

CLINICAL CASE - TEST YOURSELF

Neuroimaging

Young woman harbouring a midline space occupying brain lesion

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PART A

Young Pakistani woman harbouring a midline space occupying lesion with conglomerate enhancement pattern.

A 30-year-old Pakistani woman presented with a one-month history of progressive left upper extremity weakness and headache. The patient was afebrile and physical examination, during which the patient was awake, alert and fully oriented, revealed minor facial paresis on the left, pupils normal in size with no deviation at primary gaze and normal oculomotor function bilaterally. The soft palate was symmetrically elevated, and the tongue mobile with a full range of motion. Left arm (brachial) paresis with pronation

and disordered fine motor skills were observed. The patient was able to walk safely, with a steady gait and normal posture. Tendon reflexes were symmetrically increased at the upper extremities and normal at the lower extremities, with a negative Babinski reflex. Hypoesthesia and hypoalgesia were also noted on the left. All CBC (complete blood count) values were normal, and a brain MRI scan was performed (Fig 1); a large, lobulated, contrast-enhanced lesion was depicted at the region of the thalamus and basal ganglia on the right, surrounded by vasogenic oedema crossing the midline and extended at the left thalamus (fig. 1 a-d). Chest and Abdomen CT-scans were normal.



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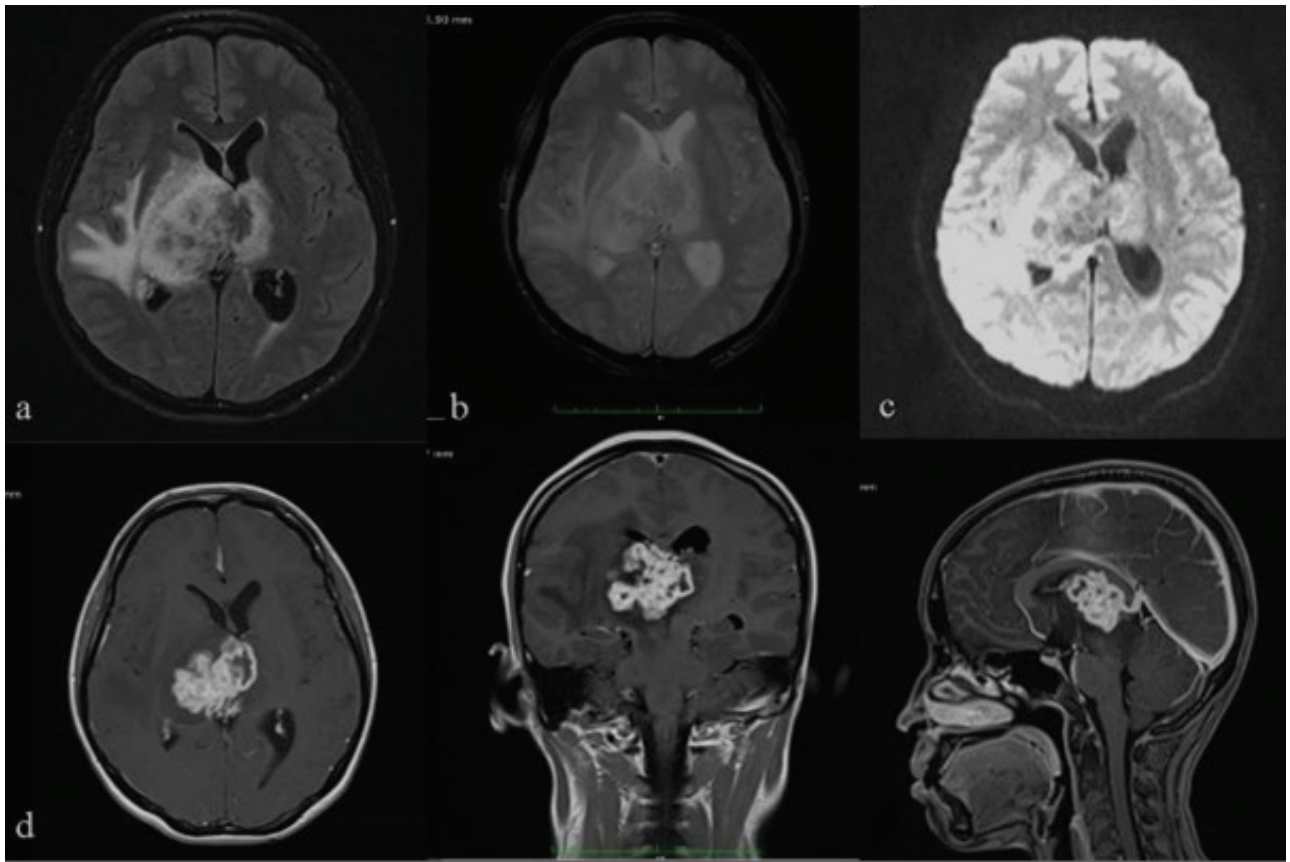


