Levodopa Non-Responsive Parkinsonism in Tuberculous Cerebral Arteritis: A Rare Occurrence

Sir,

Tuberculous meningitis (TBM) is the most severe form of tuberculosis (TB).[1] Tuberculous meningitis (TBM) has been associated with movement disorders. Tremor, dystonia, chorea, ballism, and myoclonus have been described in TBM.[2,3] The occurrence of parkinsonism in TBM has not been reported. Hereby we report a 42-year-old lady who was diagnosed with TBM based on brain imaging and cerebrospinal fluid (CSF) analysis. She developed symmetrical parkinsonism after improvement in the level of consciousness. Brain imaging showed bilateral basal ganglia infarcts.

A 42-year-old lady was admitted to a local hospital with a history of low-grade fever of 3 weeks' duration and an altered level of consciousness of 2 days' duration. She was evaluated with computed tomography of brain and CSF analysis, which showed raised protein and lymphocytic pleocytosis. She was diagnosed with TBM and was started on four-drug (isoniazid, rifampicin, pyrazinamide, and ethambutol) anti-tubercular treatment (ATT) with oral prednisolone. She had improvement in her level of consciousness over 1 week. However, she developed slowness in walking, hypophonic speech, and slow limb movements. She was referred to us for further management. There was no history of seizures. She did not have vascular risk factors. On examination, she was conscious and oriented. Her mini-mental status examination was 28. Speech was hypophonic, monotonous. Extraocular movements, saccades, and pursuits were normal. She had bradyphrenia, symmetrical appendicular bradykinesia, and rigidity. There were no rest or action tremors. She had grade 2 postural instability. There were no pyramidal signs. Her gait was narrow with bilaterally reduced arm swing and unsteady [Videos 1 and 2]. Complete blood counts, renal, hepatic, and thyroid function tests were normal. Brain magnetic resonance imaging (MRI) showed infarcts in bilateral basal ganglia, medial thalami with exudates in bilateral Sylvian fissures, and dilation of the third ventricle [Figure 1]. Brain MRI was suggestive of TBM with cerebral arteritis. Repeat CSF showed raised protein and normal glucose with 36 cells (all lymphocytes). CSF cultures were negative. Ziehl–Neelsen stain was negative. Serological test for human immunodeficiency virus was non-reactive. She was given levodopa challenge (200 mg) with no improvement. Her pre-levodopa Unified Parkinson's disease rating scale-part III (UPDRS-III) score was 42 and with levodopa was 37. She was discharged on ATT and oral steroids with levodopa (600 mg/day). At the 3-month follow-up, there was no significant improvement requiring support to get up with persistent bradykinesia and hypophonic speech.

TBM represents approximately 1% of all cases of TB. It is the most severe form of TB with a mortality rate as high as 60%.[1] Stroke has been reported in 13%–57% of patients with TBM. Cerebral infarction in TBM is due to vasculitis.[4] Basal ganglionic infarctions are the most common area of infarction in tuberculous cerebral arteritis due to the involvement of medial striate, thalamotuberal, and thalamoperforate arteries.[5] The common manifestation of TBM-related stroke is focal weakness such as monoplegia, hemiplegia, or quadriplegia. The other manifestations include sensory impairment, ataxia, seizures, movement disorders, cranial nerve palsy, and aphasia, depending on the location of infarctions.[5]

Movement disorders usually occur due to basal ganglia and its connection with the thalamus, cerebral cortex, and

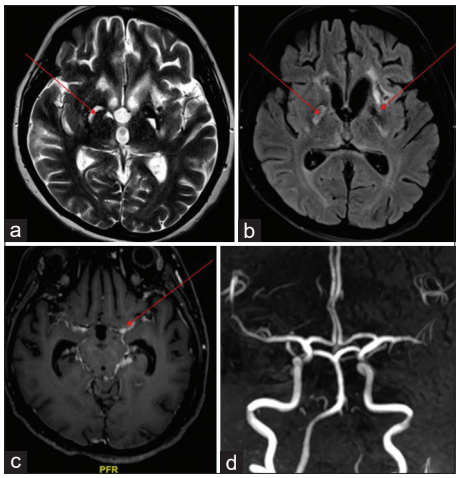

Figure 1: Brain MRI axial T2 image (a) showing old infarcts in bilateral basal ganglia (red arrow); axial fluid-attenuated inversion recovery image (b) showing old infarcts in bilateral basal ganglia (red arrows); axial T1 gadolinium-enhanced image (c) showing basal exudates (red arrow); MR angiogram (d) showing no occlusion or stenosis

cerebellum dysfunction. The movement disorders in TBM have been reported to be due to tuberculoma, ischemic infarction, arachnoiditis, and edema. Most of the literature on movement disorders in TBM is limited to anecdotal case reports. In a large study of 30 patients of TBM with abnormal movements by Alarcón *et al*.,[3] there was a higher frequency of deep vascular lesions involving the internal capsule, basal ganglia, diencephalon, and mesencephalon than those without abnormal movements. They found tremor as the most common movement disorder in their cohort. Twenty patients had postural tremor, 10 had kinetic tremor, and three had tremors at rest; seven patients had chorea, and three patients had dystonia. Hydrocephalous and infarcts in basal ganglia, mesencephalon, and internal capsule were the cause for the tremors in their cohort. There is no data regarding the presence of rigidity and bradykinesia in patients who had rest tremors.[3] Pandey *et al*.[6] from India reported a 35-year-old woman with slowness in daily activities and left-upper-limb tremor. Brain MRI showed multiple conglomerated tuberculomas in the right temporoparietal region extending up to the midbrain and right cerebellar peduncle.

The occurrence of parkinsonism in tuberculous cerebral arteritis has not been reported. Our patient was diagnosed with TBM based on the clinical features, brain MRI imaging findings, and CSF lymphocytic pleocytosis. She developed features of parkinsonism in the form of bradykinesia and rigidity, and had postural instability. There were no rest or action tremors. The parkinsonism did not respond to an adequate tolerated dose of levodopa.

In conclusion, tuberculous cerebral arteritis is known to cause infarcts in basal ganglia. The most common movement disorder reported in TBM is isolated tremors followed by dystonia and chorea in the pediatric population. The occurrence of parkinsonism has not been reported in TBM despite the involvement of basal ganglia due to arthritis.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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