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Spontaneous gastric rupture in a 22-month-old boy: Case report and review of the literature
The diagnostic work up and management of a rare pediatric emergency

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ABSTRACT

We report the case of 22-month-old boy who sustained spontaneous gastric rupture. The patient had an episode of nausea and vomiting after a large meal the day before admission to our emergency ward. Abdominal ultrasonography revealed free and corpuscolate fluid in the abdomen. Abdominal X-ray showed free air in the abdominal cavity, leading to diagnosis of gastrointestinal perforation. Blood examination revealed metabolic acidosis. An emergency laparotomy detected a wide perforation of gastric wall involving fundus and greater curvature. A sleeve gastrectomy was performed with two layers closure of abdominal wall. Idiopathic gastric rupture, beyond neonatal period, is extremely rare in childhood. Early diagnosis and surgery are mandatory to avoid fatal complications.

Gastric rupture caused by acute distension is a rare event when occurring in children beyond neonatal period; its clinical course progresses rapidly and its mortality is high [1,2]. Few cases of spontaneous gastric rupture in infants over 1 year have been reported recently in particular in Chinese and Japanese literature [1,3]. Early diagnosis and prompt treatment are crucial to avoid life-threatening complications as metabolic acidosis and ipovolemic shock [2–4]. We report the case of 22-month-old boy who sustained spontaneous gastric rupture and a short review of the literature.

1. Case report

A 22-month-old boy was admitted to our emergency department because of lethargy, nausea and vomiting after a large meal the day before admission. No history of blunt trauma was reported and this was confirmed by the absence of hematoma or bruises at abdominal level. His past medical, perinatal and family histories were unremarkable. The infant weighed 15 kg. Physical examination on admission revealed a well-developed child who appeared lethargic, with respiratory distress, tachypnea and gross abdominal distension. Laboratory examination on admission showed leukocytosis (GB 17480/mmc, N 74,1%), marked acidosis and hypoglycemia. An abdominal ultrasonography revealed free and corpuscolate fluid in the abdomen. Abdominal X-ray showed free air in the abdominal cavity, leading to diagnosis of gastrointestinal perforation (Fig. 1). An emergency transverse supraumbilical laparotomy was performed. After removing approximately 2000 ml of light-brown fluid containing gastric juice and partially digested food particles, a gastric rupture (15 × 2 cm) was detected on the fundus and along the greater curvature (Fig. 2). The gastric wall, at level of perforation, showed hemorrhage, erosion and necrosis. The absence of gastric duplication and of any abnormality at the outlet of the stomach was confirmed by manual exploration. A 4/0 Monocryl two layers closure, the first interrupted and the second running, was performed after trimming of the not viable gastric wall at rupture level. Postoperative treatment included fluid infusion, correction of acidosis and electrolytic imbalance. Total parenteral nutrition was established for six days postoperatively.

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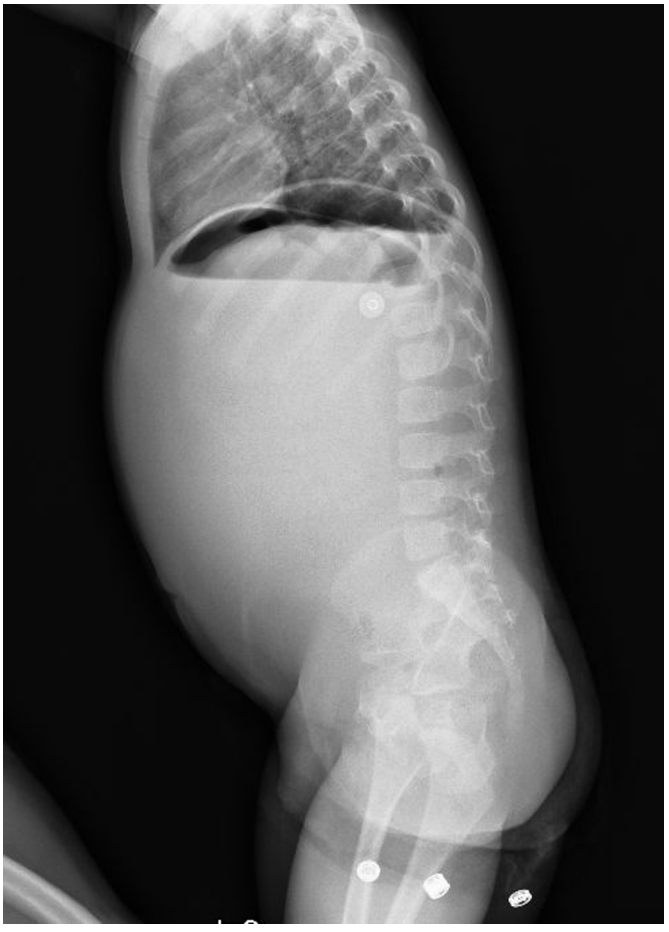

