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A Rare Case Report of Progressive Supranuclear Palsy Secondary to Tuberculous Vasculitis Following Tuberculous Meningitis

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ABSTRACT

Most of the strokes in tuberculous meningitis are usually multiple, bilateral, and located in the basal ganglia, especially the 'tubercular zone', which comprises of the caudate, anterior thalamus, anterior limb and genu of the internal capsule. Mechanism of stroke in tuberculous meningitis is usually attributed to vasculitis. Tuberculous meningitis (TBM) is fatal in approximately one-third of patients, and the sequelae of the disease in those who survive is challenging.

KEYWORDS: TBM- Tuberculous meningitis TB-Tuberculosis

INTRODUCTION

Tuberculous meningitis is an extrapulmonary form of tuberculosis characterised by subacute or chronic inflammation of meninges. Tuberculous meningitis constitutes 10% of all cases and is responsible for 40 % deaths in developing countries. Complication of tuberculous meningitis includes cerebral stroke, hydrocephalus, meningoencephalitis and tuberculoma formation.

In a case of TB vasculopathy, vasculitis, venous thrombosis and aneurysm may be the underlying events leading to a stroke. Tubercular meningitis causes necrotising arteritis of cerebral vessels.

Stroke in TBM is seen in the tubercular zone which encompasses internal capsule, thalamus and caudate nucleus. I report an unusual case of 42year old female patient with ischaemic infarction of basal ganglia presenting as a type of atypical Parkinson's i.e., progressive supranuclear palsy secondary to tuberculous vasculitis following tuberculous meningitis.

CASE REPORT

A 42 year old female patient with no known comorbidities came with complaints of low grade intermittent fever associated with headache and vomiting in March 2021.In May 2021 she developed weakness of left upper limb and lower limb, CT brain done outside showed infarct in right basal ganglia region and lumbar puncture showed increased protein ,lymphocytic pleocytosis following which ATT was started in outside hospital .In September 2021 patient developed GTCS following which repeat CSF analysis showed 120 cells (90%lymphocytes) ,protein 450mg /dl with CT brain showing infarct in bilateral basal ganglia with nodular enhancement in leptomeninges ,enhancement of bilateral middle cerebral artery and anterior cerebral artery.

Over a period of one to two months patient had progressive difficulty from getting up from chair, later she noticed slowness of activity in daily activities which later progressed to requiring assistance to do her daily activities with history of recurrent falls

over a period of 6 months. Patient later developed posturing wherein bilateral upper limb elbow flexed and left lower limb was everted since 6 months. Currently she cannot walk without any assistance. History of increased drowsiness over the past 2 months. No history of cognitive or behavioural symptoms. History of picking the bed clothes for past 15 days. History of slowing of speech, emotional lability noticed since 15 days. No history of vision disturbances or difficulty in swallowing.

On examination: patient is conscious, oriented to place, person but not to time

Emotional lability present with increased crying spells present. Facial dystonia with right side torticollis present, associated with posturing -left hand flexed and adducted, left lower limb everted. Right hand dystonic posture of elbow flexed; finger clenched. No cranial nerve involvement. Extraocular movement normal. Bilateral plantar - withdrawal

Axial rigidity present, bilateral upper limb grade 4 rigidity, bradykinesia of all 4 limbs, gait: stooped posture, require support of one person for walking, bilateral plantar - withdrawal

Investigations: Complete blood count-showed neutrophilia with lymphocytopenia, random blood sugar-89mg/dl, renal function test -normal, liver function test showing mild transaminitis, HIV-Negative,HBsAg-negative, anti-HCV-negative, VDRL-negative, serum electrolytes-showed hyponatremia of 118mEq/l, CSF analysis-CSF protein -68.40mg/dl,CSF chloride-116mmol/l,CSF glucose-57mg/dl,CSF lactate-19.2mg/dl, CSF -CBNAAT-positive, CSF India ink is negative. Fasting lipid profile- Normal, Bilateral carotid artery doppler-no stenosis

