CASE SNIPPETS

Esophago-gastric dissociation complicating devascularization procedure for portal hypertension

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Devascularization surgery for portal hypertension, indicated in selected patients, is considered safe. A 15-year-old boy with cirrhosis underwent elective modified Sugiyama’s devascularization procedure for secondary prophylaxis of variceal bleeding. He developed esophago-gastric dissociation, which was successfully managed with emergency feeding jejunostomy and restoration of gut continuity after 5 months later. [Indian J Gastroenterol 2002;21:76-77]

Key words: Portal hypertension surgery

Devascularization for portal hypertension has undergone many modifications to reduce the risk of sutural insufficiency due to ischemia. We report an unusual complication of devascularization procedure for portal hypertension in the form of esophago-gastric dissociation.

A 15-year-old boy with Child B cirrhosis underwent elective modified Sugiyama’s devascularization procedure for secondary prophylaxis of variceal bleeding. The modification consisted of one-stage surgery done by an upper midline incision. While performing devascularization of the stomach, the veins were ligated very close to the wall of the stomach, as in highly selective vagotomy, thereby saving the vagal innervation to the pylorus, obviating the need for a drainage procedure. Devascularization of the lower 7-10 cm of esophagus was achieved transthoracally. Instead of esophageal transection, all the veins were ligated transmurally 1.5 cm above the esophago-gastric junction from the serosal side, by placing interrupted 2/0 silk sutures (3-4 in each quadrant), with a nasoesophageal tube in position for guidance. These sutures were placed alternately higher and lower by half a centimeter difference, to avoid ischemia to the wall of the esophagus. This was followed by applying continuous interlocking sutures through the wall with 2/0 silk in the stomach 3-5 cm below the esophago-gastric junction. The spleen was not removed. A floppy Nissen fundoplication was performed to provide coverage for any minor esophageal leaks and to prevent gastro-esophageal reflux. A liver biopsy was taken before closing the abdomen.

On the fifth postoperative day, the patient developed distension of abdomen, mainly in the left flank. On ultrasound-guided aspiration, 300 ml of milky white thick pus came out and a provisional diagnosis of subphrenic abscess was made. On recurrence 2 days later, a tube drain was put in the left flank; this drained 500-600 ml thick pus every day for the next 3 days. The patient continued to deteriorate with the picture of uncontrolled intra-abdominal sepsis. Gastrograin study revealed leakage in the area of the esophago-gastric junction and a gastric fistula from the greater curvature. Exploration revealed almost complete esophago-gastric disconnection (with only the posterior wall intact) and a gastric fistula from the greater curvature. Because of the precarious condition of the patient, no definitive surgery was performed at this time. The lower end of the esophagus and cardiac end of stomach were closed with 2/0 silk: a feeding jejunostomy was performed after closing the gastric fistula on the greater curvature. The abdomen was closed by tension suturing after thorough peritoneal lavage. The patient was nursed intraluminally, in Trendelenburg position with a Ryle’s tube in situ for aspiration of saliva. He gained weight and was discharged after 4 weeks.

He underwent restoration of gastrointestinal continuity 3 months later by refreshing the edges of the esophageal stump and re-implanting it in the gastric cardia. There was minor leakage from this anastomosis, which healed on conservative treatment. Gastrograin study after 21 days showed no leakage, and the patient was allowed gradual resumption of oral feeds. The jejunostomy was removed 6 weeks after resumption of oral feeds. The child is now gaining weight on a normal diet. His only complaint is of postprandial heavyness that responds to domperidone.

