April - June 2019 Vol 1 Number 2

www.ispghan.org

PGLJ

Pediatric Gut and Liver e Journal

Official E-Journal of
Indian Society of Pediatric Gastroenterology Hepatology and Nutrition

ispghan
INDIAN SOCIETY OF PEDIATRIC GASTROENTEROLOGY, HEPATOLOGY AND NUTRITION

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Message from Editorial Board

Welcome to the second issue of PGLJ! We hope that the first issue of PGLJ did stand up to your expectations. We will continue to strive and work hard so that PGLJ becomes a resource that you can depend on to keep up with the rapidly evolving field of Pediatric Gastroenterology.

The second issue of PGLJ brings you a steady supply of high-quality, peer reviewed papers that are relevant and readable.

This second issue is blessed to have a **Reflection** from **Prof. SK Yachha** entitled "**Way forward for Trainees in Pediatric Gastroenterology and Hepatology in India**". A must read for all trainers and trainees in the field of pediatric Gastroenterology.

We have planned the following sections:

- **Clinico Pathological Conference** where in authors have in detail discussed about an infant with colitis.
- We have four **Case Reports** where in authors have provided us unique cases and rare presentations of common disease that will enhance our knowledge.
- **Social Edge**: This section will highlight the social issues in pediatric gastroenterology. Through this we wish to ignite the light in our readers towards the social aspects of pediatric GI disorders so that we can plan a holistic management. In this issue we discuss on corrosive and button battery ingestion and how aspect of prevention is better than cure can work beautifully here.
- Section of **Journal Watch** has two parts. First section dealing with international publications related to different aspects of Pediatric Gastroenterology. The second section incorporates article published by ISPGHAN members in between February 2019 - May 2019. We would request all members to send us information about their publication so that we can continue to incorporate them in future issues.
- **ISPGHAN Kaleidoscope**: Section deals with activities done under the ISPGHAN banner both at state and national level. Our society is growing and actively doing many activities throughout the country which needs to be shared with the rest of the pediatric community.

We would like to take this opportunity to thank all the reviewers for the effort and expertise that they have contributed to reviewing, without which it would be impossible to maintain the high standards of peer-reviewed journals.

We request our readers to pledge their support to make the upcoming ORS Day & Week (25th July -31July 2019), World Hepatitis Day (28th July) and Breast Feeding Week (1st August – 7th August 2019) a grand success.

We request you the reader to become an author and share your thoughts and research with the national and international community through this journal. We also request you to pass on the message to Trainees (DMs, FNBs, PDCCs, and Fellowships) for it will be a great place for them to start their academic venture.

Kindly submit your contribution in MS word only at: *pglj.ispghan@gmail.com*

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Way Forward for Trainees in Pediatric Gastroenterology and Hepatology In India

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Fortunate are those pediatricians who get in to various super specialty training programs. In this country we are lagging behind adults in development of such super specialists. Questions that cross the minds of doctors before joining such courses are those of short and long term benefits. There is no doubt that today' India demands best and specific treatment of the child. In a way specialized care is required which is possible if the doctor is trained in a particular specialty, possess confidence and expertise. Limited option of seat availability in pediatric specialty in India is a reality at this stage. Therefore those doctors who get selected should utilize their training period to best of their learning.

Medicine is an art where in applied clinical skills have to be strong that entails problem oriented approach and clear plan of investigations and management. One needs to develop acumen of analysis and evaluation of a given clinical problem coupled with background knowledge of the subject. Management planning is the key and should be focused on immediate requirement and finding out etiology. Always prioritize the diagnostic possibilities based on clinical evaluation, and common conditions and also the spectrum of disorders prevalent in that geographical location. Investigations that can give clue to diagnosis have to be specific/ minimal and not drawn in a vertical fashion the way they are given in book tables particularly from foreign countries. Management line adopted should depend upon availability and cost-effectiveness.

Wisdom of a doctor is of equal importance. Parents of a given patient usually approach you with a bundle of investigations / prescriptions and want you to see them in the first go of interaction with doctors. The best approach for a good doctor would be to tell parents that I will see all your papers at the end." Let me understand the illness of your child- similar to writing on a fresh clean board. Any illness how so small it may look to you as a doctor is big for parents. Therefore one needs to give importance to all those pointers while processing the relevant information of importance in your own mind for evaluation. While conversing with parents or child you can ascertain their bent of mind. Their inclination should be taken in to consideration for implementation unless you find this not in the interest of the patient. In that situation the pathway of conversation can be tactfully and wisely distracted. A good human behavior cum interaction is vital for success. You need to understand patient's socioeconomic status, family structure, affordability and willingness of treatment. A great deal of confidence and firmness is required to have an impact on the patient's satisfaction in you. Too rigid, too hard talking and too lenient approach are not useful. A soft, smiling and helpful attitude; patient friendly and convenience in obtaining things done without a long wait are factors of great patient / parent satisfaction.

As a trainee doctor you face a great deal of stress: be it limitation of scientific knowledge, fear of seniors/ teachers, academics, patient-family retaliation, suboptimal nursing care, non-cooperation of other paramedical staff or colleagues and more importantly your own difficulties such as separation from family or domestic pressure. This phase of life is real but manageable with wisdom and some adjustments keeping your ego at the lowest level.

Trainees should possess appetite for skillful leaning from seniors including teachers. Trainee at times feels that the senior is no good, does not possess adequate knowledge or is boring. The trainee then carries an impression within that he/she knows better than teachers. This kind of notion is not correct. Every person in this world is talented in one or the other domain. Recognize and identify good qualities in that person and try imbibing them for your good. Your attitude should be such that you change yourself for absorbing meaningful knowledge from your seniors.

A good trainee is one who is punctual, targeted towards work, honest, a good team worker, adjustable to surroundings, devoted and has the passion of delivering the best to the best of satisfaction of seniors and patients. Having exercised such attitude will give you inner happiness that is priceless. Trainees should partly engage themselves in to some kind of healthy entertainment/relaxation, prayers and take reasonable care of their health, finances as well as family and loved ones. All these aspects are important for holistic living in this universe.

Teachers have also to realize that trainees are an integral part of health care delivery and they are expected to train them. Trainees are our investment of future. Our generations will remain secure if they are nurtured by seniors respectfully, with great deal of dignity and are empowered with genuine knowledge and ethical practices.

The writer of this message has also been a trainee himself and also a trainer for a long time. Whatever has been expressed in this article here is my own experience. I enjoyed being a trainee as well as a trainer. Having served public sector for several decades I have now moved to private sector for development of pediatric super specialty and new training programs. March towards positive developments is my passion.

An infant with colitis

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Clinical Protocol

Six-month old male child, presented to the OPD with a history of bleeding per rectum. Symptoms had started at around 3.5 months of age. The child used to pass loose or semi-formed stool mixed with blood almost every time he defecated (4-8 times/day) Occasionally there was some mucus too. There was also a history of intermittent non – bilious vomiting. There was no bleeding from any other site, no fever or constipation. No history of recurrent infections, wheezing or atopy.

This child was first by order of birth of a non-consanguineous marriage. He was born pre-term (35 weeks) with a birth weight of 1.75 Kg. There was no family history of allergies or inflammatory bowel disease. The child was developmentally normal.

He was on mixed (breast milk + formula feed) since the early neonatal period. Complementary feeding had just been introduced.

He had been treated with oral/ IV antibiotics/ probiotics before he presented to us but there was no response. He had also been given a trial of 7 days of a milk/ milk product free diet (was fed a soy formula) but there was no response.

On examination, the child weighed 4 kg. (- 4.00 z), and height 60 cm. (-3.42 z). The child appeared lethargic. Pallor was present. There were no clubbing or lymphadenopathy. Skin was apparently normal.

Abdominal examination revealed no distension or hepatosplenomegaly. Perianal examination was normal, with no fissure or tag seen.

The investigations with their results are enumerated in Table 1.

Table 1. Investigations

Investigation	Result
Hemoglobin(> 11.5 g/dL)	9.7 g/dL
Total Leukocyte Count (4000 – 11000/ mm^3)	6400/mm^3
Differential Leukocyte Count (N/L/E/M)*	28/52/11/6
Platelet count(150000 – 400000/ mm^3)	440000/ mm^3
Peripheral blood smear	Microcytic hypochromic anaemia
Erythrocyte Sedimentation Rate (< 20 mm/hr)	25 mm/hr
Albumin(3.5 – 5.5 g/dL)	3.5 g/dL
AST/ALT(< 40 U/L)	24/29 U/L
C – Reactive Protein (< 6 mg/dL)	19.6mg/dL

*N – Neutrophil, L – Lymphocyte, E – Eosinophil, M - Monocytes

Discussion

The infant's history *i.e.* presence of loose stools with blood and mucus is suggestive of colitis. Colitis in children can have multiple causes such as infection, inflammatory bowel disease, allergic, ischemic/ vascular or colitis secondary to immune deficiency disorders. The approach to a child with colitis would be to first narrow down the differentials based on the age of the child. The causes of colitis in children stratified according to the age of the child are tabulated in Table 2.

Table 2. Common causes of colitis categorized by different age groups

Age	Cause of Colitis
Neonates and infants	Necrotizing enterocolitis* Food protein induced allergic proctocolitis Hirschsprung associated enterocolitis Primary immunodeficiency disorders Infectious colitis Infantile onset inflammatory bowel disease
2-6 yrs	Infectious colitis Henoch-Schönlein purpura Acquired immunodeficiency disorders Early onset inflammatory bowel disease Colitis in GSD 1b
>6yrs	Infectious colitis including Tubercular & *C. difficile* colitis Pediatric onset inflammatory bowel disease Vascular-ischemic colitis (associated with rheumatologic disorders) Collagenous colitis Eosinophilic colitis

*To be considered only in preterm infants (rarely term infants with predisposing factors like sepsis, cardiac disease etc.) in the first few weeks of life.

The index case is an infant and clinical features of the causes of colitis in an infant are listed in Table 3.

Food protein induced allergic proctocolitis is the most common cause of rectal bleeding in infants occurring in around 2-5 % formula fed infants. (1,2) Our patient was exposed to cow milk protein since early infancy. However, there are a few unusual points - the presence of severe failure to thrive, raised inflammatory markers and lack of response to a week-long trial of a milk – free diet being unusual.

An infectious cause per se is unlikely given the protracted course and lack of response to antibiotics. However, it is possible in the setting of an underlying immunodeficiency disorder. Even though there is no history of recurrent infections in this child it is a possibility as a child with an immunodeficiency may initially present with just an isolated GI infection. (3) Amongst the primary immunodeficiencies, in patients with common variable immunodeficiency (CVID) chronic colitic changes have been reported in 20 – 38% patients. (4) Colitis is also

Table 3. Common causes and clinical features of colitis in an infant

Cause of colitis	Clinical Features	Diagnosis suggested by
Hirschsprung associated enterocolitis (HAEC)	Presenting either before or after definitive surgery for HD, abdominal distension and explosive diarrhea, along with emesis, fever, lethargy, and even shock	Intestinal 'cut-off sign' on abdominal X-ray - 74% sensitivity & 86% specificity
Primary immunodeficiency disorders	A history of bacterial or fungal infections with unusual organisms, or unusually severe and recurrent infections with common organisms	Lymphocytopenia neutropenia Abnormal low serum immunoglobulin levels Low B-cell and T-cell lymphocyte subsets Defective oxidation burst in neutrophils by NBT or DHR
Infectious colitis	Acute onset of watery or bloody diarrhea, abdominal pain and fever	Stool examination and culture
Food protein induced allergic proctocolitis	Bloody stools in a well-appearing infant	Allergen elimination and challenge procedure
Infantile (and toddler) onset inflammatory bowel disease	YOUNG AGE onset Multiple family members, consanguinity Autoimmunity Thriving failure Treatment with conventional medfails Endocrine concerns Recurrent infections or unexplained fever Severe perianal disease Macrophage activation syndrome Obstruction and atresia of intestine Skin lesions, dental, hair abnormalities Tumors (Acronym - YOUNG AGE MATTERS MOST)	Diagnostic ileocolonoscopy, histopathology.

