

Gastric pneumatosis in a preterm infant following initial empiric antibiotic therapy

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DESCRIPTION

The case is of a twin A male infant born preterm vaginally at 31 weeks as a product of dichorionic diamniotic twins gestation. The mother was 23 years old, gravida 3 para 0, with gestational diabetes on a diet regimen. She did not have any risk factors for infection. She was seen in the antenatal clinic 5 weeks before the onset of labour with no maternal or fetal concerns. Her twins were growing normally with no congenital anomalies seen in the antenatal ultrasound scans. On the delivery day, she arrived at the emergency room fully dilated with premature rupture of membranes (PROM) at the onset of labour. She did not receive any antenatal steroids before the delivery.

The infant was born in a good condition with an Apgar score of 9 and 10 at 1 and 5 min, respectively. He was admitted to the neonatal intensive care unit (NICU) together with his twin due to prematurity and respiratory distress syndrome. Regarding his respiratory support, he was supported by nasal continuous positive airway pressure (NCPAP) since birth for 11 hours and by nasal cannula for another 2 days. The treating team started empiric initial antibiotic therapy at birth by ampicillin and amikacin based on the preterm labour and the PROM. The antibiotic therapy was continued for 5 days despite the sterile blood culture at 48 hours of age and repeatedly negative C-reactive protein (CRP).

The other twin B received ampicillin and amikacin at birth and it was stopped after 2 days due to negative cultures. He was supported by NCPAP for 12 hours and by nasal cannula for 13 days. He had a smooth NICU course with no complications.

On the fifth day of life, this infant (twin A) was noted to have poor activity, lethargy with frequent attacks of bradycardia and desaturation. Based on the clinical presentation, the neonatal team considered late-onset sepsis and necrotising enterocolitis (NEC) as possible initial differential diagnoses.

Complete blood count (CBC) and peripheral smear were of normal values and blood culture was negative at that time. CRP was elevated to 49 mg/L. Capillary blood gases showed partially compensated metabolic acidosis while the coagulation profile and serum electrolytes were normal. Anteroposterior (figure 1) and cross table (figure 2) abdominal X-ray was done and showed pneumatosis intestinalis, portal venous gas and gastric pneumatosis with no free air. Ultrasound of the abdomen showed scanty free ascites with tiny bright

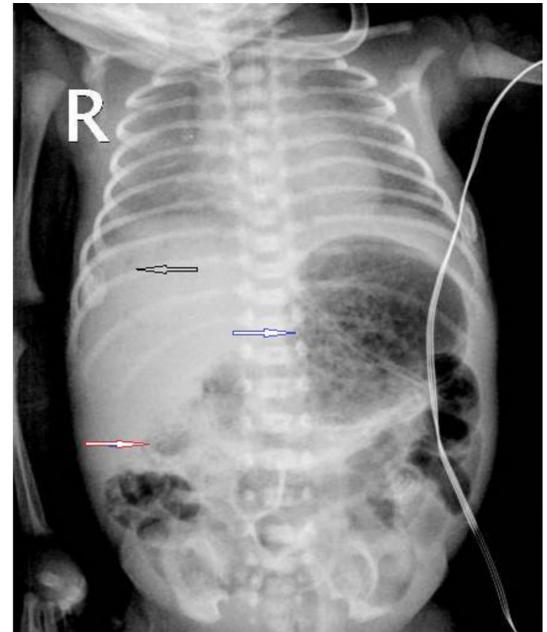


Figure 1 Antero-posterior X-ray of the abdomen shows gastric pneumatosis (blue arrow), pneumatosis intestinalis (red arrow) and air in the portal tract (black arrow).

foci within hepatic parenchyma, indicating portal tract air foci (figure 3).

The infant was supported by NCPAP for few hours which was escalated to conventional mechanical ventilation for 2 days before extubation to a nasal cannula for another 2 days. The paediatric surgery team was consulted and the infant was kept nil per mouth for 14 days. A peripherally inserted central catheter was placed and the infant received intravenous teicoplanin, intravenous amikacin and intravenous metronidazole for 14 days.

Late-onset sepsis was excluded based on the negative blood culture and normal CBC parameters. As a final diagnostic aetiology, the neonatal



Figure 2 Cross table X-ray of the abdomen shows gastric pneumatosis.



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Figure 3 Ultrasound of the abdomen shows air in the portal vein (PV).

team labelled this patient as a grade II B NEC based on Bells's staging criteria.¹

Close follow-up showed gradual clinical and radiological improvement which started 2 days after the initiation of therapy till complete recovery. The infant was discharged home safely on the 35th day of life after achieving full oral feeding with steady weight gain.

The absence of NEC symptoms and signs in twin B that was seen in twin A can be explained by the shorter duration of the antibiotic therapy which is 2 days only in the healthy twin.

Gastric pneumatosis is a rare presentation of NEC in the first week of life. It develops as a result of altered gut microbiota following empiric initial antibiotic administration for preterm infants leading to overgrowth of pathologic virulent organisms over the commensals.²⁻⁵

Other causes of gastric pneumatosis include isolated gastric pneumatosis, sepsis, gastritis, congenital hypertrophic pyloric stenosis, displaced gastric tubes, following steroid and cyclooxygenase inhibitor (COX-inhibitor), for example, Ibuprofen exposure, jejunal atresia and non-invasive positive pressure ventilation, especially in babies with cyanotic congenital heart disease and large patent ductus arteriosus.⁶⁻⁹

Learning points

- ▶ Initial empiric use of antibiotics should not be routinely administered for preterm infants on delivery as it is strongly associated with intestinal dysbiosis and increased incidence of NEC.
- ▶ The use of early antibiotic therapy for preterm infants after delivery should be restricted to indicated cases with risk factors of early-onset sepsis. It should be stopped as soon as the cultures and the rest of the sepsis workup is negative.

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