Fig. 1. Abdominal X-ray shows free air in the abdominal cavity, leading to diagnosis of gastrointestinal perforation.

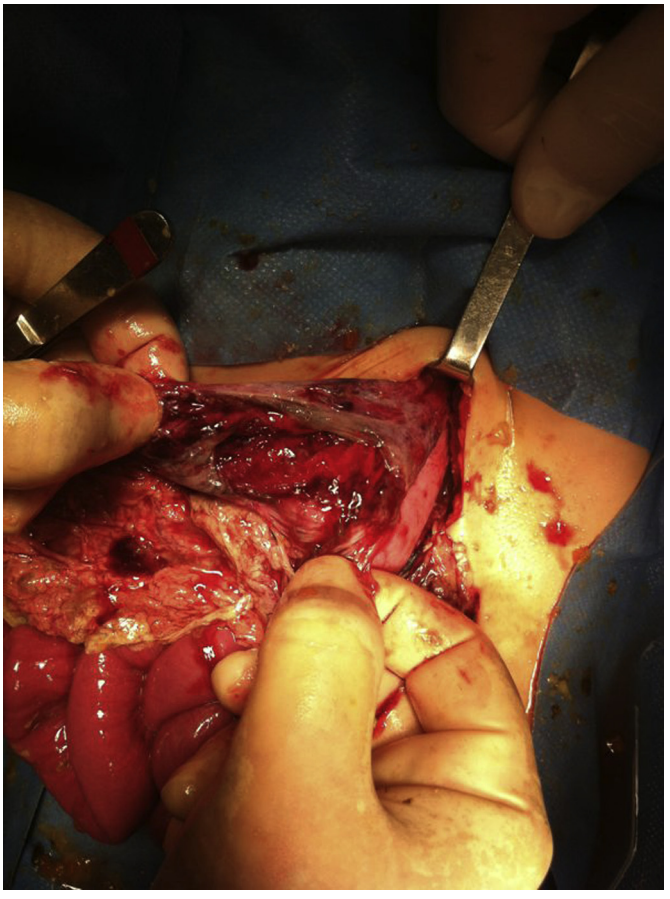

Fig. 2. Intra-operative findings of wide gastric rupture (15 × 2 cm), detected at the level of the fundus and along the greater curvature.

Antibiotic therapy with cephalosporin, metronidazole and gentamycin was employed. Oral food intake was restored on VI post-operative day without complications. Microscopy study revealed a normal gastric wall structure with necrosis, hemorrhage and acute inflammation; and there were no findings such as ulceration, a muscle defect, or thrombosis. We were unable to determine the causative factor of the rupture from histologic examination.

2. Discussion

Gastric rupture is a potential fatal condition occasionally encountered in adults and neonates but it rarely occurs in children over 1 year of age [3]. In children in pre-school age this condition is more frequently reported in Chinese and Japanese literature [1,3]. In most adults, the cause of gastric perforation is local carcinoma or peptic ulcer, although a few cases of spontaneous gastric rupture were reported [1]. Recently it has been reported that people with Prader–Willi syndrome are at significant risk for gastric perforation, in particular this pathology accounts for 3% of deaths. Vomiting and abdominal pain, although rare in Prader–Willi syndrome, were frequent findings in this cohort [5]. Our patient weighted 15 kg and he was 92 cm tall at 22 months. However no clinical features typical of Prader–Willi syndrome were identifiable.

