

Miliary Intracranial Tuberculomas Presenting as Rapidly Reversible Encephalopathy

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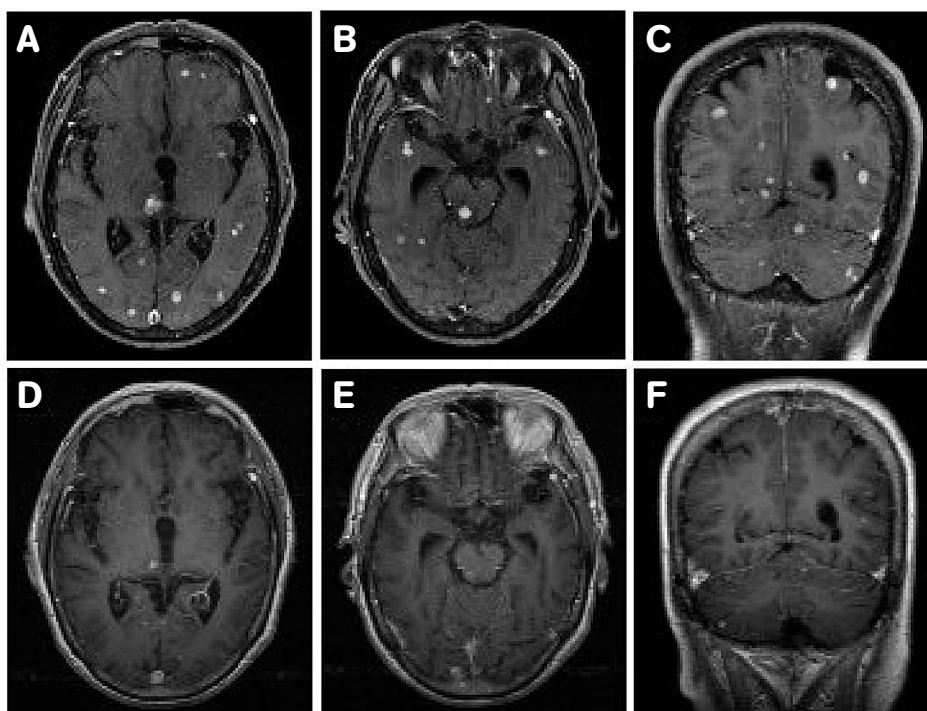


Figure. Brain MRI on admission and follow-up. Pre-treatment T1-weighted images with contrast enhancement in axial views (A, B) and coronal view (C) demonstrated multiple enhancing nodules in both supratentorial and infratentorial compartments. The follow-up MRI (D, E, F) on the 57th day of anti-TB therapy showed remarkable remission of tuberculomas.

A 70-year-old woman was admitted for rapid progression of anorexia, apathy, and akinesia within one month. She had a minor brain concussion from a traffic accident two weeks before the onset of symptoms. She was previously healthy without exposure history to

tuberculosis (TB). On admission, her vital signs were normal. Physical examination was unremarkable. Neurological examination revealed akinetic mutism and prominent grasp reflexes bilaterally. Neck stiffness was absent. Laboratory investigations including hemogram,

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liver and renal panels, and lipid profiles were within normal limits. Serum levels of cortisol, folic acid and vitamin B12, tumor markers, and thyroid function tests were all normal. The only abnormalities were hyponatremia (serum sodium: 128 m mol/L) and mildly elevated C-reactive protein (CRP: 35 mg/L, normal < 5 mg/L).

Brain CT showed several contrast-enhancing nodules in the pons, right thalamus and left frontal lobe. Brain MRI demonstrated multiple enhancing nodules in the midbrain, thalamus, cerebrum, and cerebellum. Those nodules varied in size with perifocal edema. Chest radiograph showed mild infiltrates in bilateral lung fields. High-resolution chest CT demonstrated ground-glass opacities with miliary nodules in both lungs. Histopathological exams from bronchoscopic biopsy and bronchial brushing showed chronic granulomatous inflammation without malignant cells.

Lumbar puncture was performed on the 7th hospital day and cerebrospinal fluid (CSF) examination revealed: cell count was 8 per high power field (50% mononuclear and polymorphonuclear leukocytes, respectively), protein 105 mg/dl, and glucose 43 mg/dL (blood glucose 145). Stains for bacteria, fungi, and acid-fast bacilli were negative. CSF cytology showed pleocytosis with monocytoïd cell predominance without malignant cells. Other screen tests for syphilis, cryptococcus neoformans, herpes simplex virus, and HIV were all negative. Bacterial cultures were negative. Open brain biopsy for pathologic proof was unavailable.

The patient was treated with intravenous methylprednisolone (500mg per 12 hours) since the 15th hospital day, followed by oral prednisolone 60mg/day. Although tuberculosis polymerase chain reaction of CSF was negative, mycobacterium tuberculosis complex was cultured from two sets of sputum. Combined therapy of rifampicin 480mg, isoniazid 320mg, pyrazinamide 1g, and ethambutol 1g once daily was subsequently administered. Repeated lumbar punctures on the 14th, 24th and 41st hospital day respectively revealed improvement in pleocytosis and protein elevation. Clinical improvement began within 10 days after anti-TB therapy. The patient was discharged on the 20th day of the treatment. A follow-up brain MRI on the 57th day of anti-TB therapy showed a marked resolution of multiple brain tuberculo-

mas. The patient had fully recovered after 6 months treatment.

Tuberculosis remains a worldwide health problem nowadays. Among central nervous system tuberculosis, intracranial tuberculoma is relatively rare and typically occurs in immunocompromised individuals or in patients with systemic TB. In developing countries, intracranial tuberculoma comprises 5% to 30% of all intracranial masses⁽¹⁾. Neuroimaging is helpful, especially with the presence of TB elsewhere in the body, and for the patients from endemic regions. Intracranial tuberculomas are usually confounded with inflammatory, malignancy, or infective lesions.

CT features of tuberculomas vary from the classical ring-enhancing lesions with an isodense center and surrounding edema to the rare non-enhancing hypodense lesions⁽²⁾. The characteristics of intracranial tuberculomas on MRI are extremely diverse. An iso- or hypointense core with a hyperintense rim on T2-weighted and fluid-attenuated inversion recovery (FLAIR) images is the usual presentation⁽²⁾. Single or multiple tuberculomas with ill-defined margins, patchy enhancement and occasionally calcifications is by far the most common⁽¹⁾. In our case, the miliary lesions diffusely disseminated in both supratentorial and infratentorial compartments were highly suggestive of hematogenous spreading.

Intracranial tuberculomas should be listed in the differential diagnoses of brain space-occupying lesions, even in immunocompetent patients without known systemic infection. Radiological findings as well as evidence of miliary TB elsewhere in the body could be a clue for diagnosis.

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