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INTRODUCTION

BACKGROUND:

42-day-old 1640 gram female born at 33 weeks 3 days gestation to a 20-year-old mother after a pregnancy complicated by limited prenatal care and history of drug abuse.

TESTS:

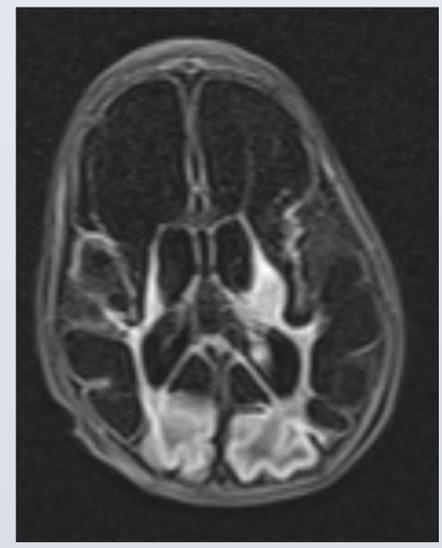
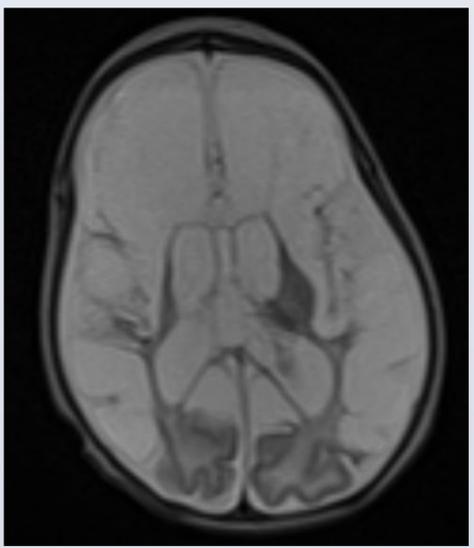
CSF, skin, and nasal samples were HSV-2 positive. Meconium drug screen was positive for buprenorphine, marijuana, and opiates.

CLINICAL COURSE:

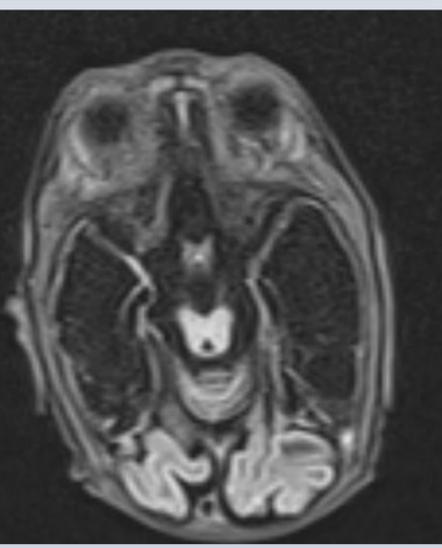
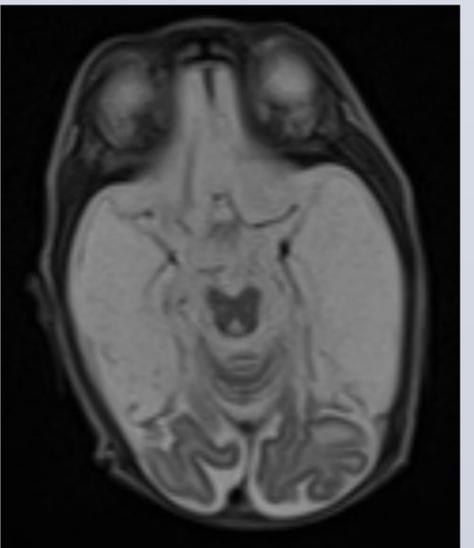
Baby was born by C-section with 9/9 Apgars and required CPAP initially. Later, the infant developed abdominal distension, hyponatremia, seizures, and was subsequently loaded with phenobarbital. After a sepsis workup and positive lab testing, the infant was started on acyclovir, ampicillin, vancomycin, and cefepime. The infant fed poorly and was unable to maintain temperature. On physical examination there was increased tone and stiffness in the periphery with decreased central tone, poor head growth with overlapping sutures worrisome for ongoing developmental delay. The baby was discharged one month later in the care of a relative.

