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Case Report

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Mass Abdomen in an Infant: A Pandora Box

Kiran B.[1], Geetha M.[2]

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Abstract

Background: Infantile hepatic hemangioendothelioma/hemangioma (IHH) is the most common type of benign tumor of Liver, contributing to 12% of all childhood liver tumors. (1) It is a neonate's common hepatic mesenchymal tumor, 90% being less than six months old. (1) The tumor is slightly more predominant in females. It may be asymptomatic or can cause serious complications like cardiac failure or coagulopathy. There is no treatment protocol established yet and increasing number of cases have been reported. There is a varied treatment options like pharmacological or interventional on the basis of the presentation. IHH is classified as a benign vascular tumor with spontaneous regression in some patients.

Case presentation: Here is a 6 month old infant presented with Infantile colic and incidentally diagnosed to have IHH. Imaging like Ultrasonography and MRI confirmed the diagnosis. The child had an asymptomatic presentation and regressed with follow up.

Conclusion: Increasing awareness about the condition needed for proper follow up. Early identification and prompt treatment is necessary to avoid fatal complications.

Keywords: Mass abdomen, Infancy, Vascular, Liver.

INTRODUCTION

Infantile hemangio endothelioma/haemangioma (IHH) is a rare benign vascular tumor of the liver. [(1)] IHH is the third most common hepatic tumor in children (12% of all Childhood hepatic tumors). It is the most common benign vascular tumor of the liver in infancy and the most common symptomatic liver tumor during the first six months of life.[(2,3)]

The tumor has slightly more female predominance.[(4)] The lesions may be single or multiple and have calcification on 50% of cases on histopathological analysis.

In most of the cases, IHH remains asymptomatic and detected in ultrasound of abdomen incidentally. But sometimes, it may cause abdominal pain, hepatomegaly, severe arteriosclerosis venous shunting with CCF, anaemia, thrombocytopenia (Kasabach Meritt syndrome), consumptive coagulopathy, Intraabdominal haemorrhage. Rarely, biliary obstruction with Jaundice, vomiting and gastric outlet obstruction has been reported.[(4)]

We report 1.5 month old child with Infantile Hepatic Haemangioendothelioma which was accidentally detected on ultrasound abdomen while evaluating for colic.

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CASE REPORT

1.5-month-old male infant, first born to non consanguineous parents, had uneventful Antenatal and postnatal period in our hospital, came with history of excessive crying and distention of abdomen noticed by parents for the past 3 days. After detailed history taking and thorough clinical examination, child was treated with anti colic medication. Child had persistent symptoms and was subjected to imaging studies. Ultrasound abdomen showed small well defined oval hypo echoic lesion in liver measuring 20*18*8.7mm in left lobe of liver (segment II) showing peripheral vascular rind with a small echogenic focus of 2mm, suggestive of high flow vascular neoplasm, hemangioendothelioma. CT Abdomen confirmed the findings. Child was referred to paediatric surgeon and was advised to follow up with annual ultrasound abdomen.

Child is asymptomatic with normal Growth, developmentally appropriate and vaccinated upto date and on regular follow up. Recent Ultrasound abdomen showed small hypo echoic focal lesion in the left lobe of Liver in subcapsular area having peripheral vascularity suggestive of high flow vascular neoplasm- Haemangioendothelioma.

DISCUSSION

Infantile hepatic hemangioendothelioma (IHH)is the most common vascular tumor of liver in children constituting 12% of all childhood hepatic tumors. About 85% become symptomatic in first 6 months of life and it is the most common symptomatic tumor. There is a female preponderance with a ratio of 1.3 to 2:1.[7]

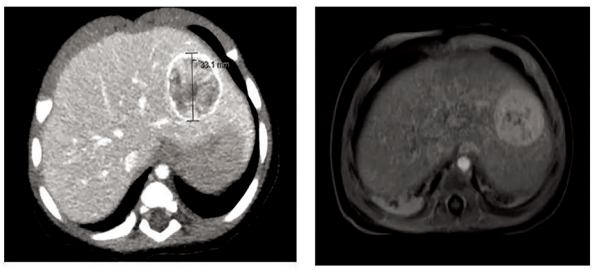
The most common presenting symptom is abdominal mass. Other presentations include hepatomegaly, high-output cardiac failure, skin hemangioma, thrombocytopenia or peritoneal bleeding. Asymptomatic lesions undergo spontaneous regression within a year, symptomatic lesions may cause heart failure or death.

Serum Alpha fetoprotein is an important tumor marker for the diagnosis and prognostication of pediatric hepatic masses. It may be used for prenatal diagnosis. It may be diagnosed in ultrasonogram.

This tumor can be incidentally found in the fetus during antenatal ultrasound. It can also be found in children who underwent abdominal computed tomography for other indications. Other presentations may be coagulopathy, heart failure or jaundice.[6,7]

IHH is divided into two types according to histology.[8] Type I IHH is composed of vascular channels lined with benign endothelial cells displaying small nuclei. Type II IHH demonstrates vascular channels with pleomorphic endothelial cells with hyperchromatic nuclei and mitosis. Type II IHH was once classified as a high grade tumor due to its aggressive histopathological characteristics than type I.[8]

Radiological evaluation is useful for patient diagnosis with sonography often being the initial diagnostic modality. On sonography, IHH is characterised by discrete, hypo echoic lesions (either solitary or multiple) within the liver that may have calcification or shunting on Doppler evaluation. A more definitive diagnosis requires a contrast enhanced CT (CECT) or a magnetic resonance imaging (MRI). While CECT shows a hypodense area which enhances with contrast. MRI may identify IHH as a low signal lesion on T1 and high signal lesion on T2 weighted images. Most cases of IHH are asymptomatic with spontaneous regression. But cases with heart failure experience a higher mortality rate (upto 70%).[5,8] (Fig. 1)



(A) (B)

Fig. 1: Showing CT/MRI image showing enhanced localised tumor seen within liver

Courtesy (image): Diagnostics 2021, 11(2), 333; https://doi.org/10.3390/diagnostics11020333

The differential diagnosis of Infantile Hepatic Haemangioendotheliomaare cavernous haemangioma, angiosarcoma, hepatoblastoma and mesenchymal hematoma.[9]

CONCLUSION

Infantile hepatic haemangioendothelioma may be an incidental diagnosis. They may be asymptomatic or symptomatic. Not all children will be symptomatic. The diagnosis is based on the symptoms and operability. Awareness is important for prompt diagnosis and management.