Gastric rupture is usually seen in premature neonates with asphyxia and low birth weight [2,3,6]. Our patient did not have a past history of prematurity, birth trauma or hypoxia. Regarding causative factors of gastric rupture in neonates there are different hypothesis, such as birth trauma, mechanical injury, idiopathic, congenital muscle defects, hypoxia and increased endoluminal pressure [3,6,7]. Common conditions associated are esophageal atresia, intestinal obstruction and annular pancreas [3]. Some authors suggest that acute gastric distension, particularly in neonates with congenital muscle defects of gastric wall, subsequent to overeating, air sucking or pylorospasm and increased intra-abdominal pressure can cause ischemia of gastric wall and cause gastric rupture [2,3]. Fukata et al. indicate that the causative factor for this condition in preschool children may be similar to what happens in neonates, because his patients exhibited, on histological examination, microscopic gastric muscle wall defects [4]. In our case, histological examination revealed no findings as ulceration, or muscle defect or thrombosis, so we were unable to determine the causative factor of the rupture from the specimen. Increased intraluminal pressure may play an important role in preschool children as well [7]; conversely, some authors suggest that ischemia of the gastric wall was a possible etiology in determining gastric rupture. In particular, organ axial gastric volvulus may result in vascular disturbance, especially at major curvature level as a consequence of the posterior wall stretching [3]. The fundus and the junction between corpus and fundus of the stomach (near to the greater curve) are the most commonly reported site of rupture [1,4]. Our patient did not show ischemic lesions or signs of gastric volvulus even if the site of rupture was the posterior wall of the greater curvature. No history of blunt trauma was reported.

Millar et al. postulated that typical signs and clinical features suggestive of gastric rupture are: tympanic abdominal distension, tenderness of the abdominal wall, subcutaneous emphysema and

evidence of shock. Vomiting is an uncommon manifestation of gastric perforation [8]. Once gastric rupture occurs, patient's condition will deteriorate progressively with: dehydration, acid-base imbalance, electrolyte disturbance (hypochloremia, hypocalcemia, hyponatremia), gradual development of pneumoperitoneum and respiratory distress [1,3]. Rupture due to mechanical injury leads to rapid hemodynamic changes thus predisposing to deadly shock. Hemoconcentration is a late manifestation of toxic shock and the main factor of decreased blood flow.

A review of literature regarding 11 cases of spontaneous gastric rupture in preschool age children highlights the following features: higher incidence in females, posterior gastric wall at level of greater curvature is the most common site of rupture, the defect observed is usually round [3]. A recent history of a common cold treated by oral medications, is reported in some patients [1,2]. Rarely subcutaneous emphysema has been observed. In our patient three of the previous characteristics were present.

When diagnosis of gastric rupture is suspected an emergency laparotomy should be performed and resection of the non-viable tissue performed. A suture in two layers is then performed and temporary gastrostomy can be performed in selected cases [1,4]. A jejunal feeding tube should be placed for enteral nutrition [1]. The patients must be carefully followed in the post-operatory period because of the risk of the delayed necrosis of the gastric wall. Broad spectrum antibiotic therapy is of great importance to avoid the risk of sepsis.

Some authors have suggested a conservative management by placing gastric tube to decompress and drain the stomach but high risk of electrolyte imbalance and acute shock should be considered [1]. The prognosis, in these cases, is generally good with appropriate surgical treatment, but delayed diagnosis and metabolic acidosis are associated with a poor prognosis.

3. Conclusion

Spontaneous gastric rupture is rare condition in childhood beyond the neonatal period. It should be considered in differential diagnosis, in children in pre-school age, in case of rapid onset of abdominal distension, dyspnea, dysphoria, coffee-ground fluid vomit, lethargy and free air on abdominal X-ray. Early diagnosis and emergency laparotomy will reduce complications and mortality.

Author contribution

G.A.M. concept and designed the study; V.D.C., R.B. and C.G. acquired and interpreted clinical data; V.D.C., F.M. and G.A.M. all contributed toward drafting and approving the final manuscript.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Conflicts of interest

There are no relationships, conditions, or circumstances that present a potential conflict of interest.

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