Suprasellar Tuberculoma Presenting as Sudden Onset Blindness in a Patient of Lupus

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ABSTRACT

Tuberculosis can be an opportunistic infection complicating the course of patients receiving prolonged immunosuppression. In these patients, the tuberculosis can involve the central nervous system and can cause diagnostic difficulty due to atypical features. Often, the diagnosis of central nervous system tuberculosis in resource limited settings is indirect, like imaging. But anti-tubercular drugs, given even on empirical basis can be life saving. A case of a young female systemic lupus erythematosus patient (on prolonged steroids) with intracranial tuberculoma is presented here. She presented with blindness and headache and her computed tomography scan showed a calcified mass in the suprasellar location. However, she responded well to anti-tubercular drugs. The differential diagnoses of such lesions are also discussed.

Key words: Lupus, Suprasellar calcification, Tuberculosis

INTRODUCTION

Systemic lupus erythematosus needs prolonged immunosuppressive therapy for control of symptoms. However, this immunosuppressed condition can predispose the patient to opportunistic infections like tuberculosis. In these patients, tuberculosis can affect different systems and can have uncommon clinical presentations. A case of a young female of lupus (on steroids) with suprasellar tuberculoma is described here. She presented with blindness and intense headache.

CASE REPORT

A 22-year-old female was admitted through emergency with sudden onset severe headache, vomiting, and dimness of vision (6/60) in both eyes. She was diagnosed with systemic lupus erythematosus one year ago by clinical and laboratory criteria (ACR) with nephropathy grade three. She had been given pulse cyclophosphamide 1 year earlier (6 doses, 21 days apart each). After that, she was put on oral steroids (prednisolone, 7.5 mg/day oral) and she continued the drug in the intervening period. At the time of admission she was still on oral steroids (7.5 mg/day). She had no other manifestations of lupus and her mental status was normal. She also had no prior visual problems.

After admission, the patient developed low grade fever and her dimness of vision progressed. By third day, she had no perception of light in either eye. Local examination of the eyes showed pupils to be dilated and non-reactive. Ophthalmoscopy showed mild papilledema bilaterally. No neck rigidity was elicited. There was bilateral sixth cranial nerve weakness; but no other neurological signs. Ophthalmologists opined that the pathology was not intra-ocular; neurologists were of the opinion that there was a vascular event in cranium. Immediate imaging was advised.

Routine laboratory tests showed mild anemia and thrombocytopenia. Her blood glucose was raised (fasting=144 mg/dL), probably due to long intake of steroids. Due to the presence of papilledema, we did a CT scan of brain [Figure 1] which showed a calcified mass in suprasellar region with perilesional edema (black arrow) with obstructive hydrocephalous. There was also mild cerebral atrophy. The Mantoux test of the patient was negative (she was on steroids), sputum did not show any acid-fast bacilli and chest X-Ray was also normal. She had contact with a sputum positive case...
of tuberculosis 3 months ago. Her hormonal profile was normal. We could not attempt a spinal tap due to hydrocephalous. The Magnetic resonance imaging scan could not be done due to cost factor. In view of the emergent nature of the illness, we started her on oral anti tubercular drugs with an increased dose of oral steroids. Her headache decreased, but vision improved only mildly. Subsequently, after 1 month, a brain biopsy was done and the lesion was found to be calcified granuloma with aggregates of epitheloid cells, calcifications, necrosis, blood vessel destructions, and few scattered caseations [Figure 2]. The lesion did not show any acid fast bacilli, but overall features were suggestive of tuberculosis. A CSF sample collected at time of brain biopsy showed increased cells (45/μL), increased protein (102 mg/dL). AFB stain of CSF was negative; a PCR from CSF was positive for Mycobacterium tuberculosis. Repeat CT scan [Figure 3] showed only a mild decrease in the size of the edema; calcified mass size was the same. She was put on anti-tubercular drugs for 1 year. At present her vision is finger counting at three feet.

**DISCUSSION**

Tuberculomas are an important cause of space occupying lesions in brain in developing countries like India. In cases like ours, with no extra cranial manifestations of tuberculosis, diagnosis can be difficult and only suggested by CT scans. For confirmation, a stereotactic brain biopsy can be attempted.[1] However, non-invasive methods like PET scan can be useful; if CSF study can be done, PCR or ELISA for Mycobacterium tuberculosis from the fluid can be attempted. Tuberculosis of central nervous system can have different forms like meningitis, abscess, tuberculoma, subdural collection or miliary form.[2] The abscess or tuberculoma may heal with calcification. These parenchymal lesions can also cause obstructive hydrocephalous.[3] Suprasellar tuberculoma can present with diabetes insipidus, visual loss, or hypothyroidism. In resource-limited settings, and when the patient is severely ill, often diagnosis is not possible and empirical treatment is needed. Steroids are usually indicated in these cases. Surgery may also be needed. Calcified mass in brain seen in CT scan can have many etiologies.[4] The following chart [Table 1] shows the different common etiologies of cerebral calcification and their differentiation with special reference to suprasellar lesions:

Suprasellar lesions can present with hypogonadism, features of raised intracranial pressure and hemianopia. But sudden visual loss is quite rare. Also, lesions with an infective cause should be differentiated from calcified tumors and aneurysms because drug treatment is helpful in tuberculoma, whereas in other cases prompt surgery is the only option. The idea of presenting this case is to draw attention on the catastrophic effects of long-term steroids. Although tuberculosis is a known complication of long-term immunosuppression, tuberculomas are rare and suprasellar tuberculoma causing visual impairment is indeed very rare. A case like ours was reported from India.
by Sharma et al in 2003. These patients should be followed up for development of epilepsy.