Figure 1

- a. MRI, axial FLAIR-weighted image
- b. MRI, axial T2-weighted image
- c. MRI, Diffusion-Weighted-Imaging (DWI)
- d. MRI, multiplane, post-gadolinium T1-weighted images

PART B

Caseating Cerebral Tuberculoma (Tuberculous Granuloma).

According to the WHO, tuberculosis ranked first among death causes by a single infectious agent and death causes in HIV-positive individuals in 2019 [1]. Although tuberculosis is a mainly respiratory condition, extrapulmonary disease is possible; CNS involvement occurs in about 1 out of 10 extrapulmonary disease cases, whose mortality and morbidity rates are remarkably high [2]. Tuberculous meningitis and granulomas are the primary CNS manifestations, and most frequently occur in immunocompromised (e.g., HIV-positive individuals, patients with malignancies, transplant recipients) and juvenile patients [3]. Granuloma development usually follows Mycobacterium tuberculosis dissemination through the bloodstream in patients with a history of known pulmonary tuberculosis [4].

Brain CT-scans usually depict round/ovoid lobulated nodules surrounded by vasogenic oedema. The lesions are initially hypodense or iso-dense to the grey matter, and progressively appear encapsulated, hypodense or iso-dense with peripheral ring enhancement and a central calcification focus (“target sign”) [5]. MRI is the modality of choice for the assessment of tuberculous granulomas; T1-weighted images show isointense lesions with a central hyperintense region of caseous necrosis, while T2-weighted images are typical of an iso-intense mass with a central hypointense locus of monocytic infiltration and gliosis [6] surrounded by vasogenic oedema [7]. On FLAIR images, the lesions mostly have a hypointense or isointense core with a hyperintense rim [8]. T1C+ images show a conglomerate ring-enhancing mass and on diffusion-weighted images no diffusion restriction is usually observed. However, in cases of liquefactive necrosis, high signal intensity is demonstrated cen-

trally; otherwise, the signal is typically low [6]. Histopathology reports are typical of firm, well-defined nodules with central caseous necrosis, enveloped in a granulomatous reaction comprised of Langhans giant-cells, epithelioid histiocytes, fibroblast and lymphocytes, frequently bacilli-free [3].

Differential diagnosis includes other ring-enhancing lesions like high grade glioma and metastases, CNS lymphoma in immunocompromised patients, neurosarcoidosis and other cerebral infections such as neurocysticercosis, cerebral toxoplasmosis, blastomycosis and cryptococcosis [7]. Central isointensity or hypointensity compared to grey matter seen centrally on T2 is helpful, as it is not seen in most other causes [9,10]. An underlying HIV infection should highlight toxoplasmosis and CNS-lymphoma as the most likely diagnoses. Multifocal manifestation in the absence of systemic neoplasia should guide towards an infectious etiology. DWI, especially when coupled with spectroscopy, may help distinguish cerebral abscess.

The absence of a relevant history of pulmonary tuberculosis or at least any signs and/or symptoms of pulmonary disease may protract an accurate diagnosis; In these cases, a brain biopsy may be unavoidable. In this context, it is additionally resonant to indicate that tuberculoma cells may possibly be dislodged and disseminated along the biopsy trajectory or dispersed adjacently to their original epicentre leading to ventriculitis development. **R**

