

Infective Parkinsonism Secondary to Thalamic Tuberculoma.

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Abstract : This is a report of a 43-year-old woman who presented with akinetic rigid type of parkinsonism with gait disturbance, and mask-like facies, with thalamic tuberculoma. She had symmetric symptoms with postural instability without rest tremor. This case highlights that infectious aetiologies should be considered in cases presenting with parkinsonism, as it is treatable and completely reversible in tropical countries.

Key words: Parkinsonism, tuberculoma

INTRODUCTION

Parkinsonism has been reported as a consequence of various central nervous system infections. The common infective agents include Japanese encephalitis, encephalitis lethargica, influenza A encephalitis, neurocysticercosis; arbovirus, enterovirus, varicella-zoster virus, measles virus/ subacute sclerosing panencephalitis, Mycobacterium tuberculosis meningitis or tuberculomas, Mycoplasma pneumoniae, salmonella typhi, Plasmodium falciparum, prion disease, and African trypanosomiasis and HIV¹⁻⁴. Here we are reporting an interesting case of parkinsonism due to intracranial tuberculoma involving thalamic region.

CASE REPORT

A 42-year-old housewife presented with insidious onset and progressive history of slowness in performing household work like cooking food and cleaning house etc. since 2 months. She also noticed difficulties in all basic activities of daily living for the last 20 days. General physical and systemic examinations were unremarkable. Neurological examination revealed normal higher mental functions and cranial nerves. Speech was low volume and hypokinetic. Ocular movements were normal. The facial expression was mask-like. There was no facial asymmetry or drooping of saliva. Motor system examination revealed generalized rigidity, severe bradykinesia in axial and appendicular muscles, normal motor power and symmetrical brisk deep tendon reflexes. Plantar responses were flexor bilaterally. There were no postural, action or resting tremors. There was gross postural instability on pull test. Routine hematological and biochemical analysis were normal. Chest skiagram and ECG were normal. HIV serology was negative. Magnetic resonance imaging (T1, FLAIR, T2 weighted, T1 contrast, MRS) of brain demonstrated conglomerate peripheral enhancing lesion in thalamic region with mass effect on third ventricle, hypointense on T2W and lipid and lactate peak on MRS consistent with tuberculoma (fig. 1 to 4). She was treated with antitubercular quadrithrapy with steroids. The parkinsonian symptoms gradually improved and at the end of four weeks she was totally asymptomatic. She is on regular follow up. At present she is able to perform all her daily activities and look after her family independently.

DISCUSSION

Intracranial tuberculomas are one of the most common extrapulmonary tuberculomas. Mycobacterium tuberculosis which is causative agent for tuberculosis involves central nervous system in two ways, first is tubercular meningitis which is more common than second form i.e tuberculomas. In developed nations CNS tuberculomas is rare occurrence but in developing nations CNS tuberculomas still constitute one-third of intracranial masses⁵⁻⁷. Approximately 1% of tuberculosis patients develop CNS tuberculoma⁸.

The manifestations of CNS tuberculomas depends upon location, size of lesion, presence or absence of surrounding edema. Intracranial hypertension and ophthalmologic symptoms are the most common

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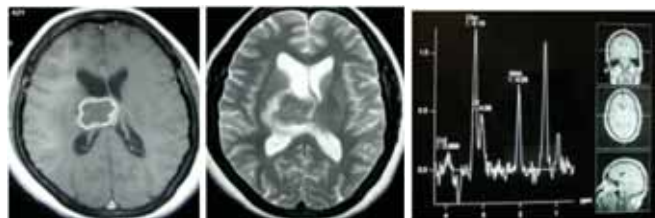


Figure 1 & 2: T1 contrast and T2 axial cuts. Figure 3: showing MRS with showing conglomerate enhancing lesion in lipid, lactate peak right thalamic region.



Figure 4: showing typical parkinsonian facies.

manifestation⁸. In our case patient presented with features suggestive of parkinsonism, without any evidence of intracranial hypertension or ophthalmological involvement. As per our knowledge CNS tuberculomas presenting with parkinsonian feature has not been reported earlier in literature. The mechanism behind the parkinsonian feature may be due to direct invasion and mass effect over the basal ganglia region as in our case. The parkinsonian features due to similar mass effect has been described in case of brain tumors involving basal ganglia region⁹.

CONCLUSION

Our case report highlights that infectious aetiologies should be considered in cases presenting with parkinsonism, as it is treatable and completely reversible in tropical countries.

REFERENCES

1. Verma R, Anand KS, Sharma BB, Garg J. Neurocysticercosis presenting as Parkinsonism. *Neurology India* 2013; 61(6)
2. Pandey S, Kumar P, Anand KS. Post Japanese encephalitis parkinsonism with predominant substantia nigra involvement. *Asian J Cog Neurol* 2014; 2(1): 63-64.
3. Wadhwa A, Anand KS. Parkinsonism associated with subacute sclerosing panencephalitis. *Asian J Cog Neurol* 2014; 2(1): 68.
4. Fernandez, Friedman. Acute Parkinsonism. <http://www.springer.com/978-1-58829-305-4>
5. Dolin, P.J., M.C. Ravighione, A. Koehi. Global tuberculosis incidence and mortality during 1990-2000. *Bull. World Health Organ* 1994; 72: 213-220.
6. Klein, N.C., B. Damsker, S.Z. Hirschman., Mycobacterial meningitis: Retrospective analysis from 1970-1983. *Am. J. Med* 1985; 79: 29-34
7. Hejazi, N, W. Hassler., Multiple intracranial tuberculomas with atypical response to tuberculostatic chemotherapy: Literature review and a case report. *Acta Neurochir (Wien)* 1997; 139: 194-202.
8. Abuhamed M, Bo X, Yan C. Central Nervous System Tuberculomas: A Review Article. *American Journal of Infectious Diseases* 2008; 4 (2): 168-173.
9. Kulali A, Tuotekin M, Utkur Y. Ipsilateral hemi-Parkinsonism secondary to an astrocytoma. *Journal of Neurology, Neurosurgery, and Psychiatry* 1991; 54: 653-660