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Case Report

Successful Diagnosis and Management in 28-Week Preterm Infant with Gastrointestinal Perforation - A Case Report

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Abstract:

Background: Gastrointestinal perforation (GIP) is a serious cause of neonatal mortality, particularly in very preterm and very low birth weight infants. Despite surgical treatment, mortality remains high. The aim of this study was to report our hospital's intensive care and surgical experience in managing gastrointestinal perforation among very preterm and very low birth weight infants.

***Case*:** A 980-gr female baby was delivered by caesarean section at 28-weeks of gestation due to maternal severe pre-eclampsia and premature rupture of membranes. She developed respiratory distress syndrome and was admitted to the Neonatal Intensive Care Unit (NICU). On the fourth day of life, she developed an acute abdomen and an abdominal X-ray showed pneumoperitoneum. Emergency surgery revealed gastric perforation. Primary repair of the gastric and omental patch was performed. She passed the critical period after surgery and improved. The baby recovered well and was discharged in good condition.

***Discussion*:** In this case, the risk factor of gastrointestinal perforation is associated with asphyxia. Early diagnosis and treatment increase the survival rate in this patient. Postoperative nutrition plays a vital role in the recovery of neonates with gastrointestinal perforation. The transition to enteral feeding must be gradual and individualized, as these infants often face feeding intolerance due to surgical stress, hemodynamic instability, and intestinal inflammation.

Conclusion: Early diagnosis, prompt surgical intervention, and careful postoperative nutritional management are essential to improve survival in preterm infants with gastrointestinal perforation.

Keywords: gastrointestinal perforation, preterm, rupture gastric, surgery

Introduction

Gastrointestinal perforation (GIP), a rare condition in preterm neonates, appears as an important reason for mortality during the neonatal period, with reported high prevalence ranging from 15 - 70%.[1] Risk factors of gastrointestinal perforation may be varied, such as iatrogenic, Necrotizing Enterocolitis (NEC), prematurity, fetal bradycardia, low birth weight, use of nasal Continuous Positive Airway Pressure (CPAP), use of orogastric tube, perinatal hypoxia, who have required resuscitation, hyperpressure in the gastric chamber, and treatment with indomethacin or dexamethasone. Some studies suggested the insertion of a peritoneal drain as a bridging procedure for surgery to gain some health and stability of the newborn, especially in low-birthweight and very low birth weight neonates who cannot tolerate the impact of surgery.[2] Studies have reported no significant difference in mortality between infants treated with laparotomy or peritoneal drain placement when the diagnosis was NEC.[1]

The current standard treatment of neonates with intestinal perforation is surgery. Despite surgical intervention, the mortality rate remains significantly high, with an incidence reached 49%, in very low birth weight and low birth weight neonates with intestinal perforation.[3] Postoperative complications may also cause morbidity and mortality, despite early diagnosis and treatment.[4]

Asphyxia at birth in infants with low Apgar scores is particularly likely to lead to gastrointestinal perforation. Redistribution of blood flow during hypoxia, hypovolemia, or other stress states, with shunting away from mesenteric vascular beds, is thought to result in microvascular injury and subsequent loss of mucosal integrity.[5]

The first signs of spontaneous gastric perforation often occur at 3 to 5 days of life. Abdominal distention is frequently abrupt and rapidly progressive. Signs of hypovolemia and decreased perfusion are usually present, manifested by tachycardia and lethargy. Respiratory difficulty from massive pneumoperitoneum may be the first sign. Infants born of pregnancies complicated by abruptio placentae, placenta previa, and amnionitis (severe fetal distress) and infants delivered by emergency cesarean section are at increased risk and should be carefully observed. Most infants with gastric perforation have evidence of free air on abdominal radiographs.[6]

We present a case of gastrointestinal perforation with pneumoperitoneum in a very preterm neonate, who was successfully treated at our hospital.

