Commentary

Parkinson's disease (PD) is a common idiopathic neurodegenerative disease of the old age. Young-onset PD (YOPD) is onset between ages 21 and 40 years.[1,2] The mainstay of PD is the pharmacologic replacement of dopamine in the form of levodopa, which is converted to dopamine in the brain. Other effective agents include dopamine agonists, inhibitors of catechol-O-methyltransferase (COMT) and monoamine oxidase-B (MAO-B) inhibitors, anticholinergics (e.g., trihexyphenidyl), and amantadine.[1] Treatment of advanced or disabling symptoms includes neurosurgical procedures such as deep brain stimulation of the subthalamic nucleus or globus pallidus. Occupational, physical, and speech therapy are often helpful.[1]

Previous studies suggest that YOPD has a slower disease progression and a greater incidence and earlier appearance of levodopa-induced dyskinesias and motor fluctuations.[1] Therapeutic strategies should take into consideration that YOPD patients face many years of gradual progression of disease and disability, a greater probability for developing various adverse effects of treatment, and worsening of quality of life. As an individually tailored treatment should be the primary goal, it should be borne in mind that the needs and expectations of YOPD patients are different from those of their older counterparts.[3]

In young parkinsonian patients with mild motor dysfunction, use of levodopa may be delayed or the dosage minimized.[2,3] However, because of levodopa's superior efficacy, when a rapid and sustained symptomatic improvement is required because of significant motor disability, levodopa may be used as the first-line agent regardless of age.[2] Although levodopa remains the most effective symptomatic drug for PD, its use is limited by the emergence of motor fluctuations and dyskinesias, particularly in young-onset patients.[4]

In comparison with patients with late-onset disease, most patients with YOPD progress more slowly in terms of motor features and have a longer disease course with preservation of cognitive function, but typically develop motor fluctuations and dyskinesias earlier and have lot of nonmotor features. Patients with YOPD have poorer social adjustment, higher rates of depression, and lower quality of life as compared to the older patients. Management of YOPD must, therefore, aim to maintain occupational, social, and daily functioning while delaying or ameliorating motor complications of treatment, providing psychological support, and where possible, preventing psychiatric complications including depression.[5]

For patients below 65 years old or above 65 years old but with preserved mental function and with no severe comorbidity, initial monotherapy with a dopamine agonist is advisable. This approach appears to delay the appearance and reduce the amount of late motor complications with subsequent levodopa treatment. All dopamine agonists have similar efficacy, which is less than that of levodopa. It is important to consider the adverse effect profile when a choice for initial or adjunctive therapy is made. When levodopa therapy is started as an adjunct in younger patients or as initial monotherapy in older patients, sustained-release levodopa preparations are preferred. They have a longer half-life and possibly stimulate the dopamine receptors more continuously. Anticholinergic drugs are appropriate for younger patients with tremor-predominant PD. Amantadine is mainly used for dyskinesias control. COMT inhibitors and neurosurgery are not treatments of choice for early PD but can be very effective for more advanced disease.[5] Because of this, levodopa use is commonly withheld until the patient experiences functional disability. Other medications are available for the treatment of early PD and can be initiated at or near the time of diagnosis. MAO-B inhibitors provide mild symptomatic benefit, delay the need for levodopa, are very well-tolerated, and may provide long-term disease-modifying effects. Dopamine agonists provide moderate symptomatic benefit, delay the need for levodopa, and cause fewer motor complications than levodopa. When compared with levodopa, however, dopamine agonists cause more somnolence and sudden-onset sleep as well as impulse control disorders.

The treatment of early PD depends in part on the individual patient's anticipated risk of side effects and the degree of motor improvement required. Physicians should also consider the early use of MAO-B inhibitors in light of their very good tolerability and the recent evidence suggesting long-term disease-modifying effects.[6,7]

Vikas Dhikav

Department of Neurology, Memory Clinic, Dr. Postgraduate Institute of Medical Education and Research, New Delhi, India

Address for correspondence:
Dr. Vikas Dhikav,
Department of Neurology, Memory Clinic,
Dr. Postgraduate Institute of Medical Education and Research,
New Delhi - 110 001, India.
E-mail: vikasdhikav@hotmail.com

References

1. Rana AQ, Saleh M, SaadYM, Mansoor W, Syed H, IqbalZ. DOPA Sparing strategy in the treatment of young onset Parkinson's disease. J Neurosci Rural Pract 2016;7:67-9.
2. Kostic VS. Treatment of young-onset Parkinson's disease: Role of dopamine receptor agonists. Parkinsonism Relat Disord 2009;15 Suppl 4:S71-5.
3. Halkias IA, Haq I, Huang Z, Fernandez HH. When should levodopa therapy be initiated in patients with Parkinson's disease? Drugs Aging 2007;24:261-73.
4. Jankovic J. An update on the treatment of Parkinson's disease. Mt Sinai J Med 2006;73:682-9.
5. Schrag A, Schott JM. Epidemiological, clinical, and genetic characteristics of early-onset parkinsonism. Lancet Neurol 2006;5:355-63.
6. Hristova AH, Koller WC. Early Parkinson's disease: What is the best approach to treatment. Drugs Aging 2000;17:165-81.
7. Hauser RA. Early pharmacologic treatment in Parkinson's disease. Am J Manag Care 2010;16:S100-7.

Access this article online

Quick Response Code:

Website: www.ruralneuropractice.com

How to cite this article: Dhikav V. Commentary. J Neurosci Rural Pract 2016;7:70-1.