*NBT – Nitroblue tetrazolium dye reduction *test*, DHR – Dihydrorhodamine

frequent in patients with chronic granulomatous disease (CGD), affecting 11% to 17% of patients. Other rarer primary immunodeficiency disorders (Severe combined immunodeficiency, Wiskott–Aldrich syndromes *etc.*) may also present with colitis. (5)

Infantile onset inflammatory bowel disease (IBD) comprises around 1% of all IBD patients and it presents and behaves quite differently from the disease that develops in older children or adults. The course of the disease is generally more severe. In up to approximately 25% of these patients, not only do they have IBD, but they also have an underlying immunodeficiency, autoinflammation disorder or epithelial barrier defect (monogenic disease). Our child does have some (young age of onset, failure to thrive) features listed in Table 3. Considering the symptoms and elevated inflammatory parameters (elevated CRP, thrombocytosis) it should be kept as a possibility.

HAEC would not be considered as there is no clinical setting for the same.

To sum up, the clinical possibilities were – allergic colitis, primary immunodeficiency disorders and inflammatory bowel disease.

Course in the hospital

The child was evaluated keeping in mind the possibilities that were narrowed down on the basis of the history. The child's HIV serology was negative, T & B lymphocyte subsets and immunoglubulin profile was normal [IgG – 864 mg/dL (Normal-309-1573), IgA-84 mg/dL(Normal-22-98),IgM-59 mg/dL(Normal -3.7-89)] and NBT Normal.

A proctosigmoidoscopy was performed which showed an edematous mucosa with nodularity and diffuse superficial ulceration (Figure 1a). Occasional apthous ulcers were also seen (Figure 1b). Biopsies were obtained and sent for histopathological evaluation.

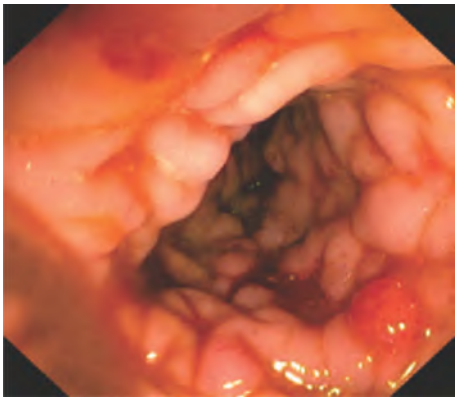

Figure 1a. Endoscopic image showing edematous mucosa with nodularity.

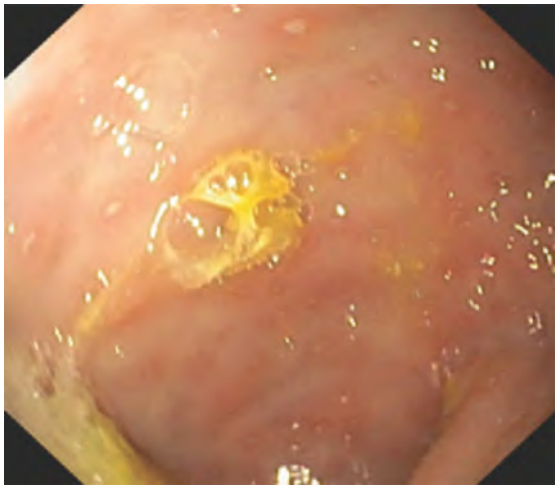

Figure 1b. Endoscopic image showing apthous ulcers

Histopathology Protocol (Figure 2)

There are multiple fragments and local denuded epithelium with surface mucodepletion. The lamina propria showed dense infiltrate of eosinophils. No crypt architectural distortion, granuloma, pigment laden macrophages, parasites or viral inclusions. The features were suggestive of allergic colitis.

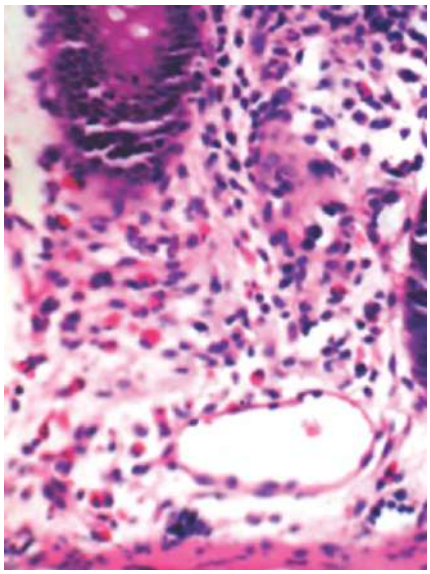

Figure 2: The rectal mucosal biopsy show edema and prominent eosinophils (>15 eosinophils/ HPF) in the lamina propria. (H&E X 200)

Discussion on histopathology

The salient histopathological features of the clinical possibilities are tabulated in Table 4.

Our patient shows a dense eosinophilic infiltrate (>15/HPF) in the absence of features of other disorders hence suggesting allergic colitis. The number of eosinophils on biopsies in patients with allergic colitis is a question of debate. Previous studies have suggested a diagnostic criteria of eosinophilic infiltration in the lamina propria of ≥ 6/HPF. (8, 9)

Table 4. Salient histopathological features of the causes of colitis in an infant

	Histopathological features
Common variable immunodeficiency (CVID)	Histopathologically, CVID is a great mimic. Features that may be seen include absence or paucity of plasma cells (evident in approximately two thirds of patients).(6) Apoptosis is much more common as compared to IBD A preponderance of eosinophils in the inflammatory infiltrate is NOT seen.
Chronic granulomatous disease (CGD)	Sharply defined aggregates of epitheloid histiocytes surrounded by a cuff of dense lymphocytic inflammation. Inflammatory cells consist mostly of degranulating eosinophils, large pigment laden macrophages & lymphocytes with paucity of neutrophils(7)
Inflammatory Bowel Disease	Moderate to severe chronic architectural changes Increased apoptosis, eosinophils in lamina propria and crypts.
Infectious colitis	Superficial ulcers with exudates, cryptitis with abscesses and increased cellularity of lamina propria with predominant neutrophils and lymphocytes
Food protein induced allergic proctocolitis	Focal eosinophilia (>6 eosinophils/HPF) in the lamina propria of one or two crypt regions or eosinophilic proctitis (focal eosinophilia and eosinophilic infiltrates in the muscularis mucosa and/ or eosinophilic crypt abscess)

Follow – up

The child was then put on a diet free of milk and milk products which included rice – dal, mashed fruits and vegetables etc. No milk substitute formula was used. Iron and calcium supplements were given.

Child gradually responded. After 2 weeks the blood and mucus subsided completely. Stool frequency reduced and the child started gaining weight.

At the last follow – up at 1.5 years of age the child weighed 10.4 Kgs (- 0.61 z-score). Anaemia had subsided and the child was gaining milestones appropriate for age.

OPEN FORUM

Food Protein induced allergic proctocolitis (FPIAP) commonly presents in infancy with most affected children presenting with symptoms by 6 months of age. Onset is rare after 12 months. It is typically manifested with rectal bleeding in well-appearing infants during the first months of life accounting for up to 60% of healthy infants with rectal bleeding. (10) Onset is usually insidious, with a latent period after introduction of the food as was seen in our child. It is typically caused by cow's milk and soy proteins and exclusively breast-fed infants may also develop clinically significant FPIAP via dairy protein transfer into human breast milk. Failure to thrive (FTT) is characteristically absent. (10)

So then why did our child have FTT? FPIAP is a part of a group of non–IgE-mediated gastrointestinal food-induced allergic disorders which also includes food protein–induced enterocolitis syndrome (FPIES) and food protein–induced enteropathy (FPE). FPIES is on the severe end of the spectrum with patients presenting with repetitive vomiting, pallor, a raised CRP and lethargy.(10,11)Chronic FPIES can lead to failure to thrive. FPE presents with protracted diarrhea, intermittent vomiting and FTT. These are all separate clinical entities but have many overlapping clinical and histologic features and at times may co - exist. (10,11) Our child presented with rectal bleeding, FTT, vomiting and a raised CRP, suggesting a possible mixed phenotype. (11) Though we didn't carry out a duodenal biopsy it is conceivable that the child had the presence of a co-existing enteropathy. The salient differences between the three types of non–IgE-mediated gastrointestinal food-induced allergic disorders are tabulated below.(Table 5)

A diagnostic elimination diet to see if symptoms improve suggests the diagnosis. Definitive diagnosis requires an oral food challenge test, however it is barely required in clinical practise. Milk protein should be avoided for up to four weeks (minimum of two weeks) until there has been a clear improvement in symptoms. A proctosigmoidoscopy and rectal biopsy is indicated only if there are atypical features or non-responsiveness to treatment. In our child we decided to go ahead with a procto-sigmoidoscopy because of the presence of protracted bleeding, failure to thrive and anaemia. Aphthous ulcers (small discrete ulcers with surrounding erythema and normal intervening mucosa) are suggestive of FPIAP. However, these findings are neither sensitive or specific. In our child the concomitant presence of diffuse loss of vascular pattern and superficial ulcerations raised the possibility of an inflammatory bowel disease but there were no suggestions of the same on histopathology.

FPIAP has a favourable prognosis, as most children will outgrow their allergy. The management comprises of the avoidance of cow's milk and cow's milk products. If the mother is exclusively breastfeeding, she should be advised to exclude all cow's milk and cow's milk products from her diet. The choice of cow's milk substitute should take into account the age of the child, the severity of the allergy. If the child is being formula fed, they should be tried with an extensively hydrolysed formula (eHF). Hydrolyzed formulae are those where the proteins have been hydrolyzed in order to remove allergenic epitopes. Soy formulae can be considered where eHF may be considered unaffordable. Soy formula is well tolerated by most individuals with Cow Milk Protein Allergy (CMPA). However, it is to be remembered that around 10-20% of children with CMPA may also have allergy to Soy. (10,12) Our child had not shown any response to week-long trial of

Table 5. Differences Between Non–IgE-Mediated Gastrointestinal Food-Induced Allergic Disorders

	FPIES	**FPIAP**	**FPE**
	Dependent on age of exposure to antigen; usually 1 d to 1 y	Days to 6 mo	Dependent on age of exposure to antigen; CM and soy up to 2 y
Family history of atopy	40% to 70%	Up to 25%	Unknown
Symptoms			
Emesis	Prominent, repetitive	Absent	Intermittent
Diarrhea	Severe	Mild	Moderate
Bloody stools	Severe	Prominent	Rare
Edema	Severe	Mild, infrequent	Moderate
Shock	15%	Absent	Absent
FTT	Moderate-to-severe in patients with chronic FPIES	Absent	Moderate
Laboratory findings			
Anemia	Moderate	Mild, infrequent	Moderate
Hypoalbuminemia	Acute	Mild, infrequent	Moderate
Acidemia	Might be present	Absent	Absent
Leukocytosis	Prominent	Absent	Absent
Thrombocytosis	Moderate	Mild	Absent
Peripheral blood eosinophilia	Absent	Occasional	Absent
Natural history	Varies by population, CM tends to resolve by age 3-5 y	Majority resolve by age 12 mo	Most cases resolve in 24-36 mo

FPIES - food protein–induced enterocolitis syndrome, FPIAP - food protein–induced allergic proctocolitis, FPE - food protein–induced enteropathy, CM – Cow's Milk

soy milk suggesting possible concomitant soy allergy too. Hence, we chose to avoid soy. In children with severe symptoms or intolerance to eHF, one may use an amino acid formula. As these formulas are expensive and as our child was already beyond 6 months of age we chose to put him on a semi – solid/ solid milk free diet and did not use any commercial formula. The unequivocal response clinched the diagnosis.

