Clinical History

- Female infant born at 25 weeks due to placental abruption.
- Head ultrasound (US) on the first day of life showed findings of prematurity but no hemorrhage.
- Six weeks into hospitalization (31 weeks’ corrected gestational age [GA]), developed methicillin-resistant Staphylococcus aureus (MRSA) sepsis and pneumonia, suffered tension pneumothorax and a hypoxic event. Head US ordered.
- Another US ordered 13 weeks into hospitalization (38 weeks’ corrected GA).
Figure 1. Coronal neonatal head US at the level of Sylvian fissures: day 1 of life (25 weeks’ corrected GA).
Figure 2. Coronal head US at the level of Sylvian fissures: 6 weeks of life (31 weeks’ corrected GA).
Figure 3. Coronal head US at the level of Sylvian fissures: 13 weeks of life (38 weeks’ corrected GA).
Figure 4. Sagittal left paramedian head US: 13 weeks of life (38 weeks’ corrected GA).
Figure 5. Sagittal right paramedian head US: 13 weeks of life (38 weeks’ corrected GA).
Figure 6. Coronal high-resolution head US: 13 weeks of life (38 weeks’ corrected GA).
Cystic periventricular leukomalacia

Periventricular leukomalacia (PVL) is a form of white matter brain injury, characterized by coagulation necrosis of the white matter lateral to the lateral ventricles. It can affect newborns and less commonly fetuses: Premature infants are at the greatest risk of developing the disorder. Affected infants generally exhibit motor control problems and other forms of developmental delay, and they often develop cerebral palsy and epilepsy later in life. This pathology of the brain has been described with various names, including “encephalodystrophy,” “ischemic necrosis,” “periventricular infarction,” “coagulation necrosis,” “leukomalacia,” and “softening of the brain,” among others.
Diagnosis

A major cause is thought to be changes in blood flow to the periventricular white matter. This area is fragile and prone to injury, especially before 32 weeks’ gestation.

Infection around the time of delivery may also play a role in causing PVL. Premature babies who have intraventricular hemorrhage are also at increased risk for developing this condition. Preventing or delaying premature birth is considered the most important step in decreasing the risk of PVL.
Figure 1. Head US on the first day of life shows findings of prematurity but no hemorrhage.

Figure 2. Head US at 6 weeks of life shows increased echogenicity in the periventricular white matter, concerning for ischemia.
Figures 3 and 4. Head US at 13 weeks of life shows interval development of severe cystic PVL, severe white matter volume loss causing “ex vacuo” dilatation of the lateral and third ventricles and increased extra-axial fluid, and severe atrophy of the basal ganglia, all thought to be related to MRSA sepsis and hypoxic-ischemic injury.
References