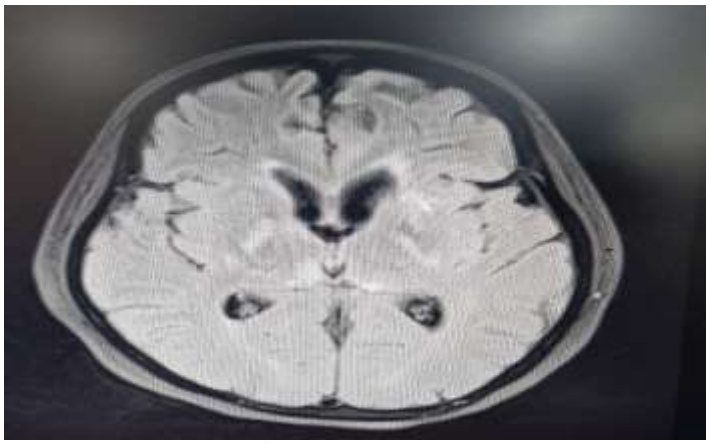

Figure -1: MRI brain-showing bilateral basal ganglia infarct and right thalamus, ependymal thickening noted in both lateral ventricles, lateral ventricles mildly dilated 2021

Figure 2: MRI brain (2021)-showing humming bird sign -flattening or concave outline to the superior aspect of midbrain

Chest x ray-showed no signs of tuberculosis of lung, 2DECHO- EF of 58%, no significant valvular or structural heart disease. Other work up of paraneoplastic neuronal antibody showed negative for all the antibodies.

Patient was treated with diagnosis of Atypical Parkinson's [Progressive supranuclear palsy] secondary to tubercular meningitis with vasculitis with tuberculous meningitis. Patient completed 9months of antitubercular treatment of:

Tab rifampicin 600mg OD, Tab INH-300MG OD, Tab ethambutol 1000mg OD, Tab pyrazinamide 750mg BD, Tab Prednisolone 30mg OD, Tab levodopa +carbidopa(100+10)mg TID, Tab Baclofen 5MG OD and Tab Aspirin 75mg 0-1-0

Following the above treatment patient can stand and walk with one person support.

DISCUSSION

Tuberculous meningitis presenting as atypical parkinsonism disease is one of the rarest of rare clinical case scenario. Tuberculous meningitis, also known as TB meningitis or tubercular meningitis, is a specific type of bacterial meningitis caused by the Mycobacterium tuberculosis infection of the meninges.

Stroke in tuberculous meningitis may develop insidiously or present as asymptomatic or silent stroke. Most vulnerable site being the basal ganglia also described as "tubercular zone"-comprising of head of caudate nucleus, anteromedial thalami and anterior limb and genu of internal capsule.[1] Dense fibrinocellular exudates may wrap middle cerebral trunks and their penetrating branches which may contribute to vasculitis and cause stroke in tubercular zone.[2]

It has been accepted that stroke in tuberculous meningitis patients was mainly caused by vasculitis secondary to the meningeal inflammation, which can be classified into three patterns: 1) infiltrative, 2) proliferative and 3) necrotizing vascular lesions.[3] Duration of the tuberculous meningitis may determine the relative frequency of infiltrative, proliferative and necrotizing changes in the cerebral vessels. Among the involved vascular territories, the most commonly affected by mycobacterium tuberculosis are those from the anterior circulation specifically the lateral striate artery, medial striate artery and middle cerebral artery.

Progressive supranuclear palsy is a type of atypical parkinsonism where in patient is characterised by slow ocular saccades, eyelid apraxia and restricted vertical eye movement. Patients frequently experience hyperextension of neck with early gait disturbance and falls. In later stages speech and swallowing is affected.

Our patient with the above-mentioned clinical features of progressive supranuclear palsy a variant of atypical Parkinson's disease was developed due to the infarction in bilateral basal ganglia region. Results from many studies have demonstrated that stroke predict poor clinical outcome, which may be disastrous consequence for young tuberculous meningitis patients.[4]

CONCLUSION

The common clinical presentations of TBM with stroke were fever, headache, neck stiffness, altered sensorium, focal weakness or hemiplegia, cranial nerve palsy and seizure. Nearly all reported cases were cerebral infarctions; lateral striate, middle cerebral and medial striate arteries were commonly affected while basal ganglia, cortex or lobar and internal capsule were the frequently injured areas. Tuberculous meningitis needs to be diagnosed at an early stage of the disease to avoid serious and life-threatening complications associated with it. Earlier the diagnosis better is the outcome of the patient as approximately 5 out of 10 patients would be estimated to have poor outcomes while 2 out of 10 may expire.

Declaration by Authors

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Conflict of Interest: The authors declare no conflict of interest.

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