minutes. One patient had perforation during the procedure, which was immediately noticed by ultrasound and underwent emergency laparotomy, manual reduction and resection of perforated bowel with end to end anastamosis. The perforation was at transverse colon and an enlarged edematous appendix and terminal ileum seems to be blocking the reduction. Patient had uneventful post-operative recovery and discharged. The other two patients who had failed reduction were also taken up for emergency manual reduction and no specific lead points were noted in both patients. Only one patient had 2 episodes of late recurrence (6 months and 14 months after first procedure) and successfully reduced non-operatively on both episodes. None of the patients in this study had undergone delayed hydrostatic reduction.

Discussion

Intussusception is one of the most common surgical emergencies in children and has been described in detail by Hunter way back in 1793[11]. Even though various treatment methods were reported in literature, surgical reduction was the mainstay of management till image guided hydrostatic or pneumatic reduction came into practice. In 1952, Ravitch and McCune used barium sulphate enema to diagnose as well as reduce intussusception, calling this "hydrostatic reduction" and reported 73.6 % success rate without mortality[12]. But lack of fluoroscopic facilities made this technique almost unavailable in most of the developing countries. In the mean time, ultrasound became the investigation of choice to diagnose intussusception with 100% accuracy. In 1982, Kim et al[13] reported the first successful ultrasound guided saline reduction of intussusception and it is followed by many articles confirming safety and high success rate of this technique[14,15].

Sudden onset of severe intermittent colicky abdominal pain which makes the babies cry with drawing up of their legs is the classical presentation of intussusception and it is usually associated with vomiting and blood and mucus (red currant jelly)stools (Fig 1). Clinical examination may reveal a sausage shaped mass with concavity towards umbilicus and blood stained fingers on per rectal exam will clinch the diagnosis. Confirmation of the diagnosis was done by picking up "target sign" (Fig 2) and "sandwich sign" (Fig 3) on high resolution (7.5 - 10 MHZ) ultrasound. Using color Doppler to check the blood flow of intussusceptum and identification of obvious lead points are the added advantages of ultrasound. None of our patients had compromised blood supply on Doppler and enlarged mesenteric nodes were present in most of the patients.

Fig 1 : Baby with Red currant jelly stools

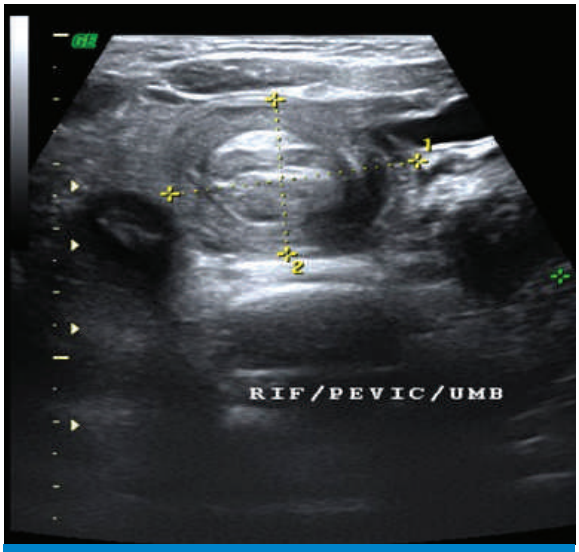

Fig 2 : Target Sign

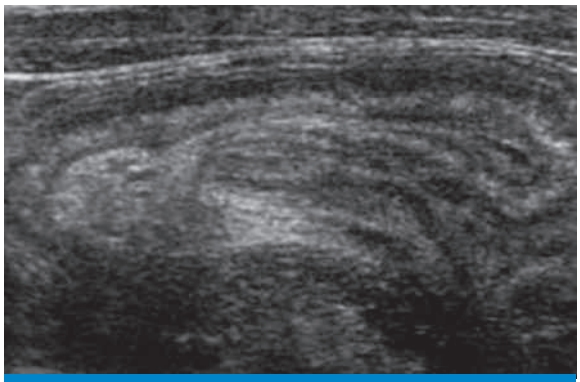

Fig 3 : Sandwich Sign

Once the diagnosis is made, patients should be adequately resuscitated and managed as early as possible as risk of bowel ischemia and perforation increases with time. In this study, we have corrected dehydration in all patients and one dose of IV antibiotics was given before the procedure to reduce the risk of sepsis. USGSER was used as the standard technique in all our patients and it has several advantages over fluro-guided pneumatic reduction[16] (Table 2).

Advantages of USGSER
• Readily available in most of the hospitals
• No Radiation hazard
• Simple, safe, effective, economical and less time consuming
• Less morbidity and shorter hospital stay
• Less complication and failure that can be diagnosed instantly
• Recurrences can also be managed safely

Table 2 : Advantages of USGSER

Even though several advantages of USGSER were quoted in the literature, the author believes that the biggest advantage of this technique is its easy availability and it can be safely done even in small nursing homes with operation theater. Even in centers with facilities, pneumatic reduction under fluro-guidance disadvantage of exposing the patient and treating team to radiation. Daneman et al reviewed the hydrostatic reduction techniques of intussusception according to success rates, complication rates, advantages and disadvantages and concluded ultrasound guided techniques are superior[17]. High success rate of this technique has been verified by many authors and our study has also proved the same with 94% success rate.

Even though routine use of sedation was not mandatory for this procedure, many studies have used minimal sedation[14]. All our patients received a mild dose (0.1 – 0.2 mg/kg) of midazolam at the beginning of the procedure, which makes the children quiet and co-operative. None of the patients developed any complications related with sedation and we believe it is definitely beneficial for our increased success rate.

Intestinal perforation during USGSER reported in the literature is very low (0.26%)[18] mainly because of uniform pressure delivered during saline flow (See Fig 4) unlike fluctuating pressures in pneumatic reduction. One our patient had perforation during USGSER, which could have been due to excessive pressures developing at the reduction of proximal most intussusceptum through ileo-cecal valve and patients invariably wakeup and strain during that part of procedure. Perforation was immediately recognized on ultrasound and the patient was immediately managed by surgical reduction. On surgery it was found that perforation was at transverse colon and an enlarged edematous appendix and terminal ileum seems to be blocking USGSER got easily reduced manually. Unlike chemical peritonitis during barium enema reduction or tension pneumoperitoneum during pneumatic reduction, peritoneal cavity was relatively clean and filled only with saline which is another advantage of USGSER. The only perforation in this study happened during the earlier part of the series and avoidance of excessive pressure while babies are straining is the key reason to stay away from this complication in rest of the patients. Even though this complication is exceedingly rare and instantly recognized, it should be managed immediately to avoid mortality. It stresses the importance of immediate availability of surgeon and operative room and the author strongly recommends that USGSER should be done preferably by Pediatric Surgeons.

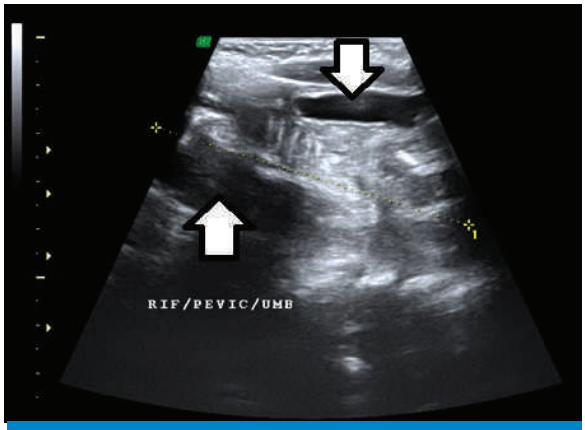

Fig 4 : Uniform flow of saline around intussusceptum

The remaining 2 failed patients were successfully managed surgically and no lead points were found to be the reason for failure (Fig 5). Only one patient in this study had 2 episodes of late recurrence (6 months and 14 months after first procedure) and successfully reduced by USGSER on both episodes. Even patients who have undergone previous surgical reduction were safely reduced by USGSER on recurrence[19].

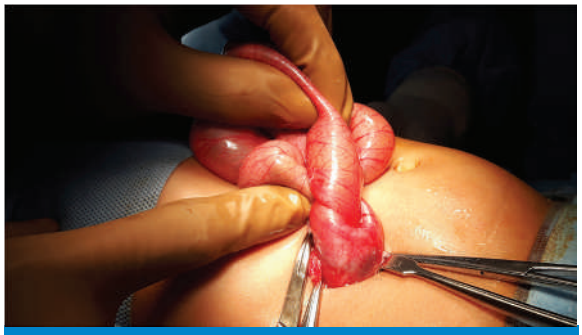

Fig 5 : Manual reduction

Conclusion

USGSER is simple, safe, effective, economical and quick with very few complications. It should be considered as the first line treatment for managing uncomplicated intussusceptions in children. This procedure should preferably be done by Pediatric Surgeons in view of serious risk of intestinal perforation.

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Original Article

Association Between Urine pH and Urinary Tract Infection in Children - A Hospital Based Cross Sectional Study

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Chettinad Health City Medical Journal 2016; 5(2): 64 - 66

Abstract

Objective: To assess the association between urinary pH and urinary tract infection (UTI), in children presenting with fever and symptoms of UTI.

Materials & Methods: Hospital based Cross sectional study was done. All children ≤ 18 yrs of age, admitted with fever and suspected urinary tract infection between September 2015 to February 2016 were included in the study with a sample size of 107 cases.

Results: The epidemiological indices - sensitivity, specificity and predictive values of the rapid screening tests (urine dipstick test) were calculated individually for urine pH and compared with a positive urine culture as the validating standard. Association between urine pH and UTI were analysed with Chi-Square test and Odds ratio. A ROC (Receiver operating characteristic) curve is plotted to illustrate the sensitivity and specificity.

Conclusions: Acidic urine pH can be taken as a good predictor of UTI in children presenting with fever and UTI symptoms with high sensitivity and specificity.

Key Words: UTI, urinary PH, urine analysis.

Introduction

Urinary tract infection (UTI) is one of the most common infections in children. It occurs in 3-10% of girls and 1-3% of boys[1]. Children are either asymptomatic or present with atypical signs and symptoms. The diagnosis and management of urinary tract infection (UTI) in young children is clinically challenging. The various tests used for the diagnosis of UTI are urine analysis, urine dipstick, esterase and urine culture. Urine culture is considered as gold standard for the diagnosis of UTI. Many studies have proved that there is a strong association between urine pH and UTI both in adults and children[2]. Urinary pH has a strong therapeutic implication in the treatment of UTI[3]. Alkalinization of urine is done as a treatment for UTI. The need for the study is that there are very few studies on Indian children and this study will have very strong diagnostic and therapeutic implication.

Aim:

To assess the association between urinary pH and urinary tract infection, in children presenting with febrile illness and symptoms of UTI.

Materials & Methods:

This is a hospital based cross sectional study with a sample size of 107 cases. This study was conducted in paediatric OPD, at Chettinad hospital and research institute, Chennai.

Study period: Over a 6 month period, between September 2015 to February 2016.

Inclusion criteria:

All children aged less than 18yrs admitted with fever and complaints suggestive of urinary tract infection were included in the study.

Exclusion criteria:

Children with concurrent infections, those on diuretics and those catheterized were excluded from the study.

A clean–catch midstream urine specimen, from these children was subjected to the standard urine analysis along with urine culture. As per routine clinical practice, the urine specimens were sent to the hospital lab in sterile containers. Urine pH was assessed using Uro-dip reagent strips (Urodip10A). Institutional ethical committee clearance was obtained. Descriptive analysis of demographic and clinical parameters were compiled into frequency and percentages. Association between urine pH and UTI were analysed by chi square test and odds ratio. A ROC (Receiver operating characteristic) curve was plotted to illustrate the sensitivity and specificity. The epidemiological indices - sensitivity, specificity and predictive values of the rapid screening tests (urine dipstick test) were calculated individually for urine PH and compared with a positive urine culture as the validating standard.The statistical analysis were made by IBM SPSS version 21.

Results:

A total of 107 children admitted to the hospital with suspected UTI were enrolled in the study, out of which 37 children were diagnosed to have UTI. Among them 17 were males and 20 were females. Among children with UTI 17 were males and 20 were females. Among children with UTI 27 % of the children were in <1 year age group, 45% in 1-5 year age group, 18 % in a 6-10 year age group and the remaining 10 % in 10 – 18 year age group as shown in **Fig 1**. The symptoms with which the children presented to the department are shown in **Fig 2.** Among 37 children with UTI, the commonest organism grown in urine culture is E coli (N=27, 72.9 %) followed by Proteus (N= 7, 19 %) and Enterococci (N=3, 8.1%) as shown in **Table 1**. The association between urine pH and suspected UTI (N=107) were analyzed. In urine culture positive children (N = 37) 77.8% of cases had acidic urine (N= 28) and 12.7 % of cases with neutral urine (N= 9). Among the children who did not have UTI, 22.2% of cases had acidic urine (N = 8) and 87.3% of cases had neutral urine (N= 62). The Chi-Square value was 44.754 and Odds ratio was 24.111 with p value <0.001 as shown in **Table 2.** The association between urine mean pH and suspected UTI (N=107) were analyzed, the mean urine pH was 6.10 in children with UTI (N=37) and 6.70 in children without UTI (N=70) with p value <0.001 as shown in **Table 3**. A ROC (Receiver operating characteristic) curve is plotted which illustrated 88.6% sensitivity and 75.7% specificity as shown in **Fig 3**.

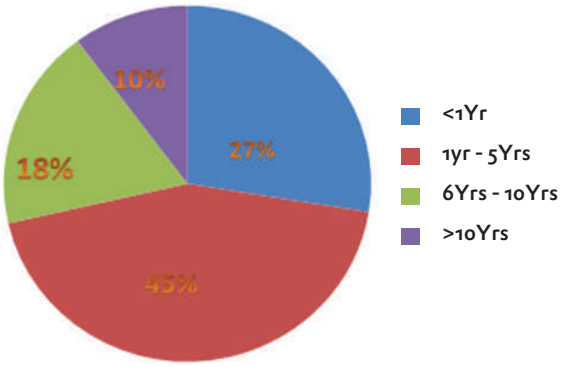

Fig 1 - Age distribution of UTI cases.
Age Distribution of Study Population (N=37)

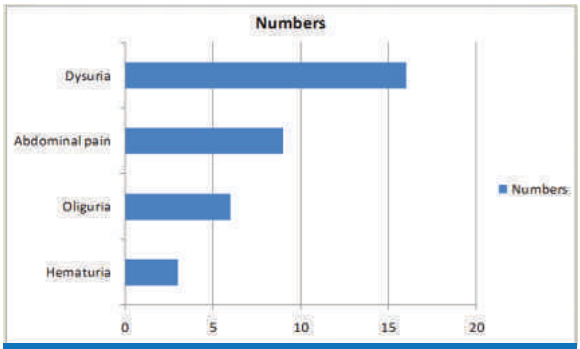

Fig 2 - Common presentation of UTI
Common Presentation N=37

Urine Culture	Frequency	Percentage
E coli	27	72.9
Proteus	7	19
Enterococci	3	8.1

Table 1 - Organism isolated in Urine Culture
Urine Culture And Sensitivity (N=37) - Organism

Urine PH	Final Diagnosis		Chi Square Value	Odds Ratio	P Value	95% CI Value	
	UTI	Non UTI				Lower	Upper
Acidic	28 77.8%	8 22.2%	44.754	24.111	<0.001	8.423	69.021
Neutral	9 12.7%	62 87.3%					

Table 2 - Association between Urine pH and UTI
Association Between Urine PH and UTI (N=107)

Diagnosis	Mean Urine PH	Mean Difference	P Value	95% CI Value	
				Lower	Upper
UTI (N=37)	6.10	0.591	<0.001	0.44	0.74
NO UTI (N=70)	6.70				

Table 3 - Association between Urine pH and UTI
Association between Urine mean pH and UTI (N=107)

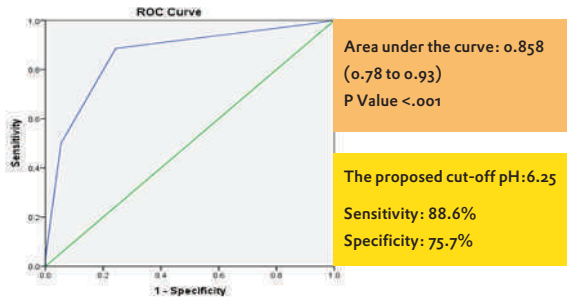

Fig 3 - ROC Analysis

Discussion:

UTI should be suspected in infants or children presenting with unexplained fever beyond three days[4-6]. In the study group of 107 febrile children 37 had culture proven UTI and 70 were culture negative. Studies by Whiting P, Westwood M et al showed that the incidence of UTI are common in female children aged less than 5years[7]. This study shows incidence of UTI is more common in females (54%) than males(45%). This study shows that 1 to 5 years being the predominant age group with UTI (55%). In this study, the commonest presentation was dysuria (43.2%, N=16) associated with fever. A study by Klinth JE et al has demonstrated that urinary pH plays a strong role in adhesion of *E.coli* to urinary tract epithelium[8]. In this study of 37 culture proven UTI, the commonest organism was found to be *E.coli*(72.9%).Studies by Mookerjee BK et al, Erdogan-Yildirim Z et al and Roasio N et al have demonstrated strong association between urine pH and UTI[11-9]. Among the N=37 culture proven UTI, 77.8%(N=28) were acidic pH with 12.7%(N=9) neutral pH. Among the N=70 non UTI,22.2%(N=8) were acidic and 87.3%(N=62) were of neutral pH. Antibiotic therapy of Amikacin or a third generation cephalosporin (cefotaxime or ceftriaxone) was given[12].Once the child showed clinical improvement, with resolution of fever and toxicity, antibiotics were administered orally based on the culture sensitivity. The current study reveals very high area under ROC curve (0.858, 95% CI 0.78 to 0.93) indicating that urine pH can be a good predictor of UTI.

Conclusion:

Acidic urine pH can be taken as a good predictor of UTI in children presenting with febrile illness with high sensitivity and specificity. Commonest organism in UTI in children being *Eschericia coli*.

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Original Article

Study of deaths due to Electrocution at Government Stanley Hospital, Chennai - An Autopsy Based Study

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Abstract

Background : Since its invention, electricity has gradually progressed from being a luxury to a necessity. Electricity and electrical appliances are indispensable in today's life. Industrialization and urbanization have greatly increased the expense of electricity. It is a source of much productivity and enjoyment. When used without adequate care and precaution the same can result in injuries resulting in morbidity and mortality. Accidental electrocution is a frequent mode of potentially preventable death in our modern civilization. Objective : To study the demographic and other features of deaths due to Electrocution, brought for autopsy. Materials and Methods : In this retrospective study done at Government Stanley Medical College, Chennai during a 2year period, we studied 28 cases of deaths due to electrocution. Results : All cases were accidental in nature. Maximum age incidence was in the age group 21-30 (32.14%) years followed by 41-50years (32.14%). All cases were male. Domestic accidents (75%) were more when compared to industrial accidents.

