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Neuroimaging of Viral Infections in Infants and Young Children

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Many viral infections can involve the central nervous systems (CNS) of fetuses, neonates, infants, and children. The spread of the infections to the CNS may occur by way of the hematogenous route through the placenta, during the peripartum period, from the respiratory system, or the gastrointestinal tract, or through the peripheral nervous system. The pathogenesis, patterns of CNS involvement, and species of viral infection may differ in the developing fetus, infancy and neonate, and early childhood. For example, viral infections of the CNS in the fetus (particularly in the first and second trimesters) may cause severe destruction of the developing brain, thus leading to a developmental anomaly. In young children, parainfectious allergic encephalitis is often accompanied by typical clinical course and reversible (mostly) white matter demyelination in the brain and spinal cord, which share certain unique characteristics in the category of viral infection in children. Although different species of viral infection may have different patterns of CNS involvement, most viral encephalitides are nonspecific. Some viral infections may have recognized patterns of encephalitic lesion distribution, especially the herpes virus group (involving the gray matter) and the enterovirus (EV) group (involving the tegmentum of brainstem and spinal cord), which may help in imaging differentiation. In this article, the authors review the clinical courses, pathologic findings, and imaging features of the most common viral infections that may involve the CNS of neonates and infants, divided into the following three categories: congenital and neonatal CNS viral infections, common CNS viral infections in infants and young children, and parainfectious encephalomyelitis.

Congenital and neonatal central nervous system viral infections

Cytomegalovirus

Congenital cytomegalovirus (CMV) disease is the most common viral infection among newborns [1]. In utero, transmission can come from primary

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maternal infection, or as a result of reactivation or reinfection of seropositive mothers. Infants can also be postnatally infected during parturition or after birth, from breast milk, saliva, urine, or other sources [2]. Primary infection occurs in as many as 2.2% of pregnant women, and serologic or culture evidence of intrauterine CMV infection has been reported in 0.2% to 2.2% of all live-born neonates [3]. The clinical symptoms and signs of the infected newborn include hyperactivity, hypotonia, jaundice, hepatosplenomegaly, petechiae, thrombocytopenia, small head size, seizure, chorioretinitis, impaired hearing, and developmental delay [2,4,5]. Nearly one half of infected infants are reported to have CNS complications [2]. Confirmative diagnosis requires viral isolation from the body fluid of infants within 3 weeks of birth or detection of CMV DNA from amniotic fluid (when intrauterine) or fetal blood by polymerase chain reaction (PCR) analysis, with the latter being reported 100% in sensitivity and 99% in specificity [2,6]. The mechanisms of CNS injury in congenital CMV have been postulated to be the affinity of the CMV virus to the rapidly growing germinal matrix cells or lenticulostriate small vessels, resulting in abnormalities of the cerebral and cerebellar cortices, periventricular calcification, and lenticulostriate vasculopathy [5].

Transabdominal or transvaginal sonography is the first-line imaging study to identify brain abnormalities in fetuses with suspected CMV, followed by fetal MR imaging. The sonographic findings of congenital CMV infection include microcephaly, echogenic intraparenchymal foci (calcifications), ventriculomegaly, intraventricular adhesions, periventricular pseudocysts, sulcation and gyral abnormalities (lissencephaly and polymicrogyria), hypoplastic corpus callosum, cerebellar abnormalities (hypoplasia, calcification), large cisterna magna, and striatal vasculopathy [3]. On CT and MR imaging, brain abnormalities depend on the degree of brain destruction and the timing of the injury. The clinical sequelae appear to be more severe if maternal infection occurs during the first or second trimester [2]. The findings include lissencephaly (injury before 16 to 18 weeks of gestational age), polymicrogyria (injury between 18 and 24 weeks of gestational age) (Fig. 1), extremely diminished volume of the white matter, delayed myelination, small cerebellum, enlarged lateral ventricles, and periventricular calcifications (Fig. 2) [7].

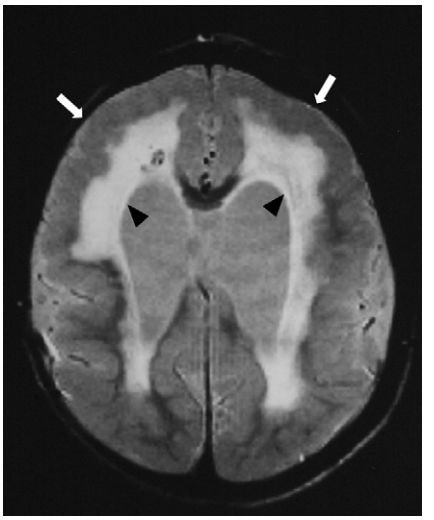

Fig. 1. Congenital CMV infection in a 5-year-old boy. Axial proton-density-weighted MR scan shows microcephaly, hydrocephalus, periventricular white matter edema (*black arrowheads*), and diffuse polymicrogyria (*white arrows*) over the frontotemporal and parietal lobes.

Herpes simplex virus

Neonatal herpes simplex encephalitis (HSE) is most commonly caused by the herpes simplex virus (HSV) type 2 (genital herpes); 85% of cases occur during the peripartum period, 10% in the postnatal, and 5% in utero. In patients 6 months of age or older, virtually all cases of HSE are caused by HSV type 1 (orofacial herpes) [8,9]. Intrauterine HSE occurs in approximately 1 in 300,000 deliveries. Infants who acquired HSV in utero typically have a triad of clinical findings, consisting of neurologic (microcephaly, encephalomalacia, hydrocephaly, intracranial calcifications), ophthalmologic

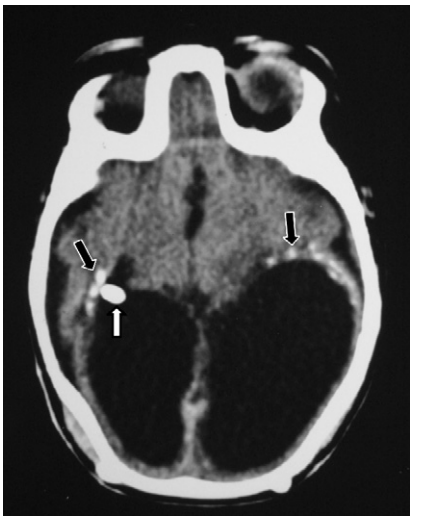

Fig. 2. Congenital CMV infection in a 1-year-old boy. Head CT scan shows marked dilatation of the lateral ventricle, with calcification along the periventricular region (*black arrows*). Note the ventriculoperitoneal shunt tube insertion in the right lateral ventricle (*white arrow*).