As cow's milk is the major source of calcium in infant diets, children on milk exclusion diets are at risk of a deficient calcium intake and should be supplemented.

Cow's milk allergy will resolve in the majority of children. Individuals should be reassessed at 6–12 monthly intervals from 12 months of age to assess for suitability of reintroduction. Most children with non-IgE mediated CMPA will develop tolerance by 5 years of age (13,14)

To conclude, FPIAP is the commonest cause of colitis in an infant. In children with atypical features one should evaluate for other possible etiologies and carry out a proctosigmoidoscopy.

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CASE REPORT

Primary Diffuse Large B Cell Lymphoma of Colon Presenting as Chronic Diarrhea in a 3 Year Old Child - Case Report

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Abstract

A 3 year old baby boy presented to hospital with a 2 months history of watery diarrhea (8-10 episodes per day) associated with abdominal pain, weight loss and swelling of face and limbs. Rehydration and electrolytic balance were restored with intravenous fluid therapy along with cover of antibiotics but diarrhea was worsening. Patient was planned for gastroscopy and ileo- colonoscopy with biopsies from duodenum and ileum. Gastroscopy was normal but colonoscopy showed ileal scalloping and transverse colon showed short segment stricture of approx. 5 cms with multiple ulcers. CT imaging showed a structuring mass involving the transverse colon and few adjacent jejunal loops with enlarged lymph nodes. Histopathological examination of the biopsies from transverse colon showed dense lymphoid aggregates. Hence Bone marrow aspiration and IHC panel of markers were advised. BMA was normal but IHC staining revealed Primary Diffuse large B cell Lymphoma of large intestine.

Keywords : GI Lymphoma, Chronic Diarrhea

Introduction:

Primary Non-Hodgkin's lymphomas (NHL) of the gastrointestinal tract are the most common extranodal lymphoma with increasing incidence in recent years, yet they are rare tumors.[1] Due to rarity and variable clinical presentation early detection is prevented while possibility of cure exists. Non-Hodgkin's lymphoma (NHL) remains the most common malignancy of the GI tract in children. They usually have different anatomic distribution and histologic appearance compared to common patterns in adult cases. Unlike adult patients in whom stomach is the most frequent site, small and large intestines are the most commonly involved sites in pediatric age group. Clinically, the patients present with varied symptoms ranging from abdominal mass to acute abdominal emergency caused by intussusception. Majority of the patients (81.4 %) present with abdominal pain as the presenting symptom, followed by abdominal swelling, vomiting, constipation, diarrhea, and intestinal obstruction. Nearly 50% of children with GI NHL have tumor infiltrates confined to GI tract with possible regional lymph node involvement.[2]

Case presentation:

A 3 year-old boy presented to hospital with a 2 month history of watery diarrhea. The child was reported to have had 8-10 episodes of stools per day which started with 5-6 episodes at presentation, watery in consistency, medium to large in quantity, associated with mucus occasionally, There was no blood, oil droplets or frothing. There was no fecal soiling of clothes ruling out anal incontinence or rectal pathology. It was associated with pain abdomen(child used to cry holding his hands around the umbilicus) prior to passage of stools and relieved after passage. No associated vomitings. Diarrhea was

not related to any specific food intake. Child also had lower limb swelling and facial swelling since 1 week along with weight loss of approximately 3 kgs(23.5% of total body weight).

His routine medical history revealed delivery via LSCS with normal development and good vaccination status. No history of recurrent chest or gastrointestinal infections. No history of previous hospitalisations.

On examination there was facial puffiness and bilateral pedal edema upto calf. There was no peripheral lymphadenopathy on examination. Respiratory, cardiac and neurological systems were normal on examination. His genital and rectal examination was normal. Abdominal examination revealed abdominal wall edema. No mass or organ palpable.

Laboratory tests showed normal blood picture with mild anemia (HB-11.7 g%). Liver function tests showed reduced protein (2.9) and albumin (1.48). His serum IgA levels were very low (<3 mg/100 ml). C-reactive protein and serum TTG (IgA)levels were normal (TABLE 1).

Wbc/pmn (*10^9/L)	7900
Hb (g/dL)/Ht (%)	11.7
Platelets (*10^9/L)	4,07,000
CRP (mg/dL)	0.501(Normal range - <0.6)
Total bilirubin (mg/dl)	0.3
AST / ALT (U/L)	26/ 38
Total protein(g/dl)	2.9
Serum albumin (g/dl)	1.48
Sodium / potassium (mEq/l)	138.2/2.92
Calcium (mg/Dl)	7.8
Stool examination	Normal
Stool for occult blood	negative
Serum IgA	< 3mg / 100 ml
TTG (IgA)	1.24 U/ml

TABLE 1 : Lab Parameter

He was rehydrated with IV fluids and started on broad spectrum I.V antibiotics along with enteral nutrition. Upper gastrointestinal endoscopy and Ileo- colonoscopy were advised along with duodenal and ileal biopsies. Gastroscopy and duodenal histopathological examination revealed no abnormality. Ileo-colonoscopy showed ileal scalloping (FIG 1) along with transverse colon showing short segment stricturous lesion extending for approximately 5 cm with multiple ulcerations and friable mucosa (FIG 2). Ileal microscopic examination revealed focal blunting and broadening of villi associated with focal cryptitis. Transverse colon lesion on microscopic examination showed large intestinal mucosa with ulcer replaced by fibrin, inflammatory exudate and bacterial colonies. Crypt atrophy was seen along with mild infiltrates of plamsa cells and eosinophils (40/hpf). A dense lymphoid aggregate composed of fairly monotonous cells having scanty cytoplasm and round to oval nuclei displaying 2-4 nucleoli is seen. No granulomas or parasites were seen. AFB staining was negative (FIG 3 & 4).

Contrast enhanced CT imaging was done which showed irregular structuring mass involving the transverse colon near the splenic flexure causing partial luminal obstruction with involvement of few adjacent jejunal loops in left upper abdomen with mild focal strictures.

Diffuse mesenteric edema is seen with multiple enlarged lymph nodes(FIGURE 5 AND 6).

Meanwhile patient was shifted to Intensive care in view of increased frequency of stools to 15-20 episodes per day and started on Total parenteral nutrition. In view of dense lymphoid aggregate with multiple nucleoli in the large intestinal lamina propria and a stricturing mass noted in CT imaging, IHC panel of markers and bone marrow aspiration was advised to r/o Lymphoid neoplasm. Bone marrow aspiration was normal and IHC was consistent with diffuse large b cell

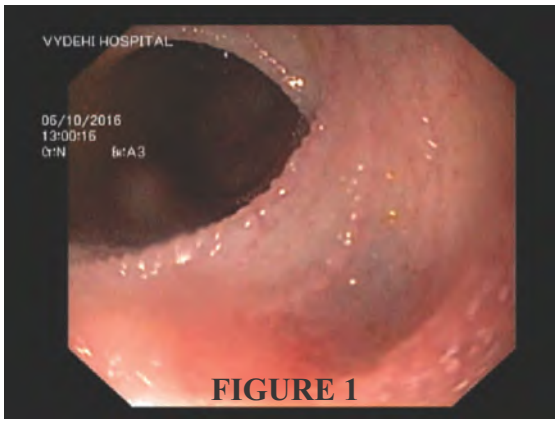
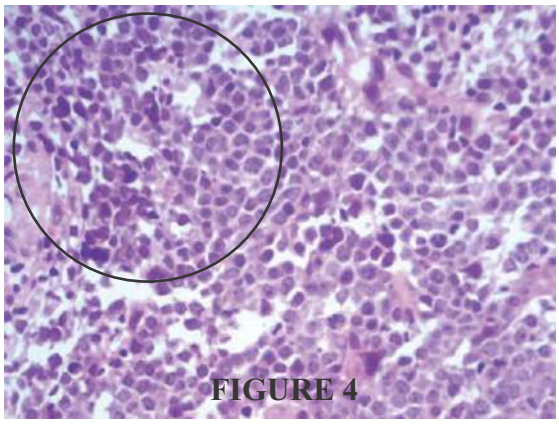


FIGURE 1



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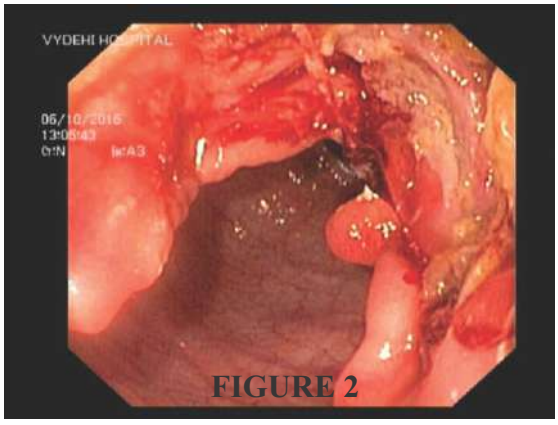

FIGURE 2

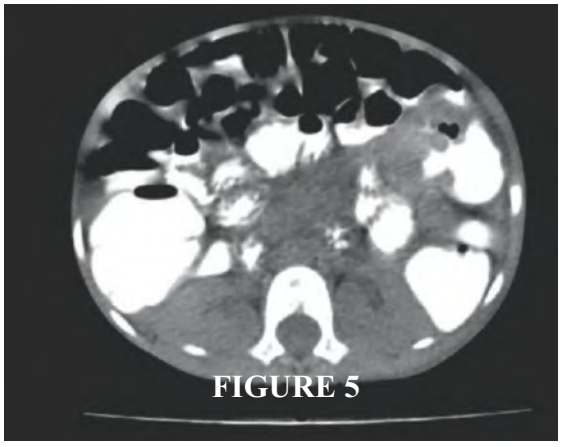

FIGURE 5

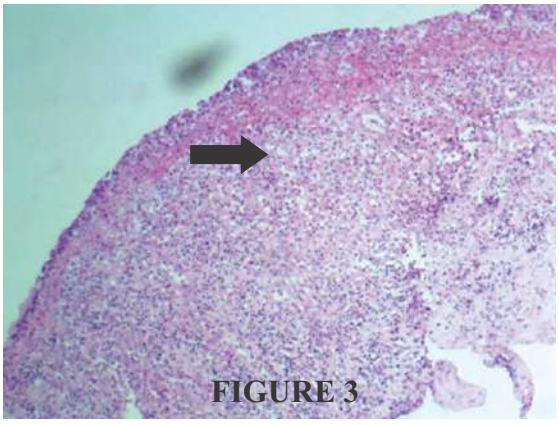

FIGURE 3

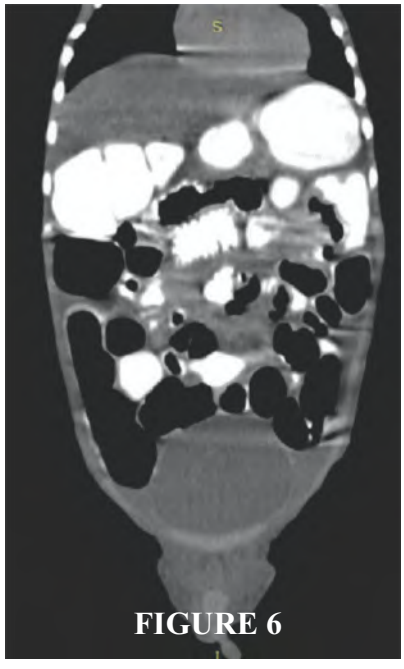

FIGURE 6

lymphoma(DLBCL)-large intestine (TABLE 2)

CD40	3+(neoplastic cells)
CD3	1+(scattered mature T -lymphocytes)
CD68	2+(background infiltrating histiocytes)
CD4	1+(normal T -lymphocytes)
BCL -8	3+(neoplastic cells)
CD30	0
CD10	0
PAX-5	3+(neoplastic cells)
CD20)	3+ (neoplastic cells)
VIMENTIN	4+(neoplastic cells)

As per Dawson criteria, patient was diagnosed to have Primary Gastrointestinal lymphoma involving large bowel with extension into small bowel causing chronic diarrhea and protein losing enteropathy.[3]

Patient was referred to medical oncologist. As there were no features of intestinal obstruction and chemotherapy being a good curative option, patient was advised chemotherapy.