Key Words: Electrocution, electric shock, electricity.

Introduction

Injuries due to electrocution are very common in today's life. Statistics of electrical injuries may not be accurate due to under-reporting of injuries. Injuries are brought to the notice of treating and investigating authorities only when they are gross or fatal. Accidental electrical injuries are common in households and industries[1]. Suicidal and homicidal electrocution deaths have also been reported in literature[2,3]. Majority of the deaths are due to lack of awareness and safety precautions while handling the appliances. Faulty devices, improper installation and wiring may be other factors contributing to electrocution.

Materials & methods

In this retrospective study done in Government Stanley Medical College, Chennai, all electrocution deaths that underwent autopsy in the Department of Forensic Medicine & Toxicology, were studied during a two year time period from January 2014 to December 2015. Socio-demographic and clinical data were studied from the inquest, clinical records and autopsy reports. Results were tabulated and analyzed.

Result and Discussion

Out of the total number of cases 9 cases (32.14%) were in the age group 21-30 years, another 32.14% were in the age group 41-50 years (Table 1). This trend of maximum cases being seen in the third decade of life has been reported by other researchers as well[1,2,4-6].

This was followed by 6 cases (21.42%) in the age group 21-40 years and 2 cases in 11-20 years and 51-60 years age group. No cases were seen in less than 10 years and more than 60 years.

During the two year period, a total of 28 cases of electrocution were observed. This constitutes 0.9% of the total 3226 cases of autopsy done during this time period.

Age group	No. of cases
11-20years	2
21-30years	9
31-40years	6
41-50years	9
51-60years	2
Total	28

Table 1 - Age wise distribution of cases

Gender	No. of cases
Male	28
Female	0
Total	28

Table 2 - Gender wise distribution of cases

In the present study all cases were male (Table 2). Male preponderance has been reported by previous researchers in their studies but total absence of female cases has not been reported.

Month	No. of cases
January	0
February	1
March	0
April	4
May	1
June	3
July	7
August	2
September	5
October	3
November	0
December	2
Total	28

Table 3 - Month wise distribution of cases

Increased number of cases were observed in the months of April, June, July and September (Table 3). This is in concurrence with other studies[1,5,4]. This could be attributed to increased demand for electricity in the summer season. Also sweating is common in summer which facilitates conduction of electricity. Furthermore, people prefer to sleep at night in rooftops of building and open terraces where there is danger of field of electric arc of high tension wires of electric poles lying close to the buildings.

Manner	No. of cases
Domestic accident	21
Industrial accident	7
Total	28

Table 4 - Manner wise distribution of cases

In the present study all cases were accidental in nature. Of the total, 21 cases (75%) were domestic accidents and the remainder were industrial accidents which occurred at workplaces (Table 4). This pattern of majority domestic accidents have been reported by other researchers as well[1,5,2,4]. Suicidal[2] and homicidal[3] electrocution cases have been reported by other researchers but no such cases were seen in this study.

Occupation	No. of cases
Skilled workers	7
Unskilled people	21
Total	28

Table 5 - Occupation wise distribution of cases

In the present study, only 25 % of cases were skilled to handle electrical devices whereas 75% of cases were unskilled people who were electrocuted while handling or fixing electrical appliances at home (Table 5).

Injuries	No. of cases
Present	17
Absent	11
Total	28

Table 6 - Presence of electrical injuries

Electrical injuries in the form of entry, exit marks or burns were seen in 60% (17 cases) of cases (Table 6). This pattern has been observed in other studies too[1,2,4]. In the 11 cases (39%) in whom no electrical injuries were seen, diagnosis was made by circumstantial evidences, treatment papers and after ruling out other causes of death. The absence of electrical injuries could be attributed to dampness of skin during electrocution which is a well documented in literature. In our study mechanical injuries were present in 9 cases (32%), apart from electrical injuries which were predominantly due to fall from height following electrocution.

Site of injuries	No. of cases
Upper limb	15
Lower limb	5
Chest and abdomen	2
Head and neck	Nil
Total	17

Table 7 - Site of electrical injuries

Of the 17 cases in which electrical injuries were seen, entry lesions were seen in all 17 cases whereas both entry and exit were seen in only 5 cases (29.4%). 88.23 % of cases had entry lesions in the upper limbs (15cases) whereas the rest (11.8%) had entry lesions in chest and abdomen (Table 7). None of the cases had electrical injuries in the head and neck region. All lesions seen in the lower limbs (5 cases) were exit marks. Previous studies also concur that maximum number of electrical lesions have been observed in the upper limbs[1,2,4].

Survival time	No. of cases
Found dead	5
Within 24 hours	7
Within 7 days	7
1-2 weeks	6
2 weeks - 1 month	3
Total	28

Table 8 - Survival time after electrocution

In the present study, survival time could not be determined in 5 cases (17%) that were found dead. 25% of cases expired within 24 hours of the incidence and 25% expired within 1 week. In a study by Gupta et al[1] 96.07% cases died on the spot and Pathak et al[4] reported that 40% of cases were brought dead to the hospital.

Conclusion and recommendations

In the present study on deaths due to electrocution, males in the productive age group (21-30 years) were found to be predominantly affected followed by 41-50 years. All cases were accidental, 75 % of which occurred at home and were observed in unskilled people. Maximum number of cases were observed in the summer season. Electrical injuries were absent in 39% of cases. Upper limbs were the predominant site involved (88.23%). 25% of the cases expired within 24 hours of the incident.

Electrocution deaths though less in number are becoming increasingly common in modern days due to urbanization and booming real estate business. It is a preventable tragedy which predominantly affects adults in the productive years of life. Knowledge of the incidence and electrical injuries and underlying causes is of primary importance, the ultimate goal being their prevention. Generally, electrical injuries that gain attention are those that are fatal or which cause disability. Accidents occurring within home are poorly documented or not reported at all. There are no national statistics available unlike road accidents.

Increasing awareness of the importance of following safety precautions in handling electrical equipments at home or workplace will greatly reduce the morbidity and mortality due to electrical injuries.

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Nap your way out of mental descent

The next time you see your senior professor taking an afternoon nap, don't be so cruel as to disturb him. Nor are you supposed smirk and squeal. You see, he is trying to revive his failing memory through nature's therapy! As we grow older, we experience gradual diminution of memory and other cognitive functions. Some may slide into dementia. One way of halting this descent is to lead a physically and mentally active life. But there is apparently another way as was discovered by Chinese investigators. In a study carried out on 2974 adults aged 65 or older, the investigators categorized the subjects into non-nappers, short nappers (less than 30 minutes), moderate nappers (30-90 minutes) and extended nappers (longer than 90 minutes) depending on the length of afternoon naps they took. All the subjects were tested for attention, episodic memory and visuospatial abilities. The moderate nappers were found to perform 4 to 6 times better than the non-nappers, short nappers and extended nappers. As this was an observational study, we are not given any insight into why afternoon nap has such a salutary effect on geriatric mind. So, in the name of work, it is unfair to deny old professor of his much needed therapy. However, his working hours may be extended so that he can put his revived mind to good use!!

(Junxin Li et al., Journal of the American Geriatrics Society, doi: 10.1111/jgs.14368, published online 20 December 2016)

- Dr. K. Ramesh Rao

Original Article

Number of Days of Controlled Ovarian Stimulation (COS) and Outcome of Assisted Reproductive Technology

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Abstract

Assisted reproductive technology (ART) has become most widely used procedure worldwide for the couples with subfertility. Also, predictability for the success rate in ART is challenging and is of paramount importance in counseling and treating the couples. Whether the prolonged period of COS would have an impact on pregnancy outcome is of concern. Hence this study has been designed to compare the number of days of stimulation and ART outcome. Aim: To assess the predictive value of number of days of stimulation in ART success rate and to use it as a counseling tool. Materials and methods: It is a retrospective study conducted in the Department of Andrology & Reproductive Medicine, Chettinad Superspecialty Hospital, Kelambakkam, Chennai. Study included 200patients from January 2009 to December 2014. Results: The proportion of women who had clinical pregnancy was 32.35%, 25.24% and 23.07% in Group A, B & C respectively. The differences in clinical pregnancy among the study groups were statistically not significant (Chi square value 1.28, p value 0.452). Number of women who had live births were 26.47%, 23.30% and 15.38% among the stimulation days 6 to 9, 10-13 and more than 14 days respectively. There is no statistical difference in Live birth rate (p-value 0.343). Conclusion: To conclude, it is clearly demonstrated that as the number of days of ovarian stimulation increases, there is a decline in the Clinical pregnancy rate and Live birth rate though it is not statistically significant.

Key Words: Ovarian stimulation, assisted reproduction, pregnancy outcome, gonadotropins, agonist.

Introduction

Assisted reproductive technology (ART) has become most widely used procedure worldwide for the couples with subfertility and it has been estimated that more than 5million babies are born out of ART worldwide[1]. Predictability of the success rate in ART is challenging and is of paramount importance for counseling and treating the couples. Ovarian stimulation is an integral part of ART programme for multifollicular growth and supernumerary embryos available for transfer and freezing. But, ovarian response to stimulation is highly unpredictable in most women and is subject to many variables like age of the couple, duration of infertility, body mass index (BMI), cause of infertility, baseline FSH level, antimullerian hormone levels (AMH), antral follicle count (AFC) etc[2]. Out of these, female age is recognized as indicator for predicting the oocyte quality[3]. However, it is not a modifiable factor in improving the success rate of ART.

The average duration of ovarian stimulation has been found to be between 8-10days. It has been noted that women with decreased ovarian reserve and advanced age group take longer time to achieve desirable follicular maturity. For predicting the success rate of ART with respect to number of days of stimulation, only few studies are available[4-6]. With variable results, whether this prolonged period of COS would have an impact on pregnancy outcome is of concern. Hence this study has been designed to compare the number of days of stimulation and ART outcome in our department.

Objective

To assess the predictive value of number of days of stimulation in ART success rate and to use it as counseling tool.

Materials and methods

It is a retrospective study conducted in the Department of Andrology & Reproductive Medicine, Chettinad Super Specialty Hospital, Kelambakkam, Chennai. Study included 200 patients from January 2009 to December 2014.

Inclusion criteria: Female age <=35yrs,Unexplained infertility, Normozoospermia, 1st cycle ART, IntraCytoplasmic Sperm Injection (ICSI) cycles, Short flare GnRH agonist protocol, Dose of Gonadotropins 225IU/300IU/375IU per day, All fresh embryo transfer cycles. Exclusion criteria: Endometriosis, Polycystic ovaries, Adenomyosis, Fibroid uterus, Hydrosalpinx , Elective freezing of all embryos, Frozen embryo transfers.

Methodology

Patients were recruited on Day 2/3 of menstrual cycle either after OCP withdrawal or natural cycle. In the

antral follicle count, which tells us about the ovarian reserve. Follicle Stimulating Hormone (FSH) test was also done. FSH is found to be the simplest, cost effective and still widely used measure for ovarian reserve test[7]. Accuracy in determining the ovarian reserve was similar with AMH and AFC as a single test and combining the two tests did not improve the prediction of ovarian response[7]. Hence, AFC has been considered as the best test for predicting the ovarian reserve[8]. Gonadotrophin dosage were decided, according to the age of the women, Body mass index, previous treatment cycle response, Number of antral follicles, Basal FSH level. All the patients included in the had short flare GnRH agonist protocol. It is an unique protocol followed in our department with the results comparable with the other standard protocols. In this protocol, GnRH agonist analogue Injection Leuprolide acetate 1mg was started subcutaneously from Day 2 of menstrual cycle and it was continued till the day of hCG trigger. It produces initial flare effect for recruitment of more follicles from the cohort and after continues administration it produces down regulation and prevents premature LH surge. Along with GnRH agonist, urinary Gonadotropins (Human Menopausal Gonadotropins) were started from Day 3 of the cycle and it was also continued till the day of hCG trigger. Patients were reviewed with first ultrasound after 5days of stimulation, according to the follicular response dosage were adjusted. Once, three or more follicles reached 18mm or more, urinary hCG trigger was given 35-36hrs prior to oocyte pick up. Timing of trigger was not influenced by weekend scheduling since our department has in house clinicians and embryologists.

Sperm preparation was done by swim up method. Intracytoplasmic Sperm Injection (ICSI) was done for all Metaphase 2 oocytes. There is a better fertilization rate obtained after ICSI as compared to IVF (68% versus 46%) and total fertilization failure following ICSI and IVF treatment was seen in 4.4% and 25% of the cycles respectively[9]. Hence, our policy is to do ICSI for all in view of referral for tertiary care. Luteal support was started with vaginal micronized progesterone 200mg thrice daily after oocyte pick up. Embryo transfer was done on Day 2,3,4 or 5 depending on the quality and the number of available embryos.

Urine pregnancy test and Serum Beta hCG were measured after 14days of embryo transfer. If the test was found to be positive, after 2weeks transvaginal ultrasound was planned to confirm clinical pregnancy. Clinical pregnancy is defined as appearance of Gestational sac detected by transvaginal ultrasound. Patients were followed up through pregnancy and delivery details either by personal or phone contact.

Measured outcomes

1. Clinical Pregnancy Rate (CPR) is calculated as number of clinical pregnancy per embryo transfer.
2. Live birth rate (LBR) is calculated as number of live births per Embryo transfer.

Statistical analysis

Number of days of ovarian stimulation was the primary explanatory variable. Occurrence of clinical pregnancy and Live birth were the primary outcome variable. The descriptive analysis of the explanatory and outcome variables was done by frequencies and percentages. Other relevant parameters like age of the female partner, BMI, Day 2 FSH level, Antral Follicle count, total dose of gonadotropin, number of Dominant follicle, Number of oocyte retrieved, total number of M2 oocytes, number of embryos transferred were compared between the study groups, using ANOVA test. Calculating the odds ratio and t's 95% Confidence Interval and p-value using binary logistic regression analysis assessed the association between number of days of stimulation and occurrence of clinical pregnancy and live birth. IBM SPSS version 21 was used for statistical analysis.

Parameter	Frequency	Percentage (%)
Group A	74	37.0
Group B	110	55.0
Group C	16	8.0

Table 1 - Number of study participants in each study group

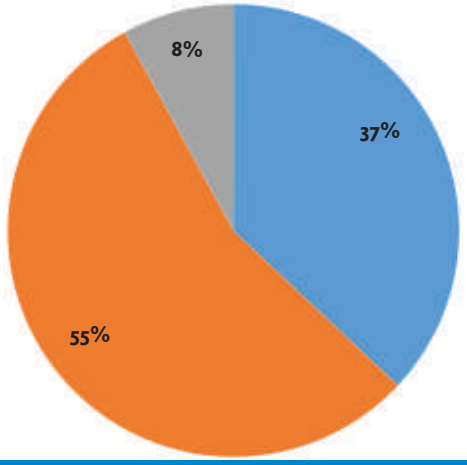

Fig 1 - Pie chart showing distribution study population among three study groups

Baseline characteristics and outcome.	Group A (N=74)	Group B (N=110)	Group C (N=16)	P value
Female age (years)	30.71	30.28	30.71	0.761
BMI (kg/m²)	25.12	25.52	26.07	0.231
Total AFC	11.18	10.24	7.07	0.006
Day 2 FSH (mIU/ml)	7.10	7.42	7.83	0.598
Total dose of gonadotropin (IU)	2445.8	3555.2	4558.9	<0.001
Dominant Follicle	8.15	8.89	6.14	0.167
No. of Oocytes retrieved	8.35	9.70	6.64	0.164
No. of Metaphase 2 oocytes	6.20	7.53	6.0	0.272
No. of embryos transferred	2.30	2.46	2.23	0.282

Table 2 - Comparison of baseline characters among the three groups.

Results

Total of 200 women were included in the study. The number of women who received stimulation for 6 to 9 days (Group A) was 74(37%), for 10-13days(Group B) was 110(55%) women and the remaining 16(8%) had received stimulation for more than 14 days (Group C). (Table 1).

The mean age and BMI of the women were about 30 years and 25 kg per m2 respectively, which was comparable among the three study groups with no statistical difference. (Table 2,2a)

Parameter	Mean ± STD	F statistic	P value
I. **Female Age**			
Group A	30.71 ± 3.05	0.273	0.761
Group B	30.28 ± 3.03		
Group C	30.71 ± 2.26		
II. **BMI**			
Group A	25.12 ± 3.70	1.476	0.231
Group B	25.52 ± 3.62		
Group C	26.07 ± 2.99		

Table 2a - ANOVA test comparing the baseline parameters among the three groups.

