SECTION EDITOR: BEVERLY P. WOOD, MD

Radiological Case of the Month

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A 36-HOUR-OLD male neonate developed rapid abdominal distention, poor feeding, lethargy, and jitteriness. The significant prenatal history included premature rupture of membranes for 1 week prior to delivery, but there was no evidence of chorioamnionitis, and results of a screening for group B *Streptococcus* were negative. The mother received intrapartum antibiotics because of premature rupture of membranes and received steroids to mature the fetal lung. The baby was delivered by emergency cesarean delivery because of fetal distress and persistent fetal tachycardia noted on intrapartum fetal monitoring. Apgar scores were 2 at 1 minute and 8 at 5 minutes. The infant received positive pressure ventilation for 3 minutes after birth. Physical examination revealed a 37-week, appropriate for gestational age male infant. The initial arterial blood gas at 30 minutes showed a pH of 7.39 and base excess of –4.8. The infant was started on formula feedings, which were tolerated well, and the first day's oral intake was 40 to 45 mL with each feeding. Meconium stools were passed 6 times after birth. At 36 hours, the neonate developed abdominal distention, lethargy, and poor feeding but did not vomit. He had tachycardia with normal peripheral perfusion, blood pressure, and oxygen saturation. The abdomen was markedly distended and tender with bulging of hernial orifices and absent bowel sounds. No abdominal discoloration, petechiae, or masses were present. An orogastric tube was inserted and yielded 10 mL of bilious drainage. Feedings were discontinued, and intravenous fluids and antibiotics were started. An abdominal supine radiograph (**Figure 1**) and horizontal beam decubitus radiograph (**Figure 2**) were obtained.

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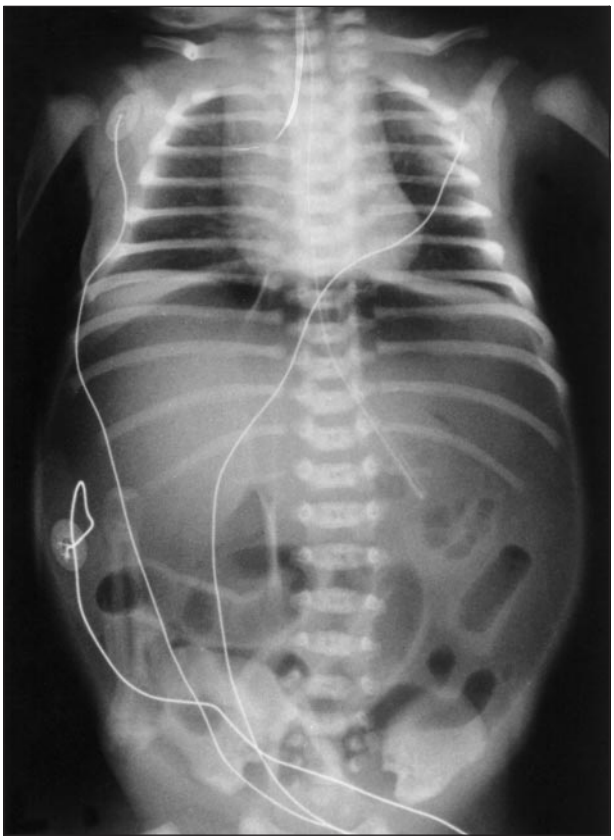

Figure 1.

