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Case report/Kazuistyka

Gastric outlet obstruction caused by duplication of duodenum – A case report and review of literature

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ABSTRACT

Gastrointestinal tract duplications are rare congenital lesions. They can occur anywhere from the mouth to the anus with a reported incidence of 1 in 18.00 to 1 in 4500 live births. Duodenal duplication cysts represent 2–12% of all duplications. Authors present the case of 5-month-old boy admitted to Pediatric Surgery Department with the symptoms of mechanical obstruction of alimentary tract and gastrointestinal bleeding (melena). US of the abdomen was suggestive of stenosis of pyloric muscle. CT scan provided correct, definite diagnosis of the duplication cyst of the duodenum. The diagnosis was confirmed during surgery and by histopathology examination, which revealed the presence of ectopic pancreatic tissue within the duplication cyst. In the presented case, it was sufficient to excise the duplication cyst alone with the stripping of the mucosal lining. Closed approach allowed for effective relief of obstruction, a rapid postoperative recovery, and the early reinstitution of feeding after 24 h. Duodenal duplication cysts are rare, and some of them can be detected after significant growth with development of compressive symptoms; concern about malignant change makes surgery and complete excision, the preferred method of treatment; because they are unique entities, the surgeon should be familiar with the anatomy and clinical characteristics of these lesions.

Introduction

Gastrointestinal tract duplications are rare congenital lesions. They can occur anywhere from the mouth to the anus with a reported incidence of 1 in 18.00 to 1 in 4500 live births [1, 2]. Gastrointestinal duplication is defined as a congenital lesion that has an anatomical relationship with some part of the alimentary tract and is lined by the alimentary tract mucosa and has a well-developed coat of smooth muscle [3]. Among gastrointestinal duplications, 75% are cystic, with no communication to the adjacent alimentary tract, while the remaining are tubular, and may communicate with the intestinal lumen [2, 4].

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Duplications of the jejunum or ileum (44–53%) comprise the most common type, followed by duplications of colon (13–15%), stomach (7–9%), duodenum (4–6%) and rectal duplications [5, 6]. Gastrointestinal tract duplications in 50% of patients are associated with other innate malformations as the following: vertebral defects, pancreatic heterotopias and duplications of other part of the digestive tract [7].

No single hypothesis can explain all possible combinations of duplications, its location and associated anomalies [2, 8]. The intrauterine vascular accident theory suggests that gastrointestinal duplications arise from an intrauterine vascular accident during early development of the fetus [2, 9]. Other proposed theories are the diverticulum theory of Lewis and Thyng, and the theory by Bremer of errors of recanalization, taking place between 6th and 8th week of embryogenesis. These theories can explain duplications in esophagus, small bowel and colon [2, 10]. According to the abortive twinning theory, gastrointestinal tract duplications represent incomplete twinning [2]. This theory could explain the colorectal duplications and duplications of the hindgut that are associated with genitourinary malformations [2, 11].

Duodenal duplication cysts are rare, and they represent 2–12% of all duplications [12, 13]. Typically, symptoms of the duodenal duplication cyst are vague, consisting of upper abdominal pain, failure to thrive or early satiety. The patient also may present with jaundice or pancreatitis, and with presence of palpable abdominal mass located in the epigastrium [2]. We report a rare case of gastric outlet obstruction caused by duplication cyst of duodenum with ectopic pancreatic tissue in a 5-month-old boy.

