Clinical picture

Disseminated tuberculosis presenting as rapidly progressive dementia

A 35-year-old right-handed man presented with complaints of holocranial nonthrobbing headache with apathy, executive dysfunction and reduced social interaction for the past 1 month. No relevant systemic symptoms were present. General physical and neurological examination was unremarkable. Hematological parameters were normal except for raised erythrocyte sedimentation rate (52 mm in first hour). Gad-enhanced magnetic resonance imaging (MRI) brain showed acute left thalamic infarct with diffusion restriction (Figure 1a–c) and prefrontal meningeal enhancement (Figure 2a and b). CSF analysis revealed 120 cells (90% lymphocytes), high protein (126 mg/dl), low sugar (32 mg/dl; corresponding blood sugar: 89 mg/dl) and Adenosine deaminase (ADA) of 25 U/l. HIV was negative. Contrast-enhanced computed tomography chest and abdomen showed diffuse miliary nodules in the lung with mediastinal lymphadenopathy. Patient was treated as disseminated tuberculosis with oral Rifampicin (600 mg/day), Isoniazid (300 mg/day), Pyrazinamide (1200 mg/day), Ethambutol (800 mg/day) daily for the first 2 months along with oral steroids (Dexamethasone 24 mg/day in three divided doses for the first week followed by slow taper over the next 6 weeks) followed by maintenance phase therapy with oral Rifampicin and Isoniazid. His neurological symptoms dramatically improved within the first month of initiation of combination therapy.

Disseminated tuberculosis presenting as rapidly progressive dementia without any systemic features is uncommon. Our patient had thalamic dementia caused by vasculitic infarct in left thalamus. Infarcts in tuberculous meningitis are commonly seen in thalamus, basal ganglion and internal capsule.1 Even though commonly seen in bilateral thalamic lesions, dementia can occur in unilateral lesions of either thalamus.2

Figure 1. MRI brain showing (a) T2 hyperintense signal in left thalamus with diffusion restriction as evidenced by hyperintense signal in left thalamus (b) on diffusion weighted imaging sequence and hypointense signal (c) on apparent diffusion coefficient sequence.
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Figure 2. Gad-enhanced MRI brain showing (a) significant prepontine enhancement and (b) leptomeningeal enhancement along the right medial temporal lobe.

References