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Diagnostic Dilemma in Diagnosis of Annular Pancreas: A Case Report

R. Subha[1], Rajeev Kumar Thapar[2], Ravi Kale[3]

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Abstract

Background: Annular pancreas (AP) is a congenital disorder of the pancreas, thought to result from the malrotation of the ventral pancreatic bud. This causes the duodenum to be encircled by the pancreatic tissue, leading to duodenal obstruction. The management of annular pancreas is usually surgical.

Clinical Description: A 10-month-old male infant presented with regurgitation of yellowish feeds for the past few months, with no other positive clinical findings. Radiological investigations revealed an obstruction after the first part of the duodenum. A barium swallow showed duodenal stenosis. The patient underwent surgery, and the diagnosis of annular pancreas was confirmed.

Management and Outcome: After clinical and radiological diagnosis, the child was managed surgically and underwent duodenojejunostomy. Postoperatively, the patient remained clinically stable and is symptom-free.

Conclusion: Annular pancreas is a rare but significant cause of duodenal obstruction, which can present from the neonatal period to adulthood. The patient may experience bilious or non-bilious vomiting. The definitive diagnosis is made via laparotomy. Surgical management is required, with duodenojejunostomy being the best treatment option.

Keywords: Annular pancreas, Duodenal obstruction, Regurgitation, Stenosis.

INTRODUCTION

Annular pancreas is a congenital disorder of the pancreas, first identified in 1818 by Tieddmann[(1)]. The endodermal lining of the foregut (duodenum) gives rise to two buds: the dorsal and ventral buds, which fuse together after the rotation of the ventral bud to form the pancreas. Annular pancreas is thought to result from the malrotation of the ventral pancreatic bud. It is a type of pancreatic fusion anomaly.[(2)]

There are multiple theories explaining the formation of annular pancreas: failure of the ventral bud to atrophy; the ventral bud adhering to the ventral mesogastrium; or the defect being in the duodenum, with the pancreas filling those defects. Any of these can lead to the second part of the duodenum being encircled by the pancreas, resulting in duodenal obstruction.[(3)]

The most common cause of duodenal obstruction, aside from annular pancreas, in infants is atresia. AP

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is associated with other congenital anomalies, such as Down's syndrome, duodenal atresia, and cardiac anomalies. The child present with typical bilious vomiting due to duodenal obstruction. Diagnosis is made by the presence of the double-bubble sign on X-ray. The definitive diagnosis can be made intraoperatively. When diagnosed before birth, the most frequent presentation is polyhydramnios due to duodenal obstruction. Annular pancreas presenting in childhood tends to be severe.[4]

Clinical Description

A 10-month-old male infant presented with regurgitation of feeds for the past few months. According to the mother, the regurgitant feeds were yellow in color and stained the child's clothes. The child was hemodynamically stable and appropriate for age in terms of growth and development.

Clinically, the abdomen was not distended, with no visible veins or scars, but visible peristalsis was observed in the right upper quadrant, moving from left to right. On palpation, the abdomen was soft, non-tender, and without organomegaly. Normal bowel sounds were present.

An erect abdominal X-ray suggested duodenal obstruction (Fig. 1). Ultrasound of the abdomen revealed a dilated stomach and first part of the duodenum, with normal wall thickness and peristalsis in the duodenum, indicating an obstruction after the first part of the duodenum.

A barium swallow showed a dilated duodenal bulb, and the second and third parts of the duodenum Barium swallow showed dilated duodenal bulb, second and third part of duodenum and presence of contrast in the jejunum which was suggestive of duodenal web/stenosis (Fig. 2a & b).

Management and outcome

Radiological investigations indicated duodenal obstruction, with the barium swallow pointing toward duodenal web/stenosis. The child underwent surgery, and intra operative findings revealed a dilated duodenum up to the third part, while the distal duodenum appeared normal with collapsed jejunal loops. The Ryles tube was able to pass into the jejunum. Annular pancreas was observed encircling the duodenum. Duodenojejunostomy was performed. The postoperative period was uneventful, and the child is doing well on follow-up