Discussion:

Dawson et al. were the first to describe colorectal lymphoma in 1961.[4] Involvement of the large intestine is rare (10%-20% of all gastrointestinal lymphomas) in comparison to the stomach or small bowel. Primary NHL accounts for 0.1%-0.5% of all malignant tumors of the colon and rectum which makes it the third most common large bowel malignancy after adenocarcinoma and carcinoid.

Malignant lymphomas are the third most common type of childhood cancer. Children typically present with diffuse extranodal disease in contrast to adults among whom primary nodal disease is common. Primary GI malignancies are a rarity in children, with limited information from Asian population. The peak age for NHL of GI tract in children is 5–15 years.

The ideal treatment approach in GI lymphoma is debatable as per literature[5]. Radical tumor resection followed by chemotherapy in early disease(St. Jude stage I and II), and limited or no resection followed by polychemotherapy in advanced disease(St. Jude stage III and IV) may be the justified approach. However, recent studies have proposed the use of chemotherapy alone as an effective treatment option in primary GI lymphoma in all stages. A study performed at AIIMS, New Delhi comparing chemotherapy vs chemotherapy plus surgery in non Hodgkins lymphoma patients showed that five years EFS(event free survival) and OS(overall survival) were 72% and 67% for CT only group compared to 60% and 64% for CT+surgery group [6].

A study from Asian population which was done in Pakistan to identify the primary GI lymphomas under 19 years age group had shown higher prevalence of DLBCL among GI lymphomas[7].

Due to rarity of presentation and non specific symptoms we are reporting the above case which was diagnosed with Primary NHL of colon (DLBCL) with extension into small bowel causing chronic diarrhea.

Conclusion:

Primary GI lymphomas is a multifarious disease varying in its staging, site of involvement, histological subtype and type of treatment offered.

Primary GI lymphomas especially of the large bowel are rare and diagnosis can be delayed (in upto 35-65 % of cases) due to variable clinical presentation , while the options for cure exist.

Among lymphoma patients, children present predominantly with extranodal disease unlike adults. Even the presentation of pediatric GI lymphomas varies with adult group as the primary organ involved is small and large intestine unlike stomach in adults.

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Dysphagia lusoria due to aberrant right subclavian artery

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Abstract

Dysphagia Lusoria is dysphagia secondary to an aberrant right subclavian (ARSA) artery that traverses behind esophagus. We present a 11-year-old female complaining of weight loss and dysphagia. The diagnosis was pointed by barium swallow followed by endoscopy and CT angiography confirmed it. The combination of the common carotid origins and the retroesophageal course of the aberrant vessel frequently contribute to symptoms in the absence of an aneurysm of the aberrant vessel. Several surgical techniques for the aberrant vessel have been described, but we did an open ligation and transposition to the right carotid artery.

Keywords : *Dysphagia,* Subclavian Artery

Case Report

An 11-year-old female presented to us with a two year history of dysphagia to solids and weight of 22 kg vs 24 in the last year. Physical examination revealed only low weight for age and routine laboratory data were within normal limits. Barium swallow demonstrated an indentation at the level of third thoracic vertebra(figure 1).

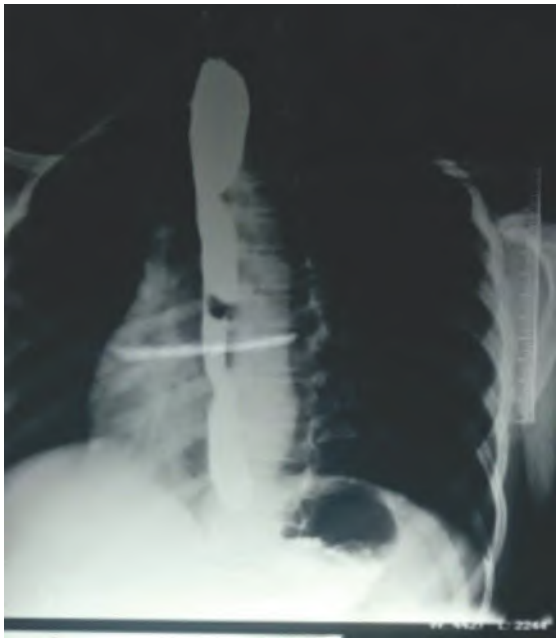

Figure 1:Barium swallow, indentation at the level of 3rd thoracic vertebra

Pulsatile narrowing was seen 10 cm from incisor teeth on upper endoscopy.

CT angiography of the chest demonstrated an aberrant right subclavian artery, originating from the descending aorta (Figure 2).

The patient was referred to the Vascular Surgery department for further management. The ARSA originated distal to the origin of the left subclavian artery and coursed through the posterior mediastinum .

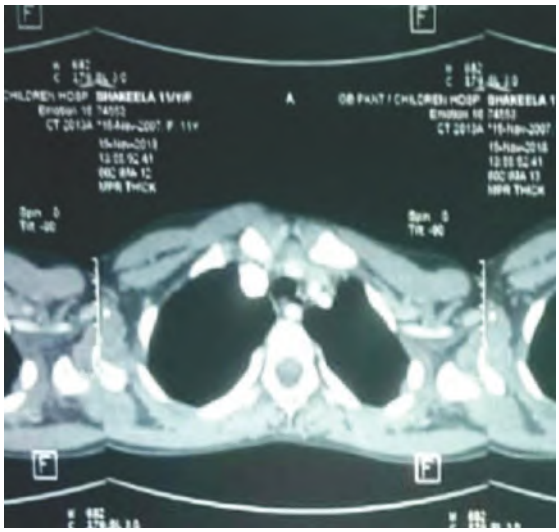

Figure 2. Arrow showing ARSA

The right anterolateral thoracotomy was done dividing artery from aorta and then its proximal end was anastomosed with right carotid artery. The patient had an uneventful postoperative course and remains symptom-free after follow up of 4 months**.**

Discussion

The first case of a symptomatic ARSA was described in the medical literature by Hanuld (1735) [1]. The term "dysphagia lusoria", however, was coined by Bayford (1794) [2]. ARSA is most common aortic arch anomaly [3]. In about 80% of individuals, three branches arise from the aortic arch: the brachiocephalic trunk, the left subclavian artery and the left common carotid artery (Adachi A). About11% of individuals have common trunk for the left common carotid and the brachiocephalic artery(Adachi B). Vertebral artery may originate proximal to the left subclavian artery as a 4th branch of the arch(Adachi C) [4]. The origin of the retroesophageal right subclavian artery as the last branch occurs in between 0.4 and 2% of individuals(incidence) [3–5].

The right subclavian artery (RSA)develops during the 6th to 8th week of gestation. The proximal part originates from the right 4th aortic arch artery, and the distal part from the right dorsal and right seventh intersegmental arteries . The aberrant right subclavian artery arises from the dorsal margin of the aortic arch, between the top of the arch and the vertebral column in mediastinum behind esophagus.

Most commonly it is asymptomatic (autopsy and retrospectively). Dysphagia is the most common symptom. Aspiration pneumonia secondary to dysphagia can occur in children.[6, 7,8] Barium swallow shows the characteristic indentation at the level of the third and fourth thoracic vertebrae. UGI endoscopy may demonstrate a pulsatile narrowing. Digital Subtraction Angiogram, CT with contrast, or MRI may confirm the diagnosis [6-9].

Symptomatic patients benefit from surgical intervention. Ruling out other cause of symptoms and after a trial of medical management (prokinetics and PPI), surgery should be considered [6].Various surgical approaches include dividing and ligating the ARSA via a left thoracotomy [11],Simple ligation and division [12], reimplanting the RSA with a graft onto the ascending arch via a left thoracotomy,anastomosing RSA to ascending aorta[13,14], endovascular or hybrid approach [15].Endoluminal grafts have also been used with some success in the presence of aneurysm of the ARSA origin.[16]

ARSA is a rare cause of dysphagia, barium swallow and endoscopy can hint towards need for CT angio chest and surgery.

Conflict of interest- none

Funding- none

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Aerophagia - An interesting cause of recurrent abdominal distension

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Abstract

Functional aerophagia involves excessive air swallowing causing progressive abdominal distension. It can present in children dealing with stress and anxiety. Early recognition and diagnosis of aerophagia is required to avoid unnecessary, expensive diagnostic investigations. Neuro cognitive evaluation and behavior therapy are accepted management strategies.

Keywords : Aerophagia, Abdominal Distension

Introduction

Aerophagia is a well-recognized condition among pediatric unctional GI disorders however it remains under recognized due to lack of awareness among pediatricians. Its defining criteria have been modified as per latest Rome IV criteria among children while it has been removed from adult classification system [1]. A large school-based, cross-sectional study conducted in North India used Rome III criteria and reported a prevalence of 1.5% while Sri Lankan study has reported 7.5% prevalence of this condition [2,3]. We report an interesting case of massive abdominal distension due to aerophagia.

Case Report

Anxious and worried parents presented to our outpatient department with their 7 year old son who was experiencing recurrent bouts of massive abdominal distension over last one month. It was sudden in onset, usually during night times after meals and resolved spontaneously after 30 minutes. As per parents child was restless and uncomfortable during that period. It was not associated with belching, increased flatulence or altered bowel habits. He had suffered similar problem of milder nature one year back. He was treated for constipation and gastritis during that time. His weight was 18 kg (weight/age < 2SD), height 118 cm (height/age < 2SD). Vitals, abdominal and systemic examination were normal. There were no identifiable stressors. His previous investigations revealed Hb 11.1gm/dl, TLC 6800/cu mm (P46 L50 E1 M3), platelet count $285x10^9$/L, ESR 20, CRP 0.8mg/dL, Blood urea nitrogen 22mg /dL, serum creatinine 0.4mg/dL, serum sodium 138meq/L, serum potassium 3.9 meq/L , serum calcium 8.9 mg/dL. Multiple ultrasonography reports did not reveal any organomegaly or intussusception. Computed Tomography of the abdomen showed no evidence of intestinal obstruction. On Upper gastro intestinal endoscopy lax lower esophageal sphincter was present. We kept the child under observation for 24 hours to record the episode as it occurred during night time. Patient had two episodes of abdominal distension during night. It was observed by resident doctor that child swallowed air for 20-30 minutes before distension occurred. There was visible epigastric distension (> 5cm increase in abdominal girth) with tympanic note on percussion. There was no tenderness or lump felt. Abdominal radiograph during that time showed distended bowel loops with increased

gas in stomach and gut without air fluid levels [Fig 1 and 2]. His distension relieved spontaneously after passage of flatus and burps. These episodes of air swallowing had gone unnoticed by parents till now. Clinical diagnosis of aerophagia was made. Psychiatric evaluation showed Attention deficit hyperactive disorder. Simethicone and behavior therapy were advised. Child improved gradually over 3months with continuous behavior therapy.

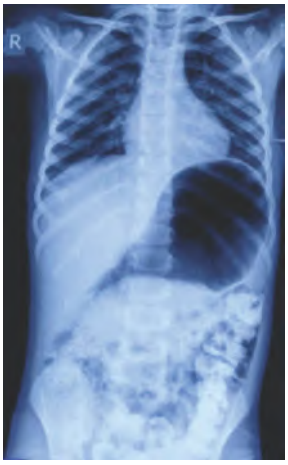

Figure 1. A plain erect abdominal radiograph showing massive gastric distension.