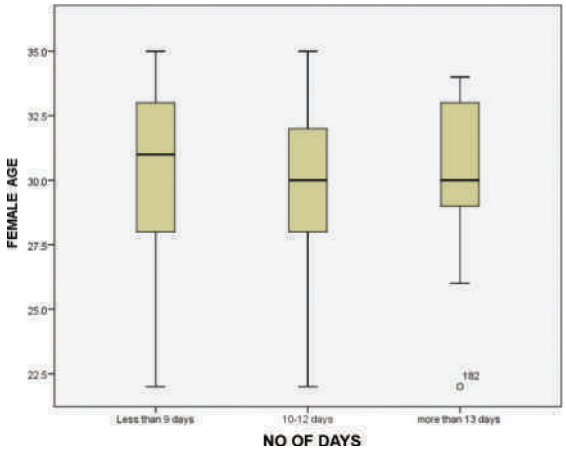

Fig 2 - BOX and WHISKER PLOT comparing the age of the women among the there study groups (N=200)

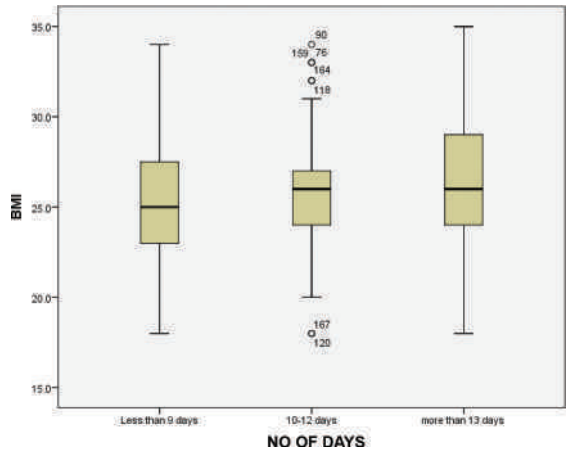

Fig 3 - BOX and WHISKER PLOT comparing the BMI of the women among the three study groups (N=200)

Statistically significant difference was observed in the mean values of total antral follicle count and total dosage of gonadotropin. The differences in the mean values of other parameters were statistically not significant. (Table 3)

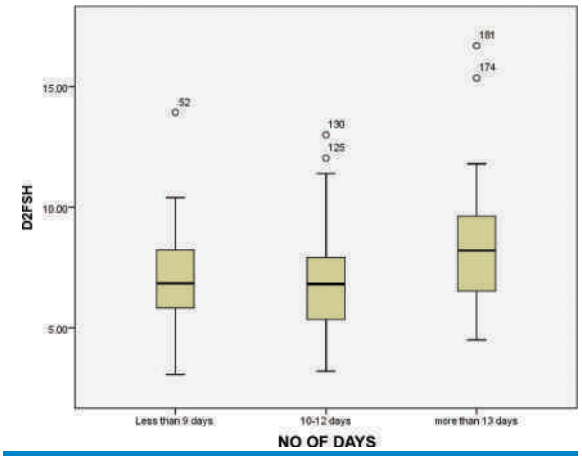

Fig 4 - BOX and WHISKER PLOT comparing D2 FSH of the women among the three study groups (N=200)

Parameter	Mean ± STD	F statistic	p-value
I. **D2 FSH**			
Group A	7.10 ± 1.82	0.515	0.598
Group B	7.42 ± 2.27		
Group C	7.83 ± 2.08		
II. **Total AFC**			
Group A	11.18 ± 4.57	5.317	0.006
Group B	10.24 ± 4.42		
Group C	7.07 ± 3.19		
III. **Total dose**			
Group A	2445.8 ± 548.6	55.81	<0.001
Group B	3555.2 ± 836.4		
Group C	4558.9 ± 1138.6		
VI. **Dominant Follicle**			
Group A	8.15 ± 4.31	1.806	0.167
Group B	8.89 ± 4.09		
Group C	6.14 ± 3.11		
VII. **Retrieved**			
Group A	8.35 ± 5.52	1.893	0.164
Group B	9.70 ± 5.71		
Group C	6.64 ± 3.60		
VIII. **M2**			
Group A	6.20 ± 4.50	1.310	0.272
Group B	7.53 ± 4.10		
Group C	6.00 ± 3.80		
IX. **Number of embryo transferred**			
Group A	2.30 ± 0.944	1.632	2.282
Group B	2.46 ± 0.661		
Group C	2.23 ± 0.842		

Table 3 - ANOVA test comparing the treatment related factors among the three groups

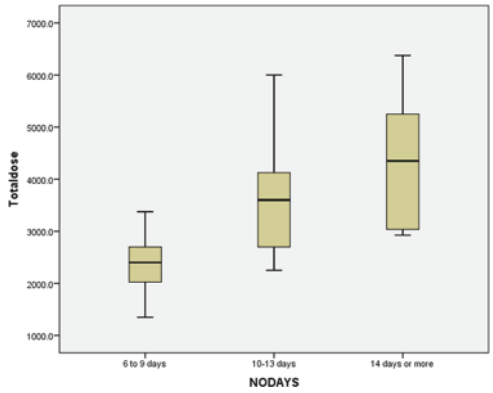

Fig 5 - BOX and WHISKER PLOT comparing total dose of FSH of the women among the three study groups (N=200)

Primary Outcomes

The proportion of women who had clinical pregnancy was 32.35%, 25.24% and 23.07% in Group A, B & C respectively. The differences in clinical pregnancy among the study groups were statistically not signifi cant (Chi square value 1.28, p value 0.452).

Parameter	Pregnancy2groups		Chi Square Value	p Value
	Yes	No		
Group A (74)	22 (32.35%)	52 (67.65%)	1.28	0.452
Group B (110)	26 (25.24%)	84 (74.75%)		
Group C (16)	3 (23.07%)	13 (76.93%)		

Table 4 - Association of number of days with clinical pregnancy (N=200)

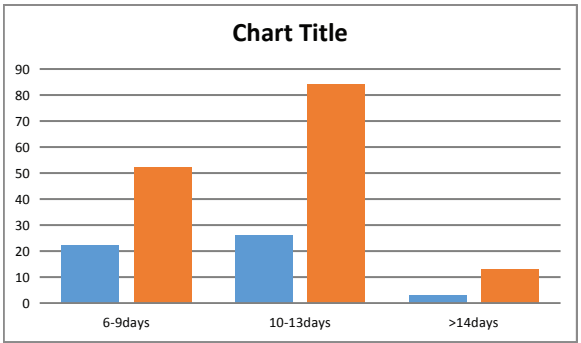

Fig 6 - Bar chart comparing clinical pregnancy among 3 study groups

Among the women who had embryo transfer, Number of women who had live births were 26.47%, 23.30% and 15.38% among the stimulation days 6 to 9, 10-13 and more than 14 days respectively. There is no statistical difference in Live birth rate (p-value 0.343)

Parameter	End Result		Chi square value (Fisher's exact test)	P-value
	Live birth	Miscarriage		
Group A	18 (26.47%)	4 (5.90%)	2.14	0.343
Group B	24 (23.30%)	2 (1.94%)		
Group C	2 (15.38%)	1 (7.62%)		

Table 5 - Comparison of end result between the study groups

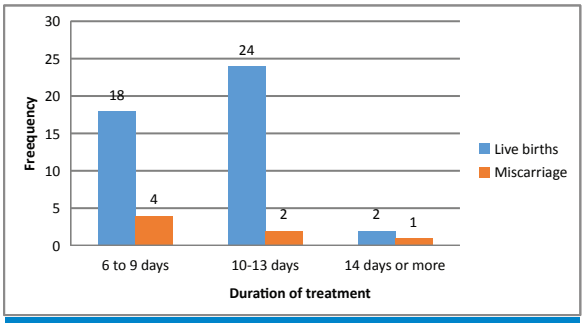

Fig 7 - Bar chart comparing clinical pregnancy among 3 study groups

Discussion

In ART cycles, duration of ovarian stimulation culminates with achieving optimum number of dominant follicle. Clinically, Ovarian stimulation is continued until two or three follicles have reached a size of 17–18 mm mean diameter[10]. Kerin et al clearly says that ideal stimulation protocols should result in development of at least three follicles beyond 17 mm so that it will yield at least two embryos for transfer[10]. As believed in practice 10-14 days cycle length is not constant in every patient and it is not an independent factor for predicting pregnancy outcome. On the contrary, the association between the daily dose of gonadotropins and outcome is well established[11].

In our study, we had 200 patients who met our inclusion criteria. In literature search, we found very few studies comparing the duration of ovarian stimulation and the outcome. The duration of ovarian stimulation ranged between 6 and 17 days. We grouped the patients as Group A, B, & C. Group A patients had stimulation between 6 and 9days, Group B patients had stimulation between 10 and 13days and Group C between 14 and 17 days of stimulation. According to Martin et al, there is no significant difference in the pregnancy rate when the duration of ovarian stimulation are less than 9days, between 10 and 11days and more than 12days[4]. Whereas Meleen et al, differ that when the number of days of stimulation exceed beyond 13days it affects the pregnancy outcome[5]. Based on the above observations, we decided to group our length of ovarian stimulation more than 14 days as Group C.

Age is considered as the best predictor of the oocyte quality and it is known to be the most important factor in determining the pregnancy potential in regularly cycling women[12]. The mean age of patients in the other two studies ranged between 34 and 36years. In our study, mean age of the patients in all groups was similar with the mean age of 30.71 +/- 3.05years. As the woman's age advances, there is an increased requirement of gonadotropins and length of stimulation, still resulting in decline in the pregnancy rate[13]. Body Mass Index is an independent factor for the number of days of stimulation. Obesity had been reported to influence both stimulation length and cycle outcome[14]. Meleen et al had mean BMI of the three groups as 26kg/m2, which was not statistically significant. In our study, we had mean BMI of 25kg/m2, which was comparable among all three groups.

There are various tests available to assess the ovarian reserve. Basal follicle stimulating hormone (FSH) levels measured on day 3 of the menstrual cycle is the most widely used ovarian reserve test to assess the ovarian response to stimulation, for over two decades now[15]. We prefer to consider day 2 serum FSH and AFC as best predictor for quantitative assessment of the ovarian reserve in our population. There was a significant difference in the total antral follicle count and the total gonadotropin dosage required among the three groups. Meleen et al and Martin et al reported similar observation in their studies. Group C women required more doses of gonadotropin in our study. From the above observation, it is clearly demonstrated that women with low antral follicle count required more doses of gonadotropin and longer days of stimu lation.

Different stimulation protocols may show wide variation in the number of days of stimulation[16]. We eliminated the confounding factor by using similar stimulation protocol for all our patients. Martin et al used long agonist protocol for all their patients and Meleen et al, included long agonist, antagonist and antagonist flare protocol.

Clinical pregnancy rate in group A, B and C were 32.35%, 25.24% and 23.07% respectively. It was comparable with the study by Meleen et al, 36%, 37.8% and 24.4% respectively among the three groups. The success of any ART programme is defined by take home baby rate[17]. The live birth rate in Group C was only 15.38% whereas Live birth rate in the other two groups were 26.47% and 23.07% were higher than group C. Though there is no statistical difference between the live birth rates among the three groups, still it is clinically higher in the Groups A and B compared to group C. Duration of ovarian stimulation may not be helpful in predicting the success rate prior to stimulation, it may still be useful in counseling the patients regarding the success rate during the cycle. Our results were comparable with the study by Meleen et al, that is 30%, 30.0% and 24.4%respectively among the three groups.

Though the dosage of gonadotropin required was more in group C who had low antral follicle count, live birth rate also remained low. Increasing the starting dose of gonadotropin stimulation in potential low responders is not an effective approach. No significant improvement in oocyte or embryo yield, or pregnancy rates were observed following such an upward dose adjustment.

Major strength of the study is that all the patients in my study population had only unexplained infertility, similar stimulation protocol and type of Gonadotropins. Limitations are small population size and it is a retrospective type of analysis.

Conclusion

From our observation, it is clearly demon strated that as the number of days of ovarian stimulation increases, there is a decrease in the Clinical pregnancy rate and Live birth rate though it is not statistically significant.

When the antral follicle count is low, the total dose of gonadotropin required is more, which was statistically significant and found to be associated with prolonged number of days of stimulation.

It is the inherent ovarian response, which decides the degree of response rather than the actual drug, dose or duration.

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Any form of physical will do

Benefits of exercise are well established. Regular exercise helps to reduce the incidence of chronic ailments and untimely death. Recommended weekly requirement is 150 minutes of moderate activity or 75 minutes of vigorous activity. While some manage to achieve the goal with a fairly evenly distributed activity throughout the week (regularly active), others may try to fulfil all their exercise needs during the weekends in one or two bursts of physical activity (weekend warriors). Yet others may be "insufficiently active". Do all these approaches lead to similar benefits? In a pooled analysis of nearly 64000 subjects aged 40 or older, researchers from Loughborough university of UK tried to find link between mortality and exercise habits over a period of 14 years (1994 to 2008). They discovered that regardless of the type or frequency, for all those who engaged in physical activity (Regularly active, weekend warriors, insufficiently active), the all-cause mortality risk was at least 30 percent lower than those who were totally inactive, although it was lowest in the regularly active. The important thing is to be physically active even if does not fulfil recommended minimum.

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- Dr. K. Ramesh Rao

Review Article

Approach to Chest Pain in Children and Adolescents

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Abstract

Chest pain in children seems to be is one of the most routinely seen presenting complaint that brings lot of anxiety and stress to patients, parents and treating physician, though most of them have a benign cause .

A simple history taking and detailed physical examination is sufficient in most of the times to identify the origin as well as the source which causes chest pain and diagnostic testing and cardiologist referral can be reserved for selected cases as routine referral still increases the family concern .

This article aims to acknowledge patients, parents fear and to give appropriate reassurance to the patients by the physician after differentiating benign from a serious cardiac condition as the possible etiology for the chest pain.

Key Words: Chest, Cardiac ,Benign

Introduction

Chest pain in children appears to be one of the most frequent cause for referral to the physicians and emergency department accounting for >6,50,000 visits every year ,especially noted in patients age group of 10-21 years.[1] It is also found to be a most common etiology after murmur, for referral to a pediatric cardiologist.[2]

Fortunately chest pain experienced by children and adolescents is found to be rarely associated with cardiac cause, with the prevalence being less than 6%.[3]

Although chest pain does not indicate serious cardiac cause in most pediatric cases, the anxiety surrounding chest pain most often is due to its association with cardiac ischemia in adults, and a society with high prevalence of atherosclerotic cardiovascular disease. and media showing sudden cardiac deaths.

There is a long list of etiologies for chest pain in children, most causes seem to be benign and also self limiting and need only symptomatic treatment and reassurance. The physicians should be aware of the differential diagnosis for chest pain in children and should make prompt efforts to determine the cause by simple history taking and physical examination before making a referral for diagnostic testing, cardiology referral and also for providing reassurance to the patients and parents .

This article helps us in understanding the various cardiac and non cardiac causes for chest pain, its approach, evaluation and need for referral to the cardiologist.

Despite the low possibility of association of cardiac disease, chest pain remains a most significant complaint in most out-patients of pediatric cardiology which needs prompt evaluation. The possible causes would be as follows:

Infectious or inflammatory causes:

Children with pericarditis, myocarditis, or endocarditis usually present with sharp retrosternal chest pain that worsens with deep breaths or when lying flat. Pericarditis, may be due to an infections like Acute rheumatic fever, coxsackie virus infection, ECHO virus, or associated with conditions such as collagen vascular disorder as in systemic lupus erythematosis, in uremia or due to neoplasm, or trauma to the chest. It can also occur as a part of the postpericardiotomy syndrome seen after heart surgery. Myocarditis again due to viral or rheumatic etiology usually occurs with pericarditis. Embolization and dissemination of infected vegetation will lead to various organ injuries and their associated symptoms.[3-5]

Coronary artery abnormalities:

Chest pain as a result of myocardial ischemia occurs in patients with abnormal coronary artery anatomy, that includes congenital anomalies of the coronary artery, coronary artery fistulas, and sometimes stenosis or atresia of the coronary artery ostium.

Coronary anomalies may precipitate myocardial ischemia or sudden cardiac death. Children presenting with chest pain along with a history of cardiac surgery or transplant are found to be at risk of myocardial ischemia or transplantation rejection leading to accelerated coronary artery vasculopathy or tachyarrhythmias. In addition, heart surgeries that potentially affect the coronary arteries (Example: corrective surgery of transposition of the great arteries) increase the risk of developing coronary artery stenosis.

Patients with a history of Kawasaki disease and associated coronary artery aneurysms are known to be at higher risk for developing coronary artery stenosis, rupture, or sometimes thrombosis. The risk of coronary aneurysm is usually seen highest during the first 5 weeks after the diagnosis of Kawasaki disease.6 Kato and associates, showed in his 10- to 21-year follow-up study, that 46% of patients with Kawasaki disease and who had history of giant coronary aneurysms were found to have stenosis or full complete obstruction and 67% had myocardial infarction, with a mortality rate of 50%. Risk factors as studied for developing coronary artery aneurysms, thrombosis, and stenosis were the extremes of age as early as < 6months , or older age (>5 years) at the time of diagnosis and males were more at risk.[7]

Coronary artery disease may also be encountered in patients who have a significant family history of hypercholesterolemia, but it is seen that children and adolescents rarely have enough obstruction so as to cause chest pain due to ischemia. Coronary artery disease presents in patients born with homozygous familial hypercholesterolemia within the first 2 decades, in contrast to those who have heterozygous familial hypercholesterolemia, in whom it presents commonly after the fifth decade of life. According to the American Academy of Pediatrics (AAP) guidelines for preventive health - care , every child needs to undergo a risk assessment for dyslipidemia usually beginning at 2 years of age, and assessment then has to be repeated every 2 years until the child is 10 years old and then annually.[8]

Structural abnormalities :

Intrinsic or structural abnormalities of the heart such as septal defects, left - to - right shunts, or cardiomyopathies [dilated /hypertrophic]may present as chest pain, but are usually associated with other symptoms like palpitations, fatigue, exercise intolerance, dizziness, or exertional syncope.[3]

Arrhythmogenic causes:

Young children who are not able to describe palpitations as a result of arrhythmias usually complain of chest pain and point to the chest. In children under 12 years, the most common etiology for Supraventricular tachycardia is usually an accessory atrioventricular pathway, whereas in teenagers, it tends to be more of atrioventricular node re-entry tachycardia. Patients with congenital QT syndrome may present in the pre-teens or teenage years with seizure or syncope along with chestpain.[3]

Syndromic causes:

Children who have Marfan syndrome, Turner syndrome, type IV Ehlers-Danlos syndrome, and homocystinuria are at a higher risk for a dissecting aortic aneurysm that presents with the sudden onset of severe chest pain.

Table 1 - Differential Diagnosis for Chest Pain in Children:[3,9-12,21-23]

NON CARDIAC CAUSES FOR CHEST PAIN [3,9-12,21-23]	
MUSCULOSKELETAL: [24-56%]	
Costochondritis	▪ Sharp stabbing , anterior chest wall pain ,involving costosternal and multiple costochondral junctions, usually unilateral, exaggerated by breathing movements, reproducible tenderness over 2 to 5 ribs is commonly seen. Self limiting ,with frequent exacerbation during adolescence.
Teitze syndrome	▪ Sharp pain localized to one costochondral junction usually affecting the second and third rib, seen in adolescents and young adults, presents as tender, swollen [1-4 cm mass] over the ribs.
Chest wall deformity	▪ Seen in Marfans syndrome ,Ehler Danlos ,chest pain associated with pectus excavatum /Carinatum.
Non specific /idiopathic chest wall pain	▪ Sharp pain lasting for few seconds to minutes, localized to mid line of sternum sometimes infra mammary area is also involved, exacerbated on deep breathing and on manual pressure on the ribs without any signs of inflammation.
Slipping rib syndrome	▪ Intense pain localized to lower chest area,due to trauma or dislocation of 8th and/or 9th and 10th ribs usually seen in athletes . It increases on sudden upward movement or on flexion of the trunk and by hooking manuouvre Pain can be reproduced along with a clicking sound.
Trauma	▪ Severe chest pain with shortness of breath and other features of contusion, hemothorax, hemopericardium.

