History: 2-month premature infant presented with rhythmic jerking of all 4 extremities at age two weeks. He had been born by Caesarian section at age 30 weeks, due to fetal bradycardia and underlying subchorionic hemorrhage. His hospital course was complicated by Group B Streptococcus pneumonia, for which he received 10 days of ampicillin and gentamicin. He also had anemia of prematurity (treated with packed red blood cells) and thrombocytopenia. Three days before the seizure, his CRP and WBC were noted to be elevated, with a marked shift to the left.

Physical exam: Afebrile, with blood pressure of 60/36, pulse of 187, and respiratory rate of 51. The patient appeared lethargic, with arms and legs were flexed. He was able to move all four extremities, jerking them in clusters of 3 to 5 jerks, every 10 to 20 seconds. Jerking was most pronounced in the upper extremities. Hypertonia was noted between episodes, especially at the right lower extremity. The anterior fontanelle was soft, and deep tendon reflexes were normal. The grasp reflex was weak and inconsistent.

Differential diagnosis: Seizures secondary to hypoxic-ischemic encephalopathy, hemorrhage, meningitis/cerebritis, mass, seizure disorder, toxic/metabolic encephalopathy, accidental trauma, non-accidental trauma.

Laboratory results: WBC of 17.5K, Hb 11.4, Hct 34.6, platelets 60. Lumbar puncture yielded bloody fluid that grew no organisms.

Imaging findings: Head ultrasound shows markedly dilated lateral, third and fourth ventricles that are filled with heterogeneous moderately echogenic material. Parenchyma is compressed. Decreased sulci due to prematurity.

Differential diagnosis based on imaging: Intraventricular hemorrhage vs pus.

Clinical course: The patient was started on phenobarbital, with resolution of tonic-clonic seizure activity. Right ventricular tap was performed to relieve intracranial pressure, with drainage of purulent material, which eventually grew Escherichia coli. The patient expired four days later.

Diagnosis: Escherichia coli meningitis

Discussion: Meningitis is a common cause of hydrocephalus and neurological problems worldwide. It usually results from seeding of the choroid plexus in the setting of sepsis. Meningeal inflammation leads to vasculitis, cortical infarcts, and diffuse cerebral edema. In infants, Group B streptococcus, Escherichia coli, and Listeria monocytogenes are responsible for most cases of meningitis and cerebritis.

If US is performed in the acute phase, it may demonstrate thick, echogenic meninges, prominent cortical vessels, and unusually wide sulci. Increased subarachnoid fluid may be seen. As the disease progresses, the ventricles fill with exudate that is less echogenic than acute hemorrhage. It evolves to appear more uniformly complex and lacy than chronic hemorrhage, allowing differentiation. Intraventricular cysts and isolated entrapped ventricles may form secondary to adhesions.
In the acute phase, ventriculitis and adhesions at the cerebral aqueduct or the fourth ventricular foramina may cause obstructive hydrocephalus. Hydrocephalus may alternatively arise from extra axial impaired resorption of fluid. In the chronic phase, hydrocephalus may be either obstructive or secondary to global parenchymal loss.

Parenchymal foci of increased echogenicity represent infection, hemorrhage, or ischemia. The entire brain may appear edematous due to extensive cerebritis. Especially in the setting of *Citrobacter koseri*, vasculitis may lead to infarction, necrosis, liquefaction, and abscess formation. Brain abscess initially demonstrates increased echogenicity and hypervascularity, evolving to a complex mass and eventual cavitation with marginal hyperemia.