Neuro-cysticercosis with Japanese encephalitis: Magnetic Resonance imaging with diffusion and spectroscopy

Sona A. Pungavkar, Jeshil R. Shah, Samir Gadani, Alok Singhai, Deepak P. Patkar, Sanjay Desai
Department of MRI, Mammography and BMD, Dr. Balabhai Nanavati Hospital and Research Centre, Vile parle, Mumbai - 400 056, India

Key Words: Japanese encephalitis, neuro-cysticercosis, MRI, MR spectroscopy, MR diffusion studies

NCC is a parasitic infection, endemic in tropical countries, caused by cysticercus cellulosae, an intestinal tapeworm. JE is caused by flavi-virus, which is mosquito-borne. Usually, JE is clinically occult but may manifest as an acute, fulminant, neurological disease with fever, focal neurological signs and unconsciousness. It has high mortality rate (25%) and has permanent neurological and psychological sequelae in the survivors. In patients with NCC, sudden deterioration of clinical condition should warrant a search for associated JE. MRS may help in detecting changes of associated encephalitis non-invasively.

Case Reports

Case 1
A 28-year-old man had history of generalized tonic clonic (GTC) convulsions two years ago. CT scan done then had revealed a ring-enhancing lesion in the left posterior parietal region with a speck of enhancing nodule within it, suggestive of NCC. He was treated with albendazole. The patient was well except for occasional headache. He presented again with history of altered sensorium for two days following GTC convulsion (4 days before presentation), minimal neck rigidity, fever and headache. No focal neurological deficit was found. Routine laboratory investigations were non-contributory. MR scan revealed a small NCC in the left posterior parietal cortex with a small scolex, appearing hyperintense with hypointense rim on T2 and hypointense on T1 weighted images (WI) with peripheral ring enhancement. Moderate amount of perilesional edema was detected. In addition, bilateral asymmetrical hyperintensities were seen within the thalami on T2WI. The left thalamus (the side with the cysticercal cyst) was affected to a greater extent (Figure 1). MRS was performed using short TE (1500 / 35). There was reduction of N acetyl aspartate (NAA) / Creatine (Cr) and elevation of Choline (Cho) / Cr ratios. No abnormal peak was detected (Figure 2). The patient subsequently became comatose. The IgM antibody titers in serum and CSF done then were positive for JE. The patient was put on medical line of treatment, but expired after two weeks.

Case 2
A 14-year-old boy had history of GTC convulsions 15 months ago. MRI done then elsewhere had revealed a ring-enhancing lesion in the right posterior parietal region with a speck of enhancing nodule within it, suggestive of NCC. He was treated with albendazole. The patient presented again with history of fever, headache, altered sensorium followed by unconsciousness for one day following an episode of GTC convulsion, 3 days before presentation. Patient was febrile and comatose (Glasgow scale of 11/15). No focal neurological deficit was found. Routine laboratory investigations were within normal limits. On MRI, the cyst was smaller and was associated with minimal edema, located in the right high-parietal cortex with an eccentric speck with peripheral ring enhancement and enhancement of the speck. T2WI revealed bilateral asymmetrical hyperintensities within the basal ganglia, midbrain and thalami, more on right side (side of the NCC). Bilateral asymmetrical cortical and focal white matter hyperintensity was seen in the temporal and parietal lobes (right > left) (Figure 3). Restricted diffusion was seen in the thalami. Basal ganglia, dorsal midbrain, temporal lobes and posterior parietal cortex regions appeared minimally hyperintense on DWI and hypointense on apparent diffusion coefficient (ADC) maps (Figure 4). Possibility of JE was considered in view of the known association with NCC, involvement of thalami and known endemicity. Diagnosis was confirmed by serologic testing using paired blood and CSF samples. Patient was put on conservative medical line of treatment but expired after six weeks.
A higher prevalence of the intestinal parasitic diseases is seen in association with viral encephalitis. Intestinal helminths are postulated to facilitate the entry or reactivate latent neurotropic viruses by carrying them into the brain or through alteration of host response. Cysticercus cysts have been found in brain specimens of patients who die as a result of JE. Pigs are efficient amplifiers of the JE virus in its natural cycle and are the intermediate host of *Taenia solium.*

The characteristic MR findings with isolated JE have been described as bilateral symmetrical hyperintensities on T2 weighted images in the thalami and basal ganglia. Although reported, the involvement of the midbrain, pons, cerebellum and cerebrum is uncommon. In cases of JE with NCC, lesions were larger and exhibited greater hyperintensity on the side harboring the cyst. In patients with multiple NCC, the severity of JE correlated with the side bearing greater number of cysts or the side lodging a degenerating cyst. Both our cases had single NCC with perilesional edema, suggesting degenerating nature, implicating disruption of the blood-brain barrier or an altered immune response, facilitating entry and/or replication of the virus in susceptible hosts. There was involvement of the thalami, more on the side of the cyst. A greater number of pathologic necrolytic JE lesions have been described in autopsy specimens with NCC, than in those specimens without NCC. Other known associations of NCC include glioma, abscess, tuberculosis and infarction.

Restricted diffusion has been reported in cases of Rassmussen’s and Nipah virus encephalitis. Restricted diffusion was also observed in our patients in the regions of encephalitis. MRS performed in the acute stage of the disease revealed decrease in NAA / Cr and increase in Cho / Cr ratios. mI peak was increased and the mI / Cr ratio was slightly elevated. Similar findings have been described with Nipah virus encephalitis and Rasmussen’s encephalitis.

The findings on MRS in many of the encephalitis are similar, but they when found in a known case of NCC, the possibility of JE is most likely, especially if the involvement of the thalami is also noted. Knowledge of this association may help in early diagnosis and better management of the more serious condition (JE). NCC is likely to predispose one to JE infection and a modulator of the encephalitic process.

**References**


Accepted on 28-09-2004