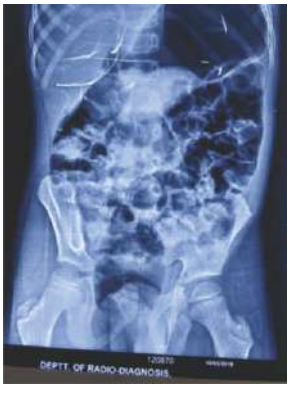

Figure 2. A plain erect abdominal radiograph showing extensive gaseous distension stomach,small bowel and colonic loops with gas seen in rectum. No abrupt transition in caliber seen in bowel loops.

Discussion

Rome IV diagnostic criteria define aerophagia as presence of all three criteria for at least two months: a. Excessive air swallowing b. Abdominal distention due to intraluminal air which increases during the day c. Repetitive belching and/or increased flatus and d. After appropriate evaluation, the symptoms cannot be fully explained by another medical condition. The word "excessive" and "increases during the day" have been added from previous definition based on the Rome III Criteria for FGIDs [1].

In daily clinical practice they can present with chronic stable symptoms or as surgical emergencies .It is mostly seen in children with severe psychiatric and/or neurologic problems. However it can affect normal children in stress situations. Pathophysiologically when air swallowing is excessive, gas fills the GI lumen, resulting in excessive belching, abdominal distention, flatus, and pain as a result of luminal distention. In few children, symptoms of distention and pain may be more severe when they are unable to belch. It is estimated that 70% of the gastrointestinal gas is swallowed, 20% is caused by diffusion of gases from the blood, and 7% to 10% is the result of bacterial decomposition. [4,5]

In extreme cases, aerophagia may lead to massive gastric and intestinal distension with the consequent development of ileus, volvulus, necrosis or even perforation due to ischemia [6]. Aerophagia may be confused with etiologies of abdominal distention and excessive flatus like gastroparesis, chronic intestinal pseudo obstruction, small intestinal bacterial overgrowth and malabsorption (celiac disease) [7, 8]. These can be ruled out on detailed history and minimal workup. Typical history of gulping sounds and movements suggestive of air swallowing or video recording

of the episodes can serve as important clues to diagnosis. In older children, large amounts of air may be swallowed while chewing gum or drinking water very quickly. This can prevent unnecessary investigations like CT abdomen in such cases.

There are no controlled studies to guide management. Therapy includes behavioral therapy and psychotherapy, speech for therapy can be considered a very important approach as it may make the patient conscious of his/her behavior [9]. A diet free of beverages containing gas may help reduce the volume of intestinal gas and alleviate symptoms. In addition, drugs such as simethicone and dimethicone can reduce gas formation in the bowel .Good communication and support to the family is needed compliance.

Conclusion

Aerophagia is a functional GI disorder characterized by repetitive air swallowing, abdominal distension, belching and flatulence .Extra intestinal symptoms of headache, sleep difficulty and lightheadedness may be present in some children. It can present as sudden acute attacks or chronically. An overlap with other FGID-like irritable bowel syndrome or constipation can be found. Detailed history and observing the episode carefully by parents or caregivers can clinch the diagnosis with minimal investigative workup. Treatment consists of parental reassurance and behavioral therapy.

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Fatal outcome of perforating lower esophageal button battery in a child

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Abstract

Impacted button battery (BB) in the esophagus can be fatal unless managed emergently. We encountered a *2-year-old* male child with respiratory distress from three weeks. Chest-X-ray suggested a radiopaque foreign body, which was confirmed to be esophageal BB during esophagoscopy and subsequently removed. Corrosive nature of BB had perforated the lower esophagus leading to pyopericardium and pyothorax. In spite of appropriate antibiotics and surgical drainage, child died of sepsis. High index of suspicion to identify esophageal BB in cases of unexplained cardio-respiratory symptoms can be helpful in early aggressive intervention.

Keywords : Button Battery, Perforation

Introduction

Foreign body (FB) in the form of Button battery (BB) ingestion is one of the important GI emergencies in children less than 5 years[1][2]. BB is lethal if not removed from the esophagus at the earliest. There are many instances of deaths reported in children after ingestion of BB. The cause of death due to BB ingestion in most of the patients is due to vascular complications such as erosion of aorta and massive exsanguination. Here we report an unusual complication of button battery with a fatal outcome.

Case report

A previously well, two-year old male child presented with 25 days' symptoms of vomiting, followed by fever and rapid breathing, who later developed dysphagia and drooling of saliva for which medical attention was sought. During evaluation, chest-X-ray (CXR) showed radiopaque round foreign body suggestive of button battery (figure 1) in the lower part of esophagus, with a retro-cardiac radiolucent shadow suggesting air in the pericardial sac. For these complaints child was referred to our center. On examination child was malnourished (weight of 10kgs [z-score of -1.75], height of 85cm [z-score of -0.89] with head circumference of 48cm), child was in respiratory distress (rate: 36/min, intercostal retractions and SpO_2 of 99% in room air), with normal cardiac examination (pulse rate 114/min, normal circulation and blood pressure of 94/60mmHg), but with an elevated jugular venous pressure. Child demonstrated micronutrient deficiencies in the form of pallor, hypopigmented hair and frontal bossing. Systemic evaluation revealed moderate hepatomegaly (span of 9cm), tachypnea, bilateral intercostal retractions and decreased air entry in the lower lung fields. Initial stabilization was done with nasal prong oxygen. In view of suspected FB (as reveal by initial CXR) in the esophagus, child underwent flexible esophagoscopy, where a metallic round FB suggestive of an impacted and partially disintegrated BB in the lower esophagus was identified (figure 2), which was removed using rat tooth foreign body forceps. After removal, a small rent suggestive of esophageal perforation at the site of impaction was noted (figure 3). In view of persistent respiratory distress and cardiomegaly on CXR, echocardiography was done, which was suggestive of pyopericardium (loculated collection of fluid). Contrast-enhanced

computed tomography was suggestive of pyopericardium and pyothorax (figure 4), due to esophageal perforation leading to esophago-pericardial and esophago-pleural fistula. Child was stabilized with antibiotics, intravenous fluids and nil per oral. In view of sepsis, worsening hemodynamics, requirement of inotropes a surgical drainage of the pyo-pericardium and pyothorax, with intercostal and pericardial drain was undertaken. However, in spite of drainage of pus, hemodynamics worsened due to uncontrolled systemic sepsis and cardiac dysfunction and child died of refractory shock after 4 days of removal of BB.

Discussion

This case highlights the lethal perforating complication of the neglected button battery in the esophagus in a young child. To best of our knowledge, this is the first case demonstrating esophageal leak leading to esophago-pericardial and pleural fistula leading to pyopericardium and pyothorax due to impacted BB.

The type of complication due to BB depends on the site of impaction in the GI lumen, duration and characteristics of the battery ingested. Similarly, the injury depends upon the type of battery ingested due to different mechanism of injury. Currently available BB are made up of either lithium, manganese dioxide, mercuric oxide, silver oxide or zinc based. Among these lithium batteries are the most lethal and unfortunately, they are more popular due to higher voltage and double the shelf life as compared to other batteries. They have a very high tissue damaging potential as compared to other chemical types. Their mechanism of injury is similar to an alkaline burn as lithium is able to generate a very high pH of upto 13 at the site of contact of mucosa within 15 minutes of impaction and deep burn upto muscular layer within 30 minutes[3]. Due to drastic shift towards lithium batteries, there is a parallel change in the epidemiology regarding the major effects of battery ingestion. A recent study has demonstrated an absolute rise in the incidence of deaths as against no mortality documented in a previous study from the same authors[2]. Reasons identified in this study for increased mortality are shift in the cell type to lithium based and increase in the diameter of BB to 20 mm, which was also the case in our patient. The previously documented fatalities are due to tracheal injury (2 patients), 1 due to tension pneumothorax, and 10 were due to fatal haemorrhage secondary to vascular erosion (7: aortic esophageal fistula, 1 each: erosion of thyroid artery, subclavian artery and a major mediastinal vessel)[4]. We believe BB should be removed when it is within the reach of the endoscope and certain high risk factors such as young child, large sized battery and fully charged unusual battery are to be identified. Even though the NASPHAN guideline mentions removal of FB anywhere in the GI lumen if >20mm and age < 5 years, one has to individualize based on the risk factors, rather than just acting on the absolute cutoffs[5][6].

Our patient highlights the need to have a detailed clinical and radiological evaluation in a child with unexplained respiratory distress and one should also consider a complicated FB as an etiology. Limitations are that the exact chemical nature of BB could not be ascertained and unfortunately the patient succumbed to complications.

To summarize, this is the first documented patient with mediastinitis, pyopericardium and pyothorax leading to death due to prolonged impacted BB in the esophagus in a young child.

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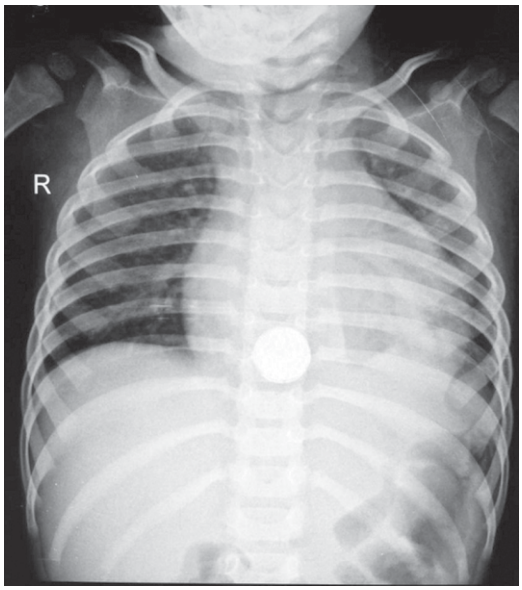

Figure 1: Chest X ray in anteroposterior view showing a radiopaque round foreign body in the lower esophagus.

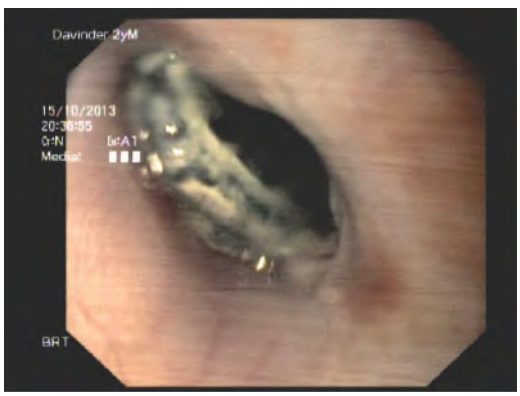

Figure 2: Esophagoscopy showing button battery

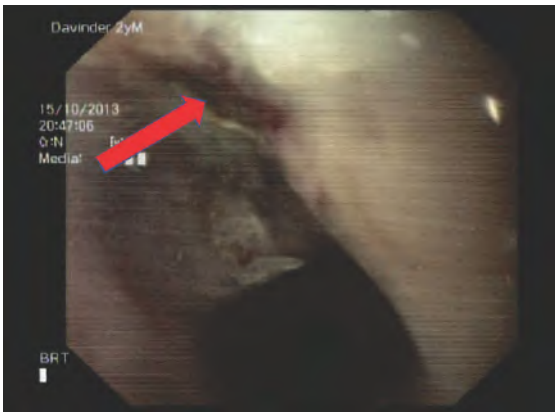

Figure 3: Esophagoscopy showing perforation in the lower esophagus (arrow)

