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Association of Jejunal Atresia with Mucosal Diaphragm and Enteric Duplication in a Newborn: A Rare Case Report

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ABSTRACT: Jejunal atresia is a leading cause of neonatal intestinal obstruction, most commonly resulting from intrauterine vascular accidents. Intraluminal mucosal diaphragms and enteric duplications are rare congenital anomalies, and their coexistence is exceptional.

We report the case of a full-term newborn presenting with early bilious vomiting and abdominal distension. Imaging suggested proximal intestinal obstruction. Surgical exploration revealed type I jejunal atresia caused by a mucosal diaphragm, associated with a cystic jejunal duplication sharing a common muscular wall. Segmental resection and primary anastomosis were performed with a favorable outcome.

This rare association highlights the importance of thorough intraoperative exploration in neonatal intestinal obstruction. Multiple embryological mechanisms may coexist, complicating both diagnosis and management.

KEYWORDS: Jejunal atresia; mucosal diaphragm; enteric duplication; duplication cyst; neonatal intestinal obstruction.

INTRODUCTION

Neonatal intestinal obstruction is a common surgical emergency, with jejunal atresia accounting for a significant proportion of cases. The incidence of jejunoileal atresia is estimated at approximately 1 in 5,000 live births.

The most widely accepted pathogenesis involves intrauterine mesenteric vascular disruption, leading to ischemic necrosis and resorption of the affected bowel segment.

In contrast, mucosal diaphragms (intestinal webs) result from failure of recanalization during embryonic development, whereas enteric duplications arise from abnormal budding of the primitive gut or split notochord syndrome. These duplications are characterized by a welldeveloped muscular wall and mucosal lining.

The simultaneous presence of these three anomalies in a single patient is extremely rare and poorly described in the literature, making this case both clinically and embryologically significant.

AIM OF THE ARTICLE

To report a rare association of jejunal atresia caused by a mucosal diaphragm with enteric duplication in a newborn, and to highlight the importance of careful intraoperative assessment and appropriate surgical management.

PRESENTATION OF CASE

A full-term male newborn was delivered at 39 weeks of gestation via spontaneous vaginal delivery following an uneventful pregnancy. The birth weight was 3100 g, and Apgar scores were 9 and 10 at 1 and 5 minutes, respectively. No antenatal abnormalities were detected on routine ultrasound examinations.

Within the first 24 hours of life, the neonate developed persistent bilious vomiting associated with progressive abdominal distension and failure to pass meconium. He was referred to our department on day 2 of life for further evaluation and management.

On physical examination, the newborn was hemodynamically stable. Abdominal examination revealed moderate distension, predominantly in the upper abdomen, with visible peristalsis.

There was no tenderness, palpable mass, or signs of peritonitis.

Laboratory investigations were within normal limits. A plain abdominal radiograph demonstrated multiple air-fluid levels with dilated proximal bowel loops and paucity of distal gas, suggestive of proximal small bowel obstruction. Abdominal ultrasonography confirmed dilated fluid-filled intestinal loops without evidence of malrotation or volvulus.

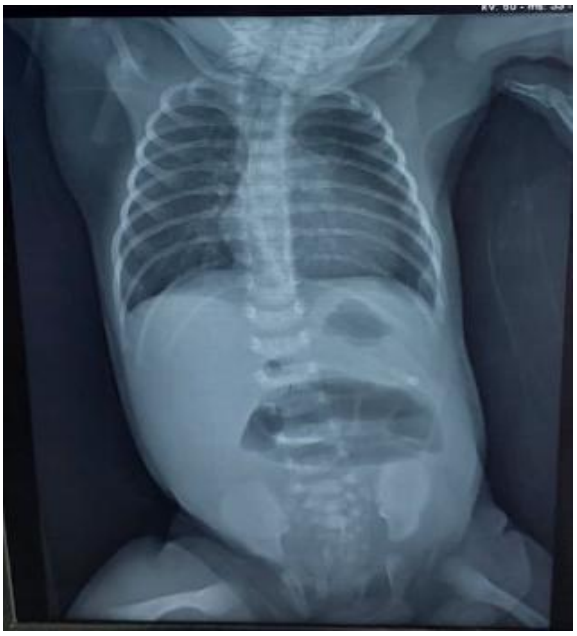

Figure 1: An Abdominal Radiograph demonstrated multiple air-fluid levels with dilated proximal bowel loops

Based on these findings, neonatal intestinal obstruction, most likely jejunal atresia, was suspected, and the patient underwent emergency surgical exploration.

A supraumbilical transverse laparotomy was performed. Intraoperative findings revealed markedly dilated proximal jejunal loops with an abrupt transition zone. Careful inspection identified type I jejunal atresia caused by an intraluminal mucosal diaphragm. Additionally, a cystic lesion measuring approximately 3 cm was identified on the mesenteric border of the jejunum, consistent with a duplication cyst. This duplication shared a common muscular wall and vascular supply with the adjacent bowel segment. No additional intestinal atresias or associated anomalies were identified following thorough exploration of the entire bowel.

Surgical management consisted of en bloc resection of the affected jejunal segment, including the mucosal diaphragm and duplication cyst, followed by primary end-to-end jejunojejunal anastomosis. Distal bowel patency was confirmed prior to completion of the procedure.

The postoperative course was uneventful. The patient was managed in the neonatal intensive care unit, with gradual reintroduction of enteral feeding beginning on postoperative day 5. Full enteral feeding was achieved by day 8, and the patient was discharged on postoperative day 10 in good clinical condition. At follow-up, the infant demonstrated normal feeding and appropriate weight gain, with no evidence of postoperative complications.

