A Real-time Cerebral Bleeding in an Extremely Preterm Newborn

A neonate was born at $26^{6/7}$ weeks of gestation in a second-level center by an emergency caesarean delivery owing to placental abruption. The newborn weighed 920 g and had an Apgar score of 2-6-7. She was intubated rapidly and an umbilical venous catheter was placed. Immediately after administering an endotracheal dose of surfactant, the newborn suffered a cardiac arrest, not responding to adequate ventilation and requiring chest compressions and adrenaline to reach return of spontaneous circulation. At 2 hours of life, she was transported to our third-level neonatal intensive care unit with a severe metabolic acidosis (pH 6.96, bicarbonates 6 mmol/L) and hypotension, needing multiple fluid boluses to reach an adequate mean arterial pressure during transport.

Upon arrival, a double inotropic therapy with dopamine and dobutamine was begun, as well as a supplementation with sodium bicarbonate and sedation with fentanyl. Cerebral ultrasound at admission (as well as at 24 hours of life) showed no intraventricular hemorrhage (IVH), only collapsed lateral ventricles. The clinical conditions of the newborn remained stable although critical (requiring subsequent administrations of bicarbonates and frequent

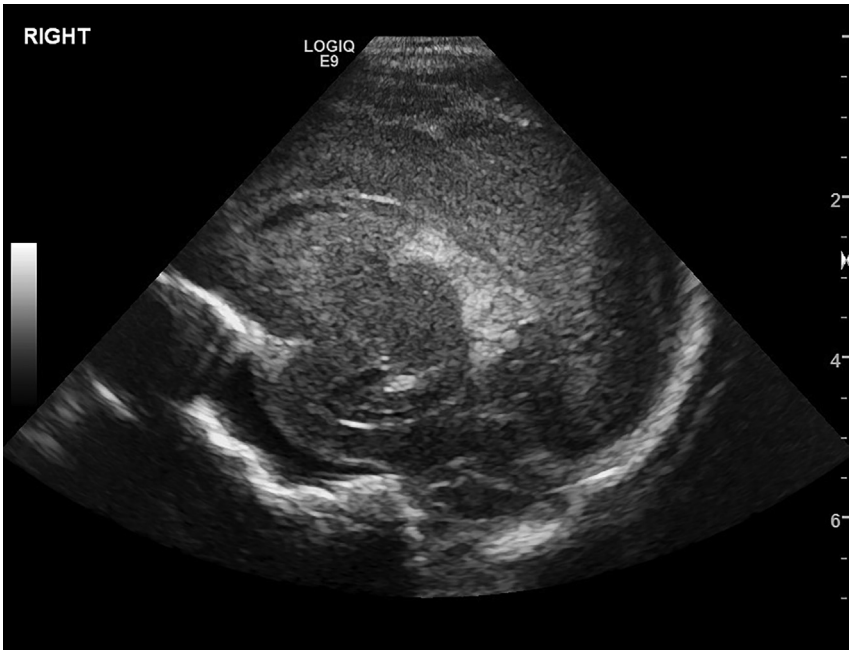

Figure 1. Sagittal view: blood occupying less than one-half of the right ventricular area at first ultrasound investigation, with no chamber dilatation.

The authors declare no conflicts of interest.

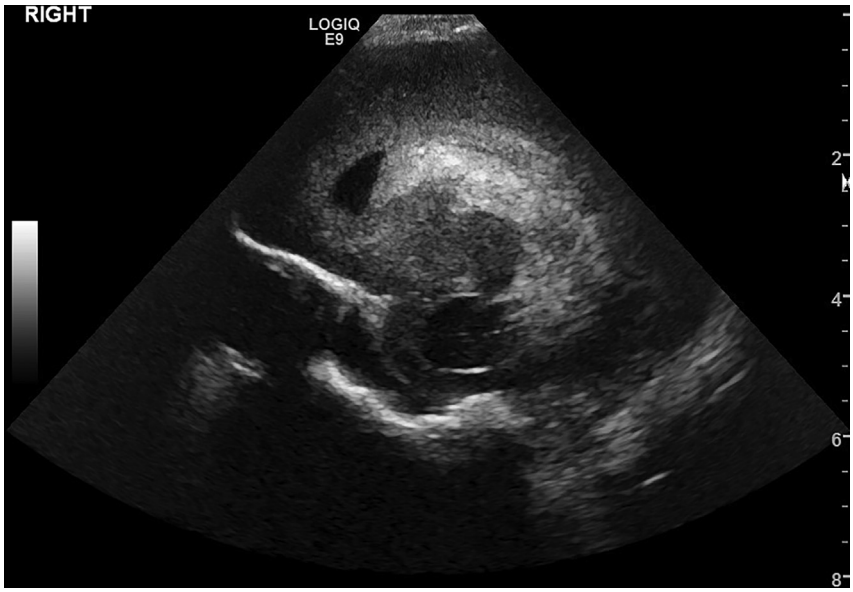

Figure 2. Sagittal view: enlarged right lateral ventricle occupied by blood during the following transfontanellar ultrasound examination.

dose adjustments of inotropes) until 48 hours of life, when a sudden appearance of eyes wide open despite sedation and a significant decrease in hemoglobin values were noticed.