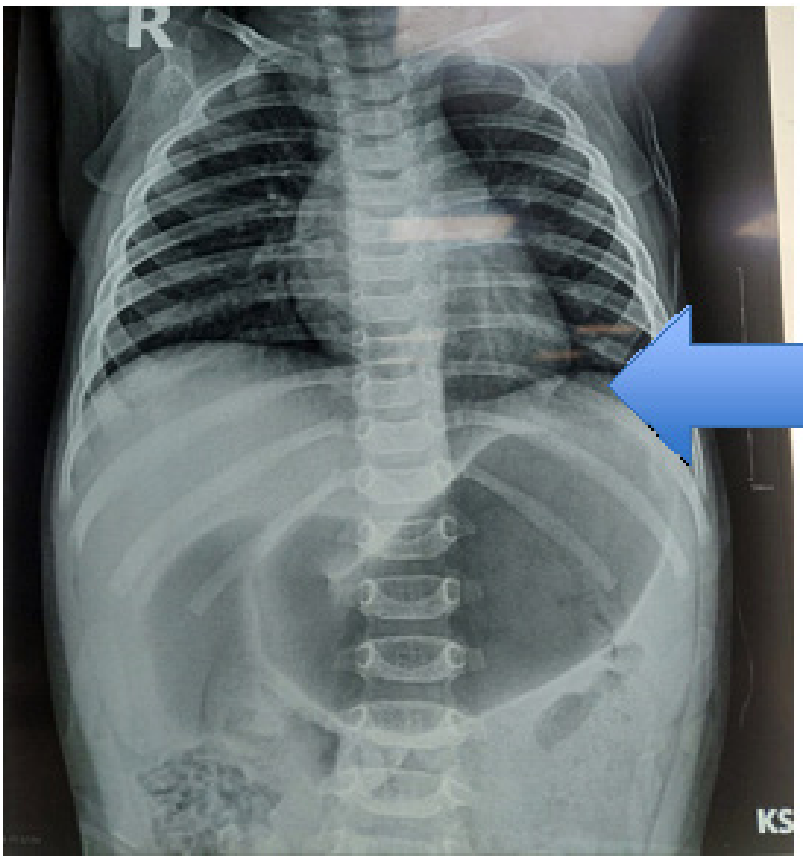

Fig. 1: X-ray erect showing dilated stomach and duodenum

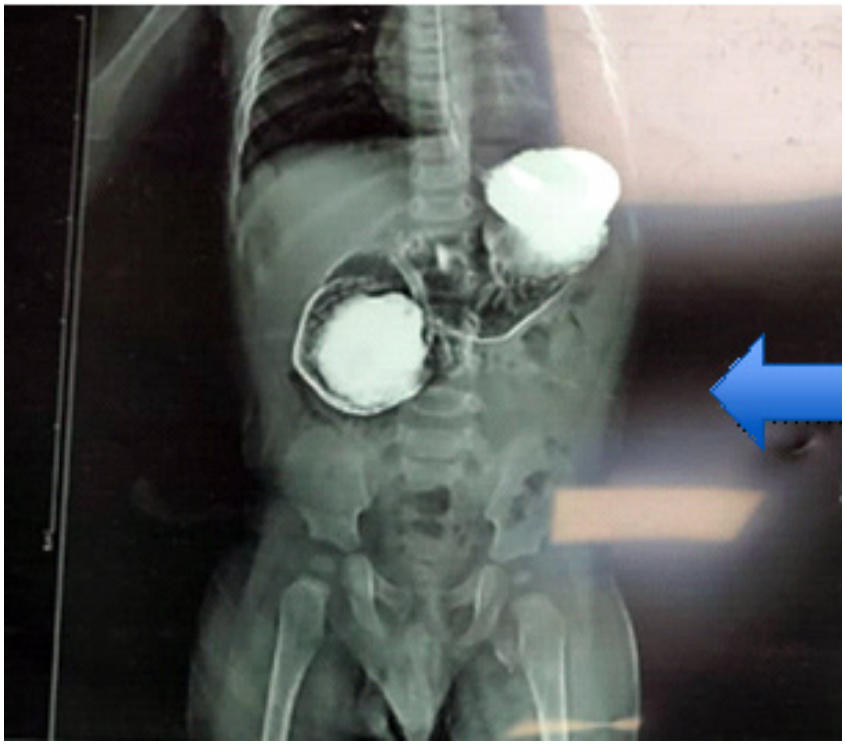

Fig. 2(a): Barium swallow showing dilatation of duodenum

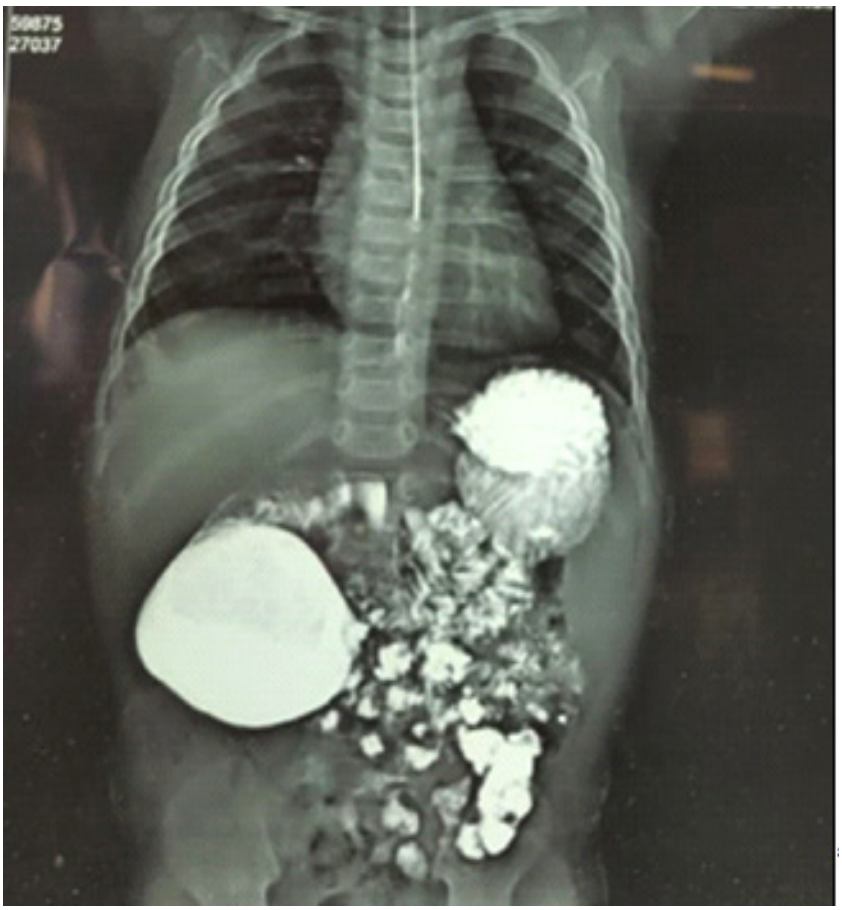

Fig. 2(b): Delayed film showing presence of dye distally

DISCUSSION

The incidence of annular pancreas (AP) is very low, occurring in approximately 1 in every 20,000 births. The clinical presentation varies based on the extent of the obstruction, with vomiting being the most common symptom in 59-94% of cases, primarily of a biliary nature. Some patients with annular pancreas may remain asymptomatic and only be discovered incidentally during imaging, surgery, or autopsy[5]. The diagnosis of duodenal obstruction is typically based on clinical symptoms and the detection of the "double bubble" sign on a plain abdominal X-ray, which appears in 90.9-100% of cases. It can be further confirmed with a gastroduodenal series that shows obstruction in the duodenum. Some also suggest the use of computed tomography (CT) and magnetic resonance imaging (MRI). However, the gold standard for diagnosis remains a thorough examination of the duodenum and pancreatic head during surgery. Sencan et al, reviewed seven cases of annular pancreas and found that most frequent abnormalities linked to duodenal atresia or stenosis is annular pancreas, associated with Down syndrome and congenital heart disease. [6] Although the pancreatic tissue surrounding the duodenum in AP may cause extrinsic compression leading to partial obstruction, the true cause of the blockage is often a duodenal atresia or stenotic web beneath the annular pancreas. Traditionally, a plain radiograph has been used to detect congenital duodenal blockage, and a contrast scan is usually not necessary, except in cases of midgut volvulus that mimic duodenal atresia or stenotic web. Management is surgical, and the conventional approach is bypassing the obstructed duodenum through duodenoduodenostomy[7].

Many recent studies report successful results with laparoscopic approaches[8,9]

CONCLUSION

We successfully diagnosed and treated an annular pancreas in a 10-month-old who presented with partial duodenal obstruction without a definitive diagnosis. A barium study helped delineate the duodenal obstruction, and duodenojejunostomy resolved the patient's symptoms. Annular pancreas, though rare in childhood, should be considered in patients with unresolved obstructive symptoms.