Case

A female infant weighing 980 g was delivered by caesarean section at 28 weeks' gestation due to maternal severe pre-eclampsia and prolonged premature rupture of membranes (>12 hours). A complete course of antenatal corticosteroids had been

administered prior to delivery. At birth, the infant exhibited clinical features of respiratory distress syndrome, including tachypnoea and chest retractions. The Apgar scores were 3, 5, and 7 at 1, 5, and 10 min, respectively.

The infant required immediate admission to the Neonatal Intensive Care Unit (NICU), where initial stabilization was performed. Respiratory support was initiated with CPAP at a positive end-expiratory pressure (PEEP) of 7 cmH_2O, fraction of inspired oxygen (FiO_2) of 25%, and flow rate of 8 L/min. The heart rate remained >100 beats/min during stabilization. Exogenous surfactant therapy was not available at the referring hospital. There were no documented pathological cardiotocography findings prior to delivery, and umbilical artery doppler studies were not available. Cord blood gas analysis was not performed; therefore, the degree of perinatal hypoxia could not be definitively quantified. However, clinical signs, including tachycardia and hypotension, suggested possible transient hypoperfusion, a recognized risk factor in very preterm infants. Early minimal nutrition was given within 24 hours of life in the form of oral care using breast milk.

On the fourth day of life, the infant developed a progressive abdominal distention and signs of an acute abdomen **(Figure 1)**. An abdominal radiograph showed free intraperitoneal air consistent with pneumoperitoneum **(Figure 2)**. The infant was transferred to our hospital for further management. Based on the infant's instability and radiographic findings, an immediate surgery was performed with a pediatric surgeon. Intraoperative exploration revealed a single gastric perforation located in the fundus extending from the lesser of curvature to greater curvature, with clean margins **(Figure 3)**. There is no evidence of intestinal necrosis, congenital muscular defect, or distal obstruction. Primary gastric repair with an omental patch was successfully performed. Postoperatively, the baby required mechanical ventilation, broad-spectrum antibiotics, and total parenteral nutrition (TPN). Nutritional management included glucose, amino acids, lipids, electrolytes, and vitamins, with total fluid requirements carefully adjusted according to clinical status. She passed the critical period after surgery and improved; she was extubated on the seventh day after the operation. Parenteral nutrition is necessary during the postoperative period, neonatologist and pediatric surgeon agreed to give adequate parenteral nutrition before the enteral nutrition is given. A trial feed was well tolerated and was slowly increased until full feeds. The baby was discharged and is doing well on follow-up **(Figures 4 and 5)**.

