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A case report of Neonatal Gastric Perforation: Is it avoidable?

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Abstract

Neonatal gastric perforation (NGP) is a rare but serious surgical emergency, with an unclear aetiology and several risk factors. Surgery remains the gold standard treatment for the management of this condition, while prevention is not prioritised in clinical practice. A full-term male newborn with a normal birth weight was transferred to our paediatric surgery unit at 4 days of life for the management of a pneumoperitoneum secondary to gastric perforation. This diagnosis was suspected preoperatively based on the striking gastric distension observed on prior radiographs. We believe that early management at the stage of gastric distension may have prevented such a complication. NGP is associated with a high mortality rate, reaching up to 70%. Although this rate has significantly decreased with advances in neonatology, more efforts are needed to reduce both the overall incidence and mortality of this condition.

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Introduction

Neonatal gastric perforation (NGP) is a surgical emergency defined as the rupture of a portion of the stomach occurring within the first 30 days of life. It is a rare entity, occurring in approximately 1 in 2,900 live births[1] and accounting for only 7.8% of all neonatal gastrointestinal perforations. Despite its low incidence, NGP is dangerous, with a reported mortality rate of up to 70%[1,2]. Even in survivors, long-term complications may include developmental delay, iron-deficiency anaemia, and steatorrhoea[3]. While the exact pathophysiology and mechanisms remain unclear and are still debated despite numerous publications, few risk factors are widely recognised, particularly in so-called “spontaneous” forms[4]. These risk factors include prematurity, perinatal asphyxia, necrotising enterocolitis (NEC), sepsis, and corticosteroid use. Awareness of these risk factors raises the question of whether NGP might be preventable through targeted control measures.

Case Presentation

A 4-day-old male newborn was delivered at term via vaginal delivery, with a birth weight of 3.5 kg and Apgar scores of 8 and 9 at 1 and 5 minutes, respectively. No resuscitation was required at birth. Placenta, umbilical cord, and amniotic fluid were all unremarkable. Notably, the mother had a third-trimester urinary infection. From birth, the newborn exhibited groans, and due to the positive infectious history and progressive onset of respiratory distress, he was admitted to the paediatric department, started on antibiotics, and underwent thoracoabdominal X-ray. The X-ray showed normal gastric appearance and adequate bowel gas distribution (Figure 1). Meconium was passed within the first 24 hours. However, by day 3, due to increasing abdominal distension, a follow-up X-ray showed massive gastric distension (Figure 2). On day 4, abdominal distension worsened with progressive respiratory distress (tachypnoea, intercostal and suprasternal retractions,

Figure 1: First X-ray (thoracoabdominal) showing normal bowel gas distribution.

Figure 2: Second X-ray (thoracoabdominal) showing massive gastric distension.

nasal flaring). There were no digestive signs, such as vomiting and bowel obstruction, and the general condition of the neonate remained stable. A thoracoabdominal radiograph revealed massive pneumoperitoneum, strongly suggestive of a hollow viscus perforation (Figure 3). Laboratory findings showed a normal platelet count (112 $\times 10^9$/L), leucocytosis (18 $\times 10^9$/L), elevated C-reactive protein (126mg/L), and normal serum sodium. Supportive measures included oxygen therapy via nasal cannula, gastric decompression via nasogastric tube, fluid and electrolyte resuscitation, and continued antibiotics. Exploratory laparotomy was performed 42 hours after the diagnosis of pneumoperitoneum.

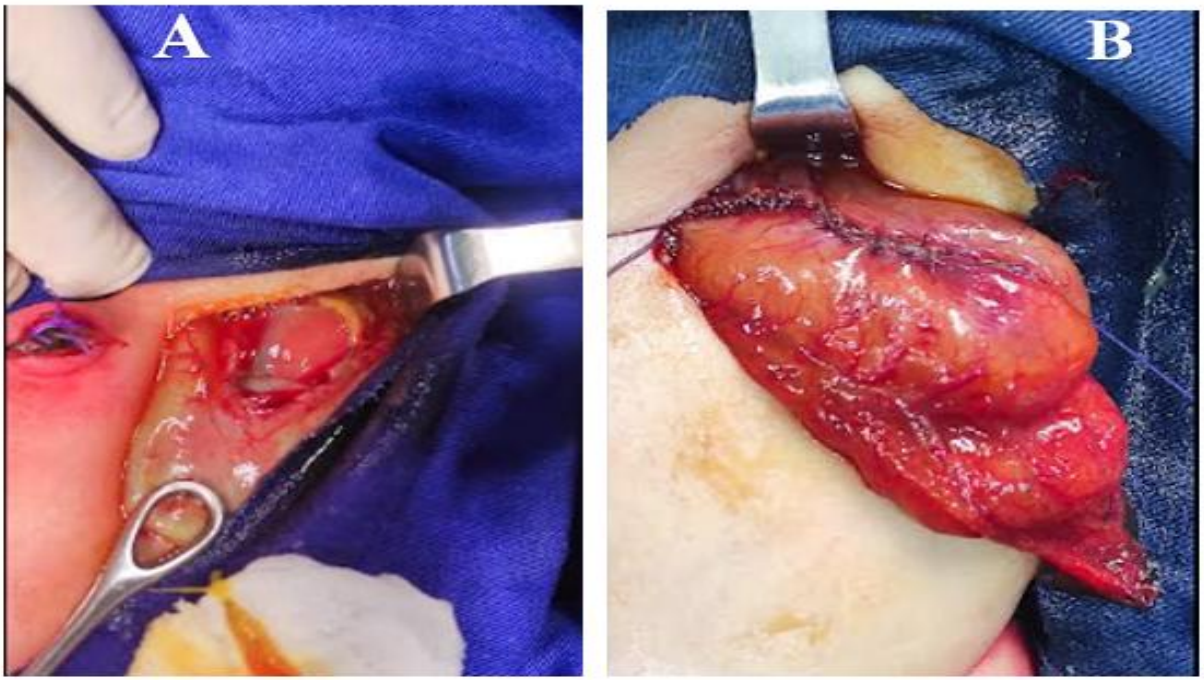
Figure 3. Massive pneumoperitoneum.

