

Movement Disorders in India

The ancient texts of the Indian system of medicine 'Ayurveda', describes Parkinsonism and tremors as early as 5000-3000 BC.¹

Parkinsonism

The Ayurvedic physician, Charaka, was possibly the first to describe Parkinson's disease (PD) in his treatise "Charaka Samhitha" where he called it 'Kampavata', literally meaning 'tremors of neurological origin'. Interestingly, the treatment recommended in Ayurveda for PD is the seeds of *Mucuna Pruriens* whose extract contains levodopa. All this was known much before James Parkinson described this disease in modern times.

The prevalence of PD in Indians is lower than people of European origin. Parsis who immigrated to India centuries ago from Persia have a much higher prevalence of PD than the Indians.² A recent epidemiological study from Kolkata showed a low prevalence (Crude Prevalence: 45.8 and Age Adjusted Prevalence: 71.6 per 100,000) of Parkinsonism.³ On the contrary, the prevalence of PD in Anglo-Indians, a mixed race from marriage between a European man and an Indian woman is about 40% lower than the prevalence in the Caucasian population.⁴

Normal Indians have 40% fewer melanised nigral neurons than British Caucasians and these neurons are not lost with increasing age.⁵ The number of melanised nigral neurons between the British and Nigerians did not differ significantly suggesting that factors other than neuronal numbers contribute to differential susceptibility to PD between non-white and white races.⁶

Genetic studies in familial PD have shown that alpha-synuclein,⁷ Parkin,⁸⁻¹¹ LRRK2,¹⁰ PINK and DJ-1 mutations occur in a small number of Indian patients. SCA2 commonly causes ataxia in the Indians but Ragothaman et al have described a family with homozygous SCA2 mutation, who presented as levodopa responsive parkinsonism. However, unlike the ataxic phenotype that have slow saccades, they had normal eye movements. These patients develop motor fluctuations and dyskinesia, hence early in the illness they can be mistaken as PD. Furthermore these SCA2 patients with parkinsonism develop slow saccades, psychosis and dementia about six years after disease onset.¹²

Manganese-induced Parkinsonism is common in the manganese mine workers in India. These patients typically walk on their toes and fall frequently as they have severely impaired postural reflexes. They typically have a pathological laughter and deep pigmentation of gums, palate and uvula, and their parkinsonism does not improve with levodopa.^{13,14} Bhatt et al reported parkinsonism on exposure to household pesticides.¹⁵ In Japanese encephalitis-endemic regions, parkinsonism occurs in ~80% of patients during the acute illness but a long-term follow up showed that it persists in only a few adults, whilst in children they develop dystonia. JE antibodies and antigen were absent in the CSF of patients who developed these movement disorders, showing that the virus

does not remain in the nervous system. These patients do not improve with levodopa.¹⁶

Surgery was the treatment of choice for PD before levodopa was introduced. In 1963, Prof Varma attempted to lesion the ventrolateral thalamic nucleus by injecting alcohol via needle inserted through the foramen ovale to control tremors.^{17,19} Using this outpatient procedure he stopped advancing the needle when the tremors stopped, thinking he had reached the planned target, the VL thalamic nucleus. However, when these lesions were mapped using the Schaltenbrand atlas, they were located in the subthalamic nucleus. This anatomical localisation was confirmed at autopsy, using a stereotaxic atlas and, recently, using MRI.¹⁸

In the modern era, surgical treatment for advanced PD has resurfaced as a treatment and Doshi and Bhatt reported depression following deep brain stimulation of the subthalamic nucleus possibly due to spread of the stimulation to the limbic region of this structure. Kishore et al have also observed motor improvement following stereotactic lesions to treat levodopa-induced dyskinesia but this is dependent on the volume of lesions in the ventral globus pallidus and suggested different anatomical substrates might be involved in controlling 'off signs' and dyskinesia.

Behari et al evaluated the quality of life (QoL) in PD patients and observed that female gender, depression, reduced independence, higher levodopa dose (>400 mg/day) and UPDRS scores were associated with worse QoL.¹⁹

Medicines and surgical interventions have improved the quality of life of PD patients but are still expensive and unaffordable to many living in developing countries. Managing PD in Indians where only 3% have health insurance is a challenge. Indian patients spend nearly 40% of their average gross income to buy medicines and despite the costs of treating PD in India being lower than in developed nations, optimal treatment is still out of reach for many Indian patients.²⁰

Chorea

Rheumatic fever is still common in India and chorea still occurs frequently. The prevalence of Huntington's disease (HD) in Indians is not known, it occurs in 1.75 per 100,000 population of Indians living in the United Kingdom.²¹ Salem et al, found that the distribution of alleles (D2642 and D4S127) in Indian HD patients are similar to West European populations and suggest that this admixture possibly occurred when British troops were located in South India during various wars.²²

Wali et al reported a large South Indian family with autosomal dominant paroxysmal kinesogenic choreathetosis (PKC) and onset in early childhood. Choreathetosis was precipitated by activities such as hyperventilation (46%), swimming (23%), exposure to cold (30.8%) and prolonged exercise (23%). This family has a second PKC mutation localised to the long-arm of chromosome 16q13-q22.1.²³



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Patients in India present with a wide variety of movement disorders and these pose challenges, especially in terms of treatment, given the therapeutic limitations due to a near total lack of health insurance

