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Duodenal web as a cause of duodenal obstruction

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INTRODUCTION

This is a case report of a preterm infant with a duodenal web causing duodenal obstruction. When the obstruction is incomplete, as it was in our case, the clinical symptoms are variable and the diagnosis can be difficult.

CASE REPORT

This male premature infant was born in a private hospital as twin B at 36 5/7 weeks by caesarean section following premature rupture of membranes. The pregnancy of the 35-year-old mother had otherwise been uneventful.

Apgar scores were 9, 9, and 10 at 1, 5, and 10 minutes, respectively. The birth weight was 1680 g, the length 42 cm and the head circumference 29.5 cm (all below the 3rd percentile). The boy was transferred to our NICU because of his low birth weight.

The child was the third of non-related parents. His twin brother and his older brother were healthy, and the family history was unremarkable.

Nutrition was initiated enterally with a preterm milk formula and supplemented parenterally (initially with a glucose/electrolyte solution, later with TPN). The infant passed his first meconium within 12 hours of life and had regular bowel movements once to twice per day thereafter.

On the 2nd day of life, some spitting and vomiting were noted. From day of life 6 onwards, he required a nasogastric tube (NGT) because of poor feeding. At that time, remarkably high amounts of gastric aspirates were noticed (up to 36 ml of predigested formula milk). Conventional X-ray showed no signs of gastrointestinal obstruction (Fig. 1). Therefore, feeding on demand was again attempted on the 11th day of life. However, the infant only drank approximately 60 ml/kg/day, beyond which vomiting reoccurred.

As this presentation was suggestive of an obstruction of the gastrointestinal tract, further diagnostic investigations were performed. An upper gastrointestinal contrast study demonstrated delayed passage of contrast medium as well as a change of caliber between part II and III of the duodenum (Fig. 2). Ultrasonography showed prestenotic dilatation suggestive of duodenal stenosis. Malrotation of the gastrointestinal tract was ruled out by a contrast study of the colon.

The results were felt to be compatible with duodenal obstruction and an exploratory laparotomy was performed. Intraoperatively, adhesions of the duodenum with the gall bladder and the abdominal wall were freed. Continuity of the duodenum was preserved and the pancreas was in its typical location. An annular pancreas was ruled out. An NGT was then advanced until it met resistance in the duodenum. An incision was made at this location leading to the discovery of

a duodenal web as the cause of the obstruction. The NGT could be advanced into the distal duodenum following resection of the web. Further inspection of the intestine was inconspicuous.

For the next seven days, the infant was fed through a trandsduodenal feeding tube. Subsequently, oral feeding was reinitiated without any problems. The boy was discharged at 5 weeks of age weighing 2340 g.

