



Primary Side-to-Side Anastomosis after Resection of Jejunal Atresic Segment is Safe and Effective in the Incomplete Type 1 Jejunal Atresia

Elroy P Weledji^{1*}, Naiza Monono² and Theophil Nana¹

¹Department of Surgery, University of Buea, Cameroon

²Department of Pediatrics, University of Buea, Cameroon

Abstract

Intestinal atresia is a life-threatening cause of intestinal obstruction in the neonate, and rarely a cause of intestinal obstruction in an infant. We report a rare presentation of an incomplete jejunal atresia in a 1 year and 7 months old girl. She presented with a sub-acute upper gastrointestinal obstruction since birth associated with failure to thrive. Laparotomy revealed a jejunal atresia (type 1) which was successfully managed by resection of the atresic segment and a side to side jejuno-jejunal anastomosis.

Keywords: Congenital; Atresia; Jejuna; Anastomosis; Technique

Introduction

Intestinal atresia is a congenital absence of the bowel lumen and death occurs from loss of gastrointestinal fluids, pulmonary aspiration and malnutrition. It can occur throughout the intestine but is common in the small intestine. The aetiology may include an ischemic event causing necrosis and resorption of the involved bowel or failure of the lumen to develop from the embryonic cord stage. Because of the mobility of the jejunum and ileum with the potential for compression or volvulus, and the anatomy of the arcades, these areas are more likely to suffer vascular ischemia. Therefore, the most likely aetiology for duodenal atresia is failure of luminal canalization after the cord stage [1]. Jejunal atresia has an incidence of about 1:1,000 live births and is more common than duodenal atresia. Jejunoileal atresia causes complete obstruction in 1:5,000 live births. In contrast to duodenal atresia's which can be associated with Down's syndrome, cardiac defects and malrotation. Jejuno-ileal atresia's can be associated with prematurity (50%), polyhydramnios (25%), cystic fibrosis (20%), gastroschisis and short gut syndrome. The site of jejunal atresia can be anywhere from the ligament of Treitz to the jejuno-ileal junction and there can be more than one atresic segment. The classification is based on the type of malformation and the presence of a mesenteric defect with the type III atresia may support the in-utero ischemic aetiology (Table 1, Figure 1) [1]. Neonates typically present with abdominal distension and bilious vomiting within the first 24 h of birth, and these symptoms do not allow for a differentiation from a duodenal atresia [2]. Early and prompt diagnosis and appropriate treatment have improved patient's outcome.

Case Presentation

A 1-year, 7 months old girl was referred from a rural area with a history since birth of intermittent bilious vomiting, and vomiting of undigested food 2 to 3 days after a meal. This was associated with colicky abdominal pain and a general failure to thrive as the symptoms worsened with time. As a neonate she required enema to facilitate defecation and as an infant her bowel frequency was less than 3 to 4 episodes per week. She was delivered vaginally at term and of normal weight (4.2 kg). She was the 5th child of a 41-year-old mother and the other siblings were normal and well. Her symptoms were managed with native herbal medications to no avail. On examination she was clinically small for age, thin and malnourished. She had a distended upper abdomen with visible peristalsis. She had no other congenital anomaly. A full blood count revealed an iron-deficiency anemia of 7 gm/dl and serum biochemistry showed evidence of dehydration with a mild hypokalemia of 3.2 mEq/l (n: 3.4 to 5.3 mEq/l), hypernatremia 159 mg/l (n: 135 to 155 mg/l) and a hypochloremia of 133 mg/dl (n: 98 to 115 mg/l) indicating some renal compensation. These were appropriately corrected with blood transfusion, intravenous hydration with potassium supplementation and nasogastric

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*Correspondence:

Elroy P Weledji, Department of Surgery, University of Buea, PO Box: 126, Limbe, S.W. Region, Cameroon,

Tel: 237699922144;

E-mail: elroypat@yahoo.co.uk

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Table 1: Classification system of jejunal atresia by Louw and Barnard (1955) [1].

