Majority patients with inflammatory pituitary lesions present with mass effect causing headaches, nausea, vomiting, chiasmal compression, and many with endocrine abnormalities and many with both the features, as in the present case. The mild elevation of prolactin level is attributable to pituitary stalk compression. Radiological findings mimic those of an adenoma and include a homogenously enhancing sellar mass that may show suprasellar extension. Pituitary stalk thickening in an appropriate clinical setting is considered a strong predictor of an inflammatory pathology. Sarcoidosis is an important differential diagnosis in this setting and could be suspected on basis of systemic involvement and appropriate laboratory investigations. It has been suggested that if suspected at presentation, inflammatory lesions of pituitary may be managed conservatively thus obviating the need for surgery. Trans-sphenoidal surgery is however both diagnostic and therapeutic and should be performed in cases with progressive compression or those which show radiological or clinical progression during conservative management. The present case highlights the importance of considering a tubercular etiology in the differential diagnosis of sellar lesions in this part of the world and the efficacy of ATT in their management.

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A solitary cryptococcal granuloma in an immunocompetent host

Sir,
Cryptococcosis or ‘cryptococcal granulomas’ are rare in
an immunocompetent host in the absence of cryptococcal meningitis. The morbidity and mortality due to cryptococcosis is high unless it is diagnosed and treated early.

A 22-year-old male presented with left focal seizures with secondary generalizations for three years. The patient had no clinical evidence of immunocompromise or meningitis. Human immunodeficiency virus (HIV) antibodies were negative and the CD4 T-cell lymphocyte counts were normal. A computed tomography (CT) scan of the brain revealed a right parietal lesion. It was isodense on plain scan and there was a peripheral enhancement on contrast administration (Figure 1). The lesion was surrounded by cerebral edema. The patient was placed on anticonvulsants and on the clinical and radiological suspicion of a tuberculoma the patient was empirically started on anti-tuberculous therapy. The patient was seizure free and had some relief from headaches.

After about one year of anti-tuberculous therapy the patient had recurrence of seizures and developed left hemiparesis. CT scan of the brain showed a slight increase in size and of the lesion and surrounding edema. The patient now had bilateral papilledema and left hemiparesis. A stereotactic biopsy of the lesion confirmed that the lesion was a cryptococcal granuloma. The mass was surgically excised. Histopathology showed characteristic features of a fungal granuloma with fibroblasts, giant cells with yeast forms of capsulated fungal elements and necrotic areas. The inflammatory response consisted mainly of lymphocytes, plasma cells, eosinophils, fibroblasts and multinucleated giant cells studded with cryptococci (Figure 2). The patient was treated with liposomal amphotericin and anticonvulsants. The patient was seizure free and the left hemiparesis improved. The follow-up is of 9 months.

Cryptococci have a strong neurotropic tendency and involve the meninges and brain.

Cryptococcosis is an infection caused by yeast like fungus, Cryptococcus neoformans. The infection occurs due to inhalation of fungus into the lungs. Hematogenous spread to the brain leads to clusters of cryptococci in the periventricular areas of cortical grey matter and basal ganglia. The leptomeninges become infiltrated, thickened and opaque. The Virchow-Robin spaces around penetrating vessels are distended with organisms; most of the patients have co-existent meningitis. Granulomatous lesions may be found in the cerebral or spinal parenchyma. In our patient a cryptococcal granuloma was identified in the parietal parenchyma in the absence of meningitis.

There are few, isolated reports of cryptococcal infection in immunocompetent patients in literature. Such previously reported cases include a cryptococcoma in the pituitary, pons, cryptococcal meningitis, midbrain, and cerebellum. Data suggest an association between cryptococcal variety and host immune status. The varieties of Cryptococcus neoformans serotypes are neoformans and gattii. These two varieties differ in their epidemiology and pathogenicity. Furthermore, symptoms, outcome, and response of cryptococcosis to antifungal therapy may vary. The two varieties also differ in immune-modulating effects. Sparse clinical data suggest the gattii variant to be more virulent and more recalcitrant to antifungal therapy. A better understanding of how cryptococcal variety influences the clinical course and response to the treatment of cryptococcosis is needed. Clinicians should be aware of the association, especially in patients with refractory disease. It may be useful to type the isolate to the variety level and administer prolonged antifungal therapy.

Early aggressive therapy has the best chances of cure. All the above patients including ours have been treated with Amphotericin B. Presently liposomal amphotericin ameliorates the adverse effects of plain amphotericin and ensures better brain penetration of the drug. Flucytosine or miconazole are alternative drugs. Surgical excision with concomitant medical therapy offers the best results.

Letter to Editor
Image based diagnosis of intracranial tuberculoma is fairly accurate in patients from endemic regions and it is not considered necessary to obtain a histological diagnosis. However, Selvapandian et al.\(^7\) have demonstrated the low positive predictive value for a diagnosis of intracranial tuberculoma on CT alone and they stress the need for confirming histological diagnosis. Surgical excision is feasible when such a lesion is easily accessible and not in an eloquent area.

In conclusion, the possibility of a fungal granuloma needs to be considered in an indolent lesion in an immunocompetent host. Histological diagnosis before initiating empirical therapy is recommended for all intracranial masses.

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**Letter to Editor**

Distress triggered cervical root compression pain by looped vertebral artery

Sir,

A 21-year-old girl presented with a 20-day history of right-sided cervicobraehialgia radiating in her hand and fingers. Whenever she was distressed, she experienced the pain associated with involuntary movements and the absence of nocturnal recrudescence and hypoesthesia of right thumb. History of familial neurofibromatosis and cervical spine trauma were absent. Both the physical and neurological examination was normal. However neither vascular bruit nor pain compliance has been detected. Cervical right oblique X-ray showed a minimal and T\(_2\) weighted axial MRI showed moderate foraminal widening at C\(_6-7\) intervertebral foramen [Figure 1]. Bright blood axial MR images revealed that vertebral artery loop compressed the C\(_7\) root in the intervertebral foramen [Figure 2]. The 3D TOF (time-of-flight) MR Angiography showed a vertebral artery tortuosity [Figure 3]. The patient refused an operation against further disturbances. Tricyclic anti-depressant Amitriptyline was given 10 mg for first 3 days, and continued 20 mg at night. All symptoms relieved and analgesia achieved within 2 days.

Progressive cervical radiculopathy due to loops of the vertebral artery is seen rare. The average age was 55 and our case is the youngest one as compared with in previous reports. However, the mechanism of how VA loop forms is unclear. A cervical trauma and spondylotic changes with the degree of VA tortuosity has been reported\(^{1,2,3,4}\) But our case has neither spondylosis nor cervical trauma.

It is difficult to distinguish the tortuosity of the vertebral artery from expected clinical symptoms with cervical myelopathy or radiculopathy in patients. It is emphasized that the frequent acropaesthesia and dysesthesia of fingers, and rare

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**Figure 1**: T\(_2\) weighted axial MR image shows moderate neural foraminal widening

**Figure 2**: Bright blood axial MR image reveals that neural foraminal widening was due to vertebral artery tortuosity, and vertebral artery loop formation is compressing to the C\(_7\) root in the intervertebral foramen