IMAGING: MRI brain without contrast showing multi-cystic encephalomalacia of bilateral frontal, temporal, and anterior parietal lobes.

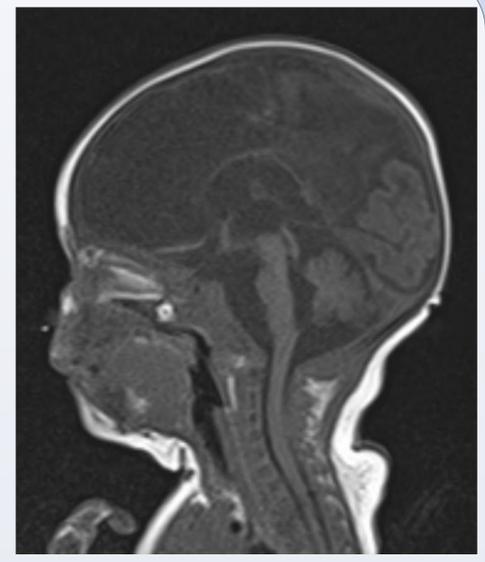
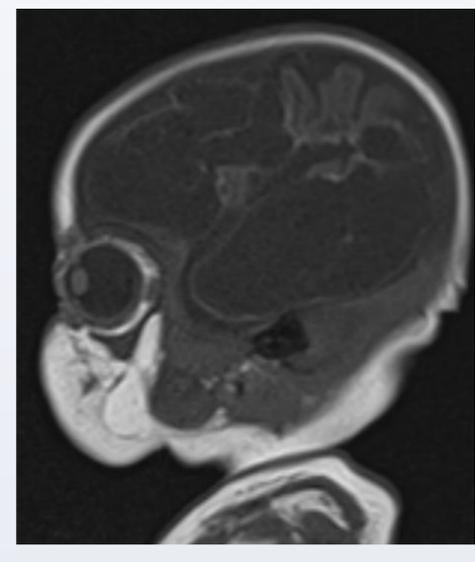
REPRESENTATIVE IMAGING



MRI brain without contrast: axial T2 (left) and axial FLAIR (right) demonstrating severe multi-cystic encephalomalacia involving the bilateral frontal and bilateral anterior parietal lobes. Minimal normal brain tissue is seen in the bilateral occipital lobes.



MRI brain without contrast: axial T2 (left) and axial FLAIR (right) demonstrating severe multi-cystic encephalomalacia involving the bilateral temporal lobes. Minimal normal brain tissue is seen in the bilateral occipital lobes.



MRI brain without contrast: sagittal T1 and FLAIR imaging demonstrates severe cystic encephalomalacia involving the frontal, anterior parietal, and temporal lobes. Minimal normal brain tissue in the occipital and posterior parietal lobes.

HSV ENCEPHALITIS & CYSTIC ENCEPHALOMALACIA

HSV encephalitis in neonates most often occurs from infection with HSV-2 obtained during descent through the birth canal (85% occur peripartum). 10% of cases occur postnatally, and 5% occur in utero. CNS infection results in diffuse encephalitis with infarction, which is fatal or leads to severe neurologic consequences. In the chronic stage of infarction, cellular debris and dead brain tissue are removed by macrophages and replaced by cystic encephalomalacia and gliosis.

Newborns are typically asymptomatic for one or two weeks. Later, symptoms include fever, rash, lethargy, and seizures in the first several weeks of life. If the patient survives, microcephaly, mental retardation, microphthalmia, enlarged ventricles, intracranial calcifications and multi-cystic encephalomalacia may occur.

Unlike HSV encephalitis that occurs in children and adults, neonatal HSV encephalitis is diffuse and does not have a predilection for the temporal lobes. Early in the course, cranial US will show increased parenchymal echogenicity. CT shows diffuse brain swelling and bilateral patchy areas of hypodensity in the cerebral white matter and cortex with sparing of the basal ganglia, thalami, and posterior fossa structures. Hypodense lesions on CT correspond to T2 hyperintensities on MRI and progress to necrosis and cystic encephalomalacia. Associated hemorrhage, calcifications, meningeal and patchy parenchymal enhancement may be seen on both CT and MRI.

HSV encephalitis is highly lethal (50%) and can cause permanent disability if left untreated. Intravenous antivirals are the treatment of choice, usually acyclovir.

REFERENCES

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