Conflict of interest: NIL

Funding: NIL

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Case Report

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Apert's Syndrome: A Rare Case Report

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Abstract

Background: Apert syndrome is a genetic disorder inherited in an autosomal dominant manner, with an occurrence rate of about 15 cases per 100,000 live births. It is caused by a mutation in the fibroblast growth factor receptor-2 (FGFR-2) gene located on chromosome 10q26. The condition mainly impacts the first and second branchial arches, leading to the early closure of cranial sutures (craniosynostosis) with fusion of fingers and toes of the hands and feet. Apert syndrome is rare in India, and a case report is presented.

Clinical Description: 21 months female toddler presented with global developmental delay along with distinctive craniofacial features. Clinically toddler exhibited an abnormal head shape and contour, characterized by turribrachycephaly, a depressed nasal bridge, frontal bossing, midface hypoplasia, and a characteristic "crossbow" appearance of the upper lip. Limb examination revealed symmetrical soft tissue syndactyly affecting all digits.

Management & Outcome: This case is notable for its rarity and the similarity of its features to other craniosynostosis syndromes, such as Crouzon and Pfeiffer syndromes, posing a diagnostic challenge. Therefore, genetic counselling for the family was recommended, along with early intervention for the child, including plastic surgery for the affected limbs.

Conclusion: Acrocephalosyndactyly is an autosomal dominant condition seen rarely, marked by craniosynostosis, craniofacial deformities, and pronounced symmetrical clubbing of fingers and toes of the hands and feet. In the majority of Apert syndrome cases, the condition occurs sporadically, often due to new mutations in the relevant gene.

Keywords: Acrocephalosyndactyly, Craniosynostosis, Midface hypoplasia.

INTRODUCTION

Apert syndrome (AS) was first described in 1894 by Wheaton, with the name later derived from a series of works by French physician Eugène Apert, who reported nine such cases. In 1906, Apert formally described the syndrome as acrocephalosyndactyly. The incidence is approximately 15 cases per 1,000,000 live births[1].

AS is a rare autosomal dominant disorder caused by a mutation in the fibroblast growth factor receptor-2 (FGFR-2) gene on chromosome 10q26. This mutation affects the FGFR-2 expression

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by suture progenitor cells, leading to a disruption in signalling that impairs the production of the fibrous material needed for normal cranial suture formation[2].

Classified as a branchial arch syndrome, AS affects the first and second branchial arches. The hallmark of the condition is the premature closure of cranial sutures (craniosynostosis), which restricts cranial growth and results in craniofacial abnormalities. Common features include frontal bossing, a narrow high-arched palate, midfacial hypoplasia, and symmetrical clubbing of fingers and toes of the hands and feet, with short, broad fused nails. While intellectual disability is often present, the exact incidence remains unclear[2,3].

AS is seen less commonly in India and one such case is reported.

Clinical Description:

A 21 months old female toddler presented with global development delay with distinctive facial features. Toddler had unusual craniofacial appearance, prompting a more detailed evaluation which led to a clinical diagnosis of Apert syndrome. A thorough examination was subsequently conducted.

The toddler was the first Caesarean section born daughter to non-consanguineous parents. Her birth history was uneventful, with no known exposure to infections, drugs, or radiation during her mother's pregnancy. There was no family history of similar malformations on either side.

On examination (Fig. 1), the child had atypical skull shape and contour, consistent with turribrachycephaly, along with a depressed nasal bridge, frontal bossing, midfacial hypoplasia, and a characteristic "crossbow" shape of the upper lip. The mouth was trapezoidal in shape, with a cephalometric dolichofacial pattern and proptosis.

Examination of the upper limbs (Fig. 2) revealed symmetric soft tissue clubbing of all digits, which almost fused into a single unit, with a single fused nail looking like a spoon-like deformity. The lower limbs also exhibited symmetrical clubbing of all toes, with a single broad fused nail. There was delayed developmental with Developmental Quotient (DQ) of 60%. No other systemic abnormalities were observed clinically.

Given the characteristic features, a clinical possibility of Apert syndrome was considered. Although AS usually has an autosomal dominant (AD) inheritance pattern, both parents were unaffected, suggesting a possibility of a sporadic mutation or gonadal mosaicism. The parents were advised to undergo further genetic testing for confirmation.

Fig. 1: Abnormal shape and contour of the head

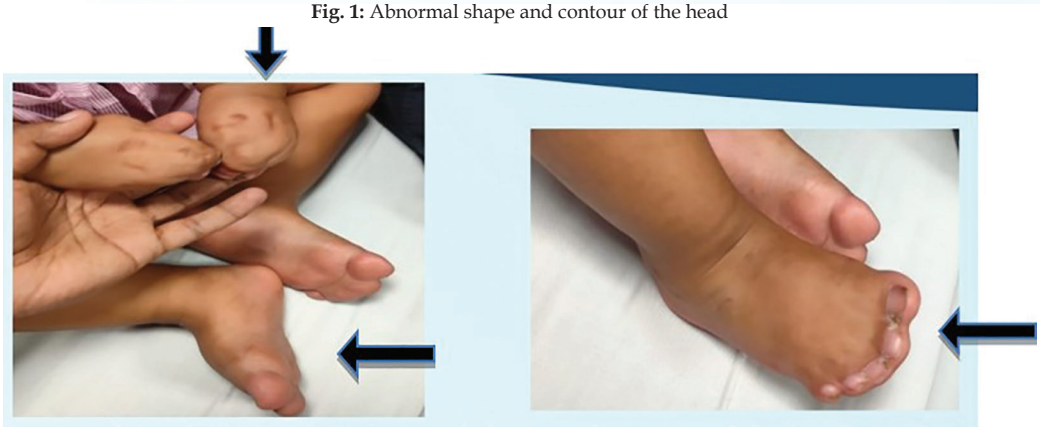

Fig. 2: Limbs showing symmetric soft tissue syndactyly of all digits

Management and Outcome:

Apart from being seen less commonly, AS has resemblance of its features to other craniosynostosis syndromes, such as Crouzon syndrome and Pfeiffer syndrome, thus pose a diagnostic challenge[2].

Management typically involves conventional surgical procedures, such as Le-Fort III osteotomyfor midface advancement. Early intervention includes optimizing hearing, potentially with hearing aids, airway management, psychological counselling, and speech therapy[3,4].

Genetic counselling was recommended for the family, along with early intervention for the child, which includes plastic surgery to address the limb abnormalities[5].

DISCUSSION

Apert syndrome(AS) is a form of craniosynostosis and is characterised by premature craniosynostosis, hypertelorism, clubbing of hands and feet, Inheritance is generally autosomal dominant but may develops due to a mutation of fibroblast growth factor receptor -2 gene (FGFR2)located on 10q26 gene locus. FGFR2 gene enables coding of a protein required for suture closure. Two different types of mutations have been demonstrated in the binding site. Most of the patients have a normal karyotype[1].