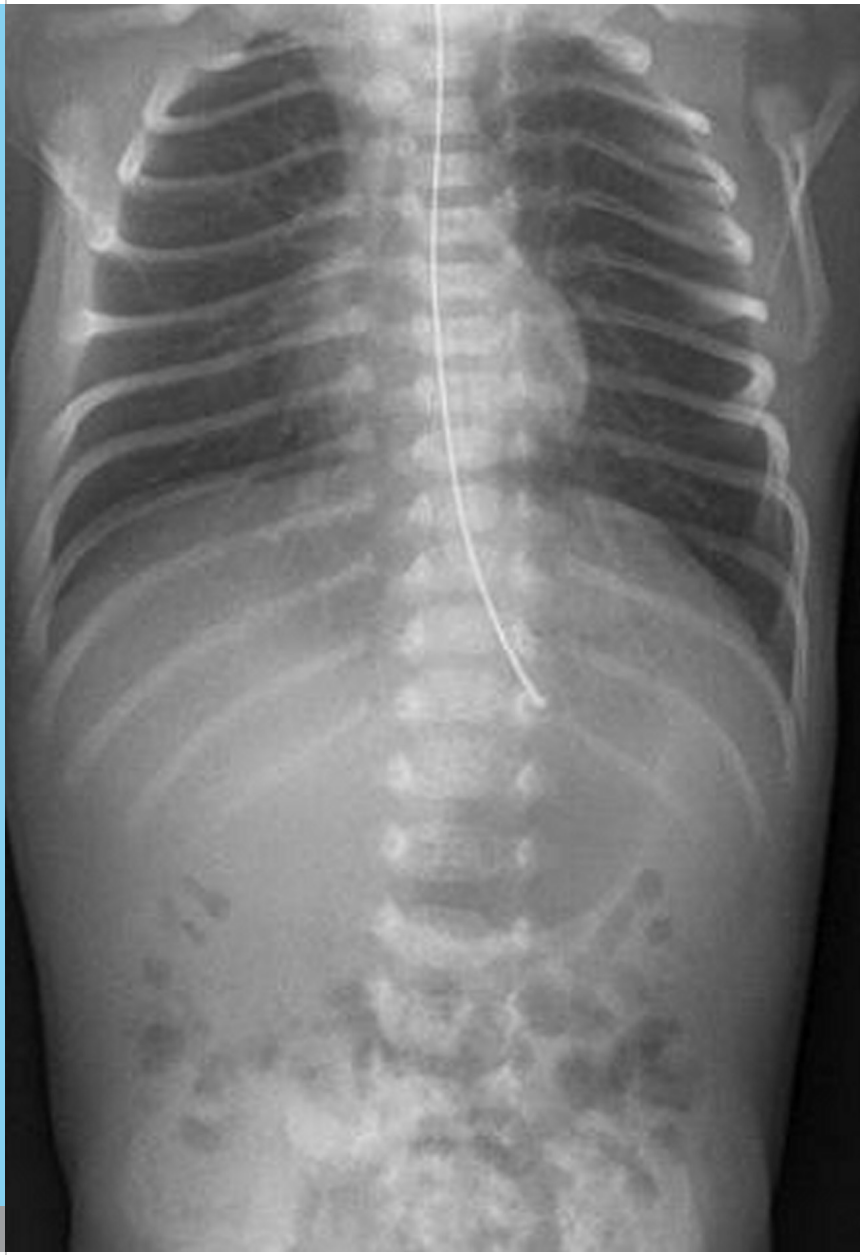

Fig. 1

Babygram without evidence of gastrointestinal obstruction.

