Neonatal Gastric Perforation – A Case Report

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ABSTRACT

Gastric perforation in neonates is a very rare surgical emergency and life-threatening condition with uncertain etiology till date. Despite having high mortality rate, pathogenesis and etiology are greatly debated. Because of its high mortality rate, prompt diagnosis and urgent surgical intervention is mandatory.

Herewith we report a case of neonatal gastric perforation having no apparent etiology. A 3 day old full term twin delivery male baby with neonatal gastric perforation presented with abdominal distention and respiratory distress. Baby was with low birth weight and was in septicemia during admission. He was diagnosed radiologically on X ray abdomen showing ‘saddle bag’ appearance and demonstrating gas out lining falciform ligament and managed by resuscitation and surgery. On exploration there was a 1 cm x 1 cm perforation seen on the fundic region along the greater curvature which was circular with pouting mucosa through it, surrounding serosa was hyperemic, no evidence of necrosis or petechial hemorrhage around perforation; primary repair was done.

Postoperatively, despite of intensive care by means of invasive ventilation, intravenous fluids and broad spectrum antibiotics, condition of baby started deteriorating and he expired 40 hours after operation. Cause of death was septicemia secondary to peritonitis in low birth weight baby.

Key words: Neonate, Gastric, Perforation, Peritonitis

INTRODUCTION

Neonatal gastric perforation (NGP) is a rare entity with just 115 cases reported till 2000[1] and was first described by Siebold in 1825 [2]. NGP is higher in black than white and at least 4 times higher in male than female [3]. Despite having high mortality rate, pathogenesis and etiology are greatly debated. Congenital absence of the gastric wall musculature, stress ulceration secondary to neurogenic difficulties, postnatal steroid therapy and ischemia of the gastric wall have been proposed etiological factors [4].

CASE REPORT

A 3-day-old male child, weighing 1960 gm, was admitted with history of abdominal distention and vomiting since one day. On history, antenatal period and parturition were uneventful. He was a full term normal vaginal twin delivery and was first of twins. Baby had cried normally, sucked breast milk well and had passed urine and meconium on first day. No congenital anomalies were detected. For first two days of life, baby was apparently alright, tolerated breast feeding well. On third day of birth, above mentioned symptoms developed, he refused feed and general condition started deteriorating and then brought to hospital. Examination revealed a lethargic newborn with marked abdominal distention, respiratory distress and cyanosis, respiratory rate was 60/ min and heart rate was 158 bpm. Bowel sounds...
were absent and liver dullness was masked. Blood reports were normal. Abdominal X-ray revealed huge free gas under diaphragm displacing viscera medially and giving ‘saddle bag’ appearance and demonstrating gas outlining falciform ligament (fig.- 1). In view of above, the provisional diagnosis of perforative peritonitis was made and urgent laparotomy was planned. On exploration, there was evidence of seropurulent free fluid with flakes, entire bowel was inspected and traced, which were normal. There was a 1 cm x 1 cm perforation seen on the fundic region along the greater curvature. Perforation was circular with pouting mucosa, surrounding serosa was hyperemic, no evidence of necrosis or hemorrhages around perforation (fig.-2). Primary repair after revising the margins was done in single layers with silk, thorough lavage was given, drain was placed and abdomen was closed. Postoperatively, baby was kept intubated, intravenous fluids and broad spectrum antibiotics were administered and nasogastric aspiration was done one hourly. On second post operative day, general condition of the patient started deteriorating and he expired 40 hours after operation. Histopathology of the margins revealed nonspecific inflammation. Cause of death was septicemia secondary to peritonitis in low birth weight baby.

**Figure 2: Per-operative image of the perforation**

**DISCUSSION**

NGP is a rare surgical emergency associated with high morbidity and mortality. Etiopathogenesis of neonatal gastric perforation is unclear but three mechanisms for gastric perforation in neonates are postulated - traumatic, ischemic and spontaneous [4, 5].

Most of the gastric perforations are caused by iatrogenic trauma either due to the vigorous nasogastric tube placement or due to positive pressure ventilation or bag-mask resuscitation [6] but in this case no such evidence of traumatic perforation was identified.

Most common cause of neonatal gastrointestinal perforation is necrotizing enterocolitis. Prematurity has been accepted as a common feature of NGP [7]. Mostly NGP is associated with primary pathology including tracheo-esophageal fistula, duodenal atresia, gastroschisis etc, while it is believed that prematurity, low birth weight, hypoxia and steroid during pregnancy are risk factors for prognosis, but not for gastric perforation [1]. In this case no primary pathology was detected.

Only 20 percent of cases are not related to any underlying risk factor and are considered as spontaneous/ idiopathic. The reported incidence of spontaneous perforation is 1 in 2900 live births [7]. It is believed that spontaneous gastric perforation is commonly found in term babies without any significant pathology, but cases have also been reported in premature, hypoxic, low birth weight babies, premature rupture of membranes and breech, cesarean or twin delivery (in decreasing order of frequency) [4]. In this case, the baby had low birth weight, so it can be assumed that this was spontaneous gastric perforation.

Spontaneous gastric perforation as reported earlier, is more common in the preterm baby with perforations most commonly occurring between the 2nd and 7th days of age. The highest reported incidence of rupture is on the 3rd day of life [9, 10, 11]. This was a full term baby and the perforation had occurred on the third day of life.

Clinical features of gastric perforation are usually those of an acute abdomen. Abdominal distension (most prominent feature) and respiratory distress (severity of which depends upon the distension of abdomen and maturity of patients) [8] are the common presenting features and same were observed in our case. Preterm and septic patients are more prone to early distress [11]. Most infants are being fed normally up to the time of perforation. Vomiting was insignificant [8] but was present in our case. Septicemia was a relevant finding and main cause of death in NGP [12, 13, 14] as noted in our case.
Most commonly the perforations have been linear tears, seen on the greater curvature, usually high and measured between 0.5-8 cm [9]. In the present case, the perforation was oval and measured 1 cm x 1 cm perforation seen on the fundic region along the greater curvature.

Prompt surgical intervention with repair of the gastric tear is the recommended management. A nasogastric tube should be placed while prompt resuscitation is undertaken [15]. Surgical repair of most perforations consist of debridement and gastroorrhaphy. Significant gastric resections should be avoided. Any delay in surgery will result in a higher mortality [7,15,16]. And the same was followed by us.

Post operative vigorous supportive therapy coupled with the use of broad spectrum antibiotics administered intravenously is necessary [8]. Despite advances in peri-operative management high mortality rate (60 -70%) has been reported [7, 17]. Degree of maturity, hypoxia and peritoneal contamination and duration of illness are risk factors for high mortality. Preterm babies are more prone to respiratory distress and septicemia and leading to very high mortality. Septicemia being the main killer [4, 6, 18, 19].

REFERENCES


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