Paternal mutations may be seen though most of the cases are sporadic, and develop because of new mutations. Incidence varies from 9.9 to 15.5 per one million live births with no sex variation.[1]

Phenotypic manifestations can be explained by premature closure of cranial sutures. Premature fusion of coronal sutures causes shorter anteroposterior diameter, high, and prominent forehead. The most prominent symptoms of this syndrome are clubbing of hands, and feet.[1,2]

This toddler had the clinical features of atypical skull shape namely acrocephaly, clubbing of fingers, and toes with Mid-face hypoplasia. Eye examination revealed hypertelorism, proptosis, and downslanting palpebral fissures. Nasal root was short and widened. This patient had all specific facial characteristics.[1]

AS children can have increased occurrence of upper respiratory tract infections, sleep apnoea and malnutrition. Respiratory difficulty can be severe requiring endotracheal intubation or tracheostomy. In patients with AS, mental disorders are rarely seen[1,2].

CONCLUSION

Acrocephalosyndactyly, is aless common inherited (autosomal dominant) disorder, characterized by craniosynostosis, craniofacial anomalies, and severe, symmetrical clubbing of the hands and feet. While many cases of AS have sporadic origin and result from de novo mutations in the FGFR2 gene, however the genetic basis remains crucial for diagnosis[1,2].

Genetic testing and counselling are essential for each diagnosed case. Advances in prenatal diagnostic techniques have made it possible to detect the condition early, enabling timely, multidisciplinary intervention. This early approach significantly improves the quality of life for affected individuals[5].

This case report aims to throw light on this rare and complex syndrome.

Lessons learnt

1. Apert syndrome is a rare inherited (autosomal dominant) disorder marked by craniosynostosis, craniofacial deformities, and severe, symmetrical clubbing of both the hands and feet.
2. The majority of Apert syndrome cases are sporadic, often arising from de novo mutations in the relevant gene.
3. Genetic counselling for the family was recommended, along with early intervention for the child, which includes plastic surgery for the facial and limb abnormalities.

Financial Conflict: Nil

Conflict of Interest: Nil

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Case Report

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Hyper Eosinophilia with Hereditary Angioedema (HAE): Case Report

Menaka J., Malarvizhi G.

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Abstract

Background: Hyperesinophilia (HES)with Hereditary angioedema (HAE), is a rare disease characterized by peripheral blood eosinophilia > 1500/mcl (> 1.5 × 10^9/L) persisting ≥ 6 months and the presence of end-organ damage. Hereditary angioedema is a lifelong illness characterized by recurrent swelling of the skin, intestinal tract, and, the upper airway. It results from insufficient activity of the C1-inhibitor protein, leading to disturbances in the kallikrein/bradykinin pathway.

Case presentation: We reviewed a case of Hypereosinophilia with hereditary angioedema (HAE) in 7-year-old boy who was referred from another hospital in Coimbatore for further management. He had elevated absolute eosinophilic count & elevated IgE. Bone marrow aspiration and biopsy were done which showed increased eosinophil. Stool for ova/ cyst was negative. He was initiated on oral prednisolone (10mg), pantaprozole (20mg) & dexamethasone (50mg).

Conclusion: Hereditary angioedema (HAE) is a genetic condition that poses a threat to life; this condition necessitates rapid diagnosis and treatment to control acute attacks and avert potentially lethal complications, especially when swelling impacts the airway, and can despite recent developments in treatment options, a child quality of life can be significantly affected.

Keywords: Hyperesinophilia, Hereditary angioedema, Protein C1-inhibitor, Kallikrein/ bradykinin, Bone marrow, Biopsy.

INTRODUCTION

Eosinophils are derived from myeloid progenitors in the bone marrow, through the action of three hematopoietic cytokines: granulocyte macrophage colony-stimulating factor (GM-CSF), interleukin-3 (IL-3), and interleukin-5 (IL-5). Of these three, only IL-5 is specific for eosinophil differentiation. The main functions of Eosinophils include host defence, inflammation modulation, and tissue destruction.[1]

Recent developments in the understanding of the underlying pathogenesis have demonstrated that hyper eosinophilia may arise from either the primary involvement of myeloid cells or the occurrence

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of an interstitial chromosomal deletion on 4q12, which is result from the *FIP1L1-PDGFRA* fusion gene (F/P^+ variant) or increased interleukin (IL)-5 production by a clonally expanded T cell population (lymphocytic variant), most frequently characterized by a $CD3^-CD4^+$ phenotype.[2]

The increased number of eosinophil's inflames tissues and causes organ damage. The heart, lungs, skin, oesophagus, and nervous system are most often affected, but any organ can be sustaining damage. Eosinophilic pneumonia arises as a consequence of lung tissue damage caused by activated eosinophil's. The substances and chemical mediators like cytokines, Leukotriene & Toxic granule product are released by activated macrophages, damage the tissues and contribute to the disease pathology[3]. The most prevalent type of hereditary angioedema (HAE) results from a lack of C1 esterase inhibitor (C1-INH-HAE), though HAE can also manifest with normal C1-INH levels. Angioedema is characterized by self-limiting tissue swelling resulting from intermittent increases in vascular permeability, which are triggered by the release of bradykinin (BK) and/or other cell–derived mediators. Typically, the recurring swellings are limited to the skin and/or the upper respiratory, gastrointestinal, and genitourinary systems.[4] (Fig. 1)