PULMONARY/RESPIRATORY CAUSES:[7-20%]	
Bronchial asthma	▪ Chest pain is bilateral described as chest tightness, associated with wheeze and dyspnea. In exercise induced asthma,exercise itself causes chest pain even in absence of wheeze.
Infections	▪ Pneumonia, pleurisy, bronchitis
Pulmonary embolism	▪ Pleural effusion, empyema all cause acute chest pain associated with fever, dyspnea, grunting and clinically on chest examination they have pertaining signs .
Pneumothorax :	▪ Acute severe chest pain , with dyspnea ,seen at risk individuals like hypercoagu-lability, immobilization, medications. ▪ Seen in tall, thin built, asthma patients, substance abuse ,sudden sharp pain ,with dyspnea and radiating to ipsilateral shoulder ,hyper resonant note on percussion and decreased breath sounds on auscultation
GASTROINTESTINAL CAUSES:[3-6%]	
Gastro esophageal re - fluxdisease, oesophageal spasm, peptic ulcer	▪ Retro sternal burning pain, water brash, ascending pain , pain associated with eating , sometimes presents with dyspepsia, epigastric tender-ness present on examination.
Cholecystis	▪ Sharp, pain with fever, vomiting, tenderness over right hypochondrium
PSYCHOGENIC CAUSES[1-9%]	
Anxiety / hyper ventilation	▪ Pain is often fleeting, vague,with history of recent stressful events, recurrent somatic complaints, sleep disturbances
MISCELLANEOUS[4-11%]	
Herpes zoster Pleurodynia [devils-grip]: .	▪ Seen in Coxsackie B infection, sudden episodes of sharp pain in the chest and abdomen
Breast related conditions .	▪ Teenage boys present usually with gynaecomastia, post pubertal females present with mastitis, fibroadenosis
HEMATOLOGIC & ONCOLOGIC CAUSES	
Acute chest syndrome [sickle cell disease]:	▪ Severe, sharp pain with icterus associated with fever, dehydration, and dactylitis, hematuria
Mediastinal/thoracic wall tumor: .	▪ Dull aching chest pain, with features of mediastinal syndrome like headache, facial edema, venous congestion
NEUROLOGIC CAUSES	
Migraine	▪ Frequent episodes of headache with vomiting with aura.
Spinal cord compression	▪ Associated with motor or sensory deficits.
IDIOPATHIC CAUSES [12-52%]	

Table 2 - Cardiac Causes For Chest Pain [3,9-12,21-23]

Condition	Presentation
INFLAMMATORY	
Pericarditis	▪ Positional chest pain ▪ Predisposing factors: Rheumatologic conditions, Malignancy, Mediastinal radiation, Infection (HIV, tuberculosis, viral), Renal failure, Recent cardiac surgery. ▪ Cardiac rub Tachycardia / tachypnea, Distant heart sounds, JVP raised.
Myocarditis	▪ Fever Viral prodrome, Short duration of symptoms , New onset heart failure symptoms. ▪ Tachycardia, Tachypnea With or without gallop rhythm, Cardiovascular collapse
INCREASED MYOCARDIAL DEMAND OR DECREASED SUPPLY	
Arrthymia	▪ Palpitations, Syncope, Positive family history ▪ Irregular rhythm
HCM	▪ Positive family history, Exercise intolerance Exertional chest pain, Syncope and / or arrythmia ▪ Dynamic systolic murmur on auscultation.
Dilated cardiomyopathy	▪ Family history Decreased exercise tolerance, syncope , Heart failure symptoms ▪ Gallop rhythm , Mitral regurgitation murmur

Severe left ventricular outflow tract obstruction	▪ Exertional symptoms Exertional syncope ▪ Loud systolic murmur
CORONARY ARTERY ABNORMALITIES	
Anomalous coronary artery origin,coronary fistula.	▪ Exertional chest pain, Exertional syncope
Coronary ischemia	Predisposing conditions ▪ History of Kawasaki disease ▪ Cardiac surgery or heart transplant ▪ Systemic arteriopathy (Williams syndrome) ▪ Severe familial hypercholesterolemia ▪ Drug use: cocaine, sympathomimetics ▪ Anginal chest pain. ▪ Tachycardia, Tachypnea, New murmur or gallop on examination
MISCELLANEOUS	
Aortic dissection, Rupture of aortic aneurysm	▪ Personal or family history of bicuspid aortic valve or connective tissue disorders (Marfan, Loey - Dietz, Ehlers-Danlos type IV, others) ▪ Acute onset sharp or tearing type of pain. ▪ Marfanoid body habitus
MVP, Atrial myxomas	▪ Positive family history ▪ Thin built, thoracic wall deformity ,mid systolic click with or without late systolic murmur

Table 3 - Findings from Various Studies Who Evaluated Only Cardiac Cause for Chest Pain .

Study [year]	Total no. of patients	Cardiac
Danduran et al (2008)[13]	263	0%
Saleeb et al (2011)[14]	3700	1%
Friedman et al (2011)[15]	406	5%

Table 4 - Findings from Various Studies Which Looked Into Causes of Chest Pain.

Study [year]	Total no. of patients	Non Cardiac cause for chest pain	Cardiac cause for chest pain
Lin CH [2011][16]	103	98%	2%
Drossner DM[2011][17]	4436	99.4%	0.6%
Carleen L. Hanson [2011][18]	135	99.3%	0.7%
Ahmet Sert et al [2013][19]	380	99.7%	0.3%
P.Babu et al [2015][20]	1126	99%	1%

Basic Approach [3,21-23]

History :

1. Description of chest pain: Typical anginal pain is retrosternal, compressive or burning type which can radiate to left arm, shoulder or jaw; associated with perspiration and worsens on exertion; sharp localising pain is usually due to musculoskeletal problems; Respiratory conditions like pneumonia can produce localised pleuritic chest pain worsening on deep breathing or coughing; acute severe pain can be due to either pulmonary embolism or a pneumothorax. Gastroeshophageal diseases can produce a burning pain, but usually show a variation in intensity with food intake or may be associated with vomiting or abdominal pain

2. Past medical history: Asthma, Sickle cell disease, Kawasaki disease, Congenital or acquired cardiac disease, hypercholesterolemia are to be ruled out to exclude specific conditions

3. Surgical history: any previous surgeries of the chest or abdomen

4. Family history: early/sudden cardiac deaths of unknown cause, arrhythmias, cardiomyopathy, hypercholesterolemia

5. Genetic disorders: Marfan syndrome, Turner syndrome, type IV Ehlers-Danlos syndrome

6. Others : History of trauma, intense physical activity, drug abuse (eg, cocaine), psychological stressors

General Physical Examination:

▪ Check Vital signs: Temperature to be recorded, Pulse rate, rhythm, character, volume, radio femoral / radio radial delay to be noted and all peripheral pulses felt, blood pressure has to be measured in all 4 limbs and oxygen saturation to be checked.

▪ Asses the general appearance of the child like colour [any cyanosis/pallor], level of consciousness, any breathlessness and is there any evidence of anxiety/ distress in the child is the child hyperventilating, are there any dysmorphic features.

▪ Range of movement tests of the arms may be done in order to elucidate any relationship to pain as seen in muscular strain.

Systemic examination :

▪ On inspection of chest look for any signs of trauma to the chest, bruising, asymmetry of the chest and localised swelling.

▪ Palpate the chest to see for tenderness (particularly at the location where they described the pain), any crepitus, parasternal eaves or thrills felt . Hooking maneuver where we hook fingers under lower costal margin and pull anteriorly as this produces pain in slipping rib syndrome

▪ Auscultate the lung fields and note air entry bilaterally, any added sounds like wheeze, crackles and pleural rub.

▪ Auscultation of precordium usually done for abnormal loud second heart sound, systolic clicks or murmurs, gallops,and pericardial rub.

▪ On abdominal examination look for signs of tenderness (particularly in the epigastric region), any trauma and palpate to see organomegaly.

Table 5 - Investigations [3,21-23]

DIAGNOSTIC TEST	INDICATIONS
CHEST XRAY	Acute onset of severe pain , Pain that causes awakening from sleep, History of drooling, foreign body ingestion, Cough, Fever, Dyspnoea, History/signs of significant trauma, Abnormal pulmonary / cardiac auscultation.
TRIAL ANTI - REFLUX MEDICATION / pH / IMPEDANCE MONITORING	Gastrointestinal - type pain, Epigastric tenderness
ELECTOCARDIOGRAM	Cardiac-type chest pain, Cardiac red flags [text] Pericarditic pain. Any abnormal chest sensation/pain in a preschool child ,Palpitations, Abnormal cardiac auscultation or diminished pulses, Abnormal heart rate or rhythm, Family history of sudden death, inherited arrhythmias, cardiomyopathy or ICD/pacemaker insertion
ECHOCARDIOGRAPHY	Abnormal physical exam, or ECG, family history, or exertional chest pain, Anomalous coronary artery origins, cardiomyopathy, myocarditis, pericarditis, pulmonary HTN, left ventricular outflow obstruction.
TROPONIN TESTING	Suspected ischemia; myocarditis or pericarditis
AMBULATORY ECG	Chest pain and palpitations.
EXERCISE STRESS TEST	Exertional chest pain and exertional syncope or palpitations

Simplified Three Step Approach [23]

❖ Considering the vast etiology ,simple three step approach which will help to arrive at a diagnosis is considered :

❖ STEP 1: Here, clinician looks for the three most common and frequently encountered cause for chest pain in children[45-65%] : Costo chondritis, other musculoskeletal disorders and respiratory causes for chest pain after complete history and physical examination.

❖ STEP2: Here ,the clinician looks for a cardiac cause [0-4%] ,if no clue from Step 1 ,he asks for an X-ray, ECG , and cardiologist opinion following a comprehensive history taking and physical examination .

❖ STEP 3:Here the clinician looks for other psychogenic cause, other systems, idiopathic causes.

Red flags that points to cardiac cause for chest pain that needs referral to cardiologist. [3,21]

- Any abnormal cardiac findings noted on Examination.
- Chest pain on exertion/strain
- Syncope on exertion /strain
- Any Chest pain associated with palpitations
- ECG showing any abnormalities
- Family history being significant like history of arrhythmias, sudden death, or genetic disorders .
- History of congenital heart disease or acquired heart disease in the past /present.
- History of cardiac surgery or interventions
- Any Orthotopic heart transplant , Any Implantable cardioverter defibrillators in situ
- History of Kawasaki disease, Connective tissue disorders

Points to remember

Chest pain in children is most commonly due to non cardiac cause.

❖ Every child who presents with chest pain justifies a detailed evaluation with , the elaborate history and thorough physical examination which is usually sufficient to diagnose the cause of the chest pain.

❖ Family and the child are to be addressed and reassurance to be given about the benign nature of chest pain .

❖ Any symptoms that suggests myocardial ischemia or any abnormal cardiac finding on examination should pave the way for immediate referring the child to a pediatric cardiologist.

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Case Report

Tuberculoma vs Neurocysticercosis - A Diagnostic Dilemma

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Abstract

A 15-year-old female patient presented with vomiting and intractable headache for a short duration of time. Neuroimaging of brain showed a single ring - enhancing lesion which could not differentiate between Neurocysticercosis (NCC) and Tuberculoma. Magnetic Resonance Spectroscopy (MRS) helped to delineate the diagnosis as tuberculoma and the girl was treated with antituberculosis drugs when conventional investigations failed to reveal the diagnosis MRS will be helpful.

Introduction

Diseases such as tuberculoma, neurocysticercosis, Neoplastic, inflammatory and demyelinating disorders presents with ring enhancing lesions of brain. In a tropical country like India single ring enhancing lesion (SEL) of brain in children is caused mostly by tuberculomas and neurocysticercosis. Shape, size, wall thickness and edema surrounding the lesion help in differentiating tuberculoma from NCC. However above mentioned characteristics of single enhancing lesion (SEL) fails in differentiating tuberculoma from NCC because of clinical and imaging similarities. MRS helps in identifying the diagnosis and starting early treatment.

Case Report

A 15 year old adolescent girl appropriately immunized for age presented with severe headache and vomiting for 4 days. There was no weight loss, seizures or fever. There was no loss of consciousness or visual disturbances. No contact with tuberculosis. History was not suggestive of migraine and there was no similar illness in the family members.

Child was well built with Pulse rate of 76/min, BP= 106/70mmHg, RR-18/min. BCG scar was present. There was no lymphadenopathy, Pallor, or Jaundice. Cardiovascular, respiratory and abdominal systems were normal. There were no focal neurological deficit or cranial nerve palsies. Deep tendon reflexes were within normal limits and bilateral plantar reflex was flexor. Power and tone was normal and there were no signs of meningeal irritation. Ophthalmological examination was normal. Hemogram, ESR, Chest X ray was normal and Mantoux test was negative. HIV serology was negative. Cerebrospinal fluid (CSF) was found to be normal (cells 3/ mm3, protein 46mg/dL, CSF sugar/random blood sugar 64/102 mg/dL, and acid-fast bacilli (AFB) stain negative). GeneXpert of sputum and CSF was also negative. MRI brain with contrast revealed single enhancing lesions measuring 17×12×9 mm involving left hypothalamus and midbrain with peri lesional edema without midline shift (Fig 1). Possibility of Intracranial tumour was ruled out; however the MRI findings were not conclusive of tuberculoma or NCC. Magnetic resonance spectroscopy (MRS) was done and it showed lipid peak in the lesion (fig 2&3). The choline/creatinine ratio was elevated and N-acetyl aspartate (NA) was reduced. So diagnosis of tuberculoma was considered. The patient was started on anti-tuberculosis therapy along with steroids. Child responded well and symptoms resolved.

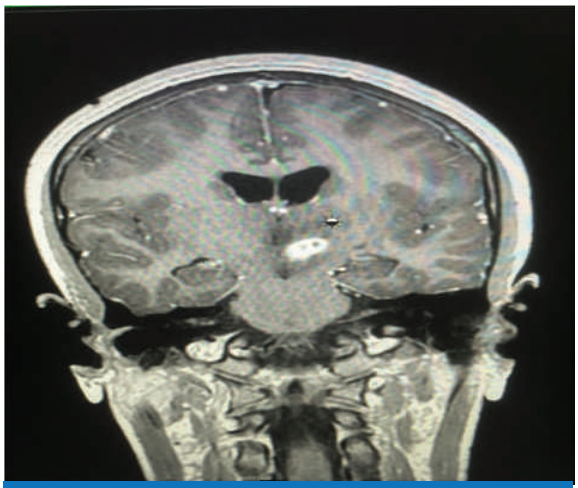

Fig 1 - MRI showing single enhancing lesion measuring 17×12×9 mm involving left hypothalamus and midbrain with peri lesional edema

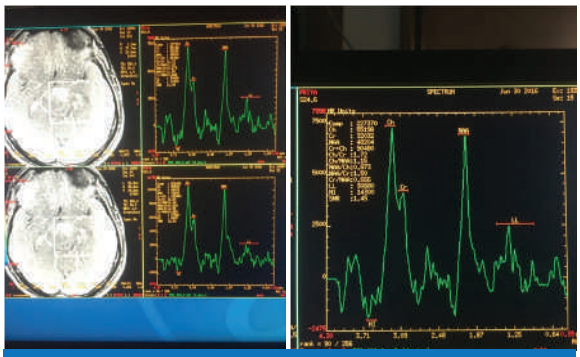

Fig 2 & 3 - MRS of the lesion showing elevated lipid and decreased N acetyl aspartate peaks with increased choline/creatine ratio favoring diagnosis of tuberculoma

Discussion

Neurotuberculosis accounts for 1% of total tuberculosis, and it carries high risk of mortality and morbidity in children. Although tubercular meningitis is the most common form of CNS tuberculosis it can also present as tuberculoma, tubercular abscess, tubercular encephalopathy and tubercular vasculopathy[1]. history, physical examination and lab investigations are important for making the diagnosis in patients with ring enhancing lesions of the brain.

Tuberculoma usually appears as hyperintense on T2-weighted and slightly hypointense on T1-weighted images[2]. With contrast they appear as nodular or ring - like enhancing lesions whereas Cysticercus granuloma appears hypointense and there will be perilesional edema on T2-weighted images in MRI and shows enhancement of ring after contrast medium administration. Usually, the lesions are regular with size < 2cm, eccentric scolex is often seen in a cysticercal lesion[3] within contrast Tuberculomas appear larger with irregular outline, cause more edema with midline shift[4,5].

MRS is superior in revealing the dilemma, in cases with single enhancing lesion of brain. MRS of tuberculoma shows elevation of lipid and choline peak elevation with decreased levels of creatinine and N acetyl aspartate, choline / creatine ratio > 1 is suggestive of tuberculoma[6]. When MRI fails to delineate the difference between NCC and tuberculoma like in our case, definitely MRS is a promising diagnostic tool to differentiate tuberculoma from other SEL[7].

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Case Report

Tuberous Sclerosis Masquerading as Febrile Seizure – A Case Report

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Abstract

Tuberous sclerosis complex (TSC) is an inherited genetic disorder that has an autosomal dominant inheritance. It can affect almost all organ systems in the body but manifestations may vary widely among individuals. Here we discuss the case of a 11 month old infant who presented with fever and seizures, initially suspected to be febrile seizures. But a detailed head to toe examination revealed multiple hypomelanotic macules and brain imaging showed subependymal nodules and cortical tubers confirming the diagnosis of tuberous sclerosis. Through this case we would like to highlight the importance of a thorough clinical examination for neurocutaneous markers, having a high index of suspicion and neuroimaging in the early diagnosis of TSC.

Key Words: Tuberous Sclerosis, Seizures

Abbreviations:
TSC- Tuberous sclerosis complex
SEGA- Subependymal giant cell astrocytomas
mTOR- mammalian target of rapamycin

Introduction

Tuberous sclerosis complex (TSC) is an inherited autosomal dominant condition with variable expression. It is a disorder of cellular differentiation, proliferation, and migration early in development, resulting in a variety of hamartomatous lesions which affects the brain, skin, kidneys, heart and other organs. The incidence of TSC is estimated to be between 1/6000 to 1/10,000 live births and a population prevalence to be around 1 in 20,000[1,2].

The hallmark of TSC is central nervous system involvement, but any organ system can be affected. The well-known cutaneous manifestation of TSC is adenoma sebaceum, which is a cutaneous hamartoma and usually does not appear until early adolescence. In some, TSC may present in infancy with cardiac rhabdomyoma or seizures, while in other affected individuals it may be diagnosed only in adolescence or adult life. There is a striking variability of clinical expression and the diagnosis of TSC is occasionally difficult, especially in those with subtle findings[3]. The age dependent appearance of the characteristic clinical features in TSC, presents challenges for the diagnosis in infancy.

Here we present a case of TSC presenting in infancy with seizures and hypomelanotic macules that signifies the importance of having a high index of suspicion and neuroimaging for diagnosis of the condition in infancy.

