Syllabus contribution to ISMRM 2012

**Imaging of Paediatric Intracranial Infections**

Simone A. Mandelstam

Academic Affiliation: Department of Radiology University of Melbourne and Melbourne Brain Centre, Florey Neurosciences Institutes.

Hospital Affiliation: Department of Medical Imaging, Royal Children’s Hospital, Flemington Road, Parkville 3052, Melbourne, Australia

**Introduction:**

The broad topic of paediatric intracranial infections encompass a wide range of organisms and processes that vary with age. These may result in significant morbidity and mortality if there is a delay in initiation of treatment. Infection may be acquired in various ways such as via haematogenous route, adjacent infection (e.g. mastoiditis) or a penetrating injury. The radiologist performs a vital role in establishing the diagnosis and excluding complications. Neuroimaging is generally requested prior to performing a lumbar puncture (LP) to avoid possible herniation in the potential setting of increased intracranial pressure. Imaging modalities include ultrasound (in infants), CT scans and MRI, each of which can be useful in different situations. It is important to stress that a normal scan does not necessarily mean that it is safe to perform an LP.

**Meningitis**

Bacterial meningitis has 10% mortality and 40% of survivors have neurological sequelae. CT and MRI may show leptomeningeal enhancement but may be normal. MRI is more sensitive than CT for demonstrating contrast enhancement (CE). Complications include subdural effusion/empyema, cerebritis, cerebral abscess, ventriculitis, arterial infarcts, venous thrombosis, venous infarcts and hydrocephalus. Organisms that commonly cause bacterial meningitis differ according to the patient’s age.

**Neonates:** Group B Streptococcus, Escherichia Coli, Listeria monocytogenes, Enterococcus, Citrobacter

Infants: Group B Streptococcus, Staphylococcus aureus, E Coli, Pseudomonas aeruginosis, Citrobacter, Listeria monocytogenes

Children < 15 years: Streptococcus pneumoniae, Neisseria meningitides, Haemophilus influenzae b (decreased in countries with Hib vaccine)

Adolescents: Streptococcus pneumoniae, Neisseria meningitides, Haemophilus influenzae b or non type b.

Brain abscess

A brain abscess is a focal intracerebral infection that begins with a localized region of cerebritis, evolving into a discrete collection of pus surrounded by a well-vascularized capsule. Such infections result from either hematogenous dissemination or local extension from an odontogenic, paranasal sinus, or otic source. The most common organisms include Staphylococcus and Streptococcus species. Brain abscess development can be divided into four stages:
1) Early cerebritis (1 to 4 days)
2) Late cerebritis (4 to 10 days)
3) Early capsule formation (11 to 14 days)
4) Late capsule formation (>14 days)
Abscesses tend to grow toward the white matter, away from the better-vascularized grey matter, with thinning of the medial walls. Diffusion imaging is helpful to differentiate abscess from cystic tumour. Proton MRS can also play a role in the diagnosis of cerebral abscess presence and type.

Tuberculosis

Intracranial tuberculosis is caused by hematogenic spread from a primary focus (usually pulmonary) elsewhere in the body. The initial tuberculous lesions may develop either in the brain parenchyma or the meninges, and are known as Rich foci. The main manifestations are TB meningitis (TBM), parenchymal tuberculomas and tuberculous abscesses. TBM is an exudative meningitis with basal ganglia/thalamic/internal capsule infarctions secondary to vasculitis/(pan) arteritis. Cranial nerve impairment may result from vascular compromise or entrapment of the nerve in basal exudates. Neurological sequelae such as marked impairment of

cognitive and motor function are reported in survivors.

The common triad of neuroradiological findings in TBM is:
1. Basal meningeal enhancement
2. Infarctions (MRI more sensitive)
3. Hydrocephalus

**Encephalitis**

Approximately 1000-2000 cases of encephalitis are reported to the Centers for Disease Control and Prevention in Atlanta per year. Infectious causes include viruses, parasites, mycobacteria, fungi and unusual bacteria such as mycoplasma pneumoniae and legionella pneumophila.