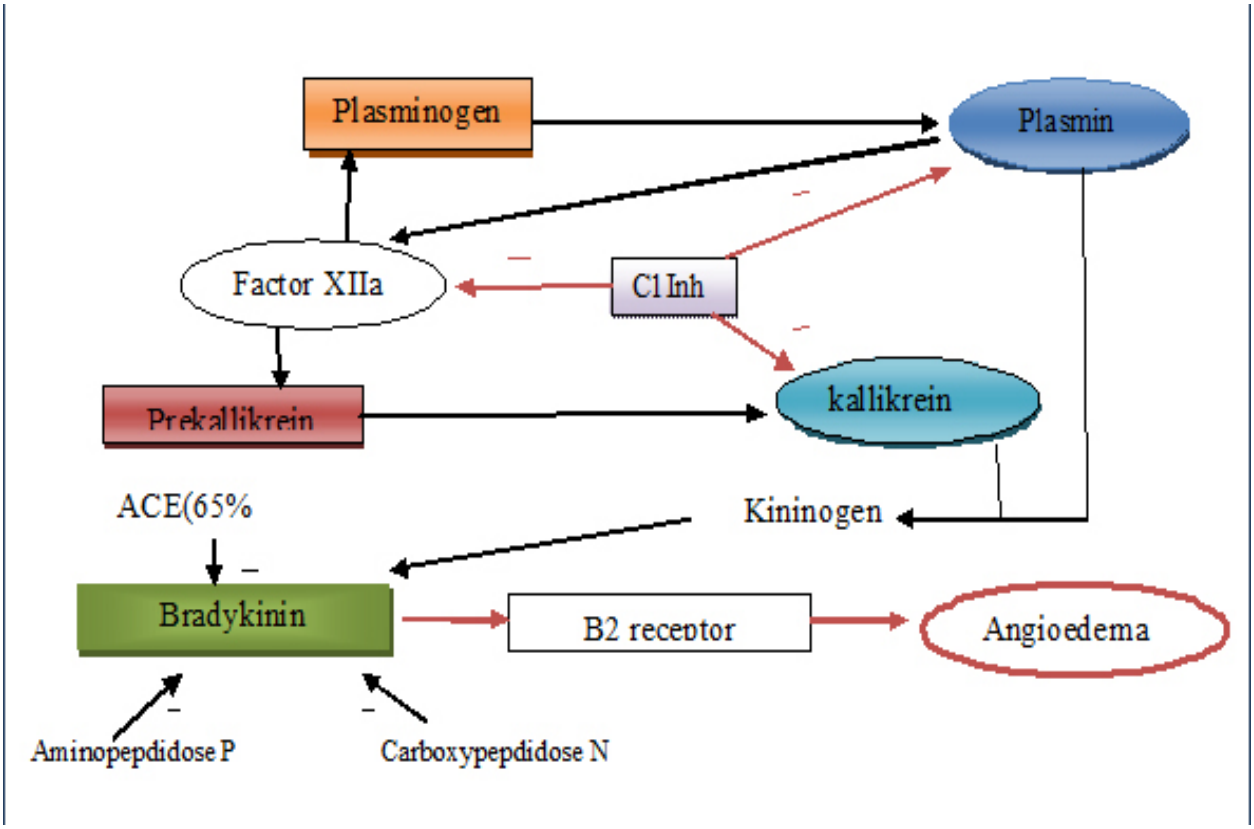

Fig. 1: Shows HAE Mechanism

CASE REPORT

A 7 year old boy admitted in paediatric medical ward and presented with the complaints of rashes on face, hand and headache, giddiness for 4 days constipation for 3 days. After admission the detailed history was collected. There was no family history of atopy or eosinophilia.

He was apparently normal till three years of age. He had a history of recurrent soft tissue swelling, migratory in nature with a symmetric involvement of lower limb, scrotum, abdomen & trunk, he was admitted in medical ward. The laboratory investigation showed that his Absolute eosinophilic count was 5800(cells/mcl) *(fig. 2), (Table 1)* He was diagnosed with hyper eosinophilic syndrome with cutaneous involvement (eosinophilic fasciitis / cellulitis) and was noted to have spontaneous resolution of swelling with fall in eosinophil count without any specific treatment. He was advised to do biopsy in case of recurrent swelling but lost to follow up in view of Covid pandemic and treated on OPD basis.

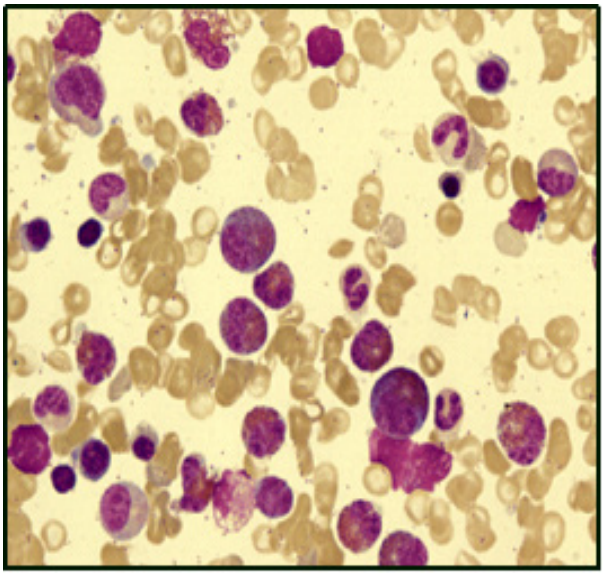

Fig. 2: Shows increased eosinophil count in blood

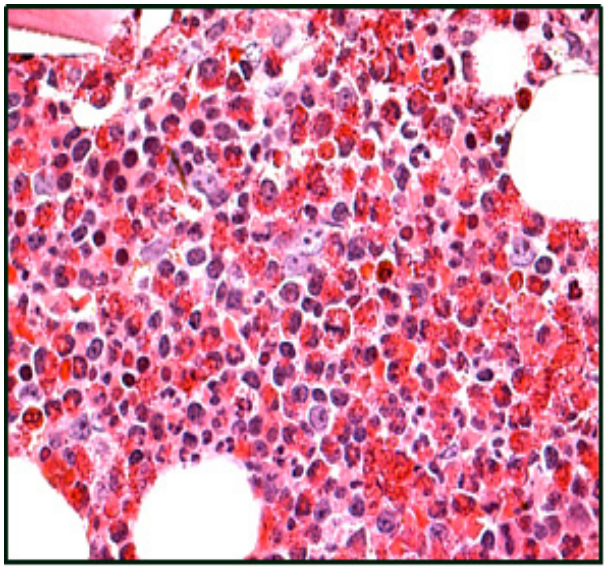

Fig. 3: Bone marrow aspiration and biopsy were done which showed increased eosinophil count

