Echogenic Bowel as an Indicator of Necrotizing Enterocolitis in a Term Newborn

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Abstract
A 3.5-kilogram infant was born at 40 weeks gestation with an uncomplicated delivery. Prenatal ultrasounds showed echogenic bowel and a ventricular septal defect (VSD), of no clinical significance. Abdominal radiographs showed pneumatosis at 21, 36, and 48 hours of life (HOL). She was treated for necrotizing enterocolitis (NEC) with intravenous antibiotics and parenteral nutrition for 7 days, before working up on feeds and discharging home with breast milk. The only prenatal finding in this case was hyperechogenic bowel, which is a soft marker and often disregarded in the absence of other signs. Chronic intrauterine gut ischemia can cause hyperechogenicity of the bowel. That same intrauterine gut ischemia may have been responsible for NEC in our patient. If a patient has persistent echogenic bowel on prenatal imaging, a critical need exists to make sure NEC is not present.

Keywords
echogenic bowel, necrotizing enterocolitis, pneumatosis

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Introduction
Necrotizing enterocolitis (NEC) is a devastating infection that causes significant morbidity and mortality in the neonatal period.1 NEC typically affects preterm infants. Term neonates account for less than 10% of reported cases.2 When NEC occurs in term infants, they tend to have a birth weight of less than 2500 grams, perinatal hypoxic events, congenital heart disease, polycythemia, sepsis, and/or endocrinopathies.2 In a 2013 study of infants >35 weeks that presented with NEC, the mean onset was 12.1 ± 11.2 days after birth.3 Typically, term infants who are more than 2500 grams and are diagnosed with NEC have major congenital anomalies lead to increased morbidity and mortality.4 Congenital heart disease is the most commonly cited risk factor for NEC in term infants.5 Congenital heart disease occurs in 18% of cases of NEC in term, normal birthweight infants.5 The pathophysiology of NEC is poorly understood. Research indicates that the causes of NEC may differ in term infants and preterm infants. We present a term infant with NEC whose only risk factor was prenatally identified echogenic bowel.

Case Presentation
A 3.53-kilogram female was born via vacuum-assisted vaginal delivery at 40 6/7 weeks gestation to a 21-year-old gravida 1 para 1 woman. The pregnancy was complicated by an abnormal quad screen 1:128, which placed the infant at risk for Down Syndrome. The subsequent Non-invasive prenatal testing (NIPT) was normal. Ultrasound at 22 1/7 weeks showed a left echogenic intracardiac focus, ventricular septal defect (VSD), and echogenic bowel. Repeat ultrasound at 27 6/7 weeks gestation showed a left echogenic intracardiac focus, ventricular septal defect (VSD), and echogenic bowel. Repeat ultrasound at 27 6/7 weeks gestation showed continued small VSD and echogenic bowel that looked “much less echogenic on imaging.” At delivery, the infant emerged with a strong cry. She required drying, stimulation, and bulb suctioning but no further resuscitation. APGARS were

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8 and 9 at 1 and 5 minutes, respectively, with points taken off for color only.

After birth, the infant was admitted to couplet care with her mother. She was breastfed and had appropriate urine and stool output. Due to the echogenic bowel found on 2 prenatal ultrasounds, a chest and abdominal X-ray were obtained at 7 hours of life. Radiographs showed a normal chest and bowel gas pattern. Repeat radiographs showed pneumatosis at 21, 36, and 48 hours of life (HOL). At 24 HOL the infant became tachypneic and had an increased work of breathing. The pulse oximetry reading was 88% to 92% on room air. She did not have abdominal distention. The infant was kept NPO and given total parenteral nutrition and intravenous (IV) piperacillin/tazobactam for 7 days. Subsequent X-rays over the next 2 weeks showed normal gas patterns with no pneumatoses. Feedings with expressed breast milk were slowly introduced after the completion of antibiotic therapy and advanced to full feedings over 5 days.

Due to the VSD seen on prenatal ultrasound imaging, an echocardiogram was obtained on DOL 2. Echocardiogram showed a moderate-sized apical VSD, a moderately restrictive VSD shunt, and a patent foramen ovale. On DOL 5, the infant had an intermittent abnormal rhythm noted on her telemetry. An EKG showed premature atrial contractions. A pediatric Cardiologist was consulted and recommended follow up with repeat imaging at 6 to 8 weeks of life.

The infant was discharged home on DOL 16 on full feeds of expressed breast milk. She was scheduled for outpatient follow up with a pediatric cardiologist.