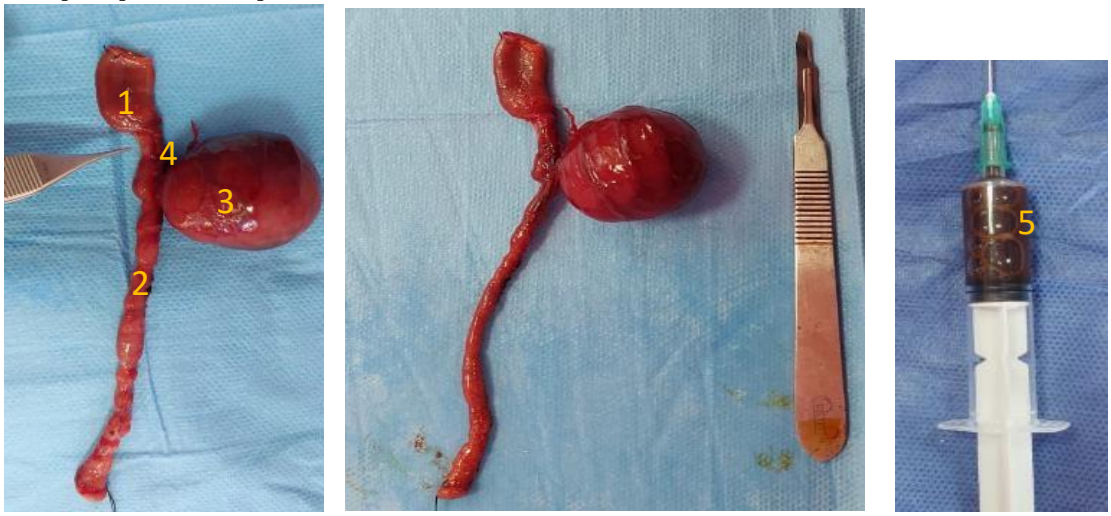

Figure 2: Operative specimen showing jejunal atresia with a mucosal diaphragm associated with enteric duplication.
1: Proximal jejunum; 2: Distal jejunum; 3: Enteric duplication; 4: Mucosal diaphragm; 5: Bile fluid within the enteric duplication.

DISCUSSION:

Jejunal atresia is a well-recognized cause of neonatal intestinal obstruction, accounting for a significant proportion of surgical emergencies in the neonatal period. Its incidence is estimated at approximately 1 in 5,000 live births. The most widely accepted pathophysiological mechanism is an intrauterine mesenteric vascular accident leading to ischemic necrosis and resorption of a segment of the intestine. However, this theory does not fully explain all types of atresia, particularly type I lesions, which are characterized by an intact bowel wall with intraluminal obstruction such as a mucosal diaphragm.

In the present case, jejunal atresia was caused by a mucosal diaphragm, also referred to as an intestinal web. This rare entity is believed to result from failure of recanalization of the primitive gut during early embryonic development. Depending on the degree of obstruction, these diaphragms may be complete or fenestrated, the latter potentially leading to delayed or intermittent symptoms. In neonates, complete obstruction typically presents early with bilious vomiting and abdominal distension, as observed in our patient.

The coexistence of a mucosal diaphragm with an enteric duplication further increases the rarity of this case. Enteric duplications are uncommon congenital malformations that may occur anywhere along the gastrointestinal tract, most frequently in the ileum. Jejunal duplications are less common and are typically located on the mesenteric border. They are characterized by a well-developed smooth muscle layer and mucosal lining, often sharing a common wall and vascular supply with the adjacent bowel. Clinical presentation varies depending on size, location, and associated complications such as obstruction, bleeding, or perforation.

The association of jejunal atresia, mucosal diaphragm, and enteric duplication is exceptionally rare and raises important embryological considerations. While jejunal atresia is generally attributed to late intrauterine vascular insults, both mucosal diaphragms and duplications are thought to arise earlier from disturbances in gut development. The coexistence of these anomalies suggests that multiple pathogenic mechanisms may act simultaneously or sequentially during fetal development, supporting a multifactorial origin.

Preoperative diagnosis of such combined anomalies remains challenging. Prenatal imaging may suggest intestinal obstruction but rarely provides a definitive diagnosis. Postnatal imaging is useful in confirming obstruction but lacks specificity for identifying associated anomalies. Consequently, definitive diagnosis is usually established intraoperatively.

This case underscores the importance of meticulous intraoperative exploration. In cases of jejunal atresia, particularly type I, surgeons must carefully examine the entire bowel to exclude associated anomalies such as additional atresias, webs, or duplications. Failure to do so may result in persistent postoperative obstruction and the need for reoperation.

Surgical management consists of resection of the affected segment, including the duplication, followed by primary anastomosis. Preservation of bowel length and vascular supply is essential to prevent short bowel syndrome and ensure optimal functional outcomes. In this case, complete resection with primary anastomosis resulted in an uneventful recovery.

Advances in neonatal intensive care, surgical techniques, and nutritional support have significantly improved outcomes in jejunal atresia. When associated anomalies are recognized and appropriately managed, prognosis is generally excellent.

CONCLUSION:

This case illustrates an exceptionally rare association of jejunal atresia caused by a mucosal diaphragm with an enteric duplication cyst. It highlights the importance of meticulous intraoperative exploration, the possibility of multiple embryological mechanisms, and the need for complete surgical resection to ensure optimal outcomes.

Early diagnosis and appropriate surgical management are associated with an excellent prognosis.

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Ethical Approval: As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

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