

EXTRINSIC DUODENAL OBSTRUCTION FROM ANOMALOUS CONGENITAL BAND

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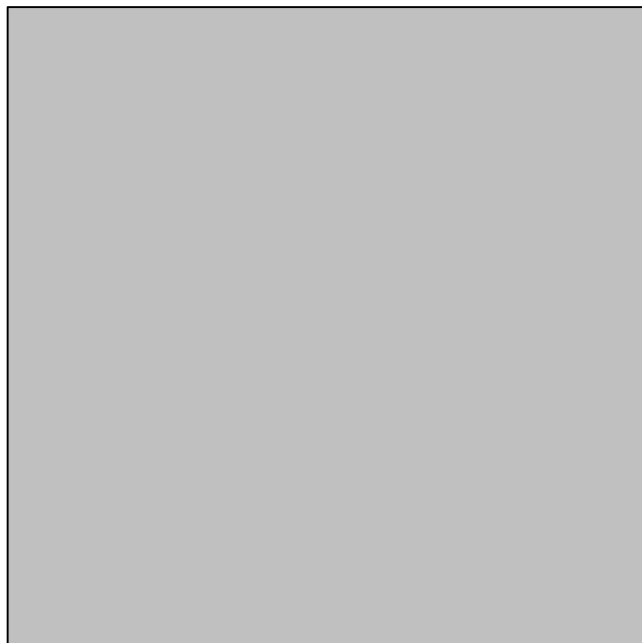
Extrinsic duodenal obstruction may be caused by congenital bands, intestinal malrotation, annular pancreas, preduodenal portal vein, and duodenal duplication in the neonate.¹⁻³ Anomalous congenital band (ACB) as a cause of intestinal obstruction in infancy and childhood is rarely reported in the literature.⁴ We report a case of extrinsic duodenal obstruction from ACB in a nine-day-old infant.

Case Report

A nine-day-old male infant was transferred from a local hospital to our institution with persistent bilious vomiting since birth. He had been delivered full term per vaginam to a 28-year-old gravida 5, para 4, and weighed 3280 g. During the pregnancy, maternal ultrasound scan revealed polyhydramnios. At presentation, clinical examination was unremarkable. A plain abdominal radiograph was normal. An upper gastrointestinal (GI) study showed a mild dilatation of the proximal duodenum, a hold-up at the third part of the duodenum, with passage of contrast into the proximal jejunum. The ligament of Treitz was in the normal position (Figures 1A-1D). The patient was kept on conservative management of intravenous fluids and nasogastric tube decompression. He underwent laparotomy, which revealed an ACB arising from the antimesenteric wall of the third and fourth parts of duodenum as far as the ligament of Treitz, and extending across the proximal jejunum to the root of the mesentery (Figure 2). The band which contained tiny vessels was lysed. At follow-up 10 months later, the infant was asymptomatic and thriving.

Discussion

Extrinsic duodenal obstruction may be caused by congenital peritoneal band, intestinal malrotation, annular pancreas, preduodenal portal vein and duodenal duplication.¹⁻³ In a series reported by Wayne and Burrington, congenital peritoneal bands were found in 39 out of 64 children with extrinsic duodenal obstruction.³ There are four types of congenital peritoneal band. Type 1



FIGURES 1A-D. Upper GI study shows a mild dilatation of the second and proximal third part of the duodenum, a hold-up at the distal third part of duodenum with flow of contrast into the proximal jejunum. The ligament of Treitz is in the normal position.

band is always associated with intestinal malrotation. The cecum, which is in the upper quadrant of the abdomen, has a band which extends across the second and third parts of the duodenum to the paravertebral gutter (Ladd's band). Duodenal obstruction may result from either compression by Ladd's band and/or from mid-gut volvulus. Type 2 band extends from the hepatic flexure of the colon across the second part of the duodenum to the right paravertebral gutter, causing duodenal compression at that site. Type 3 band is a hypertrophied hepatoduodenal ligament which obstructs the duodenum at the junction of its first and second parts. Type 4 band is a dense fibrous band which binds the distal part of the third part of the duodenum to the prevertebral fascia, causing extrinsic obstruction, and is always associated with an incompletely rotated duodenum.

The band in our case not only caused extrinsic duodenal obstruction, but also compressed the proximal jejunum. Although the duodenal obstruction was at the same site as a type 4 band, the following differences were noted: the band

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FIGURE 2. View at laparotomy: ACB from the third and fourth parts of duodenum (arrowed) to the root of mesentery. Forceps points to the ligament of Treitz.

in the present case contained tiny vessels and not a fibrous band; it arose from the antimesenteric wall of the duodenum and was inserted into the root of mesentery; and there was no intestinal malrotation, which is associated with type 4 band. We conclude that the duodenal obstruction in our case was from ACB. Akgur et al. reported eight children with ACB which did not cause duodenal obstruction. Each band contained tiny blood vessels, as was so in our case.⁴ On histological examination, the ACB contained blood vessels and nerve plexi. Based on the origin and insertion of ACB, we agree with Akgur et al. that it may have been a mesenteric anomaly.⁴

Proximal gut atresias are associated with maternal polyhydramnios due to obstruction to the normal passage of liquor through the GI tract. Intrinsic duodenal obstruction is associated with polyhydramnios, and the incidence ranges from 32% to 46%.⁵ These include both duodenal atresia and stenosis. The incidence of polyhydramnios in extrinsic duodenal obstruction, however, is unknown. In a review of infants and children with extrinsic duodenal obstruction, Tryfonas and Young found no polyhydramnios in 31 pregnancies.² It is suggested that polyhydramnios develops

in bowel atresia when there is complete obstruction for a long time.⁵ It is possible that in the present case, there was significant extrinsic duodenal and jejunal obstruction to cause polyhydramnios.

Bilious vomiting in a neonate, although not pathognomonic, warrants investigation for intestinal obstruction, especially if associated with polyhydramnios. If a plain abdominal radiograph is not conclusive, an upper GI series should be performed to exclude intrinsic duodenal obstruction such as stenoses or extrinsic duodenal obstruction such as duodenal band, and more importantly, intestinal malrotation with volvulus.¹ Duodenal band including ACB may present beyond the neonatal period in a child as repeated episodes of vomiting with upper abdominal pain.⁴ Abdominal distension may not be present.

After initial conservative management of intravenous fluids and nasogastric tube decompression, the treatment of the duodenal band, e.g., ACB, is simple lysis. The infant or child is usually cured thereafter.

If an infant or a child presents with repeated episodes of vomiting with or without upper abdominal pain, extrinsic duodenal obstruction such as ACB should be excluded. An upper GI series would show duodenal hold-up from the extrinsic compression. ACB should be considered as one of the causes of extrinsic duodenal obstruction in infancy and childhood.

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