Case Report

A 11 month old female infant, second born to non-consanguineously married couple, with normal developmental milestones presented to the paediatric casualty with 2 episodes of left sided focal seizures and history of fever for 2 days. The seizure was controlled with a single dose of midazolam and she recovered from the post ictal state within half hour. The vital parameters and neurological examination were within normal limits. Our initial diagnostic possibilities were febrile seizure and meningitis. Work up in the form of blood counts, serum electrolytes, blood glucose, cerebrospinal fluid analysis and electroencephalogram were within normal limits.

However a detailed head to toe examination of the baby revealed 8 hypomelanotic macules over the face, chest, thigh and buttocks with the largest measuring 3cm x 1 cm (Fig 1). Thus a possibility of a neurocutaneous syndrome was kept in mind and neuroimaging done. Non-contrast Computerized Tomography brain showed multiple subependymal calcified nodules adjacent to the bilateral lateral ventricles representing subependymal hamartomas, and hypointense areas in frontal areas suggestive of cortical tubers (Fig 2). MRI brain revealed multifocal, T2 hyperintense areas in the subcortical aspect of bilateral cerebral hemispheres representing cortical tubers (Fig 3). Child was diagnosed with Tuberous Sclerosis as 3 major criteria were fulfilled (cortical tubers, subependymal nodule, more than 3 hypomelanotic macules). Further investigations were undertaken to look for

involvement of other organ systems in the form of ultrasound abdomen, echocardiography, dental, skeletal and ophthalmological evaluation which did not reveal any abnormality. The child was started on oral valproate for seizure control and discharged with advice for regular follow up.

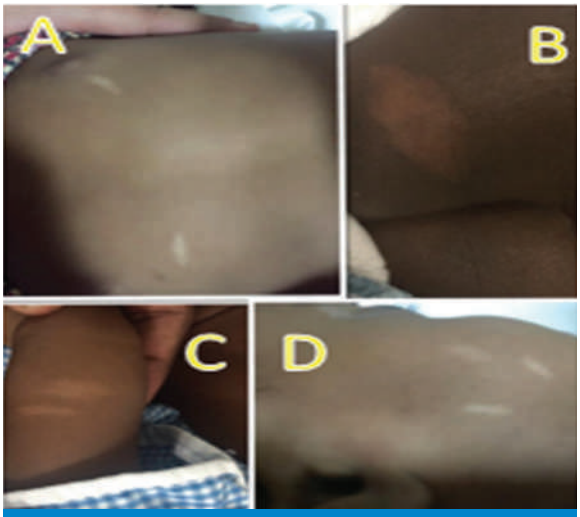

Figure 1 - Hypomelanotic macules noted over the chest(A) , Buttock (B), Arm (C) , Face (D)

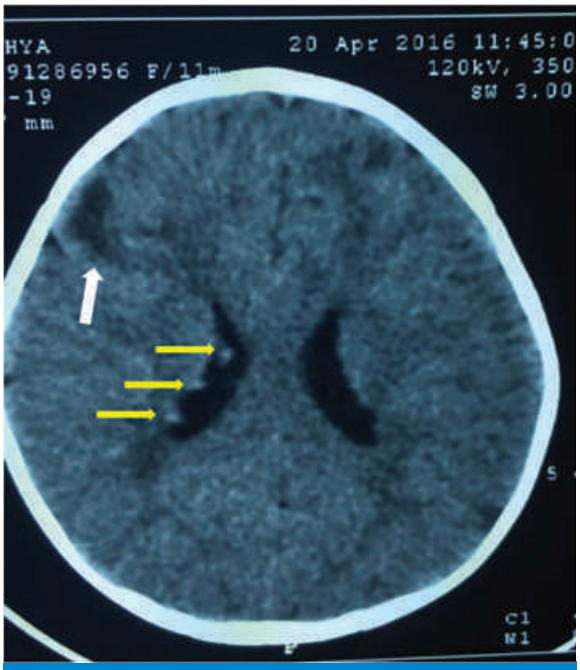

Figure 2 - CT Brain showing multiple subependymal nodules (yellow arrow) and cortical tuber (white arrow)

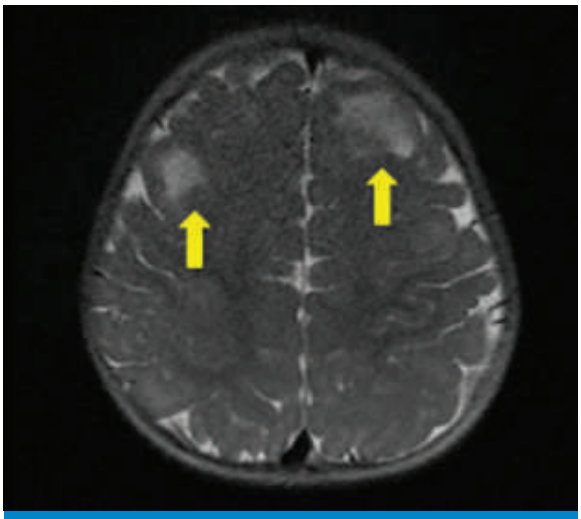

Figure 3 - MRI Brain showing T2 hyperintense areas in B/L subcortical region in frontal hemispheres.

Discussion

Tuberous sclerosis complex (TSC) is a progressive neurocutaneous disorder that involves multiple organs mainly brain, heart, kidney, lung, liver, skin and eye[4,5]. It was first described as "sclerose tubereuse" by Bourneville in 1880. In 1908, Vogt described the triad of intractable epilepsy, mental retardation, and adenoma sebaceum. TSC is now known to be a genetic disorder that is inherited in an autosomal dominant manner.

Mutation in one of the two tumour suppressor genes is responsible for tuberous sclerosis: TSC1 on chromosome 9q34, which encodes the protein hamartin and TSC2 on chromosome 16p13.3 which encodes the protein tuberin. Both of these proteins act together as a unit via mTOR signalling pathway[6].

TSC is a heterogeneous disease with wide clinical spectrum even within the same family and individuals carrying same mutation. The age dependent appearance of various clinical features poses a challenge to early diagnosis, like in the index case. Diagnosis is based on the updated 2012 criteria[7] as in table 1.

Table 1 - Updated diagnostic criteria for tuberous sclerosis complex 2012

A. Genetic diagnostic criteria
The identification of either a TSC1 or TSC2 pathogenic mutation in DNA from normal tissue is sufficient to make a definite diagnosis of tuberous sclerosis complex (TSC). Note that 10% to 25% of TSC patients have no mutation identified by conventional genetic testing, and a normal result does not exclude TSC, or have any effect on the use of clinical diagnostic criteria to diagnose TSC.

B. Clinical diagnostic criteria

Definite diagnosis: Two major features or one major feature with 2 minor features

Possible diagnosis: Either one major feature or 2 minor features

Major features

1. Hypomelanotic macules (3, at least 5- mm diameter)
2. Angiofibromas (3) or fibrous cephalic plaque
3. Ungual fibromas (2)
4. Shagreen patch
5. Multiple retinal hamartomas
6. Cortical dysplasias
7. Subependymal nodules
8. Subependymal giant cell astrocytoma
9. Cardiac rhabdomyoma
10. Lymphangioleiomyomatosis (LAM)
11. Angiomyolipomas (2)

Minor features

1. "Confetti" skin lesions
2. Dental enamel pits (>3)
3. Intraoral fibromas (2)
4. Retinal achromic patch
5. Multiple renal cysts
6. Non renal hamartomas

Central nervous system involvement is classical of TSC. Most common neurologic manifestations are epilepsy, cognitive impairment and autism[8]. Cortical tuber is the characteristic lesion best diagnosed in MRI. Sub ependymal nodules are usually asymptomatic lesions that are calcified and found projecting into the lateral ventricle, giving the 'candle dripping' appearance. But they can grow into sub ependymal giant cell astrocytoma(SEGA) that can cause hydrocephalus by obstruction. Studies show that neuroimaging resulted in a definitive diagnosis of tuberous sclerosis complex in 95% of patients in infancy[9].

Skin manifestations include, Hypomelanotic macules, that usually appear at birth and are found in about 90% of individuals. Facial angiofibromas occur in about 75% of TSC patients between ages 2 and 5 years. Shagreen patches are commonly seen as large plaques on the lower back with a bumpy or orange-peel surface and is specific for TSC. Confetti skin lesions are numerous 1 - to 3 - mm hypopigmented macules scattered over the body. During adolescence or later, subungual fibromas may form[7].

Cardiac rhabdomyoma may be the presenting sign of TSC in early infancy and is seen in 50% children. Around 50% of patients have ocular abnormalities in the form of retinal astrocytomas. Renal manifestations are seen in 75- 80% of children > 10 years of age in the form of angiomyolipoma which are usually benign. Pulmonary involvement occurs almost always in women aged 30 or older, in the form of multifocal micronodular pneumocyte hyperplasia, pulmonary cysts and lymphangio leiomyomatosis. Other manifestations include pitting of the dental enamel, bone cysts, hamartomas of stomach, intestine, and colon[7].

Medical management with mTOR inhibitor, everolimus, is effective for Subependymal Giant Cell Astrocytoma, renal angiomyolipoma, lymphangioleiomyomatosis and facial angiofibroma. Routine follow up of affected individuals with MRI brain and renal MRI every 1 - 3years, neurodevelopmental screening at key developmental stages, yearly dermatologic and ophthalmologic evaluation and bi - annual dental evaluation is recommended[10].

In conclusion, we would like to emphasise the importance of a thorough general physical examination and having a high index of suspicion for neurocutaneous syndrome among primary care physicians and paediatricians in order to diagnose these disorders early. Care must be exercised in looking for neurocutaneous markers like café-au- lait spots, hypomelanotic macules, cutaneous hamartomas, axillary freckling, subungual fibromas, facial hemangioma, nevi, pigmentary changes etc in all children presenting with epilepsy. Neuroimaging should be done in all suspected cases as it is the preferred modality for diagnosing TSC in infancy. Early diagnosis and regular follow up would significantly improve the outcome and quality of life in these children.

Conflict of interest-The authors have no conflicts of interest relevant to this article to disclose.

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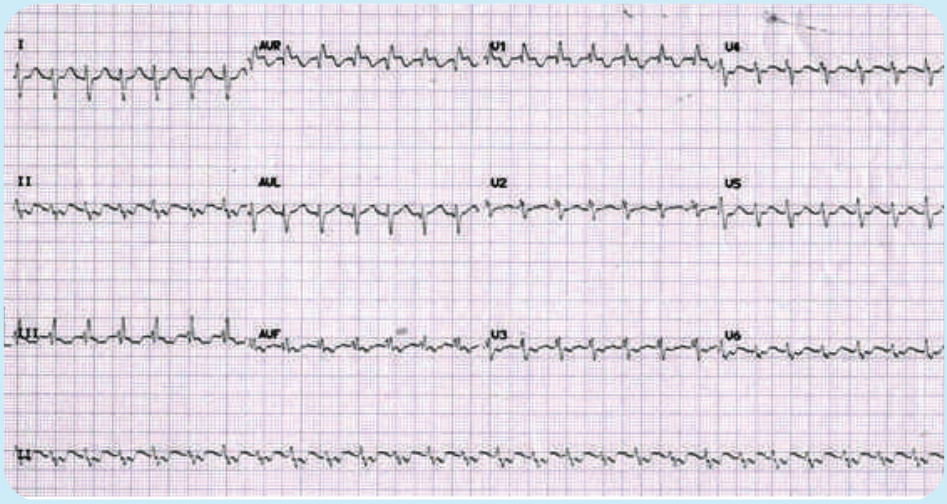
Diagnose the condition

A 26 year old female with complaints palpitations for 6 hours



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Answer in page : 102

Case Report

Anaesthetic Management of Airway Laser Surgery for Laryngomalacia

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Abstract

Laryngomalacia is a condition where there is intermittent resistance to airflow due to collapse of supraglottic structures. The Anaesthetic management during a Supraglottoplasty or Tracheostomy is quite challenging. Here, we present a case of laryngomalacia in an infant with stridor, managed with intermittent apnoea technique of ventilation for Laser Supraglottoplasty.

Key Words: Laryngomalacia, Supraglottoplasty, Anaesthesia

Introduction

Laryngomalacia accounts for 45-75% of congenital stridor. There is resistance to airflow intermittently due to the collapse of supraglottic structures. The symptoms depend upon the severity of the disease. Mild form of disease is associated with cough, regurgitation, emesis which is managed symptomatically and usually resolve by 2 years of age.[1] Severe forms of disease present earlier with stridor, respiratory distress and failure to thrive. This type of disease requires aggressive treatment in the form of Supraglottoplasty or tracheostomy, Anaesthetic management is a formidable challenge in this case in account of shared airway during surgery, risk of airway compromise and poor general condition of the patient. Paediatric airway management poses a unique set of challenges in special situations. The options need to be individualized and tailor made according to the patient profile, surgical conditions and available airway equipments. The anaesthetic plan has to be formulated with careful consideration in a resource limited setting.

Hence, a holistic approach with vigilant preoperative assessment and optimisation, a detailed airway management plan with back up options and a good rapport with the surgical team form the corner stone for successful management of airway surgeries. We present a case of laryngomalacia in an infant with stridor managed successfully with intermittent apnoea technique of ventilation for laser supraglottoplasty.

Case Report

A 4 month old female baby presented with complaints of noisy breathing and failure to thrive since birth. Baby was born at 40 weeks period of gestation by normal vaginal delivery, weighed 2.5kg at birth. There is history of admission to NICU in view of poor sucking and sepsis for 10 days. Baby was treated with IV antibiotics and discharged at day 10 of birth. She continued to have noisy breathing and poor feeding. She was brought to the casualty with complaints of increase in intensity of noise and increased respiratory rate. Baby failed to attain normal development milestones. There was absence of head control, grasp, social smile and failure to follow light. Mother gives history of occasional posturing and stiffening of limbs. Examination revealed a malnourished stunted baby weighing 4kgs with features of microcephaly, retrognathia, low set ears and low posterior hairline with widely spaced eyes. Pulse rate was 130/min regular, respiratory rate of 26/ min saturation 98% room air. Respiratory system examination showed visible retractions of suprasternal and sub costal region with audible stridor on inspiration which classically decreased in prone position. There was head lag, hypotonia in all four limbs and presence of Moro's reflex. Blood investigations like haematology, renal function tests, liver functions, and electrolytes were within normal limits. During the PICU admission baby had history of recurrent opisthotonus posturing and apnoea. MRI brain was done to rule out any central cause, however it showed no abnormality. Upper GI endoscopy showed significant reflux. Baby was managed symptomatically with oxygen therapy, Ryle's tube feeding, T. baclofen 1.25mg BD and syp lansoprazole 15mg OD. A direct laryngoscopy demonstrated an omega shaped epiglottis with overhanging aryepiglottic fold. A diagnosis of laryngomalacia was confirmed and patient was planned for a CO_2 laser excision of the epiglottis and aryepiglottic folds. Parental consent was taken for surgery and the risk of procedure and need for post op ventilation was explained.

Baby was kept fasting 4 hrs for milk and shifted to the operation theatre with an IV line secured. Baby was received in a pre warmed table and standard monitors were applied. Anticipating difficult airway small sized endotracheal tube (ET), tracheostomy tubes, resuscitation equipment were kept ready. Baby had a 24G intravenous cannula in situ, after preoxygenation with 100% oxygen Fentanyl 4mcg was given followed by induction with Thiopentone 20mg. After demonstrating adequate mask ventilation Atracurium 2 mg was given. Due to unavailability of small size laser tube, obstruction of operative field with a normal tube and risk of airway fire with continuous ventilation, an

alternative strategy for ventilation was necessary. Intermittent apnoea technique was used. Under visualisation using a direct laryngoscope, 4.0 inch PVC uncuffed tube was passed through the vocal cord. Tube placement was confirmed by auscultation and a capnography trace. Anaesthesia was maintained with sevoflurane 2% and 100% oxygen. After 3 minutes of ventilation and achieving a deep plane of anaesthesia, endotracheal tube (ET) was removed and the laser excision was initiated. When saturation dropped to 94% the surgeon was asked to stop the laser and the ET tube was reinserted and ventilation continued for 3 minutes with 100% oxygen. This cycle was repeated till the completion of surgery. Each time the tube placement was confirmed and the surgeon was informed 30 seconds before expected time for desaturation. The surgery lasted 70 minutes. A 4.0 uncuffed tube was used to secure the airway at the end of surgery and baby was electively ventilated for two days in view of possible airway edema and compromise. On postop day 2 baby was weaned from ventilator, extubated and put on CPAP at 3-5cm H_2O. There was no inspiratory stridor; saturation was maintained at 99%.

Discussion

Laryngomalacia is the most common cause of stridor in infancy. The characteristic stridor usually begins several weeks after birth and worsens till about 8 months of age. The most common coexisting condition is reflux disease however there are reported cases with neurologic disease, congenital syndromes and anomalies, and heart disease. Supraglottoplasty is amongst the common surgeries performed for severe laryngomalacia with stridor.[2] This procedure has a high success rate in healthy children. However associated conditions have the potential to worsen the surgical outcomes, necessitating their recognition and timely treatment when possible[1].

Supraglottoplasty necessitates an obstructed airway, clear view of the structures and use of laser for excision. This requires a well charted airway management plan considering a shared airway by the surgeon and anaesthesiologist and risk of airway fire and trauma. The options for ventilation include spontaneous ventilation, intermittent positive pressure ventilation, apnoeic ventilation and jet ventilation.[3] The appropriate strategy needs to be implemented based on the resources, facilities and the requirement of the surgeon. A small size laser tube with controlled ventilation would have been the ideal option in the present case. This technique has the advantage of minimum hypoxemia considering the low functional reserve in infants. However due to unavailability of appropriate size laser tube intermittent apnoea technique with controlled ventilation was used in this case. Studies have shown that with apnoeic oxygenation in children mean SpO_2 was maintained at up to 5 minutes in majority of patients and this technique can safely be used up to 10 minutes in children. However in our case we had episodes of desaturation at 3.5 to 4 minutes, hence the apnoeic period was restricted to 3 minutes for safety. This can be attributed to poor general condition of our patient with a compromised pulmonary reserve, the baby was continuously monitored with SPO_2 at all times and any fall in saturation was treated with immediate ventilation with 100% oxygen. Infant studies have shown hypoxemia as a good safety indicator during apnoeic technique.[4] In our case after first few cycles we were able to predict approximate time to desaturation and based on that we were in communication with the surgeon to insert the endotracheal tube and ventilate appropriately. This demonstrates the coordination and timely communication which is important especially in apnoeic ventilation.