With a strong suspicion for ongoing bleeding, a transfontanellar ultrasound was performed: a II grade (according to Volpe classification) bilateral (greater in the right ventricle) (**Figure 1**), IVH was discovered.[1] Moreover, a hyperechoic flow was clearly distinguishable inside the right ventricle (**Video 1**; available at www.jpeds.com), consistent with real-time active bleeding. Although the 2-hour echographic control was unchanged, a couple of hours later the newborn experienced an episode of bradycardia associated with whole body rigidity, consistent with a generalized tonic seizure. After starting a loading dose of phenobarbital, cerebral ultrasound examination was repeated. As expected, the IVH had gotten worse and both lateral ventricles were filled with blood and dilatated (**Figure 2**); likewise, the third ventricle contained blood (**Video 2**; available at www.jpeds.com). The patient died owing to hemorrhagic shock 1 hour later.

IVH represents the second most frequent cause of death in infants born prematurely and our patient owned most of the risk factors known to be associated with IVH: prematurity and low birth weight, being outborn, hypotension requiring inotropic support, cardiopulmonary resuscitation in the delivery room, and severe metabolic acidosis.[2-7] To date, there are still no specific therapies to limit expansion of IVH once it has occurred, but visualizing ongoing bleeding might suggest a tighter ultrasound monitoring to respond more promptly to possible complications, such as seizures and massive hemorrhage.[8] ■

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Eosinophilic Bloody Stool in a Neonate

A 5-day-old boy with a birth weight of 2276 g, born to a gravida 1 para 2 mother at 37 weeks of gestation, was transferred to our hospital because of bloody stools and vomiting. He had bloody stools after taking a formula made from cow's milk since 2 days of age. He had no fever and had normal vital signs and sucking ability, but he did not gain weight. On admission, blood tests showed peripheral blood eosinophilia (1224 cells/mm^3) and mild elevation of C-reactive protein level (3.2 mg/L), without anemia, hypoglycemia, and electrolyte derangements. The coagulation screen was normal.

Abdominal imaging showed no evidence of anatomical gastrointestinal obstruction. His stool was mucoid and bloody (**Figure**, A, *arrow*). Microscopic examination of Hansel-stained stool revealed eosinophil aggregation (**Figure**, B) and Charcot–Leyden crystals (CLCs) (**Figure**, C, *arrowheads*). The specific IgE levels for cow's milk were negative. After the infant was shifted from cow's milk to a hydrolyzed formula, bloody stools disappeared and his weight increased steadily. Oral food challenge (OFC) test was not attempted due to concern that that may trigger the recurrence of bloody stools and hence impede weight gain. We tentatively diagnosed him with non–IgE-mediated gastrointestinal food allergies (non–IgE-GI-FAs) due to cow's milk. One month after birth, the infant continued to gain weight while on the hydrolyzed formula and breast milk.

Non–IgE-GI-FAs are a subtype of food allergies that classically present with gastrointestinal symptoms despite the absence of food-specific IgE level elevations. Bloody stools and vomiting that occur in the neonatal period are classified under chronic food protein–induced enterocolitis syndrome.[1,2] This non–IgE-GI-FA phenotype was suggested to be involved in a Th2-type eosinophilic gastrointestinal inflammation and shows eosinophil-containing stools in some patients.[3] CLCs are needle-like structures that are detected at the infiltration sites of eosinophils.[4] The presence of CLCs in our case indicates that eosinophilic inflammation was occurring in the gastrointestinal tract of our patient.

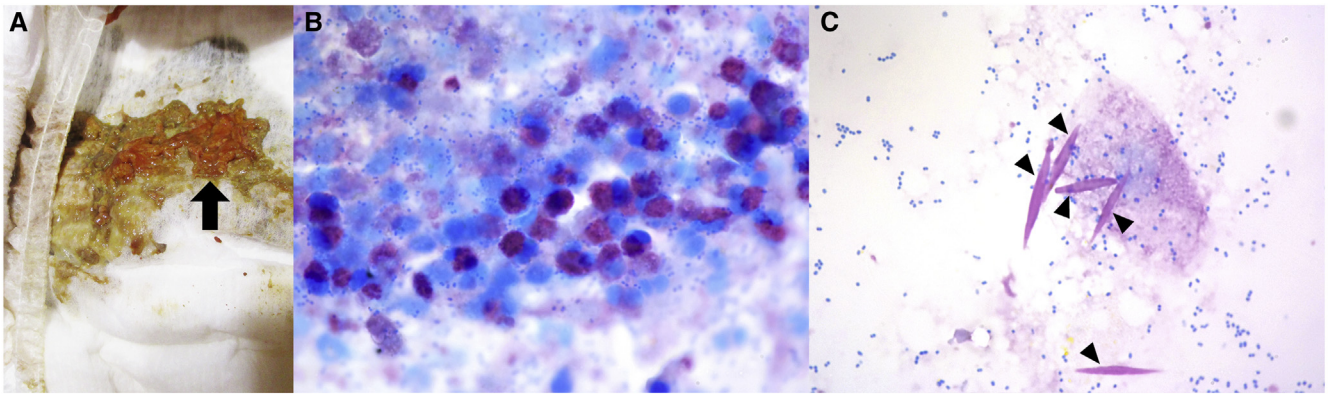

Figure. **A,** Bloody stools (*arrow*) and microscopic imaging of stool sample from a 5-day-old boy with non–IgE-GI-FAs due to cow's milk showing **B,** eosinophil aggregation and **C,** CLCs (*arrowheads*). **B** and **C** are Hansel-stained samples; original magnification: ×1000.

The authors declare no conflicts of interest.