Portal venous gas and biliary sludge in infantile pyloric stenosis

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Background: A 1-month-old infant with recurrent vomiting for 2 days was diagnosed with gas in the portal vein. This finding is usually regarded as an ominous sign.

Methods: Physical examination, plain abdominal X-ray, abdominal ultrasonography and pyloromyotomy were performed.

Results: Hypertrophic pyloric stenosis, gas in the portal vein and sludge in the gallbladder were diagnosed.

Conclusions: The benign course of this patient and the patients reported previously shows that portal gas associated with hypertrophic pyloric stenosis preoperatively may not be an ominous sign and the definite surgical treatment should not be delayed.

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Key words: portal vein; venous gas; pyloric stenosis

Introduction

as in the portal vein during infancy is usually regarded as an ominous sign, typically being diagnosed by plain abdominal film or more often by ultrasonography. Classically, this finding has been described in necrotizing enterocolitis, Hirschsprung's disease, and intestinal obstructions involving bowel distention. [1,2]

Pyloric stenosis is the most commonly encountered gastrointestinal obstruction during infancy, and ultrasonography is the primary choice for diagnosis. However, the association of pyloric stenosis with gas in the portal venous system has been reported only twice^[3,4] and no case of gas in the gastric veins has been described.

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Case report

A 4-week-old female infant was brought to the emergency department because of vomiting of stale milk after every breast-feeding for two days. Her mother had a normal pregnancy of 39 weeks, but the labor was complicated by premature rupture of membranes. The infant was delivered by Caesarian section owing to breech presentation, and was treated with ampicillin and gentamycin until blood cultures were negative.

Physical examination showed that the infant was well nourished and well developed but in a state of mild dehydration. Blood test revealed metabolic alkalosis pH=7.6, bicarbonate 30 mmol/L, Na 133 mmol/L, K 3.3 mmol/L, Cl 88 mmol/L, Urea 6.7 mmol/L (Normal: ≤8), and diastase 10 U/L (Normal: <100). Hemoglobin and white blood cell were normal. The history and blood test results were compatible with those in the clinical diagnosis of pyloric stenosis. Olive was not palpable.

Imaging studies included abdominal ultrasonography and plain films. Abdominal ultrasonography revealed a severely thickened pylorus, with a diameter of 12 mm, a length of 20 mm, and a muscular wall thickness of 4.5 mm. During the study, no opening of the pyloric canal was observed. A sonographic picture was compatible with hypertrophic pyloric stenosis (Fig. 1). [5] Ultrasonography was performed by a radiologist using a Philips HDI 5000 System (Philips Ultrasound, Bothell, WA) with a C7-4 MHz and an L12-5 MHz probe.

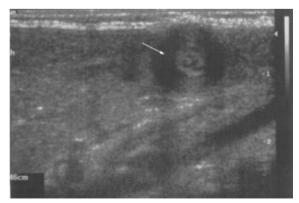


Fig. 1. Longitudinal view. A transverse cut of the thickened pyloric muscle is seen. This target like structure minimally pushing out the thin abdominal wall is often referred to as the pyloric "olive" on physical examination.

The liver was of normal size and echostructure, remarkable only for a multitude of echogenic foci studding the liver. A sonographic picture was compatible with portal venous gas (Fig. 2). Echogenic foci were also detected in the extra- and intra-hepatic portal veins and in the splenic and left gastric veins (Fig. 3). Sonographic findings were compatible with moving gas bubbles in the portal system. Echogenic foci were not demonstrated in the superior mesenteric vein. The relative positioning of the superior mesenteric artery to the superior mesenteric vein was normal.

The gallbladder was of normal size and wall thickness, but its content was not clear fluid. The multitude of internal echoes was suggestive of very thick bile and/or sludge.

Plain abdominal films showed a distended stomach but did not show signs of gas in the stomach wall or in the portal system.

The infant was transferred to the pediatric surgical department. Laparotomy with pyloromyotomy was



Fig. 2. Transverse view of the liver. A classic ultrasonographic picture of portal venous gas in the liver. Bright echogenic spots, brighter and more outstanding than the echogenic wall structures of the normal liver vasculature.



Fig. 3. A transverse view of the midline at the level of the celiac artery. Bright white (echogenic) foci corresponding to portal venous gas are seen. Long arrow points to proximal segment of the splenic vein. Short arrow points to the proximal main portal vein.

performed on the following day. An olive was palpated after anesthesia on the operating table. During the laparotomy, the stomach, duodenum and bowel appeared normal. The operative and post-operative course was uneventful. The infant recovered and resumed breast-feeding from postoperative day 2. Ultrasonography performed 48 hours postoperation detected no signs of gas in the portal vein and the splenic or gastric veins. The infant was lost to further follow-up.

Discussion

Portal echogenic foci, representing portal gas, are classically observed in association with necrotizing enterocolitis in premature babies, Hirschsprung's disease and volvulus. They are thought to be related to the loss of mucosal integrity caused by ischemic and inflammatory processes in the intestinal wall. In these and other conditions that portal gas is observed, the gas is thought to enter the portal system from the veins or lymphatics of the intestinal wall. In the present case we detected the moving portal gas bubbles in unusual proximal locations, both in the splenic and in the left gastric veins pointing to the gastric wall as the "source" of the gas.

On plain abdominal films, no intramural gas was observed in the gastric wall. We assumed that gastric distention and recurrent vomiting that resulted from gastric obstruction caused wall injury and loss of integrity in the gastric mucosa and an increased diffusion of gas in the gastric veins and the portal system.^[6]

In cases of portal gas related to stomach distention, intramural gas was not seen in the stomach wall and portal gas resolved in a day.^[7] This is the third case reported with portal gas in an infant with pyloric stenosis and the first report describing air in the splenic and left gastric veins.^[3,5]

Biliary sludge presumably evolves as a result of 48 hours of recurrent vomiting. This rapid process of the disease reflects the high lithogenicity of infantile bile^[8] and clearance of the gallbladder sludge 48 hours after operation.

The benign course of our patient and those reported previously shows that portal gas associated with congenital hypertrophic pyloric stenosis preoperatively may not be an ominous sign and should not delay the definite surgical treatment.

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Contributors: KA proposed the study and wrote the first draft.

GE analyzed the data. All authors contributed to the design and interpretation of the study and to further drafts. AY is the guarantor.

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