Anaesthesia in this case was maintained with sevoflurane and ventilation was controlled. Reports comparing controlled and spontaneous ventilation for airway surgeries have been equivocal and the technique followed is based on the experience and practice of the consulting anaesthesiologist. Controlled ventilation has been shown to have lower incidence of laryngospam[5], however the risk of loss of airway control and unable to ventilate situation with use of muscle relaxant need to be considered and back up airway plan must be kept ready while using this technique. Sevoflurane offers more stable hemodynamics and faster recovery times when compared with TIVA with propofol and remifentanil. However sevoflurane also has an additional advantage of relaxing the jaw thereby aiding placement of rigid laryngoscope[6].

Conclusion

Anaesthetic management of laryngomalacia requires a meticulous preoperative assessment in addition to a well formulated airway management strategy. A close communication with the surgeon with careful planning of the operative steps and time is vital. In absence of ideal airway equipments, knowledge of various options available for airway control and utilising the most feasible and safe option is critical.

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Case Report

Life Threatening Vasculitis in a Child- Kawasaki Disease – Case Report & Literature Review

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Abstract

Kawasaki disease(KD) though not very common , is a clinical condition which needs a high degree of suspicion for diagnosis. The child presented to our hospital with history of fever of 12days duration and a laboratory evidence of thrombocytosis. Child was afebrile with minimal peeling of skin of both soles. Based on the retrospective history and clinical presence of unilateral cervical lymphadenopathy with a lab evidence of raised ESR , platelet count and CRP child was diagnosed to have KD and started on immunoglobulin. Echo revealed giant coronary aneurysms . Child is on follow up with aspirin, clopidogrel and warfarin.

Key Words: Kawasaki disease, coronary aneurysm, vasculitis.

Introduction

Kawasaki disease is a mucocutaneous lymph node syndrome that affects infants and children. High index of suspicion supported by laboratory investigations, leads to early diagnosis and treatment that can prevent coronary aneurysms and long term morbidity and mortality.

Case report

This is the case report of a 5 year old male child who was referred to our institute with 12 days fever and thrombocytosis. There was no other complaint . On examination the child was conscious, oriented, febrile and hemodynamically stable. Child had right cervical lymphadenopathy (2cm). systemic examination was not contributory. Examination of the extremities revealed peeling of the skin over the soles (Fg-1). Retrospective history revealed that the child had fever with erythematous rashes and a red tongue during the first week of the illness. A clinical diagnosis of kawasaki disease was suspected. Laboratory evaluation revealed total count of 6000 cells/cumm, differential count of P64 L33 E3, Platelet count: was 8 lakhs/cu.mm. CRP was 24 mg/dl, ESR was 40mm in 30 minutes and 70 mm at 1 hour. Repeat platelet counts were 10 lakhs and 12 lakhs over the next 3-4 days. Blood widal test, non enteric culture, urine routine, urine culture, serology for scrub typhus and MSAT were negative. Echo revealed giant aneurysm of Right Coronary artery, Left main coronary and Left anterior descending artery. (Fig 2) Right coronary artery was dilated with a measurement of 5.8mm. Left Main Coronary Artery measurements at the origin and at the distal ends were 3.8 mm and 11*16 mm respectively - aneurysm. Left Anterior Descending artery was hugely dilated with a measurement of 10 mm. X-ray chest and ECG were normal. Child received 2g /kg IVIG as an infusion over 12 hours. Child was also treated with T. Aspirin 100 mg/kg/day until ESR was normal, T. Clopidogrel and T. warfarin . Repeat ECHO after 8 weeks revealed no regression. Child is now on follow up with oral medications.

Discussion

Kawasaki disease also known as mucocutaneous lymph node syndrome is a vasculitis in infants and children KD predominantly involves the small and medium sized arteries. Coronaries are the commonest to be involved. The exact cause of this disease is not known but is a consequence of exaggerated immunological events following a pathogen in a genetically predetermined individual[1]. The first week is characterized by neutrophilic arteritis which forms saccular aneurysm followed by fusiform aneurysm in months. Over the years stenosis and thrombi may occur. Diagnosis of kawasaki disease is based on clinical and laboratory criteria. Fever for 5 days with 4 of the following criteria

1. Bilateral nonexudative conjunctival injection with limbal sparing;
2. Erythema of the oral and pharyngeal mucosa with strawberry tongue and red, cracked lips;
3. Edema and erythema of the hands and feet;
4. Rash of various forms and
5. Nonsuppurative cervical lymphadenopathy, usually unilateral, with node size >1.5 cm.

Alternate criteria for diagnosis includes Fever for at least five days and two or three principal features; coronary artery abnormalities on transthoracic echocardiography[2]. Incomplete (atypical) Kawasaki disease occurs in children with fever lasting five or more days and with two or three of these findings[1]. KD is a systemic illness with involvement of gastro intestinal, musculoskeletal, central nervous system , genito urinary and other systems. The acute phase presents with fever and other manifestations of the illness for the first 2 weeks. This is followed by desquamation and thrombocytosis with development of coronary aneurysms for 3 weeks. Sudden death has been reported in this phase of illness. Convalescent phase shows resolution of the signs and continue till the ESR becomes normal. The laboratory findings of KD include, neutrophilic leucocytosis, anemia,

thrombocytosis in the second week, elevated liver transaminases, sterile pyuria, pleocytosis of CSF. Common viral ,bacterial infections, rheumatologic illness, Echo features include lack of tapering, perivascular brightness, ectasia decreased ventricular function, mild valvular regurgitation, pericardial effusion and aneurismal changes . Management is aimed at prevention of coronary involvement. In untreated the occurrence of coronary aneurysm varies from 15- 25 %. In children with treatment, transient changes are reduced by 5 % and giant aneurysm by 1% .

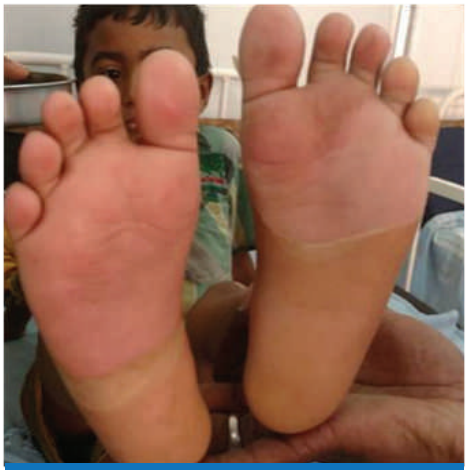

Fig 1 - peeling of skin over the soles

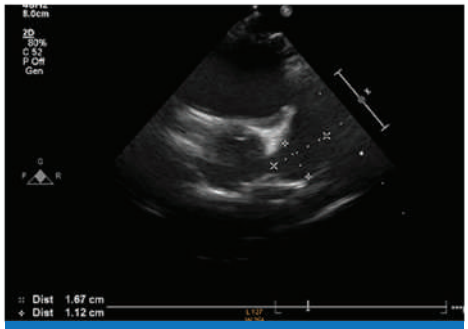

Fig 2 - Left main coronary artery dilatation

Intravenous (IVIG) at a dose of 2g/kg given over 10-12 hours is the treatment of choice to prevent the coronary complications. Recent meta analysis has revealed that addition of corticosteroids to IVIG help to prevent coronary abnormalities[3]. If the child continues to have persistent symptoms or persistent fever after 36hours of IVIG and high dose aspirin then a repeat dose of IVIG may need to be given. Therapy is most useful in the first week of illness. However the therapy with IVIG can still be tried if the child presents with fever and raised ESR and CRP. Aspirin at 80-100 mg/kg /day in 4 divided doses till afebrile for 48-72 hours and then at a dose of 3-5 mg/kg/day for 6-8 weeks. If coronary abnormalities are seen then aspirin is continued along with oral clopidogrel and warfarin. Child needs to be followed up with repeat echo at diagnosis, 2 weeks and 6-8 weeks if initial echo is normal[4]. Children on long term aspirin should receive the influenza vaccine, and varicella vaccination. The long term follow up and intervention are similar to adults with coronary artery disease. KD may recur in 1- 23% of children. The aneurysms regress in 50% by 1-2 years. However giant aneurysm are less likely to regress and may lead to stenosis and thrombosis. Coronary artery aneurysms more than 8mm size are unlikely to regress[5]. Options for second-line treatment include additional IVIG, Intra Venous methylprednisolone pulse (IVMP), prednisolone (PSL), IFX, ulinastatin (UTI), Cyclosporine A, Methotrexate and plasma exchange (PE).But for IVIG , none has been recommended with strong evidence[5] . All children with KD need to follow heart healthy diet, exercises,limit injury prone activity, monitor lipid levels and avoid narcotics.

The child described had giant aneurysm and was a delayed referral to the Institute. The child had elevated ESR and CRP and hence was given IVIG. Literature evidence shows that IVIG can be given up to 10 days of illness or later if a patient has persistent fever, aneurysms, or inflammation[1]. Since this child is at high risk for stenosis and thrombosis leading to myocardial infarction the child is on long term aspirin, clopidogrel and warfarin.

Prevention of coronary aneurysm in KD needs high degree of suspicion for an earlier diagnosis .

Acknowledgement

We thank the faculty of the Department of Cardiology, Institute of Child Health and Hospital for Children, Egmore, Chennai for their help and guidance in the management of this child.

Conflict of interest : Nil

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Case Report

An Interesting Case of Salbutamol Overdose

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Abstract

Accidental ingestion of salbutamol respiratory solution has not been reported in children. The case reported here had junctionaltachycardia,hypertension with wide pulsepressure and hypokalemia following ingestion of 1.25mg/kg of salbutamol two doses at 6 hours interval. The child improved completely with gastric decontamination and oral propranolol.

Key Words: Hypertension, Hypokalemia, Junctional tachycardia, Propranolol, Salbutamol

Introduction

Salbutamol is commonly used as rescue medication in the treatment of childhood asthma. Lower concentrations are available as oral medication in syrup (1-2mg/5ml) or tablet(2-4mg) formulations and as metered dose inhalational medication(100mcg/puff). Higher concentrations are seen in respirator solutions(5mg/ml) which are delivered using nebulizers. The respirator solutions are available commercially in respules and small volume bottles. Accidental overdose of salbutamol is reported in children[1,2]. Toxicity occurs after ingestion of more than1mg/kg/dose[3]. Common symptoms and signs of overdose are agitation, vomiting, tachycardia, widened pulse pressure, hyperglycemia, low serum carbon dioxide and hypokalemia[3]. Fever[4] and metabolic acidosis[5] are infrequently reported and hypertension has not been reportedin salbutamol overdose.

Case Report

A 9yrs old female child, an intermittent seasonal wheezer presented to the casualty early in the morning with palpitation of three hours duration. The child is a known case of intermittent asthma since 3yrs of age. She was not on any preventive therapy or any alternative medicine for her illness. She was prescribed rescue dose of oral salbutamol in a dose of 0.1mg/Kg to be used at six hours interval during previous episodes of acute exacerbations. For the present exacerbation, the mother had accidentally given the same volume of salbutamol respirator solution (5ml=25mg) two doses 6 hours apart instead of oral syrup(5ml=2mg).The total dose ingested was 2.5mg/Kg over 6 hours.

At the time of presentation, the child was conscious,dizzy, tremulous without pallor, icterus, cyanosis or clubbing. Pulse rate was 170/min without variation when observed with a cardiac monitor, regular, normal volume. Respiratory rate was 21/min abdominothoracic without intercostal or subcostal retractions. Blood pressure was 126/78mmHg in right upperlimb and 124/78mmHg in left upperlimb in supine posture without significant fall in standing position. Temperature was 98.9°F recorded orally. She weighed 20Kg and her height was 129cms. Systemic examination revealed tachycardia, normal heart sounds, without any cardiomegaly or murmurs. Respiratory system examination did not reveal any rhonchi or rales. Abdominal examination was normal without tenderness or distension. Bowel sounds were normally heard. Nervous system examination was normal except for fine tremors of both hands. There was no ataxia or nystagmus. Rhomberg's sign was negative. Basic investigations revealed normal complete blood count, random blood glucose, urea, creatinine. Serum potassium was low- 2.1mEq/L. Other electrolytes including sodium, chloride were within normal limits. ECG (Figure 1 and 2) showed paroxysmal junctional tachycardia with poorly delineated P wave and narrow QRS.

The child was admitted, clinical parameters, vital signs and ECG were continuously monitored. Blood glucose and serum potassium were monitored 2ndhourly. As the child presented within one hour of ingestion of the second dose and had not vomited previously, she was given single dose of 20g of activated charcoal followed by propranolol 0.25mg/Kg (5mg) two hours after charcoal as the junctional tachycardia persisted. The same dose was repeated once 8 hours later with which the ECG changes reverted. Blood pressure fell to 108/64mmHg at 2hours after the first dose of propranolol which was maintained for 72 hours without a precipitous fall after the second dose.Hypokalemia was asymptomatic all through and serum potassium was 3.6mEq/L 18 hours after the second dose of propranolol. Blood glucose remained within normal limits throughout the course of hospital stay.There was no wheezing after propranolol use. Child was discharged after 72 hours when her vital signs and ECG were normal.

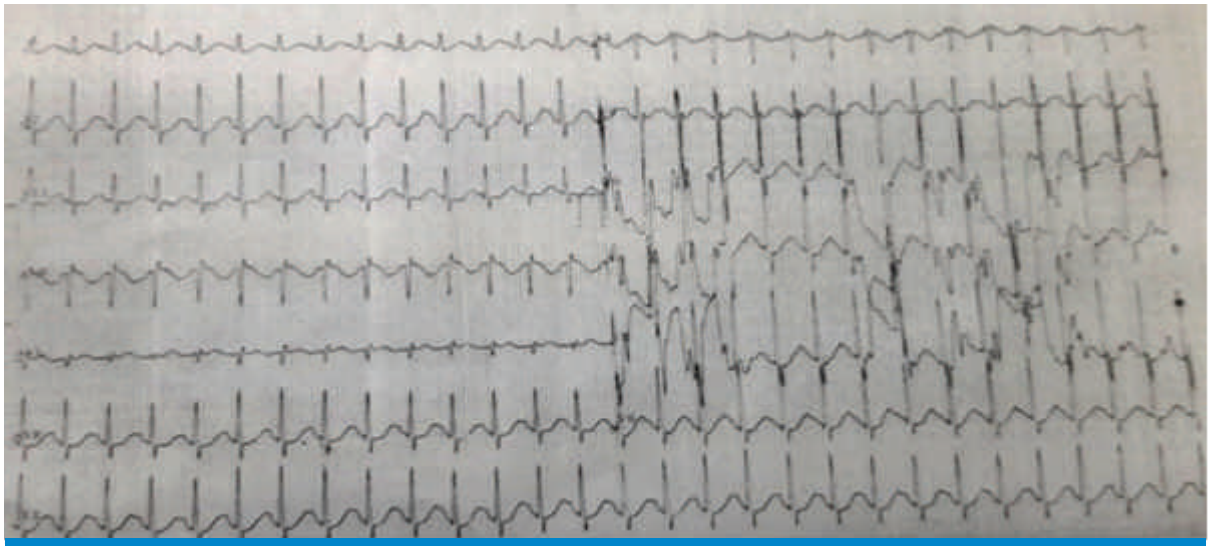

Figure 1 - Ecg Showing Tachycardia , Absent P Waves, Narrow QRS Complexes

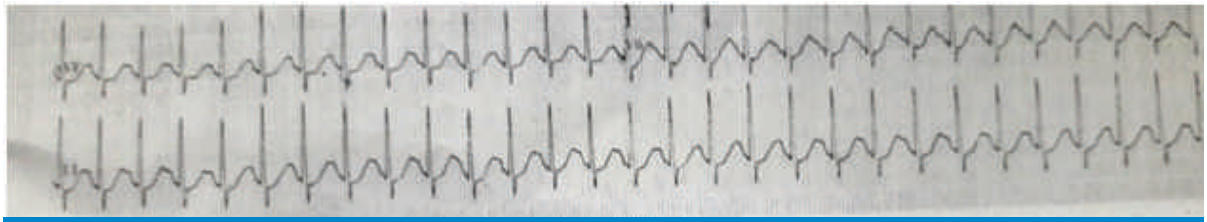

Figure 2 - Rhythm Strip Showing Junctional Tachycardia

Discussion

Salbutamol poisoning is less common in children because of low concentrations in commercially available oral preparations. Use of ß-adrenergic blockers has been suggested as helpful in patients with severe toxicity. But it is very rare that a child with salbutamol ingestion will ever need ß-blocker administration[6]. Junctional tachycardia and hypertension are hitherto unreported in salbutamol overdose in children. Heart rates less than 180 beats per minute need to be differentiated from simple sinus tachycardia as junctionalrythms require treatment and sinus tachycardia due to salbutamol overdose needs only monitoring until the drug is eliminated from the body. The lack of variation of heart rate with time and respiration and typical superimposed "p" waves in ECG suggested junctional rhythm in the present case. Hypertension reported in this case is not a direct effect of salbutamol but could be a response to maintain the peripheral vascular resistance. Propranolol is superior to atenolol in alleviating the cardiovascular and hypokalemic effects of salbutamoloverdose[7] despite the risk of bronchospasm in asthmatic children. The benefit of propranolol in reverting arrhythmia overweighed the risk of bronchospasm and hence used in this case. But there was no bronchospasm as feared.Hypokalemia though was severe did not present with symptoms. Correction was not attempted as there were no symptoms or signs of hypokalemia clinically and shift of intracellular potassium to the extracellular compartment was expected. In the presence of tachyarrythmia, supplementation of oral potassium may also be hazardous.Higher concentration solutions and similarity between various drug delivery formulations predispose to unintentional poisoning in children.

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Case Report

Terlipressin Induced Ventricular Tachycardia

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Abstract

Terlipressin, a vasopressin analogue, is commonly used to treat oesophageal variceal bleeding . Ventricular Tachycardia, a fatal arrhythmia due to Terlipressin, is a well known, but under reported complication. We report a case of broad complex Ventricular Tachycardia in a 53 year, male patient during treatment with Terlipressin for bleeding oesophageal varices, which reverted to normal sinus rhythm after withdrawal of drug and usage of anti arrhythmic drugs . Electrolyte disturbances, long QT interval as seen in alcoholic liver disease were found to be underlying causes for this.

Key Words: Ventricular tachycardia, Long QT, Hypokalemia, Terlipressin

Introduction

Terlipressin, a synthetic analog of triglycyl lysine vasopressin is effective in controlling bleeding varices[1-3]. The adverse effects of terlipressin include abdominal cramping, headache, arterial blood pressure elevation, acute myocardial infarction and arrhythmias.[2] Arrhythmias including ventricular tachycardia and bradycardia are known adverse effects of vasopressin, but are infrequently noticed with terlipressin. Hence, we are presenting this rare case of ventricular tachycardia following a therapeutic dose of terlipressin for oesophageal variceal bleeding, which reverted to normal sinus rhythm after withdrawal of drug and anti-arrhythmic drugs usage.

