CASE REPORT

Neonatal gastric perforation in a preterm infant: A case report from northern Uganda

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Abstract

Spontaneous neonatal gastric perforation is a rare but life-threatening entity, with only a few hundred cases described in the literature. No cases have been documented in Uganda. The mortality rate from neonatal perforations is high, but with early diagnosis and surgical treatment, survival rates can be improved even in low-resource settings. This pathology should be part of the initial differential diagnosis whenever a neonate, especially if preterm, presents with abdominal pain and distention in the relevant clinical setting. We report a case of a preterm neonate who presented on the third day of life with abdominal distention, fever, and refusal to breastfeed. On examination, she was found to have features of a visceral perforation. This was corroborated by findings of pneumoperitoneum on x-ray and ultrasonography. She underwent gastrorrhaphy and had a stable postoperative recovery. She was discharged on postoperative day 12 in good condition.

Keywords: neonatal surgery, stomach perforation, preterm birth, gastrorrhaphy, Uganda

Introduction

A few hundred cases of spontaneous neonatal gastric perforation (NGP) have been described in the literature. No such cases have been described in Uganda, and gastric perforations commonly encountered by surgeons in this region are secondary to peptic ulcer disease.[1] The pathogenesis of NGP is not well established, but risk factors include prematurity and low birthweight.[2],[3] The mortality rate from neonatal perforations is high; however, with early diagnosis and surgical treatment, survival rates have improved.[3] The incidence of NGP is approximately 1 in 2 900 to 5 000 live births.[3],[4] It is critical that this pathology be considered whenever a neonate presents with abdominal distention and peritonitis.

Case presentation

We describe a female neonate who presented on the third day of life as a referral from a peripheral health centre. The mother reported that the baby had a gradual onset of abdominal distention and pain with the passage of dark stools, intermittent low-grade fevers, refusal to breastfeed, and non-projectile vomiting of feeds that progressed to bilious vomiting. The mother reported anuria the day prior to admission.

The mother was a 28-year-old P4+0 with 3 prior normal deliveries. Two children were healthy, and 1 child died in infancy following the extraction of a false tooth. She attended antenatal care at 20 and 28 weeks and reported no complications during pregnancy. The baby was born at approximately 35 weeks gestation with the assistance of a traditional birth attendant. The delivery was reportedly uneventful. The cord was ligated with glove cuff bands. The mother and baby were transferred to a peripheral health facility, where the baby was referred to our hospital.

On examination, the baby weighed 2.61 kg. She was in obvious respiratory distress with an oxygen saturation of 84% to 90% on room air. Her respiratory rate was 66 breaths per minute, and she had mild intercostal retractions. She was afebrile (body temperature, 36.1 °C), and her pulse rate was 150 beats per minute. Her blood pressure was not measured due to the unavailability of an appropriate cuff. The baby appeared jaundiced and acutely ill. Lung sounds were clear on auscultation, and no gross chest wall abnormalities were observed.

The abdomen was symmetrical with moderate to severe distention and visible superficial vessels. The cord stump had some septic foci, and hernia orifices were intact. The abdomen moved with respiration. On palpation, there was generalized peritonism with no obvious masses. The abdomen was tympanic with no clear shifting dullness. Bowel sounds were not heard. Digital rectal examination revealed an empty rectum. Cardiovascular, genitourinary, and nervous system examinations were unremarkable.