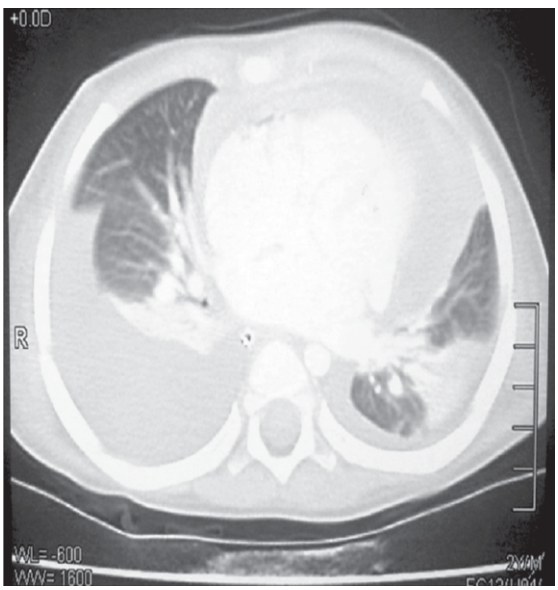

Figure 4: CECT chest showing pyopericardium and pyo-thorax

Corrosive and Button Battery Ingestion in Children

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As an MBBS student, acute substance poisoning was a sure-shot full mark question in forensic medicine (jurisprudence). We remember avidly preparing for the same. As an intern and later pediatric trainee, we learnt to manage some of them with variable confidence, some of us less keen than the others. As a pediatric gastroenterologist, of the acute emergencies that challenges us, the most dreaded perhaps is an acute corrosive ingestion and we are duty-bound to manage them. As we climb the ladder of super-specialisation, do we become more fearful; do we expect the worst outcome? Possibly, our experiences shape our fears. With regard to corrosives and foreign bodies, a gastroenterologist lives to tell the vivid tales of heroism, precision, decisiveness or of dismay, futility and catastrophe to his starry-eyed residents. Unwritten experiences and tips for management are passed on through generations. But what persists at the end of the day is a perpetuating scenario, a vulnerable community, the horrors of acute ingestion and the burden of its sequelae.

Problem in India

Acids and alkali, the two extreme ends of chemistry spectrum are physiologically available in gastric and pancreatic juices. They are gifted by nature and serve a purpose in the human body. When synthesised for commercial use, they are ultimately designed for our domestic use or materialistic pleasure. Alkaline household caustic agents are drain openers and oven cleaners (sodium hydroxide and hypochlorite), household cleaners (ammonium hydroxide, ammonium chloride), bleaches (hydrogen peroxide) and dishwashing agents (sodium carbonate, sodium silicate). Acidic domestic agents are toilet bowl cleaners (hydrochloric acid, phosphoric acid), metal cleaners (hydrochloric acid), battery fluids (sulfuric acid), jewellery cleaners (nitric acid). These products are colourfully packaged, scented, shelved and ubiquitously available in all grocery stores and urban supermarkets costing Rs 200-400 per litre. The same in their raw forms are sold at Rs 15-30 per litre in the rural and impoverished societies. Illiteracy and unawareness being major deterrents in our country, these caustics are loosely and irresponsibly stored in empty plastic bottles (mineral water or soft drinks) and not kept away from the reach of children. The scenario is further complicated due to the lack of strict government regulations that allow free unrestricted sale, improper packaging and lack of biohazard labeling. Most of the ingestions are accidental, especially under the age of 5 years as they are inquisitive in nature. Children are lured to the soft drink bottle or mistake the clarity of the caustic as potable water. Adolescents may have suicidal intent due to academic challenges, peer discordance, familial disputes or behavioural issues. Button battery, a unique foreign body has intensely alkaline and electrical properties that causes persistent tissue necrosis even after removal. They are found in toys, watches and other electronic goods. Toy safety is a major concern in the West where other than material quality and toxicity, it is mandatory to screw or fasten the battery

compartments. Changing of batteries is hence responsibly performed by the guardian. In India, cheaper toys have accessible battery compartments which can be easily unlocked or broken. Hence young children are prone to accidental ingestion of the battery.

Burden of the disease

Once the caustic ingested, a chain of agony ensues. Scientifically we debate acid versus alkali, coagulative versus liquefactive necrosis, predominant esophageal injury versus stomach injury, so on and so forth. Practically, it does not matter…the damage is done! The child is scarred for life. In an acute setting, fortunate patients are optimally managed, unfortunate ones are mismanaged. Their luck is further dependent on the physician's awareness, timely referral, patient's accessibility to the centre and expertise available. By the time a gastroenterologist receives the case, chronicity would have ensued. Vocal cord and laryngeal stenosis results in voice problems, respiratory issues and chances of life-threatening aspirations. Long, tenacious esophageal strictures cause various grades of dysphagia. The stomach capacity is reduced after fibrosis. Antropyloric stenosis produces gastric outlet obstruction. Compromise in nutrition leads to cachexia, dyselectrolytemia and apathy. Daunting endoscopies, endless complex dilatation procedures, struggle for adequate nutrition and managing the complications are the challenges faced. In refractory cases, when the esophago-gastric tract is replaced by an alternative conduit, newer problems arise as the normal physiology is disturbed. Moreover, compromises are governed by the affordability of patients. An emaciated toddler with a nasoenteric tube lying listlessly on your endoscopy table is a sorry sight. When the weeping parents in their tattered clothes helplessly approach you with folded hands, you are left perplexed as to how you would sustain the case. I was recently baffled by a 10 month old baby with a 2 cm button battery impacted in the esophagus. The 4 year old elder sibling found the shiny battery on the road side, played with it and left it on the bed. Attracted by the object, the infant who had just attained his pincer grasp, made the full use of his skill. The damage done was irreversible. I wished the infant would have rather swallowed a coin! Dealing with button batteries is perhaps a far worse nightmare than corrosives due to rapidly progressive necrosis of gut wall and surrounding structures. The larger the battery, the more is the charge and greater is the damage. Ingestion of smaller batteries may not be witnessed always by caretakers. They may pass out of the gastrointestinal tract and yet have delayed presentation. Most of the damage occurs above the diaphragm. Invariably the child will require multiple chest surgeries. The surgical planes of resection are challenging for even expert surgeons due to unhealthy friable tissue in the proximity of great vessels and nerves. Leaks, gaping wounds, suture line dehiscence and restenosis of lumen are the recurrent problems. Replacement grafts are expensive. Vascular catastrophes, perforation and tracheo-esophageal fistulae are notorious complications. Registries in United States of America (USA) report 46% short term and 70% long term case fatality.

Action plan

Pediatric caustic ingestion is presently unheard of in developed countries. One unfortunate infant death in USA from a button battery sent shock waves throughout the nation to bring in consensus guidelines for management. Unlike USA which registers battery ingestion, India does not have any registry to record these events. At the national level, corrosive ingestion and its sequelae may seem a minute problem as compared to community diarrhea, respiratory infections and malnutrition. In India, other than anecdoctal case reports, no facts can be

presented to the government for a national action plan. Hence a dedicated registry is one of the foremost strategies. Tougher acts such as the Federal Hazardous Substance Abuse Act (USA) will also need to be brought in to our country making it mandatory for every stake-holder (from manufacturer to seller) to comply with rules. As brilliant researchers in scientific forums, we continue to argue timing of endoscopy, role of adjuvant therapy, newer drugs, stents and techniques of dilatation. Is that solving our problem? Are we doing enough justice to our profession? We promote organ donation for cadaveric transplants but we do not speak of simpler preventive medicine for issues such as caustics. This is the glaring paradox in India. As sedulous pediatric gastroenterologists and pediatricians, we need to spread awareness at all levels. Fortunately, India is media-receptive. We must encourage safe handling, storage and disposal of caustics and battery ingestion at domestic and community level. Through newspapers, television and social media, the awareness must percolate to masses. The effort has to be individual and collective, immediate and sustained.

Of all the acquired diseases we deal with, caustic ingestion is definitely an uncalled for condition. The rewritten modified Hippocratic Oath of 1964 states "I will prevent disease whenever I can, for prevention is preferable to cure". As conscientitious doctors, we must rise to this challenge. It is time to bring a permanent change to our system.

This is an author's informal and personal viewpoint with no conflicts of interest with any individual or organisation.

Hepatology Update: Neonatal Cholestasis Syndrome

STUDY 1: Yang, L. , Zhou, Y. , Xu, P. , Mourya, R. , Lei, H. , Cao, G. , Xiong, X. , Xu, H. , Duan, X. , Wang, N. , Fei, L. , Chang, X. , Zhang, X. , Jiang, M. , Bezerra, J. A. and Tang, S. (2018), Diagnostic Accuracy of Serum Matrix Metalloproteinase-7 for Biliary Atresia. Hepatology, 68:2069-2077. doi:10.1002/hep.30234

Early diagnosis and treatment is required to improve clinical outcome in neonatal cholestasis syndrome. Neonatal Hepatopathy and Cholangiopathy are predominantly considered in this spectrum. Recent advances in this field are making the algorithmic approach more decisive. Serum matrix metalloproteinase-7 (MMP-7) is suggested to have discriminatory features for infants with Biliary Atresia. MMP-7, a protease involved in intercellular signaling through breakdown of extracellular matrix. The study concludes that serum MMP-7 assay has high sensitivity and specificity to differentiate Biliary Atresia from other neonatal cholestasis. The area under the curve of MMP-7 for the diagnosis of Biliary Atresia(BA) was 0.9900 with a cutoff value of 52.85 ng/mL; the diagnostic sensitivity and specificity are 98.67% and 95.00%, respectively, with a negative predictive value of 98.28%.

The diagnosis of BA was made by the presence of fibrosing obstruction of extrahepatic biliary remnants excised after intraoperative cholangiography. The authors studied MMP-7 among healthy controls (n=72 with 54 <6 months) and among 135 with cholestasis (75 with BA, 60 with non-BA). Median concentration for MMP-7 was 2.86 ng/mL in healthy controls, 11.47 ng/mL for non-BA cholestasis, and 121.1 ng/mL for BA. The predictive value for MMP-7 was particularly impressive, 74 of 75 BA subjects were correctly identified as having BA. Only 3 non-BA patients were incorrectly assigned a BA diagnosis based on MMP-7 values. Study also compares GGT with MMP-7 which showed a superior performance for MMP-7.Further studies with larger sample size are needed. One of the limitations includes insufficient data for the differentiating with MMP7 other rare cholestatic liver diseases such as Alagille syndrome, alpha-1-antitrypsin deficiency, citrin deficiency, and ductal plate malformation.

STUDY 2: Large-scale proteomics identifies MMP-7 as a sentinel of epithelial injury and of biliary atresia *Science Translational Medicine* 22 Nov 2017:Vol. 9, Issue 417, eaan8462 DOI: 10.1126/scitranslmed.aan8462

Using large-scale proteomics, the authors screened 1129 proteins in a cohort (n=70) of patients with BA. They identified several proteins that are increased with BA. Slow off-rate modified aptamer scan (SOMAscan, SomaLogic Inc.) applied to 70 serum samples from a cohort of infants at the time of diagnosis of BA (at Kasai operation, $n = 35$) and to age-matched infants with neonatal intrahepatic cholestasis (IHC; $n = 35$)

Matrix metalloproteinase-7 (MMP-7) is the lead biomarker. MMP-7 is constitutively expressed by normal cholangiocytes, increases in the serum upon biliary injury, and modulates the clinical phenotype in an experimental model of BA as concluded in the study.