| Type 1 | Web with complete or incomplete obstruction |
|-----------|---|
| Type II | Two blind ends often connected by a fibrous strand with slightly shortened intestinal length (complete obstruction) |
| Type III | Flaws in the blood supply |
| Type IIIa | Blind ends separated by a mesenteric defect |
| Type IIIb | Distal end - 'apple peel', rare, high mortality |
| Type 4 | Multiple intestinal atresias |

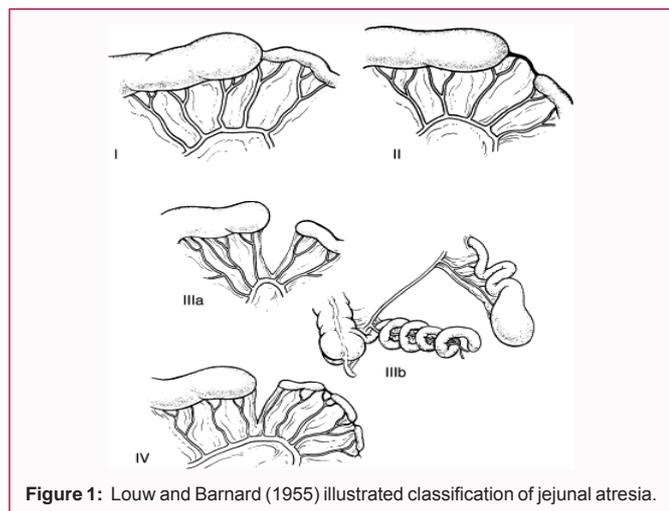


Figure 1: Louw and Barnard (1955) illustrated classification of jejunal atresia.



Figure 2: Jejunal atresic segment (formalin-fixed).

decompression. An abdominal ultrasound had suggested a pyloric obstruction with a dilated stomach, but a plain abdominal X-ray could only show some dilated loops of proximal small bowel and constipation in the distal colon. From the clinical examination the differential diagnosis of the sub-acute high small bowel obstruction included a duodenal atresia, stenosis, web or annular pancreas. A laparotomy through a (R) transverse, supraumbilical incision revealed a grossly dilated stomach and an enlarged duodenal bulb. The pylorus appeared normal and there was no abnormality seen in the mobilized 2nd part of the duodenum. Further inspection of the rest of the small bowel revealed a dilated and very thickened jejunum of about 5 cm in diameter with increased peristalsis towards the atresic segment situated at about 1 m from the duodenojejunal flexure. The atresic collapsed segment was patent on palpation and the distal intestine was of normal length and diameter. This incomplete atresic segment was excised en-bloc with about 3 cm of proximal and distal jejunum (Figure 2). Both ends of small bowel were closed with 3.0 vicryl and a wide isoperistaltic side to side anastomosis fashioned in 2 layers with a hemostatic full thickness continuous suturing of the inner layers using 3.0 vicryl. She had a prolonged postoperative ileus

Table 2: Causes of intestinal obstruction in neonate and infant.

| Neonate (< 28 days) | Infants (<1 yr old) |
|---------------------------|------------------------|
| Necrotizing enterocolitis | Malrotation |
| Intestinal atresia | Meckel's diverticulum |
| Meconium ileus | Peritoneal bands |
| Hirschsprung's disease | Hirschsprung's disease |
| Duplication cysts | Duplication cysts |
| | Volvulus |

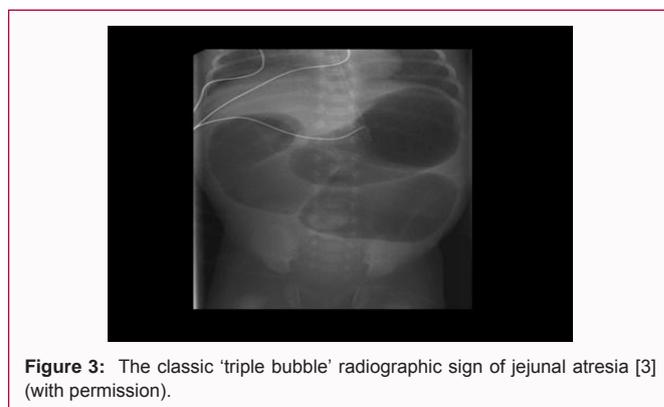


Figure 3: The classic 'triple bubble' radiographic sign of jejunal atresia [3] (with permission).



