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Case Report

Spontaneous neonatal gastric perforation in a very preterm neonate

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ABSTRACT

Gastric perforation in neonates is a rare, serious and life-threatening problem. The precise aetiology is obscure in most cases. By virtue of its high mortality rate, it requires prompt recognition and surgical intervention. We report a case of gastric rupture in a very preterm/very low birth weight neonate with antenatal reversal of flow in umblical artery.

Keywords: Gastric perforation, Very preterm neonate, Gastric rupture

INTRODUCTION

Gastric perforation accounts for approximately 7% of all gastrointestinal perforations in neonates; it has poor outcomes and a high mortality rate.[1] Over 300 cases have been reported in the literature since Siebold described the first case in 1825.[2] The probable causes of gastric perforation include asphyxia, anatomical abnormalities of the stomach (e.g. congenital agenesis of the gastric musculature) secondary to iatrogenic traumas (e.g. endotracheal and feeding tube insertion), vigorous respiratory resuscitative measures, ventilator use, increased intragastric pressure.[3,4] However, the exact mechanisms of neonatal gastric perforation remain unclear and is greatly debated. We present a case of gastric rupture in a very preterm/very low birth weight neonate presenting on day 3 of life with a rapid increase in abdominal girth and hemodynamic instability.

CASE REPORT

A male baby born at 29+3 weeks with birth weight of 1.34 kg was born by emergency caesarean section in view of severe pre-eclampsia in mother and reversal of flow in umblical artery on Dopplers. Baby required resuscitation at birth in view of respiratory distress and also required non-invasive ventilation for the same. On day 3 of life baby had sudden increase in abdominal girth for which x-ray was done and showed air under diaphragm (Figure 1). Baby was electively intubated and connected to ventilator. Pediatric surgery opinion was sought and exploratory laparotomy was done.

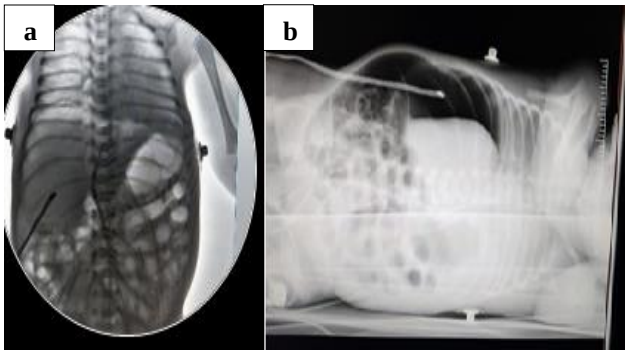

Figure 1: X-ray showing (a) gas under diaphragm, and (b) pneumoperitoneum.

On laparotomy baby was found to have gastric rupture from greater curvature all along up to gastroesophageal (GE) junction to pyloric region with peritoneal contamination (Figure 2). The rupture was sutured in layers and gastrostomy, jejunostomy and a peritoneal drain was placed (Figure 3). Post-surgery abdominal x-rays were reported normal with no evidence of obstruction or perforation.

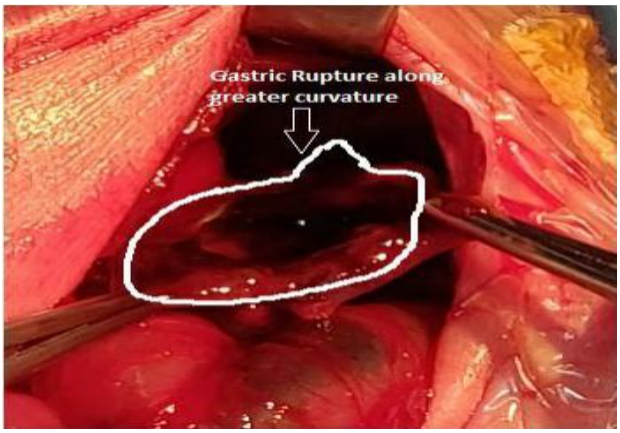

Figure 2: Tear along the greater curvature till gastroesophageal junction.

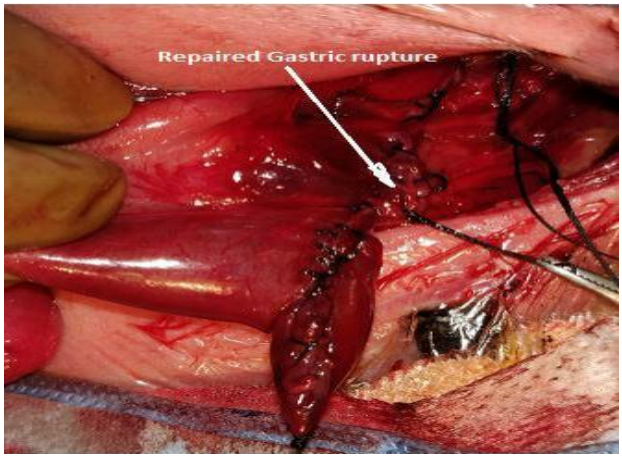

Figure 3: Repair of the tear along greater curvature.

