

Imaging of congenital central nervous system infections

TORCH

- toxoplasmosis
- other (syphilis, enteroviruses, VZV, parvovirus B19, Zika)
- rubella
- cytomegalovirus
- herpes simplex virus

Mécanisme lésionnel:

1. Pathogen-specific endotoxins
2. host's inflammatory response

Early interfere with normal brain development

1. Cytomegalovirus

most common across the placenta.

10% only are symptomatic at birth (epilepsy, neurodevelopmental delay, cerebral palsy, vision loss and sensorineural hearing loss)
25% of asymptomatic still develop sequelae by age 2, such as sensorineural hearing loss.

Imaging findings

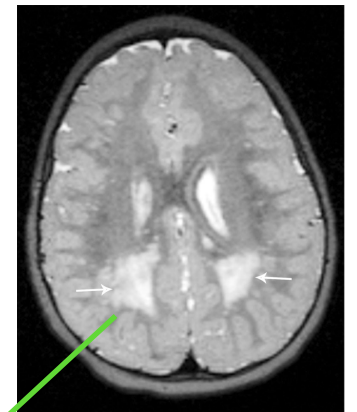
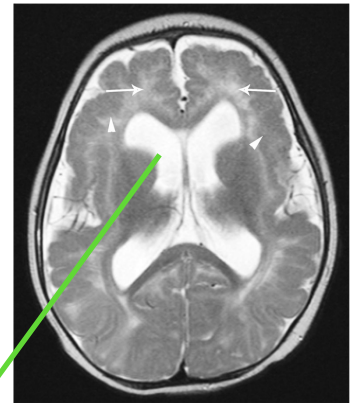
CMV neurotropic hematogenously (choroid plexus, ependyma, germinal matrix and capillary endothelium)

=> interfere neuronal migration

=> Capillary involvement can lead to thrombosis, ischemia

1. ventriculomegaly,
2. migration disorder (microcephaly agyria/pachygyria, cerebellar hypoplasia polymicrogyria schizencephaly)
3. white matter disease (posterior)
4. calcifications (periventricular & deep gray nuclei)
5. Periventricular cysts (anterior temporal lobes)

In isolation, cerebral calcifications, migrational abnormalities, white matter disease and anterior temporal cysts are nonspecific, though in combination these findings raise concern for congenital CMV disease.



2. Toxoplasmosis

protozoan parasite

classic triad (10% of cases!)

- chorioretinitis
- hydrocephalus
- intracranial calcifications

Imaging findings

1. calcifications
2. large ventricles
3. macrocephalus or microcephalus
4. hydrocephalus
5. parenchymal destruction/volume loss
6. orbital (microphthalmia)



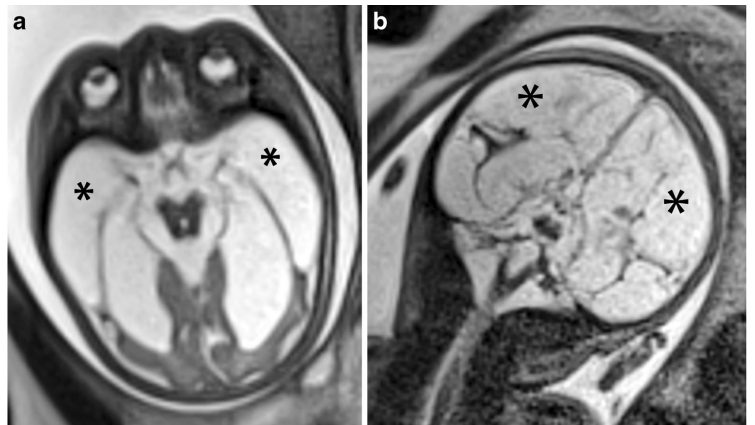
In contrast to congenital CMV disease, hydrocephalus is frequently associated with congenital toxoplasmosis, while migrational abnormality is very rare.

3. Herpes simplex virus

most common route is in perinatal maternal genital tract

Imaging findings

1. Infection intra-uterine
 - encephalomalacia
 - ventricular enlargement
 - calcifications



2 Encéphalite postnatale

edema / multifocal DWI restriction/ hemorrhage

=> severe parenchymal destruction rapidly (cystic encephalomalacia, cortical thinning, atrophy, calcifications, ventricular enlargement)

Unlike older, no predilection for the temporal lobe => white matter, cortical gray matter, basal ganglia, temporal lobes, brainstem, cerebellum.

Patchy parenchymal and meningeal enhancement