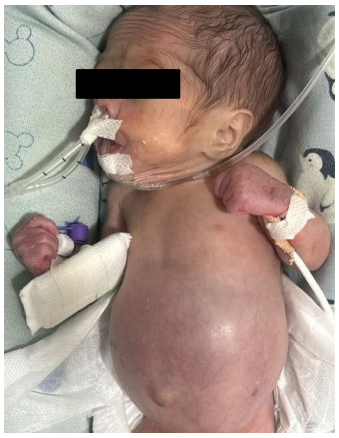

Figure 1. The baby with abdominal distension

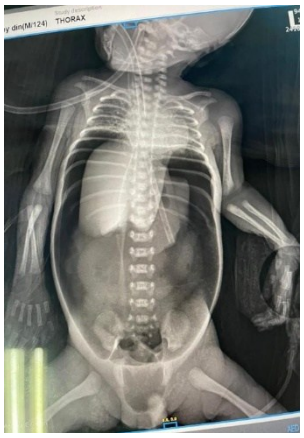

Figure 2. Radiographs revealing a pneumoperitoneum

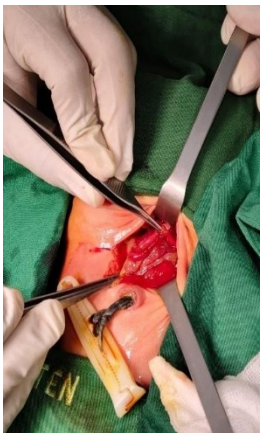

Figure 3. Intraoperative found a gastric rupture

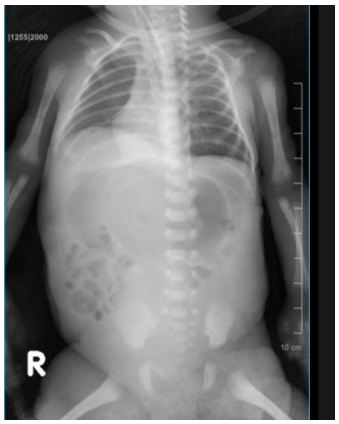

Figure 4. Radiographs 1 day after surgery

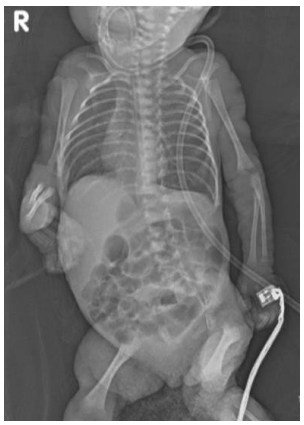

Figure 5. Radiographs 10 day after surgery

Discussion

This case demonstrated a successful diagnosis and management of gastric perforation in a very preterm infant, a condition associated with high mortality despite advances in neonatal care. The major risk factors in this infant included extreme prematurity, very low birth weight, respiratory distress syndrome, and physiological instability during early postnatal adaptation. In this case, the absolute indication for operative intervention was findings a pneumoperitoneum on an abdominal photograph. The indication for surgery is also associated with the best outcome.

Gastrointestinal perforation is a major life-threatening complication in preterm infants, with high morbidity and mortality.[5] It is associated with prematurity, low birth weight, asphyxia at birth or placement of an orogastric tube, ischemia, and exposure to indomethacin or dexamethasone.[1] In our case, the baby was premature, asphyxia at birth, and very low birth weight. The incidence of GIP increases with decreasing gestational age, and the median age of onset is seven days, with a range of 0-15 days.

That implies that GIP usually presents earlier in life, with varying risk factors according to the time of presentation. In comparison, NEC occurs later, usually after the introduction of feeds.

It is important to differentiate neonatal gastric perforation from spontaneous intestinal perforation (SIP) and NEC, as their pathophysiology and risk factors differ significantly. NEC is characterized by intestinal inflammation and necrosis, frequently accompanied by pneumatosis intestinalis and portal venous gas. SIP, in contrast, usually presents within the first week of life as an isolated intestinal perforation, most commonly involving the terminal ileum without surrounding necrosis or significant inflammation. In our patient, intraoperative findings revealed a localized gastric wall perforation without evidence of intestinal necrosis, pneumatosis intestinalis, portal venous gas, or diffuse inflammatory involvement characteristic of NEC. The anatomical location and absence of small intestine involvement also excluded SIP.

The most common cause of neonatal gastric perforation is congenital dysplasia of the muscular layer of the gastric wall, which typically occurs early after birth. The defect is usually localized in the anterior wall of the stomach body. Unlike NEC, gastric rupture is characterized by a large amount of free intraperitoneal air and rapid clinical deterioration due to peritonitis and sepsis.[7] Differences between NEC, SIP, and GIP are presented in **Table 1**.

Diagnosis of gastrointestinal perforation is confirmed by radiographic evidence of pneumoperitoneum. In low-resource settings, differentiation relies heavily on clinical presentation, timing of onset, radiographic findings, and intraoperative evaluation, as advanced imaging and histopathology may not be readily available.[12] A newborn with spontaneous gastrointestinal perforation and peritonitis may initially present with feeding intolerance and physiological instability, including lethargy, temperature instability, recurrent apnea, bradycardia, and delayed capillary refill. Progressive abdominal rigidity, absent bowel sounds, and signs of sepsis strongly suggest intra-abdominal viscus perforation.[13] Operative management remains the accepted treatment for neonates with pneumoperitoneum due to gastrointestinal perforation. Although some infants with medical NEC may initially be managed conservatively, pneumoperitoneum on abdominal radiograph is generally considered an absolute indication for surgical intervention. In some very low birth weight infants who are hemodynamically unstable, primary peritoneal drainage may be considered as a temporizing measure. Early recognition and timely surgical intervention were crucial to achieving a favorable outcome in this high-risk preterm infant.[9] The only absolute indication for operative intervention is pneumoperitoneum on an abdominal radiograph.[14]