A left supraumbilical transverse incision was made. Intraoperative findings revealed peritonitis due to a linear 5 cm perforation on the anterior surface of the stomach, midway between the lesser and greater curvature (Figure 4). Primary repair (gastrorrhaphy) was performed using interrupted 5-0 sutures in a single layer, followed by peritoneal lavage. No drain was placed. Postoperative recovery was uneventful.

Discussion

NGP is a rare condition, classically divided into two types: idiopathic and traumatic. It predominantly affects male newborns, with male prevalence of 76.5%[5], 62.5%[4], 72%[3] and 61.9%[6] reported by various studies. Prematurity and low birth weight are recognized predisposing factors[2,7], along with maternal infection, early-onset neonatal sepsis, perinatal asphyxia, hypotension, nasogastric tube insertion and corticosteroid therapy[4]. In our patient,

Figure 4. Intraoperative picture (A) showing perforation of 5cm between greater and lesser curvature (B) final appearance after repair.



the identified risk factors were maternal infection and early neonatal infection. The classical clinical presentation begins with abdominal distension and respiratory distress, followed by deterioration and signs of sepsis. Radiologically, pneumoperitoneum on abdominal X-ray confirms the diagnosis. In our case, gastric distension was detected prior to perforation, which prompts reflection on whether early intervention at this stage could have prevented progression to NGP. While surgical repair remains the mainstay of treatment, peritoneal drainage can be considered as a temporising measure in unstable neonates[2]. Surgical treatment involves gastric perforation repair (gastrorrhaphy) after debridement of necrotic edges. Some authors recommend associated gastrostomy or, in extensive necrosis, interposition of small bowel or colon segments to improve perfusion and surgical success[8]. Although classic laparotomy repair is most commonly used, laparoscopic repair is possible with all its advantages linked to its minimally invasive nature[9]. In our patient, primary repair was performed by laparotomy, without drainage, despite a 42-hour delay.

Prognosis is influenced by several factors. Lacusso identified sepsis as the only factor significantly associated with mortality[2]. Babayigit highlighted perforation size ≥1.5 cm as a poor prognostic factor[4], while Garge emphasised intraoperative findings, particularly the extent of gastric necrosis[8]. The roles of prematurity and low birth weight are debated; some studies report them as poor prognostic indicators[4,8,10], while others do not[1,5]. This discrepancy may reflect differences in the quality of perioperative care. Huang identified laboratory predictors of mortality: leukopenia (<5 ×10⁹/L), hyperleukocytosis (>20 ×10⁹/L), thrombocytopenia, elevated serum lactic acid (>2.5 mmol/L), hyponatremia, and metabolic acidosis (pH <7.3)[3]. Additional poor prognostic indicators include male sex[1], signs of shock and hypovolemia, and sclerema neonatorum[3]. Delay between symptom onset and surgery also worsens outcomes. Our patient presented with three negative prognostic factors (male sex, sepsis, and a 5 cm perforation) however recovered favourably. Overall mortality has decreased significantly over time, likely due to improved neonatal intensive care, despite therapeutic approaches changing minimally. Preventing NGP could be achieved by identifying and controlling known risk factors such as: the insertion of a large-bore nasogastric tube in cases of gastric distension, cautious insertion of nasogastric tubes (gentle insertion, appropriate size, lubrication, measuring the necessary length), intensified antibiotics in sepsis, enhanced monitoring of

preterm and low-birth-weight newborns, ensuring adequate perfusion, administration of drogues to ensure good blood circulation preventing perinatal asphyxia, and controlling airway pressures in ventilated neonates. It is also important to improve the factors that influence the overall survival rate by optimising time to surgical intervention, which is crucial to improving outcomes.

Neonatal gastric perforation remains a life-threatening surgical emergency, with significant morbidity and mortality. While its aetiopathogenesis is still debated, diagnosis and treatment are usually straightforward. Prevention, however, is underexplored in the literature despite its potential to reduce incidence. Further studies focused on preventive strategies are warranted.

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