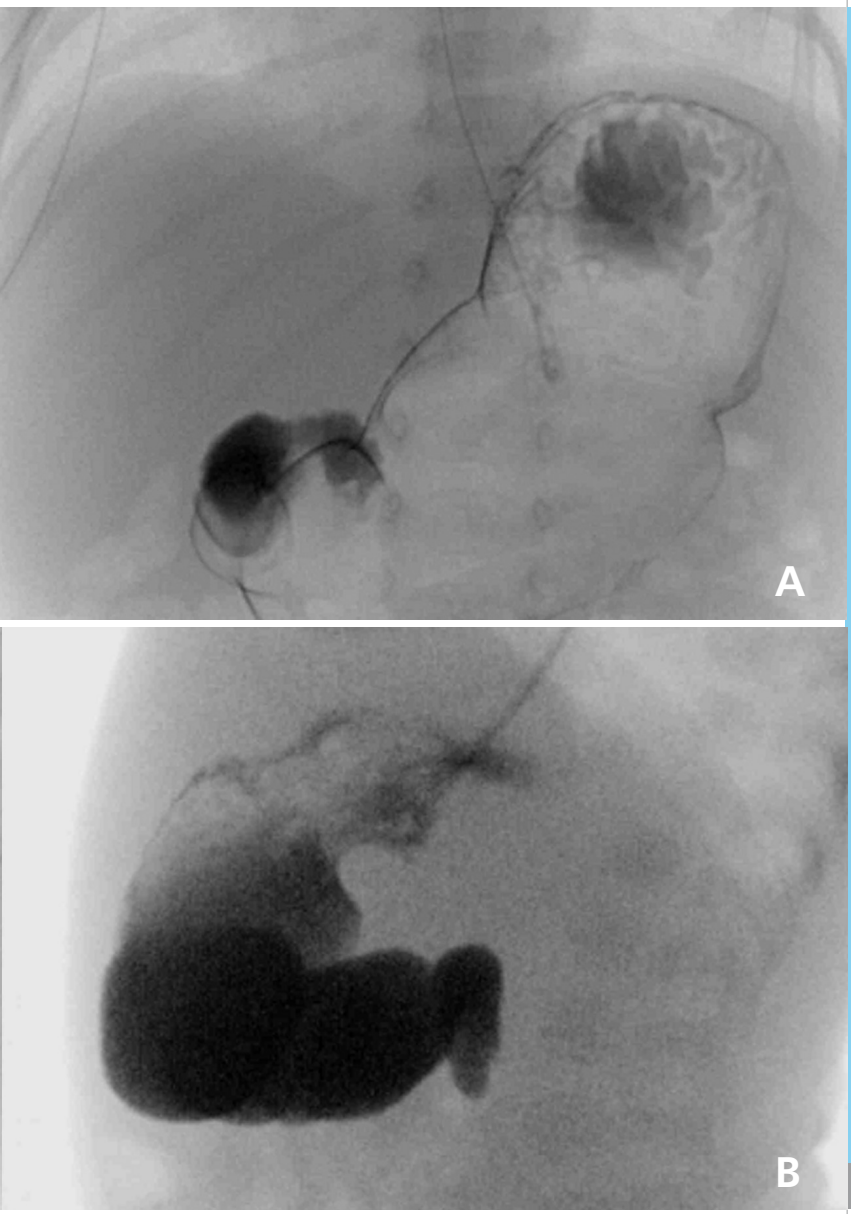

Fig. 2

Upper gastrointestinal contrast study demonstrating failure of the contrast medium to progress beyond the distal part of the duodenum (A: ap view, B: lateral view).

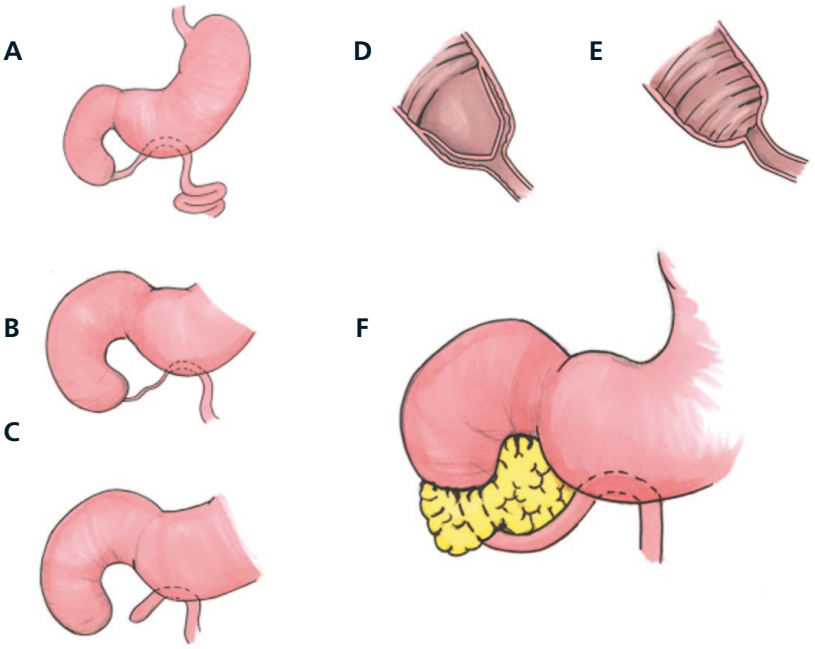

Fig. 3

Classification of anomalies leading to duodenal obstruction: A) atresia with an intact intraluminal membrane leading to marked discrepancy in size between proximal and distal segments of the duodenum (type 1); B) blind ends of the duodenum are connected by a fibrous cord and the mesentery is intact (type 2); C) blind ends of the duodenum are separated and the mesentery is absent at the site of the separation (type 3); D); windsock anomaly; E) intraluminal membrane with a perforation F) annular pancreas (redrawn after (1) by F. Berger)

