Congenital infections

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Infections of the fetal nervous system differ from those of older children because they act on the nervous system while it is developing. The manifestations and outcomes of these infections differ depending upon the age of the fetus at the time of infection. In general infections during the first two trimesters will result in congenital malformations, whereas those that occur during the third trimester usually manifest as destructive lesions.

The primary etiologic agents include cytomegalovirus (CMV), toxoplasma gondii, rubella virus, herpes simplex virus, varicella zoster virus, lymphocytic choriomeningitis virus (LCMV), syphilis, and human parvovirus B19. In most regions CMV infection is the most frequent congenital infection followed by toxoplasmosis.

Transmission of infections of the fetus occur via two main pathways. Bacteria usually ascend from the cervix to the amniotic fluid. In contrast, toxoplasmosis, syphilis, rubella, CMV, and other viruses are transmitted via the transplacental route.

Neuroimaging techniques include ultrasonography (US) and MRI during the prenatal period. In the neonates US, MRI and very often CT are performed in combination because CT is highly accurate to detect calcifications. Intracranial calcifications are mostly seen in CMV infection and toxoplasmosis. Neuroimaging features of CMV infections include intracranial calcifications, ventriculomegaly, neuronal migration anomalies, temporal, germinal matrix and-or cerebellar cysts, and white matter abnormalities. Polymicrogyria is the primary cortical malformation seen in CMV infection, and its diagnosis is highly challenging in fetuses below the age of 26 weeks. Diffusion tensor imaging is necessary to look at the cortical plate carefully. Toxoplasmosis is characterized by less extensive intracranial calcifications than CMV, hydrocephalus most often caused by ependymitis occluding the aqueduct, and cortical malformations that are less frequent than in CMV infections. Therapy by antibiotics may be prescribed during the prenatal period to improve the long-term prognosis. Herpes simplex virus infection usually manifests in the neonatal period with extensive brain damage as
multifocal lesions, deep gray matter injury, hemorrhage, and watershed pattern of injury. Immunization programs have decreased the epidemics of rubella. However it is still a major concern in developing nations. Rubella virus is responsible for lobar destruction, extensive encephalomalacia, lenticulostriate vasculopathy, and intracranial calcifications especially in the periventricular white matter and the basal ganglia. Congenital syphilis occurs in children born from untreated mothers. However the early clinical manifestations usually appear in the first two years of age. Neuroimaging features are basilar meningitis causing leptomeningeal enhancement and hydrocephalus, and infarctions. LCMV infection may mimic CMV infection or toxoplasmosis. Congenital varicella infection is rare and is responsible for hydrocephalus, cerebellar aplasia, polymicrogyria, necrosis of deep gray matter and cerebellum. AIDS encephalopathy occurs in infant born from untreated mothers. Radiologic features include lenticulostriate vasculopathy, progressive calcifications of the basal ganglia and diffuse white matter abnormalities. Proton MRS shows low NAA and elevated choline and myo-inositol. The effectiveness of preventing mother-to-child transmission of HIV by perinatal administration of antiretroviral therapy is now established. The risk of infection of the neonate has decreased to 1-2% in industrialized countries. Many prevention programs in developing countries are based on the use of zidovudine (azidothymidine, AZT) alone or in combination with other drugs. The drug is generally prescribed during the second half of pregnancy and then to the neonate for 6 weeks after birth. Transient mitochondrial damage is reported in neonates exposed to zidovudine: approximately one-third of children have hyperlactatemia during the first months of life, sometimes persisting until 6-12 months. Mitochondrial DNA depletion and ultrastructural mitochondrial pathology are also described in peripheral blood lymphocytes. MR signal changes are described in uninfected children exposed to antiretroviral treatment with and without mitochondrial dysfunction. These MR changes consist mainly of periventricular white matter and brainstem (tegmentum) high signal intensities on T2 and FLAIR images. Other neurologic complications include the higher risk of early febrile seizures for children perinatally exposed to antiretrovirals compared to non-exposed children.

References