MMP-7 is more accurate than gamma glutamyltranspeptidase (GGT). The combination of MMP-7 and GGT has a AUROC of 0.94 in validation cohorts. The authors further studied the role of MMP-7 by immunostaining and found it primarily was detected in cholangiocytes of intrahepatic bile ducts in infants with BA. MMP-7 expression in the liver do not correlate with fibrosis. Immunostaining showed minimal or no expression of MMP-7 in parenchymal or nonparenchymal cells of the normal liver. In liver biopsies obtained at the

time of diagnosis of BA, MMP-7 is detected in cholangiocytes of intrahepatic bile ducts (IHBDs). Livers from BA subjects had higher *MMP-7* mRNA expression compared to Intra Hepatic Cholestasis (fold change relative to normal controls, 9.98 ± 7.65 versus 2.54 ± 1.73; $P < 0.0001$) The data defined a primary localization of MMP-7 in cholangiocytes of extrahepatic bile ducts (EHBDs), with minimal or no expression in intrahepatic cholangiocytes of normal livers and detectable expression in intrahepatic cholangiocytes of diseased livers (but lower than the level seen in EHBDs).

Luminal Gastroenterology Update: GERD. (Gastroesophageal Reflux Disease)

STUDY 1: Mechanisms of Aerodigestive Symptoms in Infants with Varying Acid Reflux Index Determined by Esophageal Manometry .Collins, Carissa R. et al. The Journal of Pediatrics, March 2019 Volume 206, 240 – 247 https://doi.org/10.1016/j.jpeds.2018.10.051

The study interprets that symptoms alone are not able to predict the degree or presence of reflux in infants. Symptomatic neonates (n = 74) born at a median of 28.9 weeks gestation using 24-hour pH-impedance to determine (Acid reflux index) ARI severity, followed by provocative esophageal manometry with graded mid esophageal infusions (0.1-5.0 mL) of air, water, and apple juice is done. The effects of 2635 separate esophageal stimuli on reflexes and symptoms are analyzed. The authors considered ARI <3% as normal, 3-7% as indeterminate ARI, and >7% as abnormal ARI. Nonacid reflux is much more likely to provoke symptoms in this population than acid reflux, further questioning blind use of proton pump inhibitors and acid suppressor. Symptoms recorded are arching, irritability, cough, gag, sneeze, gasp, bradycardia, desaturation, throat clearing, startle, grimace, grunting, mouthing, and yawning. GERD severity plays no role in the generation of symptoms. The study concludes that symptoms and peristaltic reflexes are manifestations of the recruitment of several neurosensory and neuromotor pathways evoked by mid-esophageal infusions. ARI severity grade plays no role in symptom generation. GERD should not be diagnosed and severity should not be assigned based on symptoms alone.

STUDY 2: Pediatric Gastroesophageal Reflux Clinical Practice Guidelines: Joint Recommendations of the North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition and the European Society for Pediatric Gastroenterology, Hepatology, and Nutrition. (JPGN 2018;66: 516 –554) doi:10.1097/MPG.0000000000018 89

The published guidelines reinforce important clinical points to be followed in the practice. Few of the important recommendations are highlighted here. The working group suggests not using barium contrast studies for the diagnosis of GERD in infants and children. Based on expert opinion, the working group suggests to use esophago-gastro- duodenoscopy with biopsies to assess complications of GERD, in case an underlying mucosal disease is suspected, or prior to escalation of therapy. The working group suggests scintigraphy should not be used for the diagnosis of GERD in infants and children. They recommends not to use positional therapy (ie, head elevation, lateral and prone positioning) to treat symptoms of GERD in sleeping infants. The guideline suggests not to use a trial of PPIs as a diagnostic test for GERD in infant while a 4 to 8 week trial of PPIs for typical symptoms (heartburn, retrosternal or epigastric pain) in children as a diagnostic test for GERD can be used. They recommend not using antacids/alginates for chronic treatment of infants and children with GERD. It is advised to avoid use of H2RA or PPI for the treatment of crying/distress in otherwise healthy infants. Based on expert opinion, the working group recommends the use of PPIs as first-line treatment of reflux-related erosive esophagitis in infants and children with GERD.

STUDY3: Eichenwald EC and AAP committee on fetus and newborn. Diagnosis and

Management of Gastroesophageal Reflux in Preterm Infants. Pediatrics. 2018;142(1):e2018 1061 DOI: 10.1542/peds. 2018-1061

Highlights of the guidelines are as follows. GER is a normal developmental phenomenon that will resolve with maturation. Signs commonly ascribed to GER in preterm infants include feeding intolerance or aversion, poor weight gain, frequent regurgitation, apnea, and desaturation bradycardia and behavioral signs, including irritability and perceived postprandial discomfort. These signs will usually improve with time without treatment. There is poor data on worsening lung disease attributable to GER and micro aspiration in mechanically ventilated preterm infants. Left lateral body position, head elevation, and feeding regimen manipulation, have not been shown to reduce clinically assessed signs of GER in the preterm infant. Supine positioning on a flat and firm surface and avoidance of commercial devices designed to maintain head elevation in the crib, should be paramount importance in practice. Drugs should be used sparingly, if at all, in preterm infants.

Luminal Gastroenterology Update: Potential celiac disease.

STUDY 1: Progression of Celiac Disease in Children With Antibodies Against Tissue Transglutaminase and Normal Duodenal Architecture Auricchio, R. et al. Gastroenterology, article in press DOI: https://doi.org/10.1053/j. gastro.2019.04.004

Potential celiac disease is characterized by positive results from serologic tests for tissue transglutaminase antibodies (anti-TG2) but normal duodenal architecture (Marsh stages 0–1). Which patients would progress to overt celiac and which would need observation is a matter of debate. This study highlights on this issue much pending issue.

This is a prospective study of 280 children (ages 2–18 years) in Italy with suspected celiac disease. Enrolled patients have 2 consecutive positive results from tests for anti-TG2, tested positive for the endomysial antibody (anti-EMA), have total serum levels of IgA in the normal range, normal duodenal architecture (Marsh stages 0–1) in 5 biopsies, and HLA DQ2- or DQ8-positive haplotypes. Follow up period is 12 years (range, 18–150 months; median 60 months). Serologic tests and clinical analyses are done in the study every 6 months and a small bowel biopsy was taken every 2 years. A multivariate analysis of clinical, genetic, and histologic data to identify factors associated with progression to villous atrophy is done. The study has a largest cohort of potential celiac disease patients with the longest follow up. Immunohistochemical staining of duodenal biopsy for CD3+, TCRγδ+, and CD25+cells and presence of extracellular deposits of anti-TG2 IgA is done.

42 of 280 children (15%) developed villous atrophy. 89 children (32%) no longer tested positive for anti-TG2 or anti-EMA on follow up. The cumulative incidence of progression to villous atrophy is 43% at 12 years on gluten consumption. Data suggest that prescribing indistinctly to all potential celiac disease patients a GFD (Gluten free diet) would be an overtreatment. Younger patient at diagnosis has a greater chance to remain "potential". Factors most strongly associated with development of villous atrophy are numbers of γδ intraepithelial lymphocyte cells followed by age and homozygosity for the HLA DQB1*02. Lower numbers of γδ positive cells in the intestinal epithelium have been noted to be protective. Marsh 0 lesions at diagnosis on histopathology have less progression to villous atrophy. HLA effect is age specific and is not generalized and needs further research.

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Publications by ISPGHAN members
March – May, 2019

March

1. *Janwadkar A, Shirole N, Nagral A et al. Citrullinemia Type 1: Behavioral Improvement with Late Liver Transplantation. Indian J Pediatr. 2019 Mar 8. [Epub ahead of print]*

Citrullinemia Type 1 (also known as classic citrullinemia) is a rare autosomal recessive urea cycle disorder due to reduced activity of argininosuccinate synthetase 1; characterized by hyperammonemia leading to neurological damage. The authors report a case of an 8-y boy who was diagnosed with Citrullinemia Type 1 at birth which was anticipated prenatally due to family history. His diagnosis was confirmed as a homozygous mutation (Exon 15: c.1168G > A (p.G390R)) of ASS gene. Inspite of being on a protein-free diet and ammonia scavenging treatment; the patient developed recurrent episodes of encephalopathy and seizures; complicated with behavioral issues. The patient underwent living related liver-transplantation from his mother (heterozygous carrier of the same mutation) resulting in the reversal of neuro-behavioral changes. It is important to consider liver transplantation in citrullinemia Type 1 as it corrects the genetic deficiency of ASS

2. *Kadyada SP, Thapa BR, Bhatia A et al. Role of Diagnostic Endoscopic Ultrasound in Idiopathic Acute Pancreatitis and Acute Recurrent Pancreatitis in Children. Pancreas. 2019 Mar;48(3):350-355*

Endoscopic ultrasound (EUS) is a minimally invasive pancreatic imaging modality. The authors evaluated children with idiopathic acute pancreatitis (IAP) and acute recurrent pancreatitis (ARP) for changes of chronicity (Rosemont criteria). Diagnostic yield of simultaneously performed transabdominal ultrasonography (TUS) was compared with EUS. In this prospective observational study. Patients underwent EUS and TUS after 2 months of a pancreatitis attack. Forty-five (18 IAP, 27 ARP) patients were evaluated. It was found that applying EUS, changes of chronicity and risk factors were noted only in ARP. Endoscopic ultrasound performed better than TUS in detecting chronicity.

3. *Alam S, Lal BB, Sood V et al. AARC-ACLF score: best predictor of outcome in children and adolescents with decompensated Wilson disease. Hepatol Int. 2019 Mar 19. [Epub ahead of print]*

Doubts have been raised about efficacy of New Wilson's index (NWI) in predicting Liver Transplant (LT) or mortality in decompensated Wilson Disease (WD) patients. APASL ACLF Research Consortium (AARC) has introduced a new score (AARC-ACLF) which has not been studied in children. In this study, sixty-six confirmed cases of decompensated WD were evaluated. Thirty-nine (59%) improved on medical management and 27 (41%) either died (20) or were transplanted (7). AARC-ACLF had the best predictive score for mortality at 90 days with AUROC of 0.939. For every unit increase in AARC-ACLF score, there was a likelihood of 66% increase in 90-day mortality. The optimal cut-off for the AARC-ACLF score to predict mortality was 11 or more.

4. *Pamecha V, Vagadiya A, Sinha PK, et al. Live donor liver transplantation for acute liver failure - Donor safety and recipient outcome. Liver Transpl. 2019 Mar 12.. [Epub ahead of print]*

In countries where deceased organ donation is sparse, emergency live donor liver transplantation (LDLT) is the only lifesaving option in select patients with acute liver failure(ALF). The aim of the current study was to evaluate live liver donor safety and recipient outcomes following

LDLT for ALF. The authors found that outcome of emergency live liver donation was comparable to elective donors. Post-operative worsening of cerebral edema, preoperative SIRS, sepsis predicted outcome after LDLT for ALF

5. *Cherukuru R, Reddy MS, Shanmugam NP et al. Feasibility and Safety of Split-Liver Transplantation in a Nascent Framework of Deceased Donation. Liver Transpl. 2019 Mar;25(3):450-458*

Split-liver transplantation (SLT) is a valuable option for optimizing the use of good-quality deceased donor grafts. It is not routinely reported outside the West because of limited deceased donor numbers, technical and organizational constraints, lack of experience, and a predominant living donor liver transplantation (LDLT) practice. The authors report their experience of SLT and compare outcomes with pediatric and adult LDLT recipients.

They found that SLT is an effective technique with outcomes comparable to living donor grafts for adult and pediatric recipients. Using SLT techniques at centers with limited deceased donors optimizes the use of good-quality whole grafts and reduces the gap between organ demand and availability.