Sutural insufficiency due to ischemia is a unique complication of Sugiyama’s procedure with minor leaks having been reported in 0%-18% of cases. This is because transection of the lower end of the esophagus results in interruption of blood flow over a wide area, and the subsequent anastomosis is nourished only by blood coming from the walls of the esophagus and stomach. It has been shown that undernourished patients (serum albumin <3 mg/dl, or total lymphocyte count <900, or 10% weight loss in 6 months, or reduction of one or more anthropometric parameters below 50th percentile of normal value) develop more complications. Modifications of Sugiyama’s devascularization procedure that avoid transection of the lower end of the esophagus have many advantages: no danger of leakage, tension or stricture at the transection site, simultaneous disconnection of gastric and esophageal varices without transecting the fundus, preservation of distal esophageal function, avoidance of opening the gastrointestinal tract, and retention of access and good vascularity for healing. In our own experience of over 40 cases, there was not a single leak. We are unable to pinpoint the reason for the complication in this case but hypothesis that hypoproteinemia (serum albumin 2.8 mg/dl) may have added to the ischemia between two suture lines, one on the esophagus and another on the stomach.

Sutures on the esophagus and stomach along with ligation of veins very close to the wall of the stomach, as in highly selective vagotomy, should be avoided as it can result in such a complication.

References

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Spontaneous duodenal perforation in a newborn
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A full-term 4-day-old male baby presented with spontaneous duodenal perforation into the lesser sac. Exploration revealed a perforation in the posteromedial wall of the second portion of the duodenum. The perforation was closed and the baby recovered uneventfully. [Indian J Gastroenterol 2002;21:77]

Key words: Neonatal gastrointestinal perforation, stress ulcers

Duodenal perforation is an unusual cause of pneumoperitoneum in a neonate. Most perforations appear to occur in stress ulcers. We report a neonate with duodenal perforation into the lesser sac, who was apparently well till the event.

A four-day-old male baby was brought to the hospital with complaints of abdominal distension for 2 days, bilious vomiting for 1 day and poor urinary output. The baby had been delivered vaginally, with no apparent birth trauma; he was well on the first day of life and had taken breast feeds. There was no history suggestive of birth asphyxia, or early neonatal sepsis. On the second day of life, the baby became lethargic and refused feeds, following which there was gradual abdominal distension and vomiting. On examination, the child weighed 2.7 Kg, was alert and active. The abdomen was soft, with mild distension and ill-defined tenderness in the periumbilical area. There was bilious aspirate in the nasogastric tube. Bowel sounds were absent. Other systems were normal.

Investigations: hemoglobin 16 g/dL, serum urea 42 mg/dL, creatinine 1.3 mg/dL, normal serum electrolytes, serum bilirubin 2.9 mg/dL. X-ray of the abdomen revealed pneumoperitoneum with a suggestion of free air in the lesser sac.

The child was resuscitated and was taken up for surgery. At surgery, there were puntaneous fibrinous flakes in the lesser sac with a perforation in the posteromedial wall of the second portion of the duodenum. The visualized mucosa around the perforation was normal. After mobilization, the perforation was closed in a single layer with interrupted 5/0 silk sutures. A gastrostomy was created and a transgastric feeding tube was inserted. The postoperative course was smooth, and a dye study done on the 7th postoperative day showed an intact duodenum. The feeding tube and gastrostomy were removed and the child tolerated oral feeds well. Presently the child is 4 months of age, and is thriving well.

Perforation of the gastrointestinal tract in the newborn is usually a consequence of necrotizing enterocolitis.1 Gastric perforations are the next important cause of pneumoperitoneum, and these are rare. Duodenal perforations are even more unusual. A relatively high acid secretion in the first week to ten days of life2 coupled with perinatal stress, is thought to lead to secondary stress ulcers in newborns. These ulcers, in contrast to those in adults, tend to occur in the duodenum and perforate rather than bleed.2

Almost all reported cases have been associated with major perinatal illness.3,4 An association with steroid use in babies with bronchopulmonary dysplasia has also been reported.5 Duodenal perforation in otherwise healthy neonate has been reported only once,2 and as in this case the precise etiology is unclear.

Perforation is usually into the general peritoneal cavity, but this patient had perforated into the lesser sac, which led to the paucity of clinical manifestations of peritonitis.

Although mortality of 40% has been reported,2 aggressive early exploration has led to better results.3,4 Thus, duodenal ulcer perforation must be kept in mind in the differential diagnosis of pneumoperitoneum in the newborn.

References

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