Duodenal atresia and stenosis are the most common causes of intrinsic duodenal obstruction in the newborn. The first description of duodenal atresia was published in 1733, and the first survivor was recorded in 1914. Gradual improvement in treatment and survival of infants with duodenal obstruction has since then been achieved (1).

Intrinsic and extrinsic pathologies are known reasons for duodenal obstruction. An annular pancreas can cause extrinsic duodenal obstruction. The pancreas develops out of two outgrowths: one dorsal and the other ventral to the duodenum. During bowel rotation, fusion of the two parts can lead to a ring shaped construction around the duodenum (Fig. 2F). An anterior portal vein crossing over the top of the duodenum and the pancreas may result in extrinsic compression of the duodenum. Finally, anomalies of fixation and rotation of the bowel are also known to cause obstructions (1).

Intrinsic obstruction of the duodenum is thought to be related to a failure of recanalization of the duodenal lumen. The duodenum is derived from the distal proportion of the foregut and has a nearly closed lumen during the embryonic period. Between weeks 8 to 10 of gestation, vacuolization begins ultimately leading to a patent lumen. Failure of this process leads to different anomalies that can be classified into three major types: 1) complete atresia caused by a mucosal/

submucosal diaphragm (Fig. 3A); 2) obstruction of the duodenum caused by a short fibrous cord connecting the proximal and distal proportion of the atretic duodenum (Fig. 3B); and 3) complete separation of the proximal and the distal part of the duodenum (Fig. 3C).

One study of 77 patients with congenital duodenal obstruction classified the patients in three groups based on their respective intraoperative findings: 42% (32 patients) had duodenal atresia, 39% (30 patients) presented with an annular pancreas and only 19% (15 patients) had a duodenal web causing the obstruction (2). With a frequency of 38-55%, patients with duodenal obstruction have further congenital anomalies (1). Trisomy 21, intestinal malrotation, situs inversus and congenital heart diseases are the most important (3, 4). Duodenal obstruction is also associated with prematurity and low birth weight.

Signs and symptoms suggesting a duodenal obstruction differ widely depending on the underlying defect. Between 30-59% of patients with proximal intestinal obstruction have a maternal history of polyhydramnios during pregnancy (1). Vomiting shortly after initiation of feeding, feeding intolerance and failure to thrive are among the first symptoms babies present with. Dehydration and changes in electrolytes are possible complications. In 85% of all cases, the obstruction is distal to the papilla of Vater, and therefore gastric aspirates and vomiting will be bilious. A gastric tube is

recommended to reduce vomiting and lower the risk of aspiration. Abdominal distention, in contrast, is rare as the obstruction is proximal.

The incidence of a duodenal web is 1 in 10'000 to 1 in 40'000 (5). Such a web consists of a mucosa and submucosa but does not have a muscular layer (Fig. 3D, 3E). The diagnosis duodenal stenosis is often delayed because of the incomplete character of the obstruction and the resulting tolerance to limited feedings.

Plain X-rays and ultrasonography are often unremarkable. A typical finding during upper gastrointestinal series is the so-called "windsock sign", peristalsis of the proximal part of the gut resulting in a prolapse of the membrane into the distal part of the lumen (6, 7).

There are, in essence, two therapeutic options: Surgery entailing resection of the obstruction followed by duodenoduodenostomy is well established. In some cases, endoscopic resection of duodenal webs has been described. However, this latter procedure is being discussed controversially. Supporters promote this procedure because of its minimally invasive character; opponents criticize the high risk of injury to the papilla of Vater.

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