Case report

A 5-month-old boy was admitted to our Pediatric Surgery Department with the symptoms of mechanical obstruction of alimentary tract. The boy was born at term, spontaneous vaginal delivery, with 3400 g birth weight, and 10 Apgar score. Five days before the admission, the boy passed one black stool, and after that, the mother of the boy observed progressive, increased, nonbilious vomiting. During those days, the boy lost 700 g, and on the admission, his weight was 6.2 kg and the patient showed symptoms of dehydration and electrolyte imbalance. The X-ray of the abdomen showed gas in the stomach, and nearly no gas in the rest of the alimentary tract. During an attempt of upper gastrointestinal series, the patient vomited up barium swallow, and the X-ray showed only enlarged stomach (Fig. 1). US of the abdomen revealed a cystic lesion in epigastric region measuring 2.4 cm × 1.6 cm, hypertrophy of pyloric muscle 5 mm, and pyloric channel length of 26 mm suggestive of stenosis of pyloric muscle. Because of the previous symptom of gastrointestinal bleeding (the patient passed one black stool), as a next step in diagnosis, the boy had had gastroscopy, and endoscopic image showed a submucosal bulge blocking the gastric outlet in the site of the pylorus (Fig. 2). Finally, computer tomography scan was suggestive of the congenital duplication cyst of the proximal part of the duodenum (Fig. 3). After achieving the hydro-electrolytic balance, the patient was operated on. We performed elective laparotomy *via* transverse supraumbilical incision. The hypertrophic pylorus was externalized. Pyloric and proximal part of the duodenal serosa and external muscular layer were opened. In the common serosa and muscular layer, beginning at the pylorus level, there was a cystic mass 2 cm × 1.5 cm, obstructing but not communicating with the lumen of the pyloroduodenal canal. The cystic mass was enucleated without damage to the pyloric channel and the duodenum, and primary repair was done (Fig. 4). The excised cyst contained clear mucoid fluid. Histologic examination revealed duodenal mucosal lining with fibromuscular tissue, with the ectopic pancreatic tissue, consistent with the diagnosis of the congenital duplication cyst of the duodenum. The postoperative period was uneventful. The patient was discharged on the 9th day after the operation. The boy was asymptomatic for 6 months of follow-up.

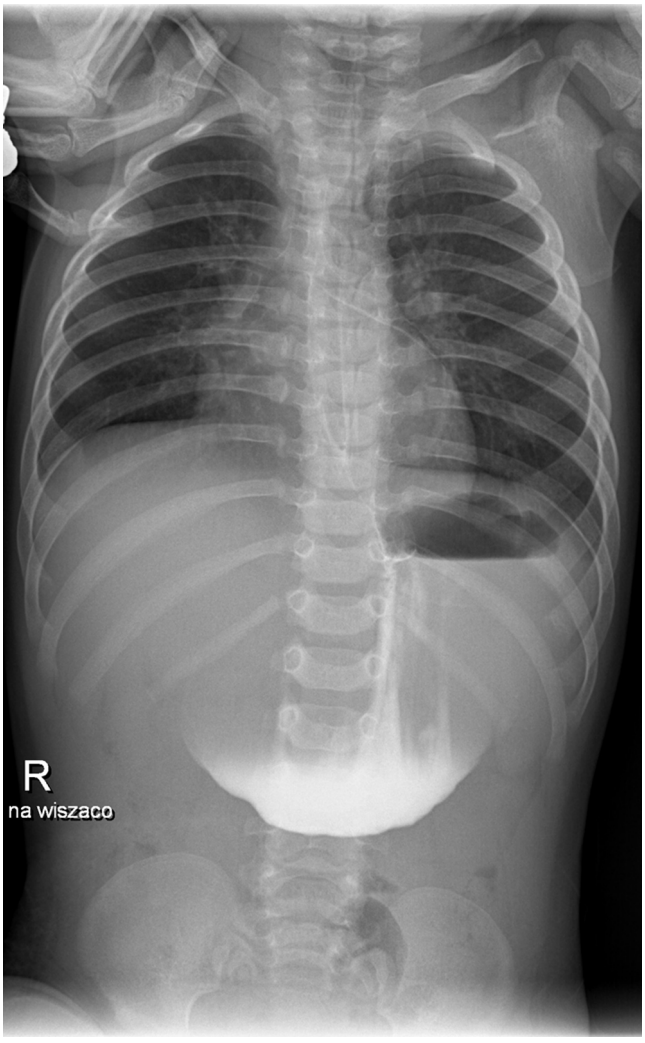

Fig. 1 – Diagnostic imaging: barium study showing enlarged stomach

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