

Case Report

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Acquired colonic atresia in a 4-month old term male infant: a rare case report

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ABSTRACT

Acquired colonic atresias are very rare but, are known in association with necrotizing enterocolitis. We report a case of a 4-month term male infant with recurrent episodes of abdominal distension, bilious vomiting and constipation off and on, without the history of necrotizing enterocolitis. Exploratory laparotomy was performed, an inflammatory mass with multiple dense interloop adhesions were found in the mid-transverse colon. These adhesions were lysed to identify the proximal dilated and distal blind end of the colon. Rest of the gut was normal. This case is unique for the fact that, it is a case of acquired colonic atresia without history of necrotizing enterocolitis, unlike other reported cases of acquired colonic atresia.

Keywords: Acquired intestinal atresia, Intestinal obstruction, Necrotizing enterocolitis

INTRODUCTION

Intestinal atresia is a condition in which there is complete obliteration of the bowel lumen. Majority of intestinal atresias are congenital. Congenital intestinal atresias are rare in the colon.¹ Acquired atresias of the intestine are extremely rare. Acquired colonic atresias have been reported as a consequence of necrotizing enterocolitis.^{2,3} We report a very unusual case of acquired colonic atresia without the association of necrotizing enterocolitis.

CASE REPORT

A 4-month-old male child presented with history of recurrent abdominal distension, bilious vomiting and constipation off and on for a period of 1 month. The child was born to a 26-year old primigravida at 38 weeks gestation by spontaneous vaginal delivery with a birth weight of 2.5 kgs. There was no significant antenatal history. He had a history of delayed crying and fever after

birth. The child passed meconium within the first 6 hours of birth and was normal until 3 months after birth.

At 3 months of age the child presented with abdominal distension, vomiting and constipation. A diagnosis of subacute intestinal obstruction was made and he was treated conservatively with IV fluids, antibiotics and suppositories. Presently, he had the same symptoms and on examination the child was severely dehydrated, abdomen was soft and distended and revealed visible peristalsis. All the laboratory investigations were done, renal function test including electrolytes were within normal limits. An erect abdominal plain radiograph revealed multiple air fluid levels (Figure 1).

A barium enema contrast study performed soon after revealed blind end of the colon, the dye was not seen beyond transverse colon (Figure 2).

A provisional diagnosis of Hirschsprung disease was made.

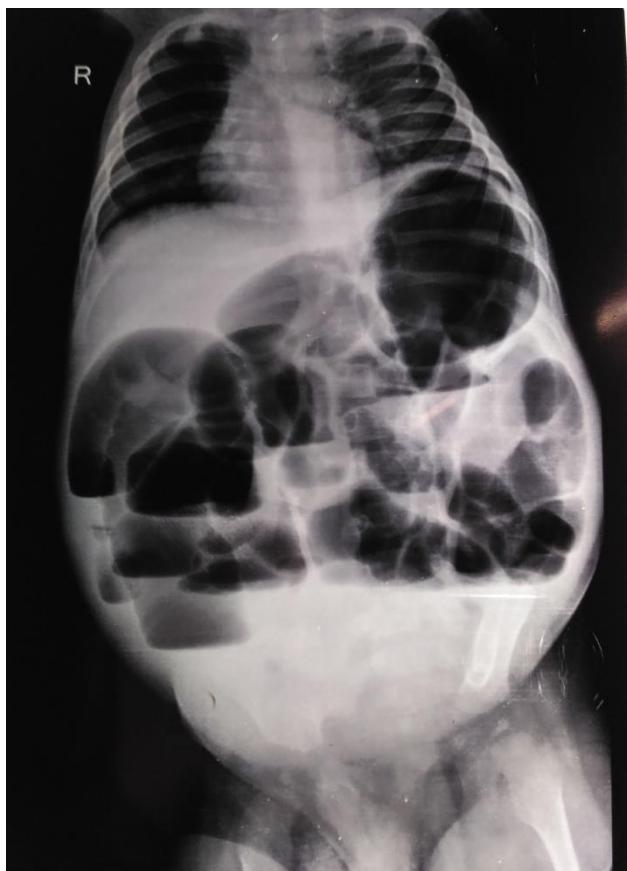


Figure 1: Plain erect abdominal radiograph of the patient showing multiple air-fluid levels.



Figure 2: Barium enema contrast study of the patient showing micro-colon and absence of the dye beyond the transverse colon.

Exploratory laparotomy revealed an area of obstruction in the mid transverse colon. The obstruction was due to an inflammatory mass with multiple dense interloop adhesions. These adhesions were lysed, the portion of colon proximal to the mass was dilated and the distal portion revealed a micro colon, both the portions ending blindly. Remainder of the gut was normal. An end colostomy was performed. A biopsy performed from the portion of colon distal to the mass and was sent for histopathologic examination, showed the presence of ganglion cells. His post-operative period was uneventful and was discharged after 7 days. After a period of two months the patient was admitted again, re-explored and an end to end colo-colic anastomosis was performed. Post-operative course was uneventful and the child recovered well.

DISCUSSION

Congenital atresias of the gut are caused by vascular compromise during early stages of pregnancy.⁴ Although majority of them are caused by a vascular event, new evidence suggests the presence of a genetic mechanism in the pathogenesis.⁵ The aetiology of congenital atresias of the duodenum is due to failure of recanalization and its co-existence with trisomy 21 is well documented.⁶ Acquired atresias of the intestine are documented rarely in premature infants after recovery from necrotizing enterocolitis. Necrotizing enterocolitis (NEC) is a severe inflammatory disorder of the bowel common in pre-term infants. Classic signs and symptoms of necrotizing enterocolitis (NEC) include bilious vomiting, abdominal distention, and bloodstained stools. The acquired atresia of intestine in cases with NEC, the affected segment was observed to be distal to an established enterostomy.^{7,8} Acquired colonic atresias are an extremely rare entity. We could only find four cases of acquired colonic atresia all associated with necrotizing enterocolitis.

Beardmore HM et al reported two cases of acquired colonic atresia subsequent to necrotizing enterocolitis, both of the patients were premature male infants one of them presented at 5 days after birth and another at 24 hours after birth with bloody stools, vomiting and abdominal distention.² Mares AJ et al reported two cases of acquired colonic atresia as a sequelae of necrotizing enterocolitis, both the patients were premature female infants presenting at 2 days of birth and 18 days of birth with classical features of necrotizing enterocolitis.³ Intestinal atresias which were not associated with necrotizing enterocolitis were very rare with only seven reported cases in English literature and all in the small intestine. The reason for atresia described by one of these authors was a thromboembolic event interrupting blood supply to a particular segment of intestine resulting in stenosis or atresia of that segment.⁹ In five cases the reason was due to the presence of adhesive bands interrupting blood supply causing ileal atresia and in one case the reason was due to ileo-colic intussusception.¹⁰⁻¹³

It is important to note that in our case the infant was born at term and there were no symptoms and signs of necrotizing enterocolitis. We failed to find in English literature a case of acquired colonic atresia without the association of necrotizing enterocolitis. The underlying pathophysiology is presumed to be either a recurrent colo-colic intussusception or recurrent local infection leading to the inflammatory process or perforation at the local site leading to the formation of thrombus followed by mesenteric vascular occlusion.

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