Some viral infections may have recognized patterns of encephalitic lesion distribution, especially the herpes virus group (grey matter) and the enterovirus group (tegmentum and spinal cord). Six members of the herpesvirus family cause neurologic disease in children. HSV-2 and congenital CMV will not be covered in this talk, as they will be discussed under congenital infections. The others are:

a] HSV-1: HSV encephalitis of childhood shows asymmetric limbic system involvement that may be bilateral and hemorrhagic. Deep grey matter is spared.

b] Varicella Zoster Virus (VZV): VZV causes cerebellitis and vasculitis that may present as basal ganglia infarct. Immunocompetent and immunosuppressed patients may develop multifocal leukoencephalopathy.

c] Ebstein-Barr Virus (EBV) shows tropism for deep grey matter structures with nonspecific involvement of cortex and hemispheric white matter. Immunosuppressed patients may develop EBV- associated CNS lymphoma.

d] Human Herpes Virus -6 (HHV-6) is neurotropic, often involving the limbic structures and medial temporal lobes. It is associated with febrile seizures in infants. It may present as meningoencephalitis, leukoencephalitis and acute necrotizing encephalitis especially in immunocompromised patients.

**Fungi**

Fungal infections occur most commonly as opportunistic infections in
immunocompromised patients. They cause meningitis, meningoencephalitis, intracranial thrombophlebitis and brain abscess. Imaging appearances may differ between immunocompetent and immunocompromised children.

Some of the more common fungal infections include:

a) **Candida**: Resembles other granulomatous diseases by invading vessel walls – vasculitis – infarcts +/- hemorrhages. Invasive form progresses to abscess and cerebritis.

b) **Aspergillus fumigatus**: Similar to Candida, causes cavitatory lesions with partial rim enhancement and hypointense T2 rim +/- haemorrhage.

c) **Coccidioidomycosis**: basal granulomatous meningitis, very similar to TBM.

d) **Cryptococcus**: hydrocephalus, vasculitis – ischaemia, cortical atrophy, non – enhancing basal ganglia pseudocysts, rarely enhancing parenchymal lesions.

**Parasites**

Parasitic CNS infections remain a significant problem in underdeveloped nations. **Neurocysticercosis** is endemic in Mexico, central/ Latin and South America, India, Africa and China. Parenchymal infestation is common. Less commonly affected areas are subarachnoid spaces, ventricles and spinal cord (rare). CT and MRI findings in parenchymal neurocysticercosis depend on the stage of development of the parasites.

There are 4 stages:

a) **Vesicular stage** (viable): small, round low density, well demarcated, no perilesional oedema and no CE. The scolex can be visualized as an internal nodule.

b) **Colloidal stage**: ill-defined lesions surrounded by oedema. Most show ring enhancement. On MRI the cyst walls are thick and hypointense on T2 with perilesional oedema.

c) **Granuloma**: Nodular hyperdense lesions on CT surrounded by oedema. On MRI, granular cysticerci are signal voids on T1 and T2 surrounded by oedema or gliosis with hyperintense rims around the area of signal void.
Calcified: (dead) Small hyperdense nodules without oedema or contrast enhancement. Difficult to see on conventional MRI sequences.

**Hydatid**

Cerebral involvement by hydatid disease occurs in 1–4% of patients infected with the parasite. Common CT and MRI findings of simple intracranial cystic hydatidosis include the presence of well defined, smooth, thin-walled, spherical homogenous cystic lesions. The intensity of the cystic contents is similar to that of CSF. On unenhanced CT scans, the cyst wall is isodense or hyperdense to brain tissue. On MRI scans, the cyst wall has low intensity on both T1-and T2-weighted images. Usually, no enhancement of the walls is noted in simple cysts however the pericyst may enhance. This finding is further substantiated by the absence of perilesional edema on T2-weighted images. The most important factors in prognosis are the localization of the focus of infection, rupture of the cyst and dissemination of its content.

**References and further reading:**


García, HH, Del Brutto OH. Imaging findings in neurocysticercosis. Acta Tropica 2003; 71-78.


Website: Centers for Disease Control and Prevention, USA http://www.cdc.gov/