Also, intracranial calcification is a vexing problem in a resource limited settings. Proper differentiation of the lesions in CT scan is important to the treating physician. In a resource limited setting, anti-tubercular drug, even on empirical basis can be life saving and should be used without delay. Newer diagnostic methods like PET scan can also help in distinguishing these lesions (tuberculosis has lower FDG uptake than lymphoma). MR spectroscopy can also help in this regard; tuberculoma shows prominent lipid peak at 1.3 ppm.

The lesion of tuberculosis will show significant change in subsequent imaging following therapy although the radiological resolution may not match with the clinical improvement, especially in central nervous system lesions. Specially calcified lesions often indicate irreversible damage.

Table 1: Table Showing the characteristics of different lesions with cerebral calcification

| Lesion                      | Site                                      | Symptoms                                      | CT appearance                                      |
|-----------------------------|-------------------------------------------|-----------------------------------------------|---------------------------------------------------|
| Infectious                  | At site of tuberculosis or abscess (cortex, basal cisterns, meninges) | Fever, headache, diplopia                      | Contrast enhancing lesion with marked edema; may mimic tumor; calcification in late stage |
| Viral (rare)                | CMV-periventricular                        | Signs of respective viral illness             | Periventricular enhancement; calcification         |
|                            | HIV-basal ganglia                          |                                               | Cortical atrophy; basal ganglia calcification      |
|                            | HSV-gyriiformii in cortex                  |                                               | thin-walled cavitating lesions with ring enhancement; asymmetric; target sign |
| Others                     | Toxoplasma: basal ganglia                  | Neurodeficit                                  | Hypo density in the temporal lobes either unilaterally or bilaterally, with or without frontal lobe involvement |
|                            | Neurocysticercosis: near cortex, brainstem | May be asymptomatic                            | Hypo density in the temporal lobes either unilaterally or bilaterally, with or without frontal lobe involvement |
|                            | Anywhere: Craniopharyngioma, germinoma, dermoid tumors, pituitary adenoma: suprasellar calcification | Seizure, headache, vomiting, focal neurodeficit | Usually well circumscribed lesion; calcification++; bone erosion; heterogeneous appearance |
| Neoplastic                  |                                          |                                               |                                                    |
| Oligodendroglioma, astrocytoma, craniopharyngioma, etc. | Cortical atrophy                            | Multiple; junction of gray and white matter; edema++; contrast study important to detect meningeval spread |
| Metastasis                  |                                          |                                               |                                                    |
| Congenital                  | e.g. Tuberous sclerosis, grey-white matter junction | Seizure, mental retardation                    | Calcified cortical tubers and calcified subependymal nodules (periventricular); cystic lesions |
| Associated with tumors      | Teratoma, astrocytoma, primitive NET       | Growth failure, congenital anomalies          | Lobulated mass; high fat content; ill localized; usually near ventricle; speckled calcification |
| Endocrine                   |                                          |                                               |                                                    |
| Hypoparathyroidism          | Basal ganglia                              | Hypocalcemia, seizures, cardiac arrhythmia    | Basal ganglia calcification; no edema; sometimes calcification of cortex |
| Hypothyroidism              | Rarely seen                                | Mainly associated with Hypoparathyroidism     | Nothing distinctive; like previous                 |
| Calcification of normal     | Pineal gland, Choroids plexus, falx cerebi, etc. | Usually asymptomatic                           | Calcification spots at the sites mentioned         |
| Vascular                    |                                             |                                               |                                                    |
| AVM, aneurysm, hemangiomas  | Usually asymmetrical, near cortex Internal carotid calcified aneurysm: suprasellar calcification | Seizure, sub arachnoid hemorrhage             | CT angiography needed; subarachnoid hemorrhage may be present; serpiginous vascular enhancement-typical of an AVM. Occasionally, CT scans can demonstrate edemas, mass effect, or ischemic changes that may be associated with AVMS |
| Misc                         | Extensive, associated with infarcts         | Symptoms of neuro lupus; or asymptomatic      | Vascilitis lesions; calcification (irregular); Edema; periventricular white matter changes; globus pallidus hypointensity |
|                             | Basal ganglia                              | Hypoxic signs                                 |                                                    |
|                             |                                             | May be asymptomatic                            | Symmetrical basal ganglia and/or thalamus calcification |
|                             | Fahn's disease or familial basal ganglia calcification | Unknown for radiation.                       | Radiation: periventricular decrease in attenuation of CT; necrosis |
|                             | Celiac disease: occipital calcifications | Celiac disease: seizures (occipital, in children) |                                                    |
|                             | Sarcoïd: suprasellar | Sarcoïd: suprasellar |                                                    |

CMV: Cytomegalovirus; HIV: Human immunodeficiency virus; HSV: Herpes simplex virus; CT: Computed tomography; NET: Neuroectodermal tumor; AVM: Arterio venous malformation
CONCLUSION

This case shows the importance of brain imaging in suspected infective disorders and the need to interpret the images quickly for maximum benefit of the patients. Especially in the background of the immunosuppressed state, any infection (like tuberculosis) can present with a catastrophe like blindness and timely therapy can prevent subsequent morbidity to a large extent. At times, empirical therapy can also be used, provided clinical suspicion is strong and diagnostic tests are unavailable or impractical. Also, clinicians need to be well versed with the differential diagnoses of intra cranial calcification, which, though a common finding, can be a diagnostic dilemma at times.

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