Case Report

A 53-year-old male, alcoholic for 30 years, presented with massive hematemesis. On admission his pulse rate was 114 beats/min and B.P was 130/90 mm of Hg. General Physical examination showed pallor but no signs of icterus, clubbing and lymphadenopathy. Abdominal examination showed enlarged liver with free fluid. His higher mental functions and cardiorespiratory status were normal. His laboratory findings include hemoglobin level of 10.5 g/dl, TLC 8900 cells/cu.mm, platelets -1,86,000, Total bilirubin 2.7mg/dl, direct 1.6, indirect 0.9, borderline liver enzymes and normal renal function tests. Serum electrolytes were Na-149meq/l, K-3.4 meq/l, Cl-118 meq/l. His ECG revealed prolonged QT interval (QTc) of 521msec. Abdominal ultrasonography demonstrates increased liver echogenicity and ascites. After stabilizing the patient, an emergency endoscopy was done which revealed bleeding oesophageal varices for which variceal band ligation was done. Post procedure patient was kept on I.V Fluids, Inj. Pantoprazole, Inj. Ondansetron and Inj. Terlipressin. Following 3rd dose of Terlipressin, after 20 minutes, patient developed asymptomatic Ventricular Tachycardia(VT), noticed on cardiac monitor (Figure 1). Cardioversion to normal rhythm was done with amiodarone bolus and infusion. Laboratory tests repeated after the episode revealed mild Hypokalemia (K-3.2 meq/l) but serum sodium, calcium and magnesium levels were normal. Electrolyte imbalance was corrected by intravenous replacement.

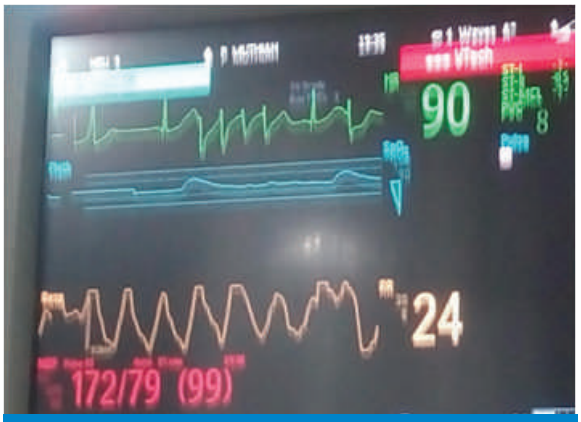

Fig 1 - Monomorphic Ventricular Tachycardia

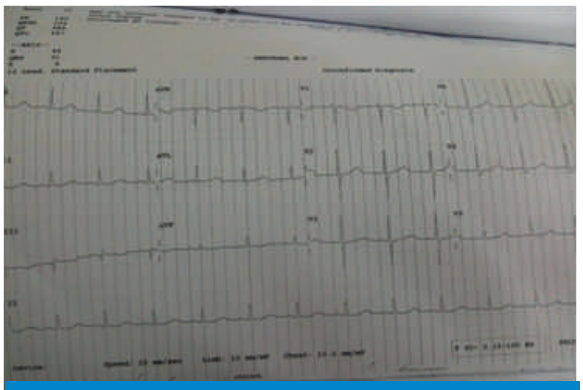

Fig 2 - Baseline ECG with Prolonged QT

ECG taken 1 hr before showed sinus tachycardia, 109 beats/ min and QTc prolonged to 547msec (Figure 2). Post cardioversion ECG revealed prolonged QTc persisting for the next 5 days (Figure 4). BP readings were 156/86 mmHg at 07.30 pm and 170/100 mmHg at 08.00pm. Corresponding pulse rates were 58 beats/min and 70 beats/min, but before the episode of arrhythmia, heart rate decreased to 52 beats/min. A myocardial infarction was ruled out with normal 2D echo and Cardiac panel study. Terlipressin was immediately discontinued and he was kept on injectable octreotide and amiodarone. Patient improved gradually and there were no further episodes of VT.

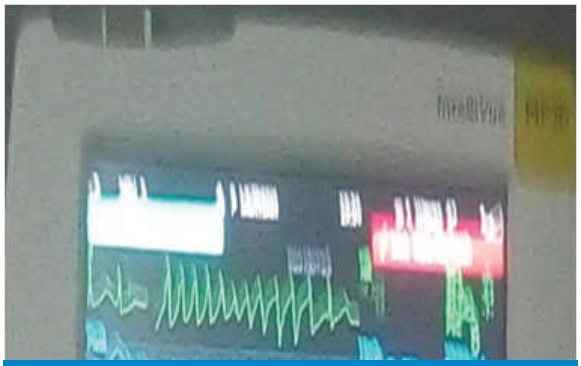

Fig 3 - VT on Cardiac Monitor

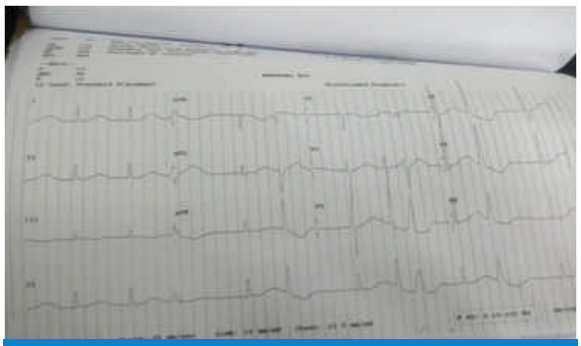

Fig 4 - Post Cardio version ECG

Discussion

A prolonged QT interval, is the most common ECG finding in patients with liver cirrhosis.[4,5] Prolongation of the QT interval predisposes the patients to polymorphic ventricular tachycardia called Torsades de pointes[6]. Delayed repolarization of cardiac muscle cells, because of potassium channel irregularities and sympathoadrenergic overactivity will lead to QT interval prolongation[7]. Major factors responsible for QT interval prolongation in cirrhosis are in Table 1[7].

Most patients who develop drug-induced arrhythmias have underlying risk factors[8,9]. Hypokalemia and hypomagnesemia, are the most important factors responsible for drug-induced long QT syndrome.

In above case he had mild prolongation of the QTc and with terlipressin, it progressed to an episode of VT. Here, the decompensated liver and the electrolyte imbalance were mainly responsible. Patient had hypokalemia, which caused VT in a patient with compromised liver function and terlipressin administration.

Conclusion

The major factor leading to pro-arrhythmogenic action of vasopressin and terlipressin is electrolyte imbalance. The present case shows the need for careful use of intravenous terlipressin and meticulous care of patients during treatment, with cardiac monitoring and correcting electrolytes, and limiting its use to proven variceal bleeding.

Factors	Examples
Decompensated liver	High Child Pugh class, High MELD score, GI Bleeding
Blood parameters	Electrolytes, Creatinine, Aldosterone , Nor epinephrine .
Fluid overload	Left ventricular end Diastolic parameters
Coronary events	Risk factor- Elder Age , DM , Smoking & Alcholism
Drugs	Terlipressin, Erythromycin.

Table 1 - Factors affecting QT prolongation in cirrhosis patients7

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Class Room

Development of Infant and Young Child : Comprehensive Evaluation

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Chettinad Health City Medical Journal 2016; 5(2): 96 - 100

Introduction

Advances in obstetric and neonatal care in the recent years has led to increased survival of babies viz preterm, low birth weight, congenital anomalies, metabolic problems, who are prone for developing long-term morbidities such as developmental delay, learning disability and visual/hearing problems. The shrinking size of families with a single child has resulted in increased concern and expectancy of their only child by their parents. The rates of infertility are also on the rise, due to working mothers,late marriages, sedentary life style, metabolic syndromes etc., go for assisted reproductive techniques or for adoption. Development of the child is again a concern.

Increased awareness among general population and greater anxiety in parents has forced us as health professionals to be very careful in labelling a child with delay in development or passing off a baby as normal.

In India, prevalence of developmental delay as extracted from various sources shows, that in children under 2 years of age it is about 1.5-2.5%[1,2].

Tools that can accurately evaluate developmental parameters must be used by caregivers during baby immunisation visits.

Certain babies who fall in the high risk group need to be followed up at regular intervals in the development clinic.

As we sow, so shall we reap! The first three years are emphasized as the foundational years in every child's life. Specific skills need to be stimulated during the respective 'windows of opportunity', to ensure positive development; for example,red squirrels if not given nuts to crack at certain age, never acquire the skill of cracking them, similarly if chimpanzees are not given bananas to peel within certain age they will never learn to peel. This is the period where the brain absorbs information at its best, like a 'super sponge' and hence, development gallops[3].

Only when there is early detection of any developmental delay, the key 'early intervention' can be utilised at its best to help the child to develop his/her potential.

Terminology[4]

Development : It is described as a dynamic process that optimally utilizes the genetic potential of the baby, within the context of the available environment, enabling its achievement of full potential.

Developmental Delay : when a child shows a significant delay in the acquisition of certain milestones or skills, in one or more domains of development (i.e., gross motor, fine motor, speech/ language, social/ adaptive), A significant delay in development has been defined as discrepancy of atleast 25 percent or more as compared to the expected rate, or if a discrepancy of 1.5 to 2 standard deviations from the norm is noted.

Global Developmental Delay : A delay in two or more of the developmental domains, (i.e., gross motor, fine motor, speech/ language, social/adaptive).

Deviance : If a child develops milestones or skills that are not following the typical acquisition sequence as in case of spastic cerebral palsy, in which the infant rolls over early as a result of increased extensor tone.

Developmental Dissociations : Rates of development differ in different developmental domains like in autistic children often exhibit normal gross motor development but have significant delay in language development.

Regression : When a child loses skills or milestones that are previously acquired it is regression as seen in serious neurological and inherited metabolic disorders.

Advanced motor development gives no indication of intellectual superiority. Sometimes one finds similar history in families. Social and cultural factors play a role. Early exposure and contact with objects allows faster acquisition.

Lateness may be due to familial, environmental factors, temperament,intellectual disability, abnormal muscle tone.

Key Developmental Milestones[5,6]

Developmental milestones are a set of functional skills or age-specific tasks that most children can perform at a certain age range.

When looking at the development of a child, four domains are considered, Gross motor, finemotor, language, social and adaptive. Each domain and a brief description of the typical developmental sequences for each are given below.

Fine motor	Age in months
Reaching for objects both hands	4
Reaching for objects with one hand, transfer objects	6
Immature pincer grasp	9
Mature pincer grasp	12
Imitates scribbling, tower of 2 cubes	15
Scribbles,tower of 3 cubes	18
Vertical stroke,tower of 6 cubes	24

Table 2 - Fine motor milestones

Gross motor	Age in months
Neck holding	3
Rolls over	5
Sits in tripod fashion	6
Sitting without support	8
Stands holding on	9
Creeps ,stands without support	12
Walks alone	15
Runs	18
Walks up and downstairs	2 yrs

Table 1 - Gross motor milestones

Language development depends on genetic, auditory,environmental factors, intellectual ability, interaction. Most common development delay is delay in language.

Development of manipulative skills not only depend on intelligence but also child's aptitudes.

Language	Age in months
Alerts to sound	1
Coos	3
Laugh loud	4
Monosyllables[ba,da,pa,]	6
Bisyllables[mama.baba,dada]	9
1-2 words	12
8-10 words	18
2-3 word sentences, pronouns,I,Me,you	24

Table 3 - Language milestones

Smile may be delayed in a blind child,autistic child, and in myotonic dystrophy.

Intellectual ability,manipulative ability and opportunity given to learn play a role in acquiring social skills.

Social and adaptive	Age in months
Social smile	2
Recognises mother	3
Stranger anxiety	6
Waves "bye,bye"	9
Comes when called,plays a simple ball game	12
Jargon	15
Copies parents in task	18
Ask for food ,drink,toilet	24

Table 4 - Social /Adaptive milestones

Hearing	Age in months
Respond by startle, blink, cry, quietening,	1
Turn head to sound	3
Turn head to one side and below the level of ears	6
Turn head to one side and above the level of ears	7
Directly looks at the source of sound diagonally	10

Table 5 - Development of hearing

Listening to music commences in utero, as the fetus is immersed in vibrations from the mother's heart beat, breathing, voice and other internal sounds. At five months, the fetus responds with movement to phonemes that it hears through the amniotic fluid, spoken by the mother and by six months, it responds to music by blinking its eyes.

Vision	Age in months
Fixate on baby's mother as she talks to the baby.	1
Fixate intently on an object shown to the baby.	3
Binocular vision	4
Follows objects of interest	6
Follows rapidly moving objects	12

Table 6 - Development of vision

Dressing :
Dresses and undresses by 3-4 years
Tie shoe laces by 4 to 5 years

Feeding :
Baby learns to chew by 6-7 months ,
Finger foods are picked by 10 months
Handle cup by 15 months
Self feeding 2-3 years

Handedness usually established by 2 years
Left handedness is usually seen in twins, epileptics, psychotics and GENIUS

Visual development depends on Intellectual ability. Blind child will be born with no facial expression

Sphincter Control

In the new born period, micturition is a reflex act, and can be conditioned from 1 month. Voluntary control begins by age 15-18 months when baby will say he/she has wet his pants, and may say no if asked whether he/she wants to pass urine. By the age of 2-2½ years he can pull his pants down and climb on lavaratory seat unaided. Most of them are dry by day at 18 mon and 90% dry by night at 5 years.

Psychological stress including over enthusiastic training during sensitive period of learning will delay control

At Risk Group

Advancement in perinatal and neonatal care has led to increased survival of infants who form a cohort who are at-risk for developing long-term morbidities such as developmental delay, learning disability and visual/hearing problems .A very effective and rigorous follow-up programme for all the neonates who are discharged from a particular health facility would be practically very difficult and not feasible also. Therefore, it is very important to categorise a cohort of neonates who are prone or are at higher risk of developing these adverse outcomes as 'at-risk' infants[7]. A well co-ordinated and appropriate follow-up program would really help in early detection of these problems thus paving way for early intervention.

Tools And Techniques To Assess Developmental Delay[3]: Developmental delay can be identified early by a simple process of using screening tools which can be administered with minimum time and equipments.

Parents can also make use of some of these tools to monitor their child's development, while some need a minimal amount of training as used by health professionals. A detailed assessment of developmental milestones should be done in accordance with the corrected age to compensate for prematurity till 24 months of age.

Screening tools: A simple developmental screening test is utilised for quick evaluation of developmental skills and is only meant for identification of children who might have a problem. If the results of a screening test is suggestive of developmental delay in the child, the child should be then referred for a detailed developmental assessment by trained personnel.

- Development observation card [DOC]
- Trivandrum development screening chart[TDSC]
- Language Evaluation scale Trivandrum [LEST]
- Denver development screening test .[DDST]

Developmental Observation Card is a self-explanatory, simple card that can be used by the parents. (Table 7).

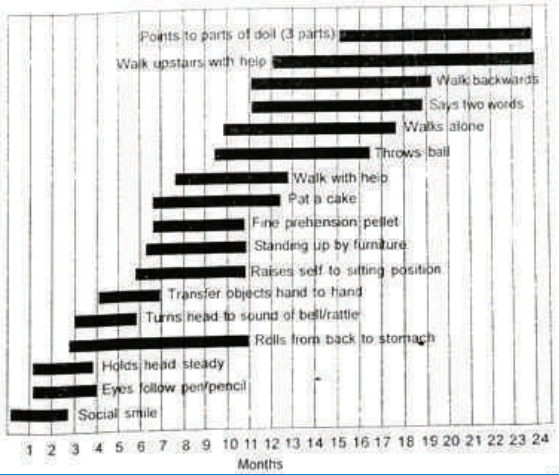
Developmental milestones	Attained age
Social smile	2 months
Holds head steady	4 months
Sits alone	8 months
Stands alone	12 months

Table 7 - Developmental observation card [8]

Trivandrum Developmemtal Screening Chart (TDSC), is a simple developmental screening test that has been designed and validated at the Child Development Centre, Trivandrum[8].

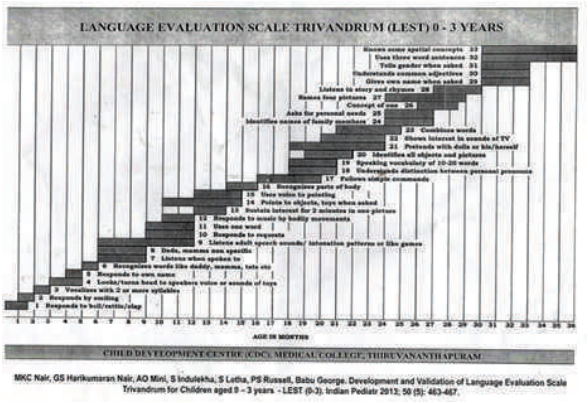
It is used for age range 0 - 2 years and it takes 5 to 7 minutes to apply the test. There are about 17 test items displayed in the chart,. The age range for each test item in the scale has been taken from the norms as given in the Bayley Scales of Infant Development (BSID). In the scale there are horizontal dark lines where the left hand side of each horizontal dark line represents age at which 3% of children have passed the item and the right end represents the age at which 97% of the children have passed the item. There are 24 vertical lines marked in the chart which represents age in months.

The test can be administered as follows where ,a vertical line is drawn or a pencil can also be kept vertically, at the level of the chronological age of the child who is being tested. If the child fails to achieve any item i.e developmental milestones that falls short on the left side of the vertical line as noted above, the child is considered to have a developmental delay.



Avertical line is drawn, or a pencil is kept vertically, at the level of the age of the child (in months) being tested. If the child fails to achieve any item that falls short on the left side of the vertical line, the child is considered to have a development delay.

Language evaluation scale Trivandrum (LEST) : LEST is a scale designed to detect language delays between 0 and 3 years and contains age appropriate developmental milestones pertaining to expressive language and receptive language. If the child fails a single item in the age group he is considered to be at risk for developing speech and language delay[9].



Denver Developmental Screening Test–II (DDST-II): This tool is used for children of age group ranging from 2 weeks to 6 years. The test comprises of 125 items, that are divided into four categories: ▪ Gross Motor ▪ Fine Motor/Adaptive ▪ Personal Social ▪ Language. These items are arranged in chronological order according to the age at which most of the children pass them. The administration of test takes 10 - 20 minutes by a trained personnel and procedure consists of asking the parent questions regarding the child's development and making the child perform various tasks using a Denver kit and observing the same[3].

Developmental Assesment Tools[3]: Developmental evaluation is an elaborate , in-depth assessment of a child's skills and is usually can be administered only by a highly trained professional, such as a developmental therapist who uses various assessment test to create a profile which gives us a view of child's strengths and weaknesses in all the developmental areas. These results as obtained after a developmental evaluation are then used to plan the treatment and also to determine if the child needs early intervention services or rehabilitation services.

