

Role of Advanced Diagnostic Imaging in Intracranial Tuberculoma: MR Spectroscopy

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A 22-year-old G1P1 post-partum female presented to the Medicine Department with single episode of sudden onset right sided complex partial seizures 17 days after normal vaginal delivery. There was history of moderate headache and non-productive cough since few days. Her antenatal course was uneventful. No history of fever, weight loss, altered sensorium or contact with a tuberculosis patient. On physical examination, vitals and systemic examination were within normal limits. BCG scar was noted on left upper arm.

Complete blood count revealed mild anaemia (Hb =9 mg/dl), while rest of the blood counts were within normal range. ESR 25 mm/

hr was raised. Serum glucose, electrolytes, chest X-ray was within normal limits. Mantoux test was also not reactive.

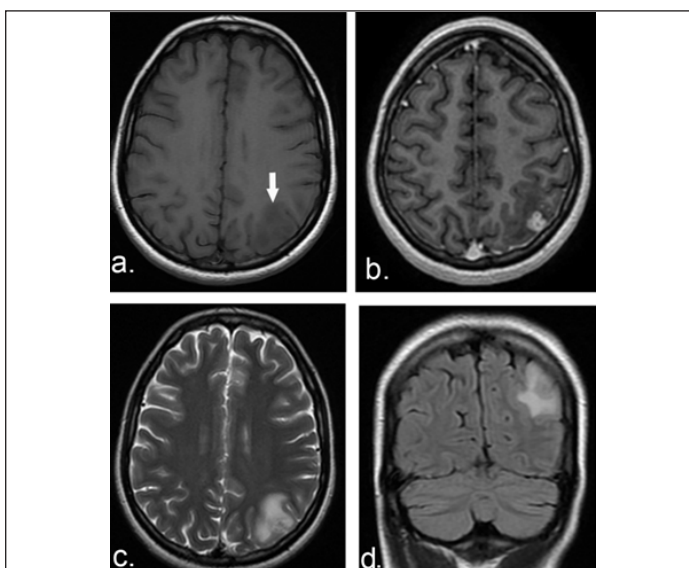
Brain MRI revealed well-defined hypo to iso-intense lesion in left high parietal lobe on T1WI. Lesion shows mixed signal intensity on T2 and FLAIR images with surrounding hyperintense oedema. Contrast enhanced T1 images revealed two conglomerated ring enhancing lesions [Table/Fig-1a-d]. No evidence of true restricted diffusion or blooming on GRE images [Table/Fig-2a-c].

On MR Spectroscopy, lesion shows 187720 MR units of lipid-lactate at 1.25 ppm (increased), 25311 MR units of choline at 3.2 ppm (increased) within the lesion with 1.39 choline-creatine ratio (increased) and 26832 MR units of NAA at 2 ppm (decreased) and 1.06 NAA-choline ratio (decreased) within the lesion. 18304 MR units of creatine at 3.09 ppm (decreased) and 1.47 NAA-creatine ratio (decreased) within the lesion [Table/Fig-3].

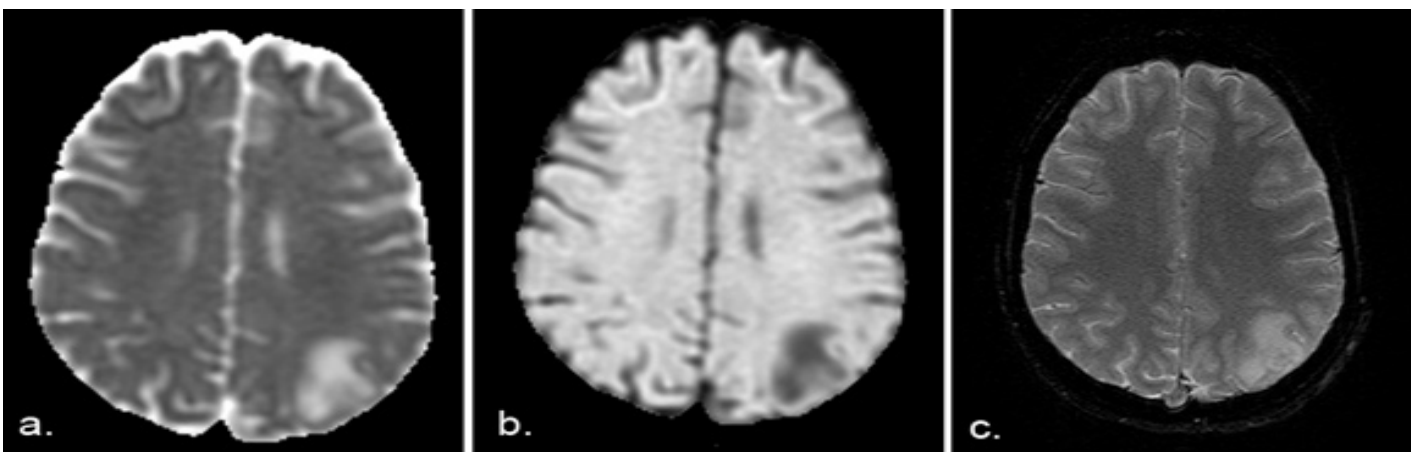
CSF analysis findings were as follows: glucose – 42 mg/dl, proteins – 61 mg/dl, white blood cells – 5/mm³, red blood cells – 0/mm³. Bronchoalveolar lavage smear was positive for acid fast bacilli [Table/Fig-4].