Figure 4: Incomplete type I Jejunal atresia with incomplete web (membrane)-formalin-fixed.

for about a week as a result of hypokalemia of 2.1 mEq/l which was corrected. She gradually tolerated feed and was discharged 2 weeks after the surgical correction.

Discussion

This case demonstrates a rare presentation of intestinal atresia in a toddler. The causes of intestinal obstruction in the neonate and the infant are listed in (Table 2). The delayed presentation for appropriate investigations and management was largely due to the sub-acute nature of the proximal intestinal obstruction from the incomplete type I jejunal atresia. It is crucially important to inspect the whole

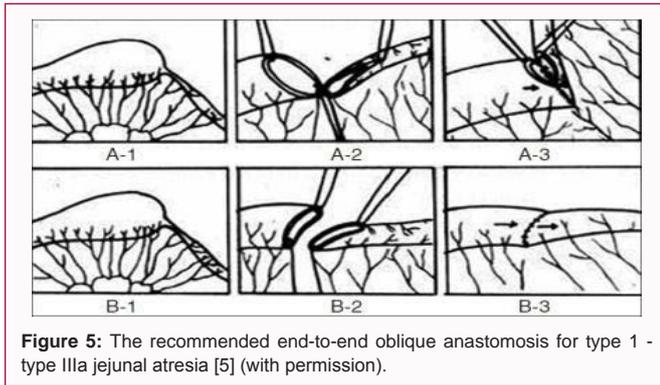


Figure 5: The recommended end-to-end oblique anastomosis for type 1 - type IIIa jejunal atresia [5] (with permission).

intestine during a laparotomy for congenital intestinal obstruction as seen in this case where the causative pathology was discovered only at operation. Other events such as volvulus, herniation, constriction and intussusception have been observed during surgery in patients with jejunal atresia [3]. Thus, rapid diagnosis is important in the treatment of this anomaly. The classic radiographic sign of jejunal atresia is that of a triple bubble appearance for a proximal obstruction which is equivalent to the double bubble sign of a duodenal obstruction plus a third bubble caused by filling and distention of the jejunum by air (Figure 3). There can be multiple dilated small bowel loops proximal to the atresia and the number of dilated loops increase as the point of atresia becomes more distal [3]. Contrast enema typically shows micro colon (small unused colon). The differential diagnosis on plain radiograph would include malrotation with midgut volvulus and on contrast enema the differential diagnosis will include a total colonic Hirschsprung disease and meconium ileus (Table 2). Antenatal ultrasound may show dilated proximal bowel loops, often greater than 7 mm, evidence of an in-utero bowel perforation, or there may be polyhydramnios, especially when the disease is proximal [4]. The goal in treatment of intestinal atresia is to establish intestinal continuity with as much functional bowel as possible. The type 1 jejunal atresia is not a true atresia as it consists of a web with complete or incomplete obstruction [1,2]. The incomplete obstruction as seen in this case has a web/membrane demarcating the proximally thickened and wider jejunum from the distal jejunum. Both sides showed a similarly structured valvulae conniventes (mucosal folds) consistent with the jejunum (Figure 4). In neonatal jejunoileal atresia (type 1-IIIa) an end-to-end oblique anastomosis is recommended as it is wide with fewer angulations, and allows a linear flow of effluent. In addition, there is less force exerted over a post-anastomotic side wall (Figure 5) [5,6]. The primary side-to-side anastomosis technique following resection of the atresic segment would suffice for the simple

incomplete type 1 atresia where the incomplete obstruction allowed a mature and visible proximal intestinal motility as in this case, and thus the decrease chance of a functional obstruction [7,8]. This technique is not advisable in neonates or preterm babies because of the failure of the dilated bowel wall to generate adequate intraluminal pressure for effective peristalsis despite a patent anastomosis. They would have a greater incidence of functional obstruction [8-10]. The side-to-side anastomosis is also useful after a failed primary repair of jejunal atresia in neonates [8]. The complex types (type IIIb and type IV) with short bowel syndrome would require more complex pediatric surgery with detailed perioperative care [11].

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