Table 1: Shows complete blood test value in each hospital admission

Complete Blood Cell Count	Reference value	First Visit	Second Visit	Third Visit
Absolute Esinophil Count	80-360 µl	510µl	362 µl	946 µl
Hemoglobin	10.7-14.7g/dl	12.5g/dl	12.61g/dl	12.8g/dl
PCV	33-45%	36.2%	37.6%	39.4%
Total Wbc Count	50014500Cells/cumms	6400Cells/cumms	9040cells/cumms	8600cells/cumms
Poly Morphs	45-75%	28%	42.19%	26.%
Lymphocytes	20-40%	63%	43.35%	62%
Monocytes	2-10%	1.0%	9.01%	1.0%
Eosinophils	2-6%	8%	6.2%	11%
Basophils	0.0-1.0%	0.0%	0.25%	0.0%
Total Rbc Count	3.7-5.7 mil/microliter	5.01 mil/microliter	4.82 mil/microliter	5.45 mil/microliter
MCH	23-31 pg/cell	24.94pg/cell	26.24pg/cell	23.4pg/cell
MCHC	32-36 g/dl	34.4 g/dl	33.5 g/dl	32.4 g/dl
MCV	72-88 μm^3	72.4 μm^3	78.1 μm^3	72.2 μm^3
RDW	11.5-14.5%	17.4%	16.4%	15.1%
Platelet Count	150000-521000 cells/µl	240000 cells/µl	239900 cells/µl	312000 cells/µl
Neutophil	1-2.5	0.4	0.95	0.5

Table 2: Shows immunoglobulin value

Biochemistry	Child Value	Reference Value
Immunoglobulins IGA	240	41 to 297mg/dl
Immunoglobulins IGE	13760	Upto 90IU/dl
Immunoglobulins IGG	1873	600 to 1300 mg/dl
Immunoglobulins IGM	65	40 to 160mg/dl

He developed sharp chest pain on right side for 3 weeks, moderate and intermittent fever lasted for 1 week and again admitted in pediatric medical ward. Child was evaluated by pediatrician, Hematologist, pathologist, dermatologist, ophthalmologist. On physical examination the child was having left side strabismus.

At admission the child had absolute eosinophil count of around 1500 (cells/mcl). Bone marrow

aspiration and biopsy were done which showed increased eosinophil and no abnormal cells. Subsequent complete blood count done showed falls in eosinophil count. Stool for ova/cyst was negative. Immunoglobulin profile done showed elevated IgE and mildly elevated IgG *(Fig. 3 & Table 2)* ANA (antinuclear antibody test) was negative. USG & CT Thorax was suggestive of moderate pleural effusion on right side & gallbladder sludge.

Diagnostic pleural tap was done and pleural fluid analysis was suggestive of exudative effusion with 82 percentage eosinophil. The child was diagnosed with Eosinophilic pneumonia and work up for TB Negative, Mild positive for C-ANCA (Anti neutrophil cytoplasmic antibody). Workup to rule out hereditary is done. C4 level are normal and C1 esterase level is high.

During hospital stay child developed high grade fever spikes followed by vesiculopapular skin rash on hand and feet and oral ulcers. He was initiated on oral prednisolone (10mg), pantoprazole (20mg). Then he was also empirically started on tablet dexamethasone (50mg). Simultaneously parental counselling was conducted.

DISCUSSION

HES is a rare, diverse illness that can cause multiple tissue and organ damage in addition to an inexplicable persistent eosinophilia. It is divided into three categories: idiopathic, primary (clonal), and secondary (reactive) HES. HES is most frequently caused by infections and drug reactions.[5]

In this case review the child presented with primary symptoms of rashes on face, hand and recurrent soft tissue swelling of lower limb, scrotum, abdomen & trunk followed by pulmonary and gastrointestinal involvement. Hyper-IgE syndrome was suspected in child because of the very high IgE level. C4 level are normal and C1 esterase level is high.

This case report is similar with a case of 10 yrs old boy admitted in the medical ward Aarhus University Hospital, Aarhus N, Denmark, the main symptoms exhibited by the child were a severe itching and a widespread papulonodular skin rash. The child did not exhibit a heightened vulnerability to skin infections or occurrences of invasive infections. No skeletal abnormalities were present, nor were there any characteristic facial features associated with hyper-IgE syndrome.[5]

The Goal of HES Management is to lower eosinophil levels in the blood and tissues, thereby preventing tissue damage–especially in the heart. Standard HES treatment includes glucocorticosteroid medications such as prednisone, and chemotherapeutic agents such as hydroxyurea, chlorambucil and vincristine. Interferon-alpha may also be used as a treatment. This drug requires administration through frequent injections.[6]

This case, Initially the child was noted to have spontaneous resolution of swelling with fall in eosinophil count without any specific treatment and they failed to follow up the treatment due to Covid pandemic. After which experienced recurring symptoms, which led to hospitalization. Following the administration of steroid medication Prednisolone 20 mg/day & T. Dexamethasone 50 mg, peripheral blood hyper eosinophilia reverted to a low level.

CONCLUSION

Early identification and intervention for children with HES may be made easier by regular follow up. Steroid management and early detection are crucial to avert major complications. Sensitizing the parents and child about prevention of infection is imperative.

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Funding: No

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Case Report

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Colonic Atresia with Rectal Atresia: The Management of a Rare Presentation: A Case Report

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Abstract

Obstructions in the lower intestinal tract, specifically colonic and rectal atresias, are distinct conditions that share similarities but necessitate different surgical interventions. Most of our understanding regarding these malformations is derived from isolated Case Report and a limited number of comprehensive investigations. Rectal atresia is a rare congenital anomaly characterized by a normal anus but an obstructed rectum. Its co-occurrence with other digestive system abnormalities, particularly colonic atresia, is even less frequent. We present an unusual case in which rectal atresia is observed in conjunction with colonic atresia, which was not detected during the initial surgical procedure. Contrast enema revealed only 2 cm of the anal canal from the distal end. The infant underwent surgical intervention for rectal atresia via the posterior sagittal approach. An end-to-end anastomosis was performed following resection of the atretic segment. Postoperatively, the patient was maintained on a regimen of regular Hegar's dilatation. Rectal atresia with colonic atresia is an exceedingly rare anomaly with very few cases reported in the literature. Awareness of such coexistence may minimize further complications.

Keywords: High divided sigmoid colostomy, Transverse loop colostomy, Colonic stricture.

INTRODUCTION

Neonatal intestinal obstruction can be caused by rectal atresia (RA), a highly uncommon condition that makes up 1% of all anorectal malformations (ARM)[1]. RA is anatomically characterized by a normal anal canal and a well-formed anal sphincter[1,2]. Another infrequent cause of neonatal intestinal obstruction is colonic atresia. This abnormality represents less than 10% of all intestinal atresia cases. The overall occurrence is estimated to be between 1:5000 and 1:66,000[3,4,5]. When these two anomalies occur together, it requires a thorough understanding of the anatomical structure and careful planning for effective treatment.