6. *Bhatnagar S, Srivastava G , Ansari A : Bowel Habits of Healthy Indian Children Less Than Two Years of Age. Trop Gastroenterol 2018;39(1):17-21 (In Print in March 2019)*
   DOI : http://dx.doi.org/10.7869/tg.455

Background: The bowel habits of children less than two years are quite varied and there is no definite hard data on stool pattern of Indian children particularly less than two years of age. Aim: To define the normal frequency and consistency of stools of healthy Indian children between 0-24 months of age. Methods: Parents of children aged up to 24 months were asked on a three day recall basis about their child's usual bowel habits and dietary history. Bowel habit was recorded in terms of number of stools the child passes per day, stool consistency, the age at which night bowel movements stopped, and the age of commencement of regular bowel movements. Feeding type was recorded as exclusive breast feed, mixed milk feeds or solid feeds. The bowel habits were correlated with the age and type of feeding. Result: Children in their first six months of life had stools which were predominantly "pasty" and "runny like cream" with high and variable frequency. Beyond six months consistency was "solid" and "pasty" stools. On analyzing the combined effect of the type of milk feed and age on bowel frequency and consistency, children beyond one month of age either on exclusive breast feed or on mixed milk feed had similar frequency and consistency of stools. By one year of age more than 90% children attained regular stool pattern with no night time bowel movements. Conclusion: This is the first report from India which describes the stool pattern of normal healthy children less than two years of age.

April

1. *Bolia R, Rajanayagam J, Hardikar W et al. Impact of Changing Treatment Strategies on Outcomes in Pediatric Ulcerative Colitis. Inflamm Bowel Dis. 2019 Apr 19. [Epub ahead of print]*

In recent years, treatment strategies for ulcerative colitis have evolved with an early step-up approach, the availability of biologicals, and therapeutic drug monitoring. This study was carried out to evaluate the effect of these changes on disease outcomes.

The authors found that a reduction in 2-year colectomy rates has been observed in patients with pediatric ulcerative colitis since biologics have become available for its treatment. However, the numbers of disease-flares rates and hospital admissions remain unchanged.

2. *Prasad D, Srivastava A, Tambe A, et al. Clinical Profile, Response to Therapy, and Outcome of Children with Primary Intestinal Lymphangiectasia. Dig Dis. 2019 Apr 26:1-9. [Epub ahead of print]*

Intestinal lymphangiectasia (IL; primary or secondary) is an important cause of protein-losing enteropathy. The authors evaluated the clinicolaboratory profile, response to therapy, complications, and outcome of children with primary IL (PIL). Twenty-eight children with PIL (16 boys, age at symptom onset-12 [1-192] months and at diagnosis 8 [1-18] years) were studied. Pedal edema (93%), chronic diarrhea (78.6%), and recurrent anasarca (64%) were the common presentations.Presence of chylous ascites suggests severe disease in children with PIL. Majority of PIL children respond to dietary therapy; only 20% need additional therapy. Long-term follow-up is essential to monitor for symptoms relapse and complications.

3. *Jain V, Sangdup T, Malik R et al. Abernethy malformation type 2: varied presentation, management and outcome. J Pediatr Surg. 2019 Apr;54(4):760-765.*

The purpose of this study was to study the varied presentations and the outcomes in children with Type 2 Abernethy malformation following shunt ligation. Five patients were included with a median age of 6 years. Hepatopulmonary syndrome was the presentation in 4 patients while one patient presented with liver tumor. At the median follow up at 14 months, good intrahepatic portal flow was seen in all patients. All patients demonstrated improvement/ resolution of symptoms.

May

1. *Phulware RH, Gahlot GPS, Malik R et al. Microvillous Inclusion Disease as a Cause of Protracted Diarrhea. Indian J Pediatr. 2019 May 2. doi:10.1007/s 12098-019-02963-y. [Epub ahead of print]*

Microvillous inclusion disease (MVID), also known as congenital microvillus atrophy, was first described by Davidson et al. in 1978. Till date, only a handful of cases with MVID have been described in English literature. These patients usually present with intractable secretory diarrhea in early days of life. The pathognomonic findings of MVID are villous atrophy along with the formation of intracellular microvillous inclusions on electron microscopy. Till date, no curative therapy exists.
Herein, authors describe a case of intractable diarrhea with MVID diagnosed in a 3-mo-old male child who presented with intractable diarrhea.

2. *Sood V, Khanna R, Alam S, Rawat D, Bhatnagar S, Rastogi A. Ductal paucity and Warkany syndrome in a patient with congenital extrahepatic portocaval shunt. World J Hepatol. 2014 May 27;6(5):358-62.*

The authors describe an eleven-year-old clinically dysmorphic and developmentally retarded male child presenting with complaints of 5 episodes of recurrent cholestatic jaundice since 3 years of age was evaluated. Imaging revealed features consistent with congenital extrahepatic portocaval shunt (Abernethy type 1b), multiple regenerative liver nodules and intrahepatic biliary radical dilatation. The presence of ductal paucity and trisomy 8 (Warkany syndrome) were identified on liver biopsy and karyotyping. In this article the authors have proposed an explanation for these unusual and previously unreported features.

Clinical Scenarios in Pediatric Liver Disease (CSPLD 2019) – 5th National Conference: 30th-31st March 2019

The Department of Pediatric Hepatology at Institute of Liver and Biliary Sciences organized its 5th national conference 'CSPLD 2019' on 30th & 31st March 2019. CSPLD was founded on the concept that medicine is best taught on the bedside. The pattern of the conference involves interesting cases presented by students, moderated by an expert who discusses the cases with audience and panelists, who are all unaware of the case and approach the case with an open mind and perspective. There were 11 such case based scenarios this year. Day 2 is usually monothematic and this year's theme was 'Biliary atresia: From pathogenesis to liver transplant'. The day had focussed case presentations raising burning issues which were then answered in intricate talks by experts in the field. Day 2 also had 5 oral presentations and posters from among the abstracts submitted by students and fellows with awards in each category. The meeting was attended by around 150 delegates from all over India and abroad and 50 faculty. There was a fair mix of basic scientists, pediatric hepatologists, surgeons, allied sciences, fellows and postgraduate students. The conference was awarded 10 credit points by Delhi Medical Council. We also released a book with a collection of the 22 most interesting and informative cases handpicked from the previous years' conferences. The book received excellent reception with immediate sellout of the 30 copies made available at the conference. The first issue of Pediatric Gut and Liver e Journal was also launcded by Prof Sarin. This is official publication of ISPGHAN and in future will be a stepping stone to print journal of the society. At ILBS, The department of Pediatric Hepatology has been running 3 years training program (DM in Pediatric Hepatology) with annual intake of 3 seats.

Compiled by : Dr Bikrant Bihari Lal
MD, PDCC, DM
Assistant Professor - Pediatric Hepatology
Institute of Liver and Biliary Sciences
New Delhi

5th Annual CME- Pediatric Gastroenterology and Hepatology and Nutrition 2019

Dear Sir/Mam,

Greetings from Jaipur,

It gives us immense pleasure to informing you that Department of Pediatrics, Surya Hospital, Jaipur, organised one day "5th Annual CME- Pediatric Gastroenterology and Hepatology and Nutrition 2019" tiin association with ISPGHAN (Indian Society of Pediatric Gastroenterology, Hepatology and Nutrition) & IAP (Indian Academy of Pediatrics) – Jaipur on 21st April 2019 – 9.30 am to 4.30 pm (Sunday).

The scientific faculty were highly skilled and experts National Faculty- Dr. B. R. Thapa, Ex Professor and Head PGI, Chandigarh, Dr. Neelam Mohan, Head, Medanta The Medicity, Gurgaon, **Dr. R. K. Gupta, J K Lon Hospital , Jaipur,** Dr. Shivani Deshwal, RML Hospital, Delhi, Dr Lalit Bharadia, SDMH, Jaipur, and many more.

Our theme for this year was Pediatric Gastroenterology and Liver diseases -tips to deal debate and differentiate. Program was made mainly to solve day to day queries for pediatricians. This year we have done 10 minute extra after each talk just for questions and answers of audience and this theme was very well appreciated by Respected faculties and audience.

We have started academics at sharp 9.30- am without a single minute delay. Topics were almost circulated to faculties and audience almost 2 month prior and both faculty and audience were ready for questions and answers respectively.

One very important point, i want to mention here that Respected B R Thapa Sir, participated even after too much significant health issues because of his commitment. He was in ICU almost for 1 month just 1-2 month before the CME. His dedication towards academics and enthusiasm was really worth watching and appreciable. All the participants were very happy and enjoyed academics of specially Dr Thapa Sir and Dr Neelam Mam.

Our all program was almost good with few shortcomings, with efforts of respected faculty and supports of our Jaipur ISPGHAN team and PED GASTRO CME Organizing team of our Surya Hospital,Jaipur.

Regards and Thanks

Yours Sincerely,

Dr. Natwar Parwal,

Organizing Secretary, Ped Gastro CME

Department of Pediatrics, SURYA Hospital, Jaipur, Mobile: 09610951425,

Email: dr.nats@yahoo.com

Indian Society of Pediatric Gastroenterology, Hepatology and Nutrition (ISPGHAN)
(Registered under Tamil Naidu societies registration act, 1975, 81 No-361of 2013)

Application Form for Membership

Kindly enroll me as a Life Member/ Associate Life Member/Affiliate Foreign Member of the Indian Society of Pediatric Gastroenterology, Hepatology and Nutrition. Eligibility of member category is given in this form*(see page 2).

1. Name (in full in capitals):.....
2. Qualifications:.....
3. Designation:.....
4. Address with pincode(for communication):.....
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5. Field of medicine connected with Pediatric Gastroenterology
(Specify here specialty such as Surgery, Pathology, Radiology, Psychiatry etc.)

6. Attachment to the Hospitals:.....
7. Modes of Payment: Either by NEFT (preferred) or by multicity cheque
a) NEFT transfer to Account name: "ISPGHAN", Account No: 048201000027026,
IFSC: IOBA0000482, MICR: 600020032, India Overseas Bank, Mahalingapuram Branch, Chennai
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(**To be completed by the person(s) proposing and seconding the membership of the application**)

To the best of our knowledge and belief the overleaf particulars of
Dr..... Place.....are correct.
We consider him/ her fit and proper person to be admitted as a Life Member/ Associate Life Member/Affiliate Foreign Member of the Indian Society of Pediatric Gastroenterology, Hepatology and Nutrition.

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Complete Registration form with Cheque/NEFT receipt should be sent by post to :
Dr Anshu Srivastava, Secretary, ISPGHAN
Professor, Department of Pediatric Gastroenterology,
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Email: ispghansec@gmail.com

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To be completed by the Executive Committee of the Indian Society of Pediatric Gastroenterology, Hepatology and Nutrition (ISPGHAN) ISPGHAN Registration Number allotted:			
• Admitted as Life Member/ Associate Life Member/Affiliate Foreign member of the Society • Application rejected for the above reasons (Delete clause which is not applicable)			
Place : Date :		Signature Designation	
Membership Fee paid:			
Life Member (Indian) Associate Life Member Affiliate Foreign Member		Rs.3000.00 Rs.2000.00 US $ 100.00	

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Membership Criteria

Membership Categories	*Eligibility criteria	Current Membership fee
*Life Member	Fresh/New Life Membership of ISPGHAN shall be open to members of the medical profession, who are residents in India and who have a postgraduate degree in Pediatircs (MD, DNB), Gastroenterology or Pediatric gastroenterology from India or abroad, recognized by the Medical Council of India, and interested or involved in the practice of Pedaitric gastroenterology, hepatology and nutrition.	Indian rupees 3000
*Associate Life Member	Associate Life Membership shall be open to members of the medical profession who are Diploma holders in Pedaitric, Postgraduate students in Pedaitric, gastroenterology or pediatric gastroenterology as well as to postgraduates in other medical disciplines (recognized by the competent authorities in India), who are interested or involved in the practice of pediatric gastroenterology, hepatology and nutrition.	Indian rupees 2000
Affiliate Foreign Member	Affiliate Foreign Membership shall be open to members of the medical profession who are not ordinarily residents of India, and have a postgraduate degree in Pedaitric, Pediatrics gastroenterology or Gastroenterology recognized in their respective country of residence	US dollar 100

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National Speakers

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Dr B R Thapa
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