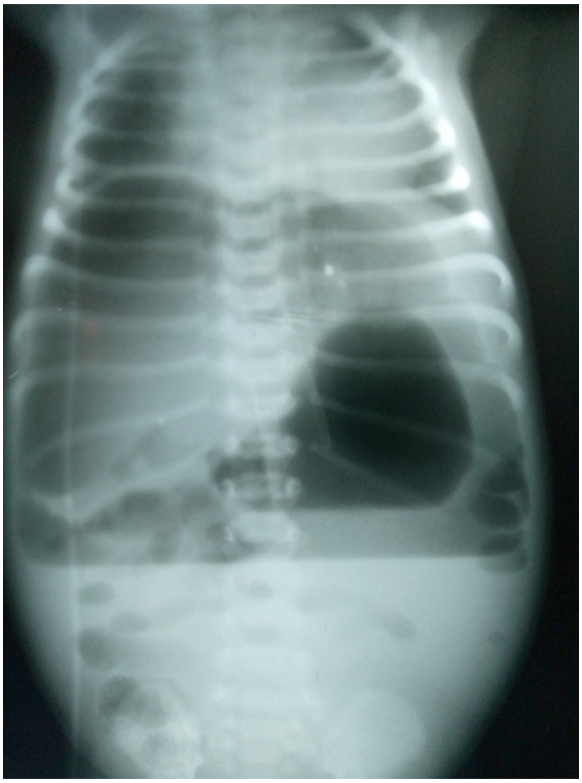

Figure 1. X-ray showing pneumoperitoneum

On admission, a nasogastric tube and urinary catheter were placed. Urine output was confirmed. Maintenance fluids (5% dextrose in normal saline) were initiated at approximately 30 mL/h through a peripheral intravenous line. Ampicillin (50 mg/kg every 12 hours) and gentamicin (5 mg/kg daily) were started. Gentamicin was subsequently discontinued due to poor kidney function. Metronidazole (approximately 20 mg every 8 hours) and scheduled rectal paracetamol were initiated for infection management and pain control, respectively.

Investigations

Initial laboratory tests revealed a normal haemoglobin concentration (13.7 g/dL), normal white blood cell count (4.25×10^9/L), and normal platelet count (269×10^9/L), along with markedly raised serum creatinine (4.25 mg/dL) and urea (103.3 mg/dL) concentrations. Total bilirubin (73.62 mg/dL) was elevated, with a direct bilirubin level of 5.64 mg/dL. Albumin (3.77 g/L) and alanine transaminase (24 U/L) levels were normal, while aspartate transaminase (134.2 U/L) and alkaline phosphatase (326.7 U/L) levels were elevated. Abdominopelvic ultrasound revealed a gaseous abdomen with complex ascites and reduced peristalsis of the small bowel. The liver, spleen, and kidneys had normal echo texture. An erect plain chest-abdominal x-ray revealed a large pneumoperitoneum (Figure 1).

Differential diagnoses

Differential diagnoses included necrotizing enterocolitis, intestinal perforation, and gastric perforation. The presumptive diagnosis of intestinal perforation was made before proceeding with an emergency exploratory laparotomy.

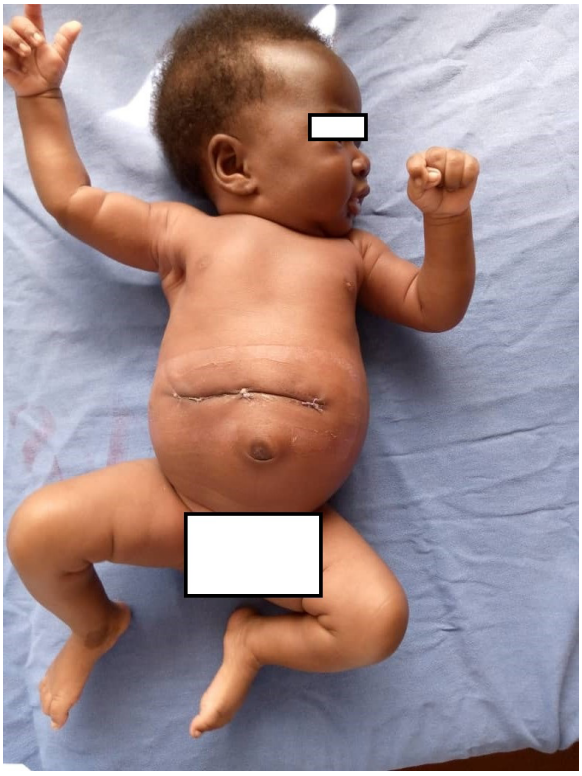

Figure 2. Patient status on the eleventh postoperative day after exploratory laparotomy and gastric perforation repair

Management

An emergency exploratory laparotomy was performed due to the severity of the presentation and laboratory findings after obtaining parental consent. Intraoperatively, an elliptical gastric perforation was found, measuring 0.8 × 0.5 cm, located on the anterior aspect of the antrum along the lesser curvature. Approximately 100 mL of bilious ascites with diffuse fibrinous exudate was noted. No features suggestive of mechanical obstruction were observed.

The perforation was repaired in 2 layers using 3.0 polyglactin 910 (continuous and Lembert) and an omental patch overlying the repair using 3.0 polyglactin 910. Thorough abdominal lavage with warm normal saline was performed, achieving clearance of the fibrinous exudate. The abdomen was closed en masse, leaving the skin open for delayed closure (Figure 2).