CASE REPORT

A three-day-old male infant presented to the emergency department with complaints of failure to pass stools since birth and abdominal distension associated with bilious vomiting. Upon clinical examination, the anal opening was present; however, it was not possible to advance a Hegar's dilator beyond 2 cm. A transverse loop colostomy was performed as an emergency procedure. At two

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months of age, a distal loopogram was planned, but the distal stoma could not be visualized due to retraction. Contrast enema revealed only 2 cm of the anal canal from the distal end. No imaging was available from the initial surgery. Two-dimensional echocardiogram and abdominal ultrasound were unremarkable. The infant underwent surgery for rectal atresia via the posterior sagittal approach. An end-to-end anastomosis was performed following resection of the atretic segment. Postoperatively, the patient was maintained on a regimen of regular Hegar's dilatation. Three months following this surgical intervention, a contrast enema was conducted (Fig. 1). This study demonstrated opacification of the entire left colon, which appeared to terminate just below the skin level. During stoma mobilization for closure, it was observed that approximately 3 cm of the distal retracted colon was atretic with a significantly narrowed lumen (Fig. 2). The bowel distal to this segment was also narrow but did not appear atretic. The distal stoma was refashioned following the excision of the atretic segment, and the patient was maintained on regular distal stoma washes to dilate the distal bowel. Stoma closure with the adequate calibre of the distal colon was achieved through washes and rectal dilatation using a Hegar's dilator after six months. The patient has demonstrated satisfactory progress during the postoperative follow-up period.

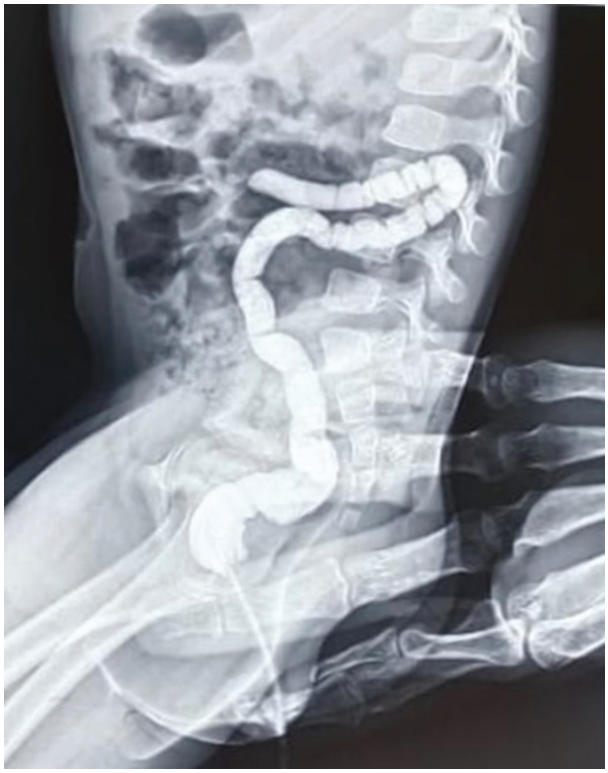

Fig. 1: Contrast enema after surgery for rectal atresia through the posterior sagittal route

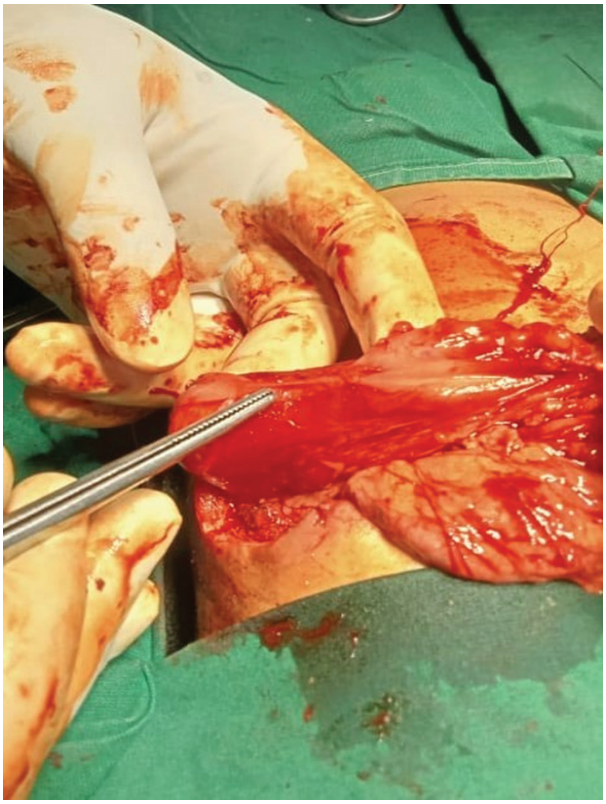

Fig. 2: Intraoperative picture showing type I colonic atresia (as pointed by the handheld forceps) after stoma mobilization.

DISCUSSION

ARM is a very common congenital anomaly in North India and in this case, we had a child with a rare variety of ARM, rectal atresia. In the hands of a general surgeon who usually doesn't do a high sigmoid divided colostomy (HDSC), transverse loop colostomy (TLC) is considered safe. In this case, there was anatresia in the distal bowel which was the left half of the transverse colon and splenic flexure. Using the Grosfeld classification, this was classified as type II colonic atresia[6]. Ismailet al reported a case of high ARM with colonic atresia and malrotation[7].

For this rare presentation, two explanations can be given. First, since the child did not have any history of NICU admission for any feeding or abdominal distension problems (to rule out necrotizing enterocolitis (NEC)), the technical problem can be the cause of the stricture in the distal bowel. As splenic flexure lies in the watershed area of the vascular supply, any undue handling or traction can cause the compromised vascular supply and stricture of the segment. Had it been a congenital or acquired stricture during the neonatal period, the TLC would have served as a lifesaving procedure as high divided sigmoid colostomy

(HDSC) would have been sited distal to the stricture and hence would have required revision in the neonatal period itself. Second, colonic atresia has a presentation with a patent anal opening. Since in this case, there was an anal opening, it might be a rare case of Colonic Atresia with Rectal Atresia[8].

If the stoma retracts before the second stage of ARM, the author believes that the stoma should be refashioned, and the correction of the ARM should be done only after a proper contrast study through the distal stoma is done. Any proximal atresia will present with a collapsed distal colon. Any attempt made at making a stoma on the collapsed segment can be catastrophic as this atresia will cause a closed loop obstruction with the ileocaecal valve. The mortality rate in such cases is reported as high as 60%.[9]

CONCLUSION

The low incidence of CA may result in delay in the diagnosis and management of this condition. Early recognition and prompt surgical intervention are crucial for optimal outcomes in these cases. Further research and collaborative efforts are needed to better understand the etiology, associated anomalies, and long-term outcomes of colonic and rectal atresias.

Conflict of interest: No

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