Patient was diagnosed as a case of the Intracranial tuberculoma on the basis of the clinical, laboratory and radioimaging findings. Patient was started on category I anti-TB therapy with injection streptomycin and discharged after 2 days with relief of symptoms. On follow-up after 15 days, patient was completely relieved of symptoms.

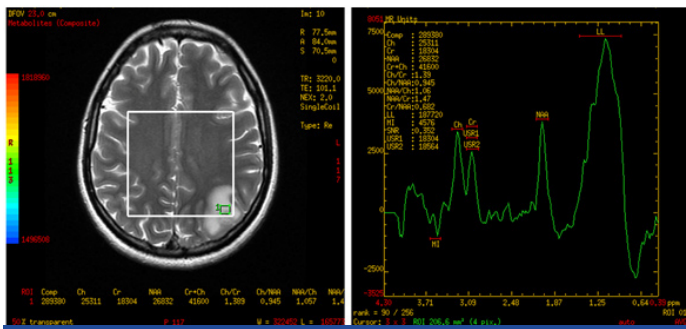
Intracranial TB occurs secondary to haematogenous spread from the focus of tuberculosis infection, most commonly in the lungs. It accounts for 2-5% of patients with TB and 10% of those with HIV-TB co-infection [1]. CNS TB can manifest either as diffuse involvement of CNS resulting in TB meningitis, or as a localized parenchymal infection resulting in tuberculosis granuloma or tuberculoma.



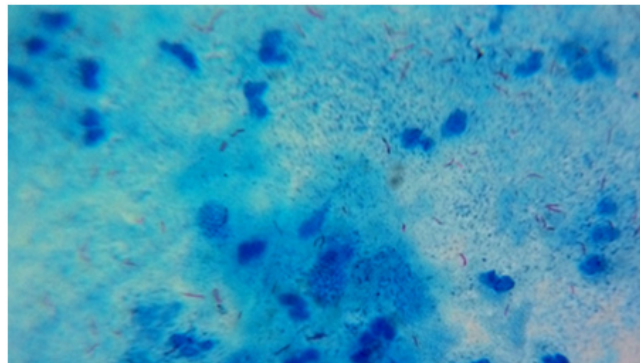
[Table/Fig-1a-d]: (a) Axial T1WI shows a well-defined hypo- to isointense lesion in left high parietal lobe (arrow). (b) Axial T1+C image shows two ring enhancing lesions. (c) & (d) Axial T2WI and Coronal FLAIR images show mixed intensity lesion with surrounding hyperintense oedema



[Table/Fig-2a-c]: (a) & (b) Axial DWI and ADC images show no evidence of restricted diffusion within the lesion. (c) Axial GRE image shows no blooming within the lesion



[Table/Fig-3]: MR Spectroscopy image shows increase in lipid-lactate within the lesion at 1.25 ppm, increase in choline at 3.2 ppm, increase in choline-creatine ratio, decrease in NAA at 2 ppm, decrease in NAA-choline and NAA- creatine ratios within the lesion



[Table/Fig-4]: Bronchoalveolar lavage smear showing tubercle bacilli on acid fast stain

Histologically tuberculomas demonstrate central caseating necrosis with a collagenous capsule containing epithelioid histiocytes, multinucleated giant cells (usually Langerhans type), plasma cells, fibroblasts and lymphocytes. The tuberculomas usually appear as lesions isointense to cerebral grey matter on T1WI and T2WI, which may or may not contain an area of central hypointensity on T1WI. On T2WI scattered hypointense foci may be noted within the iso-intense tuberculomas. Contrast enhanced T1WI usually demonstrate ring enhancement, which may be either a single ring or multiple conglomerate rings. The conglomerated ring enhancing lesions usually indicate a granulomatous disease rather than a neoplastic one. These types of the lesions were present in our case. According to the A Akhaddar et al., a 37-year-old multiparous

woman was presented with the recent headache and dizziness which was increasing in the severity over the period of time, which turned to be a brainstem Tuberculoma in the MRI [2].

Neurocysticercosis is the most common cause of inflammatory cerebral granuloma followed by tuberculosis. MRI is usually performed to differentiate cysticercal granuloma from a tuberculoma. However, due to similar MRI features, it is not much useful. MR Spectroscopy can complement MRI to differentiate between these two conditions [3]. Tuberculomas usually demonstrate large lipid peaks on MRS with increased choline levels and decreased levels of NAA and Cr. A Cho/Cr ratio of greater than 1 is typical of tuberculoma. Tuberculomas also demonstrate a prominent decrease in NAA/Cr ratio and slight decrease in NAA/Cho ratio. While neurocysticercosis demonstrate a combination of elevated levels of lactate, alanine, succinate and choline and reduced levels of NAA and creatine. MRS can also be used to differentiate between various other causes of ring enhancing lesions [Table/Fig-5].

MRS can complement as a useful non-invasive technique to differentiate between various ring enhancing lesions of brain, especially two most common conditions, viz. neurocysticercosis and tuberculomas.

	Tuberculoma	Neurocysticercosis	Pyogenic Abscess	Fungal Lesions
Lipid	↑↑	-	-	+/-
Lactate	+	+	+	+
Cytosolic Amino Acids*	-	-	+	+/-
Succinate	-	↑↑	+/-	-
Trehalose	-	-	-	↑↑

[Table/Fig-5]: Characteristics of different ring enhancing lesions on MRS

Legends - ↑= elevated, (+)= present, (-) = absent
*includes leucine, isoleucine, and valine

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