Table 1. Differences between NEC, SIP, and GIP [8-11]

	NEC	SIP	GIP
Definition	Inflammatory bowel disease with ischemic and necrosis	Isolated focal intestinal without presentation	Perforation anywhere in the GI tract
Site	Small intestine	Terminal ileum	Gastric, or GI segments
Timing	Later, often after enteral feeding starts	Early, first week of life	Very early, often the first days of life
Population at risk	Preterm infants, especially low birth weight	Extremely preterm, ELBW/VLBW infants	Preterm infants, birth stress, unstable neonates
Main risk	Prematurity, formula feeding, sepsis, dysbiosis	Prematurity, indomethacin, steroids, hypotension	Prematurity, asphyxia, ischemia, positive pressure ventilation, orogastric tube trauma, congenital muscular defect
Pathology	Diffuse intestinal inflammation with necrosis	Small isolated hole with healthy surrounding bowel	Gastric wall rupture/perforation
Necrosis	Yes, common	Yes, common	No
Radiograph	Dilated bowel, pneumatosis, portal venous gas, and free air if perforated	Free intraperitoneal air	Often massive pneumoperitoneum
Clinical presentation	Feeding intolerance, distension, bloody stool, sepsis	Abdominal distension	Sudden distension, tense abdomen, rapid collapse
Operative findings	Necrotic/inflamed bowel	Solitary ileal perforation, viable bowel	Hole in the stomach wall
Management	Medical + surgery if perforation/necrosis	Surgery or drainage, depending on stability	Urgent surgical repair
Prognosis	Significant morbidity and mortality	Variable, often better than NEC	High mortality if delayed

NEC = Necrotizing Enterocolitis; SIP = Spontaneous Intestinal Perforation; GIP = Gastrointestinal Perforation; GI = Gastrointestinal; ELBW = Extremely Low Birth Weight; VLBW = Very Low Birth Weight

Alternative management depends on diagnosis and clinical stability. While laparotomy remains definitive treatment for neonatal gastric perforation, primary neonatal drainage may be considered in extremely preterm or unstable infants as a temporizing measure when immediate surgery is not feasible. In infants with NEC without perforation, conservative management with bowel rest, gastric decompression, intravenous antibiotics, parenteral nutrition, and close monitoring may be appropriate.[15]

The nutrition of the neonate before and after surgery was very crucial. In this case, the pediatric surgeon and the neonatologist consider giving a trial feeding and see how the neonate responds. Both the pediatric surgeon and the neonatologist agreed to start feeding slowly and resting the GI tract after the surgery. Parental nutrition is also very necessary during fasting and recovery from post-operative periods. Nutrition during recovery is exclusively provided by TPN, the same as that for infants being treated for NEC. The most prevalent risk associated with prolonged TPN exposure is cholestasis. The adequate feeding of these infants remains a challenge, as they often cannot tolerate enteral feeding and/or cannot metabolize nutrients properly due to surgical stress. They suffer from hemodynamic instability, preoperative complications, metabolic stress, and inflammation, which can delay nutritional support. An individualized feeding strategy that considers the specific surgical needs and the healing process is, therefore, essential for optimal postoperative recovery.[16]

Conclusion

Neonates with gastrointestinal perforation in very preterm infants remain a life-threatening condition with high mortality. Early recognition, timely surgical intervention, meticulous postoperative care, and individualized nutritional management are essential to improve survival. This case shows that successful outcomes are achievable through a multidisciplinary approach, even in settings with limited diagnostic resources.

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This case report has received consent for publication from the subject and parents.

Conflict of Interest

This case report has no conflict of interest

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