Tools : Developmental Assessment Scale for Indian Infants (DASII), Bailey scales of infant development Third edition[BSID III].

Approach[4,10]

Screening Test (Doc/Tdsc/Ddst)

Detailed history

- Past medical history such as any ,biological causes like prematurity,kernicterus genetic as in , Down syndrome,environmental hazard like exposure to lead and psychosocial factors viz., maternal education, family income, marital status etc.
- Protective factors such as supportive family, opportunities given to interact with other children in a safe environment should be ascertained.
- Detailed Developmental history that helps us in evaluating gross motor, fine motor, expressive and receptive language, as well as social skills.
- Family history of any developmental delays, learning disabilities, hyperactivity, and any other behavioural and psychiatric problems has to be asked for.

Detailed examination

- Growth assessment by evaluating Height,Weight , with due attention paid to head circumference, we have to look for macrocephaly, microcephaly, or if there is any increased growth velocity.
- Detailed Dysmorphologic examination that includes minor and major anomalies that might give a clue for the etiology of the developmental delay should be noted.
- Neurologic examination that includes assessing the strength of muscle, tone by measuring the angles [Amiel Tison angles], symmetry and also the presence or absence of primitive reflexes to be noted.
- Developmental assessment to be done using either of the available scales : DASII or BSID III
- Hearing assessment to be done with: OAE,BERA
- Assessment of vision includes : ROP screening,Visual acuity, colour vision and Fundus Examination.

Investigations

- Cytogenetic studies like karyotyping and DNA testing to rule out Fragile X syndrome, and microarray-based chromosome analysis for arriving at the diagnosis.
- Neuroimaging with an MRI is indicated.
- An EEG should be obtained in cases that are associated with epilepsy.
- Metabolic screening is usually indicated for those who present with pertinent history or physical findings and also those who have not undergone universal newborn screening .

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Empathy and PTSD

Post-traumatic Stress Disorder (PTSD) is precipitated in 7 to 8 percent of people when they experience a shocking or dangerous event. Such individuals may suffer from flashbacks, negative thoughts and from a tendency to avoid places, events and objects. Sometimes, PTSD may not manifest immediately after the event but may develop much later. The problem with PTSD is that it is not limited to those who experience a painful event; recent evidence suggests that even people such as relatives, loved ones or caregivers, who on their own have not gone through the traumatic experience but merely have interacted with such individuals, may become afflicted by PTSD. Studies on mice have revealed that observing fear or distress in others or even hearing about it may bring about changes in the brain leading to increased flow of information in the deeper layers of cerebral cortex. Empathy may not simply be an ability to comprehend and share the feelings of another person but a phenomenon associated with changes in brain connectivity.

(Lei Liu et al., Neuropsychopharmacology, doi: 10.1038/npp.2016.273, published online 7 December 2016)

- Dr. K. Ramesh Rao

From the Pages of History

The Discovery of Insulin

Compiled by Dr Jaishree Vasudevan
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The Nobel Prize money is obtained from the fortune of Alfred Nobel for his invention of dynamite. It was thought that the Nobel Prize was instituted because of an earlier publication of an obituary 'The Merchant Of Death Is Dead' in a newspaper which confused his brother's death with his own. Whatever be the trigger for establishing the Nobel Prize, winning the Nobel Prize signifies achieving excellence in one's field.However, even the Nobel Prize has its share of controversies. In 1923, Frederick Grant Banting (1891-1941) and John James Richard Macleod (1876-1935)won the Nobel Prize for physiology or medicine for the discovery of insulin and this has not been free from controversy since then. Frederick G. Banting and John Macleod won the Nobel Prize in Physiology or Medicine in 1923 "for the discovery of insulin."

Introduction

Oskar Minkowski (1858-1931) in 1889, discovered incidentally that removing the pancreas in dogs caused a serious form of diabetes[1]. A medical student Paul Langerhans (1847-1888) discovered the islet cells in the pancreas, the secretion of which was thought to be involved in diabetes[2]. But as for the discovery of the active component, numerous scientists followed the work of Minkowski but were unsuccessful in their their attempt to extract it. Between 1914 and 1916 it was the Romanian physiologist Nicolas C Paulescu who first extracted a pancreatic anti diabetic agent that healed dogs but these experiments would be overlooked in favour of work by other scientists[3]. In 1922, the Lancet expounded that a simpler method of measuring blood sugar might have led to earlier discovery of insulin ; 25-50 ml of blood and 3 hours were required to test sugars then[4].

Fred Banting started his study of medicine in 1912 at the University of Toronto. He wanted to become a surgeon at the Toronto Hospital but became a demonstrator at the University of Ontario. He read an article on 'The relation of the Islets of Langerhans to diabetes' while preparing a student lecture on carbohydrate metabolism and he proposed in his notes:

'Diabetus:

Ligate pancreatic ducts of dog. Keeping dogs alive until acini degenerate leaving islets.

Try to isolate the internal secretion of these to relieve glycosurea.'[4]

As the University of Toronto had good research facilities under John James Richard Macleod ,a famous physiologist and expert in carbohydrate metabolism , Banting was referred to him . During their first meeting on 7th November, 1920, Macleod was unreceptive to Banting's idea and suspicious of his abilities as a researcher . Macleod wrote, " I found that Dr.Banting had only a superficial textbook knowledge of the work that had been done on the effects of pancreatic extracts in diabetes and that he had very little practical familiarity with the methods by which such a problem could be investigated in the laboratory. "For his part Banting wrote ,"Macleod put me off saying that many men had worked for years in well equipped laboratories and had not even proved that there an internal secretion of the pancreas[4]. "However in the end , Macleod said that even negative results would be of great value. In April 1921 ,Banting was given a small unused room in the department of physiology. Macleod had insisted on measuring the blood sugar as the experiment 's end point for which Banting required an assistant. Moreover Banting had never done a pancreatectomy and was shown how to do so by Macleod on May 16.Macleod's students Charles Best and Clark Noble were offered the chance to earn money by helping Banting. Best won the coin toss and was the first to work with Banting[4].

In June 1921 ,Macleod went to Scotland on a holiday, thereby giving rise to the controversy of how much advice Banting obtained from him. Banting pressed on with great determination and on 30thJuly, he and Best injected a pancreatic extract into a depancreatized dog and observed a sharp fall in its blood sugar .In their efforts to make the pancreatic extracts work consistently Banting and Best were joined by James Bertram Collip (1892-1965), an associate professor of biochemistry. Banting 's presentation of his and Best's results in December however met with substantial criticism[1-3].

Banting, right, and Best, left, with one of the diabetic dogs used in experiments with insulin.
Credits: University of Toronto Archives

On 11th January 1922, Banting and Best made an extract from the pancreas which was injected into a 14 year old diabetic boy, Leonard Thompson. This however had disappointing results. However Collip worked furiously to modify the extract by removing toxic contaminants from it and the experiment was resumed on January 23rd with spectacular success.

When asked by Banting for details of the effective extract , Collip would not tell him. This resulted in a fight in which Collip was grabbed by the collar by Banting . On 22nd March 1922, after peace had been brokered, the group published an initial report of its results on Thompson and other patients in the Candian Medical Association Journal. On May 2nd 1922 ,on presentation of the results of the clinical trial by Macleod to the Association of American Physicians ,he and his associates were given a standing vote of appreciation .

In 1923, Banting and Macleod were awarded the Noble Prize. Banting was angry at Macleod being nominated along with him . He was persuaded to accept the Nobel Prize by the Chairman of Insulin Committee since this was the first time a Canadian had been awarded the Nobel Prize. Banting shared his money with Best as did Macleod with Collip.

That the treatment of diabetes was revolutionised overnight was what was popularly believed after the discovery of insulin. However a lot of doctors were scared to use it and the editor of the Medical Journal of Australia called insulin an unproven therapy which was dangerous to patients. Insulin was used only as a last resort as not only were the most visible effects were seen in the previously fatal diabetic ketoacidosis and coma, but it was also very expensive. Long acting insulin was introduced in 1936-37. But in the next two decades,doctors found that while insulin saved the lives of juvenile diabetics , they were however developing complications such as loss of vision and kidney failure. It was not until 1993 that the results of the American Diabetes Control and Complications Trial (DCCT) were published which showed that good glycemic control could prevent or delay the onset of complications.

Connaught Laboratories, later called Eli Lilly and Co., took over manufacture of insulin in Toronto .Insulin from cattle and pigs was used for many years to treat diabetes .The first genetically engineered synthetic human insulin was produced in 1978 . The technique used *E.coli* bacteria to produce insulin. So now what's next? Oral insulin? Scientists are working hard on it!

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Answer to : **Diagnose the Condition**

ECG showing narrow complex tachycardia. It is regular with Atrial and ventricular rate of 160 per minute.Inverted P wave seen after the QRS complex. The RP interval is shorter than the PR interval. So a short RP tachycardia. The RP interval is aroung 70 to 80 ms, suggestive of AVNRT (AV nodal re-entrant tachycardia). The SVT reverted with I.V Adenosine.

Dr. M. Chokkalingam, Consultant Cardiologist, CSSH.

Dialogue with the Stalwart

Interview With Professor Mohammed Thambi

Uma Devi L*, Kathir Subramanian**
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Prof. Mohammed Thambi is one of the senior most practicing pediatrician in Tamil Nadu today. He graduated from Stanley Medical College (1954-60), did his DCH at Christian Medical College, Vellore (1960-62), MD PAEDIATRICS at Madras Medical College (1965-67), Diplomate in AB Paediatrics at Cook County hospital, Chicago (1975-77) and Fellow in Paediatric Neurology from Cook County (1977-79).

He was the personal physician and paediatric neurologist to Sahib Abdul Abib Kaki in Saudi Arabia.He worked as Assistant Professor and Professor of Paediatrics at Thirunelveli Medical College for 15 years. He was the most popular teacher at Thirunelveli and has been the paediatrician for three generations.

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INTERVIEW

1. Which is the best cry in humans?

The first cry is the best cry. When a baby is born crying it makes all the family members smile. If the baby does not cry immediately blood supply and oxygen to the brain is compromised. The neuron needs glucose as glucose and oxygen as oxygen; Unlike other cells neurons cannot convert other substrates into glucose. Because of this the neurons gets damaged and a lot of problems are produced - Hypoxic ischaemic encephalopathy. This makes the parents cry forever.

2. Tell us your clinical experience over the past 60 years

Advances in medicine, hightech investigations, new immunizations and various social programs have made our country free from protein energy malnutrition, small pox, polio, diphtheria, neonatal tetanus and blindness due vitamin A deficiency. However thorough clinical examination is paramount even today and unnecessary investigations should be avoided. I would like to mention a few advises for the young doctors:

a. Frequent stools in exclusively breast fed babies: Motion examination reveals reduction positive - Don't stop breast feeding. This is the most common mistake done by colleagues.

b. Child crying during micturition- the same child passes urine without cry more than five times - no need of elaborate investigations.

c. Baby fell down from the cradle- not from the first floor- CNS examination normal- only reassurance is needed

d. Breath holding spell : child cries - momentary unconsciousness - stops breathing for few seconds - During first episode, the whole village comes; the next episode the whole street comes; the successive episode, the neighbours come and the following one no one comes. It is a harmless condition.

3. Kangaroo mother care (KMC):

Mother can give three types of contacts- eye contact, verbal contact and tactile contact. These are very important to prevent psychological problems like autism. Though the father makes the child study, gives higher education, helps him get a job and makes him financially sound. But in an emergency he/she calls the mother first. That's because of KMC.

4. What is school fever?

Media mentions about mystery fever, Dengue fever and all other new fevers but not about the school fever. In my experience I've seen 3-4 children coming for fever wearing school uniforms everyday. If the child vomits, the school phones up. If the child passes loose stools, they pack him home. School authorities and educational authorities should take care and not allow sick children entering school. Sixty years ago, I was writing leave letters for gazetted officers, police officials and high officials but now I am giving the same for pre-KGs and LKGs.

5. Obesity?

Most of the children sit in front of the TV all the time or play with a mobile. They attend tuition both morning and evening and take all sorts of junk foods with no physical exercise. This system should change. It is in the hands of parents.

6. Children with stridor

Most of them are diagnosed as wheezing bronchitis and would be on multiple drugs.

There will be no cough and they sleep comfortably. This resolves by itself by around a year. Noisy breathing may be due to laryngomalacia which is self limiting.

7. Why parents change the doctors often?

When the child gets fever, parents want medicine for fever to stop at once. They change doctor next day if

the fever does not subside. The other doctor prescribes the same medicine in different names, for 3 days. In most of the countries paracetamol is available with one or two names, which applies for other medications also. For example, USA is three times larger than India, but paracetamol is available by only one name for 100 years. In our country everyday one new brand is coming up. Similarly all antibiotics also have more than 100 names. My advice to doctors, when they see a patient, ask which medications they have and convince them to continue the same medications instead of changing the brand.

8. Parental attitude towards the diagnosis of seizure in a child.

The word seizure or fits is considered as a family stigma. Most of the parents accept as fits only if the generalized tonic, clonic form is present. The other seizures like absent seizures (petit mal), tonic seizure, subtle seizure and myoclonic jerks are usually not accepted as seizures by parents. The doctor has to explain to them and reassure them. Only then the parents will accept long term treatment.

9. What is food faddism?

Most of the parents come with the history of child not taking food at all. The child seems to be active, weighing more than the average weight. But the children prefer colored and attractive looking food. But the parents are satisfied only if the child eats idly and rice. But the children prefer to takes colored rice, crispy dosai, chappathi, vermicelli flavored milk etc. The parents should be counseled that as long as the baby takes any nutritious homemade food they should be satisfied.

10. Your advice to the parents. Sir,

Breast milk is the best milk. Breast feed without anxiety- all mothers will have adequate breast milk. Don't introduce bottle early as nipple confusion is the first important cause of lactation failure.

Immunisation schedule keeps changing every year. Follow your pediatrician's advice and immunize accordingly. The efficacy by immunization is obvious (Reduction of many infectious diseases and eradication of small pox and polio).

Highway to mindlessness

Dementia currently affects nearly 47.5 million people worldwide. In addition to three known non-modifiable risk factors (age, family history and hereditary predisposition), several modifiable risk factors such as hypertension, diabetes, high cholesterol, head trauma etc., have been implicated in its causation until now. Now a new collaborative study conducted in Canada by Canada Health and several Universities has unveiled a new risk factor: living close to major highways. The study, in which 6.6 million subjects aged between 28 and 85 were followed up for more than a decade (2001 to 2012), showed that between 7 and 11 percent of subjects with dementia lived within 50 mitres of a major highway and the risk of developing dementia was directly related to the proximity of their dwellings to the highway. Similar association was not observed for Parkinsonism or multiple sclerosis. Although when high levels of nitrogen dioxide and fine particulate matter found in highways were factored in, the strength of aforesaid association diminished but did not disappear. If you intend to spend your advanced years with a functionally intact mind, stay far from madding highways!!
(Hong Chen et al., The Lancet, doi: http://dx.doi.org/10.1016/ S0140-6736(16)32399-6, published online: 4 January, 2017, abstract.)

- Dr. K. Ramesh Rao

Chettinad
Academy of Research & Education
(Deemed to be University Under Section 3 of the UGC Act 1956)

Chettinad
Hospital & Research Institute

National conference on

"Recent Advances in Cancer Research and Therapy"(RACRT-2016)

- 8th & 9th July , 2016

Organized by **Department of Pharmacology & Faculty of Allied Health Sciences**

CHETTINAD OGCON - 2016 (16.07.2016)

Organized by **Department of Obstetrics & Gynaecology**

CHETMEDICON - 2016 (20.8.2016)

Organized by **Department of General Medicine**

Chettinad Academy of Research & Education
Courses Offered 2016 - 17

I. Under Graduate Courses

M.B.B.S. (4 ½ years + 1 year internship)
B.Sc. (Allied Health Sciences) (3 years +1 year internship)
B.Sc. (Medical Bionanotechnology) (3 Years)
B.Sc. (Medical Biotechnology) (3 Years)
B.Sc. (Medical Genetics) (3 Years)

II. Post Graduate Courses

Medical Post Graduate Courses - M.D. Pre and Para Clinical (3 years)

M.D. (Anatomy)
M.D. (Physiology)
M.D. (Biochemistry)
M.D. (Pathology)
M.D. (Microbiology)
M.D. (Pharmacology)
M.D. (Community Medicine)

Medical Post Graduate Courses - M.D./M.S. Clinical Courses (3 years)

M.D. (General Medicine)
M.D. (Anesthesiology)
M.D. (Dermatology, Venerology and Leprosy)
M.D. (Respiratory Medicine)
M.S. (Orthopedics)
M.S. (Ophthalmology)
M.D. (Paediatrics)
M.D. (Psychiatry)
M.D. (Radio-diagnosis)
M.S. (Otorhinolaryngology)
M.S. (General Surgery)
M.S. (Obstetrics and Gynecology)

Post Graduate Courses (2 years)

M.Sc. (Tissue Engineering & Regenerative Medicine)
M.Sc. (Medical Genetics & Molecular Diagnostics)
M.Sc. (Medical Bionanotechnology)
M.Sc. (Medical Biotechnology)
M.Sc. (Clinical Research & Experimental Medicine)
M.Sc. (Marine Pharmacology)
M.Sc. (Human Nutrition)
M.Sc. (Occupational Health & Industrial Safety)
M.Sc. (Health & Yoga)
M.Sc. (Computational Biology)
M.Sc. (Bioinformatics)
M.Sc. (Counseling Psychology)
M.Sc. (Pharamaceutical Chemistry)

Post Graduate - Super Speciality Courses D.M. / M.Ch Courses (3 years)

D.M. (Cardiology)
D.M. (Neurology)
D.M. (Neonatology)
D.M. (Gastroenterology)
M.Ch. (Cardio Vascular & Thoracic Surgery)
M.Ch. (Neuro Surgery)
M.Ch. (Urology)

M.Sc. - (Allied Health Sciences) (2 years)

1. (Echocardiography & Cardiovascular Imaging Technology)
2. (Radiology and Imaging Science Technology)

Post Graduate Courses in Nursing (2 years)

M.Sc. Medical Surgical Nursing - (Cardio Vascular and Thoracic Nursing)

III. POSTGRADUATE DIPLOMA COURSES

Postgraduate Diploma in Clinical Embryology (1 year)

IV. NURSING COURSES

B.Sc. (Nursing) (4 years)
Post Basic B.Sc Nursing (2 years)

V. Ph.D Program

(1) Ph.D CARE Fellowship (JRF) Full time & Part time Ph.D. Under the Faculty of Medicine, Nursing & Allied Health Sciences.
(2) Post Doctoral Fellowship