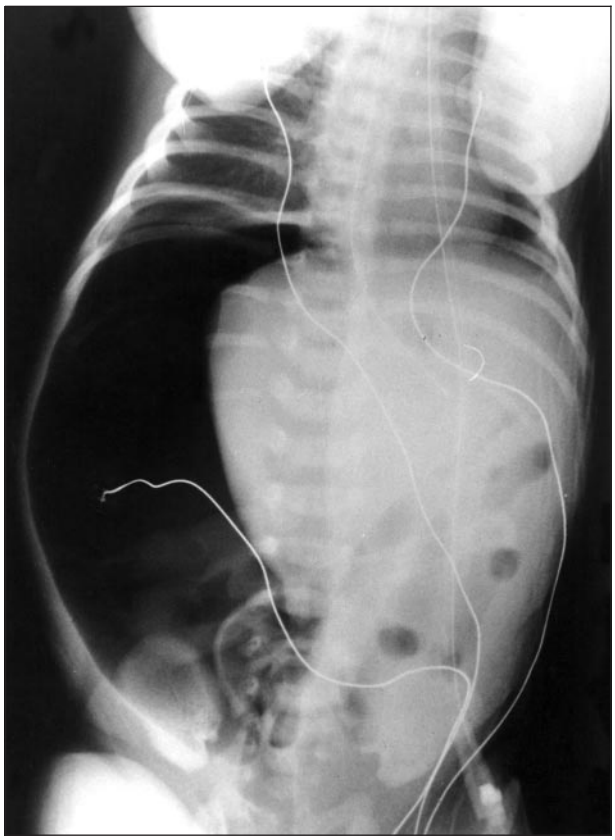

Figure 2.

Denouement and Discussion

Spontaneous Neonatal Gastric Perforation

Figure 1. *Abdominal radiograph, abdominoperineal view, shows pneumoperitoneum with "football sign."*

Figure 2. *Abdominal radiograph horizontal beam decubitus shows massive pneumoperitoneum.*

An emergency laparotomy was performed. A massive release of air occurred with peritoneal incision, and formula was present in the peritoneal cavity. A 1-cm linear perforation on the greater curvature of the stomach in the cardiac region was observed. No necrosis was present around the site. All layers of the stomach wall were separated by air. The perforation was sutured in a double layer. Peritoneal fluid cultures grew coagulase-negative staphylococci and enterococci species. The postoperative course was uncomplicated. The patient started feeding on postoperative day 7 and was discharged home on day 10.

Gastric perforation is a rare abdominal catastrophe in the full-term newborn. It accounts for 10% to 16% of all gastrointestinal perforations. The usual age at presentation is 2 to 7 days[1-5] in term infants. Incidence in African American infants is higher than in white infants[1,2] and is higher in males than females.[2,4] Usually the infants have been fed before they become symptomatic.[1]

Certain risk factors are associated with gastric perforation:

1. Prenatal. Premature rupture of membranes; toxemia; breech; diabetic mother; group B *Streptococcus*–positive mother; amnionitis; placenta previa; placental abruption; emergency cesarean delivery.[1]
2. Postnatal. Prematurity[1,2,6]; low birth weight[1,2,6]; small for gestational age infants[2]; low Apgar scores at birth; respiratory distress with resuscitation or ventilation.[1]
3. Exchange transfusion.
4. Surfactant deficiency; mechanical ventilation.[6]
5. Patent ductus arteriosus; indomethacin treatment.

Gastric perforation is associated with significant mortality and morbidity if it is not recognized early. There are several proposed causes for spontaneous gastric perforation: congenital deficiency of musculature in the gastric wall[1]; thinner stomach wall near the cardiac than the pylorus[1]; gaps in the circular musculature of the newborn stomach at the fundus near the greater curvature[1]; and acute gastric dilatation secondary to pylorospasm and atonic stomach in a stressed neonate. Some of these factors may have contributed to gastric perforation in this neonate despite the absence of notable risk factors.

Signs and symptoms include sudden onset abdominal distention,[1,2,4-6] poor feeding,[1-4] lethargy, and listlessness. Vomiting is an uncommon manifestation of gastric perforation. An idiopathic gastric perforation occurs on the anterior surface of the cardiac region along the greater curvature.[1-4] No necrosis is visualized around the site when diagnosis is early.

Early diagnosis is essential to avoid catastrophic consequences. The abnormality is characterized radiologically by the presence of free air in the peritoneum-pueumoperitonem[2-4,6]; absent gastric gas bubble[2]; absence of bowel air-fluid levels and fixed dilated loops of intestine-positive "football sign"[2]; and an air-fluid level in the peritoneum. Differential diagnosis includes acute necrotizing enterocolitis, septicemia, intestinal obstruction, and spontaneous pneumoperitoneum without gastrointestinal perforation.

The surgical treatment is primary gastric wall closure in 2 layers.[1-3,6] Occasionally, gastrostomy is needed for postoperative decompression. The prognosis is excellent with almost 100% survival if early diagnosis is made. Delay in diagnosis, prematurity, metabolic acidosis, and hyponatremia are associated with poor prognosis and increased mortality.[1,4,5]

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