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Not applicable

Conflict of interest

The authors declare no conflicts of interest

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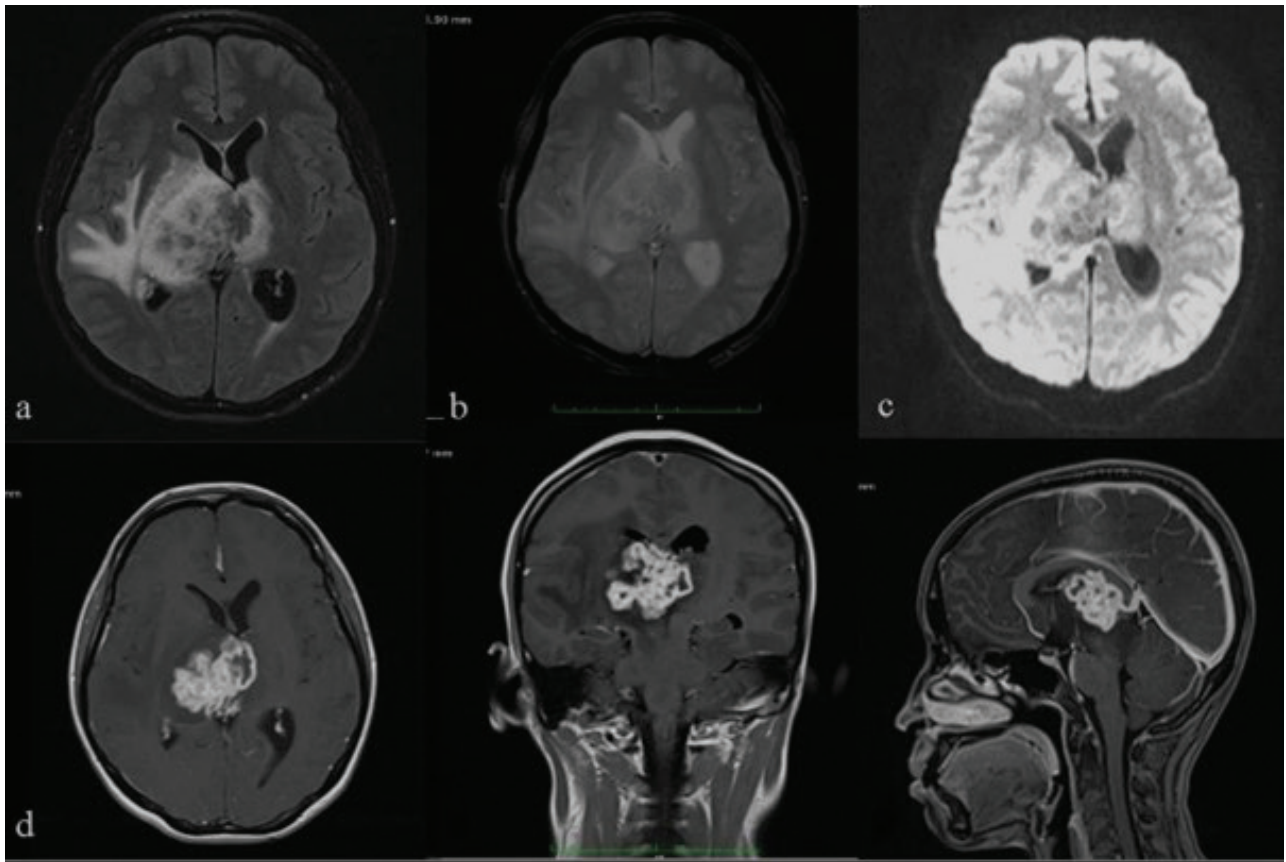


Figure 1: Brain MRI scan with intravenous paramagnetic contrast administration depicting a large, irregular, oval-shaped, ill-defined, lobulated space-occupying lesion at the region of right thalamus, internal capsule, pallidum. The mass is surrounded by moderate vasogenic oedema which extends to the external, extreme capsule, insula and the temporoparietal cortex. The lesion crosses the midline and extends to the left thalamus and third ventricle.

- a. Axial FLAIR sequence: The mass is moderately hyperintense with central hypointensities, surrounding vasogenic oedema, midline shift to the left and transependymal edema as a hyperintense band around the ventricular margins.
- b. Axial T2-weighted sequence: The lesion demonstrates heterogeneous signal intensity with several central spots of iso-hypointensity surrounded by a hyperintense rim.
- c. Axial DWI: There is no diffusion restriction.
- d. Axial, coronal and sagittal post-gadolinium T1-weighted sequences: The lesion shows intense, heterogeneous enhancement with areas of central necrosis, irregular outlines with peripheral contrast enhancing and moderate peri-lesional oedema.



KEY WORDS

tuberculosis; granuloma; tuberculoma; caseous ; brain

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