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Neonatal gastric perforation

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This male infant, twin A, was born to a 34-year-old G3/P3 by Cesarean section at 37 0/7 weeks of gestation after an uneventful pregnancy. The newborn adapted well, with an arterial cord-pH of 7.23 and Apgar scores of 7, 9, and 10 at 1, 5, and 10 minutes, respectively. His birth weight was 2470 g. Physical examination was unremarkable, except for hypothermia, for which the patient was admitted to our neonatal ward. Adaptation of twin B was uneventful.

Initial routine laboratory studies revealed a low white blood cell count (WBC) of 8500/mm³ with an absolute neutrophil count of 2590/mm³. Blood glucose, C-reactive protein and capillary blood gas analysis were normal. Body temperature stabilized, the clinical exam remained unremarkable and the patient was discharged back to the maternity ward.

At 72 hours of age, the leukocyte count had decreased to 5100/mm³ with an absolute neutrophil count of 970/mm³. When the leukocyte count fell to 4600/mm³ with an absolute neutrophil count of 780/mm³ at 96 hours of life, with still normal values for CRP and blood sugar, the patient was admitted to the NICU.

Over a period of 6 hours, the infant became increasingly irritable, tachycardic and tachypneic. The abdomen was tender and distended. He developed emesis, which subsequently turned bilious. A nasogastric tube was inserted, feedings were discontinued and intrave-

nous fluids and antibiotic therapy were started. Abdominal radiographs were obtained (Fig. 1, 2) which demonstrated extraintestinal air, consistent with the diagnosis of intestinal perforation. Over the next hour, while preparations for laparotomy were under way, the abdominal distension continued to worsen. Shortly before laparotomy, WBC diminished further to a value of $1300/\text{mm}^3$ with an absolute neutrophil count of $380/\text{mm}^3$, a CRP value was 2.5 mg/l and clotting studies were normal.

At emergency laparotomy, extensive necrosis and perforation of the stomach extending from the greater curvature to the cardia were observed. Peritoneal fluid cultures grew staphylococcus aureus as a mono-culture. Pathologic examination of the resected stomach tissue showed acute hemorrhagic necrosis (Fig. 3).

The postoperative course was complicated by bilateral small scrotal abscesses, one of which perforated spontaneously. The patient was discharged home after 21 days of hospitalisation.



Fig. 1

Abdominal X-ray in supine position (vertical beam).



Fig. 2

Abdominal X-ray in left lateral decubitus position (horizontal beam).

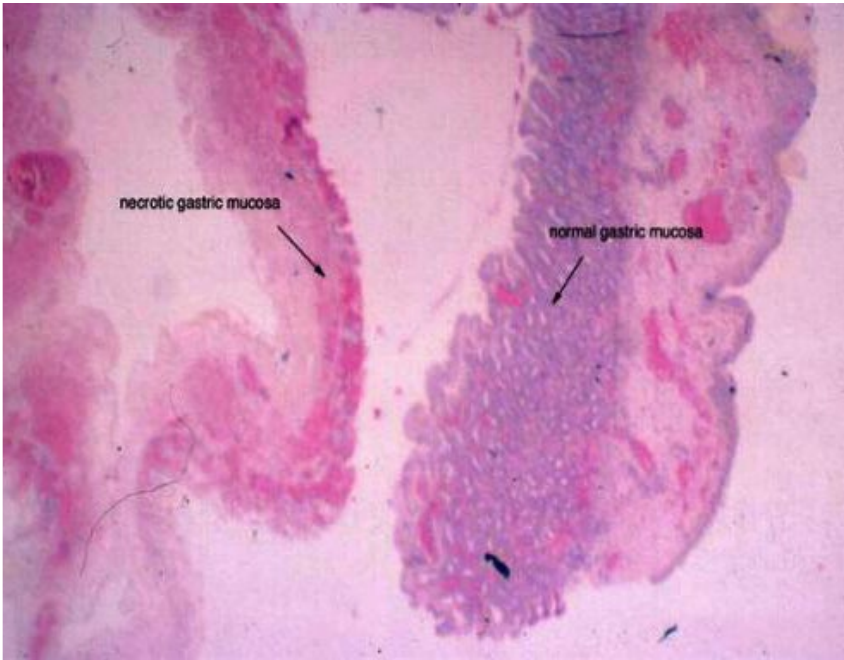


Fig. 3

H&E stain of gastric specimen showing areas with necrotic gastric mucosa.

DISCUSSION

Gastric perforation is a rare condition that accounts for 10 – 16% of all gastrointestinal perforations (1). The usual age of presentation is 2 to 7 days in term infants. The incidence is higher in black than in white infants and is at least 4 times higher in males than in females (2). Most cases of gastric perforation were considered spontaneous in the older literature, whereas more recent retrospective reviews reveal several associated gastrointestinal lesions and contributing factors (3).

Deficiencies in the gastric muscle layer have been described by a number of authors, but it is probable that muscle layer deficiencies are a result of perforation rather than the cause. In 1965, Shaw et al (4) experimentally proved the latter theory in dogs by tying off the esophageal and duodenal ends of the stomach and insufflating the organ with air until it perforated. All perforations occurred on the greater curvature, and when examined histologically, all specimens showed lack of musculature close to the site of perforation. He concluded that this was a non-specific finding produced by mechanical rupture of the stomach, as a result of increased intragastric pressure. When distension is sufficient to force muscle bundles to separate from one another, rupture can occur in the weakened area.

Traumatic causes are described, especially secondary to feeding tube placement or vigorous respiratory resuscitation (2). Drug associated gastrointestinal perforation

ration has been noticed under treatment with dexamethasone or indomethacin (4,5). Prematurity, low birth weight, necrotizing enterocolitis and isolated gastric ischemia due to hypoxia are considered to be additional risk factors (6). There seems to be an association with prenatal conditions such as premature rupture of membranes, chorioamnionitis, gestational diabetes or maternal group B streptococcal infection leading to emergency cesarean delivery (2).

In our patient, none of the above-mentioned maternal, prenatal or postnatal risk factors were identified. Nonetheless, we believe that our patient must have suffered from isolated gastric ischemia leading to extended necrosis and neutrophil consumption. Pathologic examination of the resected stomach tissue showed acute hemorrhagic necrosis. Early diagnosis and rapid management allowed this infant to survive this rare abdominal catastrophe.

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