Outcome and follow-up

Postoperatively, the baby showed no complications. Her respiratory rate and oxygen saturation improved, and she remained afebrile. She continued to receive intravenous fluids, and the nasogastric tube remained on continuous free drainage. On day 3, bowel sounds returned, and she was started on warm 5% dextrose with water, 10 mL by mouth every 3 hours. On days 4 and 5, the nasogastric tube was clamped

and aspirated before each feed, and expressed breast milk was gradually introduced. By day 6, the nasogastric tube output had significantly decreased, and the baby was passing stool and tolerating larger feeds. The nasogastric tube was removed, and breastfeeding was successfully initiated. The surgical site healed well, and she was discharged home in stable condition on day 11.

Discussion

We report a case of spontaneous NGP in a preterm infant with an umbilical stump infection, complicated by neonatal sepsis and delayed care due to community birth assisted by a traditional birth attendant.

Risk factors for NGP include prematurity, infection, low birthweight, perinatal hypoxia, neonatal resuscitation, conditions leading to increased pressure within the stomach, medications like indomethacin and dexamethasone, and nasogastric tube use.[5]-[7] Secondary gastric perforations may occur with congenital conditions, such as intestinal atresia, oesophagal atresia with distal fistula, diaphragmatic hernia, and amniotic gastritis.[2]

Our patient's physical examination findings of significant abdominal distention, tympany, and peritonism strongly suggested perforation. This clinical suspicion was corroborated by imaging: abdominal ultrasonography revealed gaseous distention and complex ascites, and an erect thoracoabdominal x-ray confirmed the presence of pneumoperitoneum.[5]

Historically, NGP had a mortality rate of 100% until the 1980s. Advances in diagnosis and treatment have improved mortality rates, which now range between 32% and 60%.[2],[3],[6],[7] Prematurity is a major risk factor for mortality, especially in low-birthweight infants.[8] Our patient was born at 35 weeks gestation.

Neonatal sepsis was another predisposing factor.[2],[5],[9] This neonate's delivery outside a health facility with nonsterile cord ligation likely contributed to infection and sepsis, independent of the perforation.

Neonates with respiratory distress have an increased risk of death during or after surgery,[10] compounded by lung immaturity in preterm infants. Early antibiotics, oxygen therapy, fluid resuscitation, nasogastric decompression, and operative management likely contributed to this patient's favourable outcome.

Spontaneous perforations typically occur in the proximal stomach along the greater curvature,[11]-[13] thought to arise from mechanical strain.[14] Lesions elsewhere or with a punctate appearance suggest iatrogenic injury, especially from orogastric tube placement.[15] Our patient's perforation was on the lesser curvature and likely not iatrogenic, as symptoms preceded nasogastric tube insertion. Infection, which can cause stress ulcers,[16] was the likely cause. Preterm neonates may also have motility dysfunction caused by an absence of interstitial cells of Cajal, predisposing them to perforation.[14]

Gastroduodenal perforations in neonates have a high mortality rate.[2],[7] This is likely due to chemical peritonitis caused by spillage of gastric acid and milk into the peritoneum. In our case, early surgery with copious lavage and antibiotic initiation likely mitigated these effects.

In high-income settings, NGP similarly affects preterm and low-birthweight infants, usually presenting at around 3 days of age.[2],[3],[6] Diagnosis is primarily clinical, aided by imaging.[3],[8],[17] Gastrorrhaphy is the standard treatment, with mortality rates ranging from 73% to 93%.[2],[18] Early recognition, intervention, and access to neonatal intensive care units enhance survival.[3],[17] Notably, Aydin et al.[19] reported successful nonsurgical NGP management in a critically ill, low-birthweight, preterm infant.

Conclusions

Although NGPs are rare, the mortality rate is high. This entity should be suspected early in neonates presenting with gaseous abdominal distention, especially in preterm or low-birthweight infants. High quality of preoperative and postoperative care improves survival. Early diagnosis can be attained even in low-resource settings with the appropriate clinical suspicion, assisted by widely available tools, including x-ray and ultrasound. This patient's clinical presentation of abdominal distention and peritonitis without a clear pattern of obstruction raised suspicion for perforation.

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