Baby was kept on nil per os (NPO), was on intravenous fluids (IVF), total parenteral nutrition (TPN) and appropriate antibiotics were added. Abdominal girth was monitored closely and bowel sounds were monitored daily. The aspirates from jejunostomy, gastrostomy and nasogastric (NG) tube were monitored and replaced with appropriate fluids. Feeds were started through feeding jejunostomy on post-operative day 5 (5th POD) after bowel sounds returned and adequate drain output were observed. Eythromycin was started on 7th POD to improve gut motility. Baby passed stools regularly after starting of erythromycin along with rectal washes. On POD 7 upper gastrointestinal (GI) dye study was done suggested small capacity stomach, reflux present normal C loop, normal proximal loop, and no leak to peritoneum. Gradually feeds were switch over to gastrostomy tube on POD 13. Peritoneal drain and jejunostomy tube was removed on POD 16 and POD 17 respectively. Baby tolerated the feeds well and the feeds were increased slowly. Oral feeds were started at POD 24 which the baby tolerated well. Feeds were seesawed with feeding gastrostomy and full oral feeds were reached on POD 32. Gastrostomy tube was removed on POD 34. Currently baby is feeding well on direct breastfeeding (DBF) and pallada expressed breast milk (EBM) feeds.

DISCUSSION

We reported a case of gastric rupture in a very preterm infant with antenatal Dopplers showing reversal of flow in umblical artery indicating ischemia to gut. However factors like prematurity and low birth weight may have also contributed to it.

Three mechanisms have been proposed for stomach perforation: traumatic, ischaemic, and spontaneous. Most of the gastric perforations are due to iatrogenic trauma by vigorous nasogastric or orogastric tube placement.[5] Perforation is usually along the greater curvature and appears as a puncture wound or a short laceration. However in our case there was a tear all along the greater curvature.

The mechanism of ischaemic perforation has been difficult to elucidate because these cases of perforation are associated with condition of severe physiological stress, such as extreme prematurity, sepsis and neonatal asphyxia. Because gastric stress ulcers have been reported in a variety of critically ill infants, it has been proposed that these perforations result from the transmural necrosis of such ulcers.[5,6] In our case baby was premature.

Spontaneous gastric perforations have been reported in otherwise healthy infants, usually within the first week of life, particularly in between the first 2 and 7 days of life.[7] One hypothesis is that spontaneous perforations are due to the congenital defects in the muscular wall of the stomach.[8] Anatomical defects of the gastric muscular wall have been suggested to potentiate perforation of the stomach among neonates, especially in prematurity. The circular muscle layer of the new-born stomach normally contains several gaps, most prominently in the fundus and along the greater curvature. These gaps are more common in premature infants.

The clinical manifestation of neonatal gastric perforation includes abdominal distention, feeding intolerance, respiratory distress, poor activity, gastrointestinal bleeding, and abdominal erythema. Hemodynamic changes such as shock which are frequent often complicate the condition and management. The most common radiographic finding in gastric perforation is pneumoperitoneum, which in this case is obvious (Figure 1).

The differential diagnosis of neonatal spontaneous gastrointestinal perforations include necrotizing enterocolitis, septicemia, intestinal obstruction, all of which were ruled out in our case.

Surgical repair of most perforation consists of debridement and two layer closure of stomach. Postoperative vigorous supportive therapy coupled with the use of broad spectrum antibiotics administered intravenously to prevent peritoneal contamination along with intravenous nutrition is necessary.

Due to the associated problems of sepsis and respiratory failure often found in premature infants, mortality rates of gastric perforation are high, ranging from 45% to 58%.[9] Jones et al reported a case series of 4 premature neonates of very low birth weight of which 3 died. This points to the fact that mortality may be higher in premature and low birth weight neonates.[10]

CONCLUSION

Spontaneous neonatal gastric perforation (SNGP) is a rare life threatening surgical emergency with characteristic clinical manifestations including severe abdominal distention, intolerance to feeds, vomiting, and respiratory distress. It has a multifactorial etiology of prematurity, low birth weight, congenital defects in muscular wall of especially premature neonates and mechanical pneumatic rupture. Success in treatment depends on early diagnosis and immediate surgical intervention.

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