Discussion

According to Abbo et al, prematurity represents the most common risk factor for NEC. With the incomplete development of all the preterm infant’s body systems, preterm infants are often unable to combat the rapidly progressive inflammation that occurs with NEC. Within 24 to 48 hours of birth, NEC can progress from initial symptoms to death. Important antecedents in the pathophysiology of medical (classic) NEC include intestinal immaturity and inflammation, microbial colonization, and dysbiosis secondary to an exaggerated host inflammatory response.

Preterm babies with NEC die much more often than their term baby counterparts. Globally, the reported cases of NEC in preterm infants range from 2% to 7.5%. NEC in the term infant is less common than in the preterm infant. Abbo et al confirmed that NEC in full term neonates remains rare. For full term neonates, the incidence approximates 1 for every 20000 births and accounts for 10% of all NEC occurrences.

Christensen postulates that for term infants and infants who weigh more than 2500 grams, conditions which predispose to NEC include a predisposing or underlying condition, such as congenital heart disease, primary gastrointestinal defects, perinatal asphyxia, polycythemia, sepsis, hypotension, endocrine disorders, and respiratory disease.

Although the underlying mechanism for NEC in term infants is unclear, the literature suggests that the hypothesized causes can be placed into 3 primary categories. The first category is maternal and gestational conditions including maternal diabetes, maternal drugs of abuse, preeclampsia, anti C Rhesus incompatibility, intrauterine growth restriction, and premature rupture of membranes. The second category is organic pathology, which includes congenital cardiac anomalies. Impaired mesenteric oxygenation due to poor perfusion or cyanosis may be a major predisposing factor that contributes to the development of NEC. The third category is medical conditions that require neonatal interventions, such as hypoglycemia, polycythemia, respiratory distress syndrome causing general anoxia, and infection, as well as formula feeding.

In a study spanning more than 5 years, Lambert et al compared 30 term or near-term neonates who developed NEC to 5847 who did not. They concluded that NEC among term or near-term neonates occurred more often when the baby was fed artificial formula. Our patient was solely breastfed. She did have a ventricular septal defect, but it was not clinically significant during the time of her hospitalization; therefore, it would be unlikely to have caused mesenteric ischemia before or after birth. She did not have polycythemia, hypoglycemia, or other medical conditions that have been noted as risk factors for NEC. No maternal risk factors put her at risk of NEC. The newborn’s only risk factor was the echogenic bowel found on 2 prenatal ultrasounds.

According to De Oronzo, there are several causes of echogenic bowel. These causes include, but are not limited to, fetal aneuploidy—especially Trisomy 21, bowel atresia, oligohydramnios, and intra-amniotic hemorrhage. The pathogenesis is poorly understood, but could be secondary to decreased fluid content of the meconium or hypoperistalsis. A complicated link exists between hyperechogenic fetal bowel and placental dysfunction, so chronic intrauterine gut ischemia may be responsible for the hyperechogenicity.

Echogenic bowel is diagnosed in 0.2% to 1.4% of second trimester ultrasonographic examinations and often occurs as a normal variant. However, echogenic bowel has also been associated with several pathologic conditions that include cystic fibrosis, chromosomal abnormalities and in utero infection.
with cytomegalovirus and toxoplasmosis. The diagno-
sis of fetal echogenic bowel in the second trimester
has significant implications for prenatal management
and should be considered an important marker of pla-
cental damage. This finding in the second trimester is
associated with adverse pregnancy outcomes.12 When
fetal echogenic bowel is present in isolation, it is a
benign condition often yielding a positive prognosis.
However, if multiple additional anomalies are
observed, the prognosis tends to be less favorable.13

Conclusion

NEC is a devastating intestinal infection that causes sig-
nificant morbidity and mortality in neonates. NEC most
commonly occurs in preterm newborns but may also
occur in term infants. The pathophysiology of NEC is
not completely understood. Research indicates it may be
different in preterm and term infants. According to De
Oronzo, chronic intrauterine gut ischemia can cause hyperchogenicity of the bowel, which was seen in our
patient.11 Intrauterine gut ischemia may be the reason
our patient developed NEC. If a patient has persistent
echogenic bowel on prenatal imaging, a critical need
exists to make sure NEC is not present. Early detection
remains the best way to ensure a positive outcome for
the affected infant.

Author Contributions

GW, CW, and MB: Contributed to the conception and design
of this manuscript.

GW and BD: Drafted the manuscript.

CB and MB: Critically revised the manuscript.

All authors: Reviewed, edited, and gave final approval for
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Disclosure

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patient was treated at the University of Arkansas for Medical
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Informed Consent

Written informed consent was not obtained due to the patient
being lost to follow up. Infant was never seen by a primary
care provider or subspecialist after hospitalization, even after
multiple attempts by the authors and the institution to contact
the family, making it impossible to obtain written consent. No
images or patient identifiable information is being utilized.

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