



General Treatment for Parkinson's Disease

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DESCRIPTION

Parkinson's disease is the condition that results in trembling, rigidity, difficulties with coordination and balance, among other unintentional or unconscious movements. Norepinephrine, the primary chemical messenger of the sympathetic nervous system, which regulates numerous bodily functions like blood pressure and heart rate, is also lost in people with Parkinson's disease. Some of the Parkinson's disease non-movement symptoms, such as fatigue, fluctuating blood pressure, slower digestion, and a sharp drop in blood pressure after standing from a sitting or resting position, may indeed be explained by the loss of norepinephrine.

Parkinson's disease is diagnosed by a neurologist, a medical professional trained to treat disorders of the nervous system, based on our medical history, an analysis of our signs and symptoms, and a neurological and physical examination.

TREATMENT

Carbidopa-Levodopa

Within two years of the onset of symptoms, levodopa medication is necessary for PD patients. Carbidopa, an aromatic acid decarboxylase inhibitor, is frequently taken with levodopa, the most effective medication for treating Parkinson's Disease (PD), to substantially lower the probability of nausea. It has been demonstrated that increasing the carbidopa and levodopa dosage from the current recommended 1:4 ratio increases on time without dyskinesia and decreases off time.

Dopamine agonists

When used early in the course of therapy, dopamine receptor agonists stimulate dopamine receptors and postpone levodopa-related side effects such as motor fluctuations and dyskinesia's. However, there isn't enough data to establish that early dopamine agonist administration reduces the disease's course or even enhances long-term quality of life. In clinical practice, common non-ergot dopamine agonists include pramipexole, ropinirole, rotigotine, etc.

MAO inhibitors (Monoamine Oxidase Inhibitors)

Selegiline and rasagiline are beneficial in patients with moderately advanced PD who have motor problems from levodopa, even though these medications are most typically used in early, mild PD. Safinamide, another MAOI, has been shown to increase mean on time without distracting dyskinesia and decrease daily and early hours off times. Safinamide is a reversible MAOI that also inhibits neurons in the brain dopamine reuptake, reduces neuronal glutamate release, blocks voltage-dependent activated sodium channel, and buffers intracellular calcium entry.

Catechol O-Methyltransferase (COMT) inhibitors

They increase levels of levodopa and dopamine in the central nervous system by blocking the central and peripheral breakdown of levodopa and tolcapone, respectively. The hepatotoxicity of tolcapone has restricted its use. Levodopa (50, 75, 100, 125, 150, and 200 mg), carbidopa, and entacapone triple-combination therapy is accessible but frequently rejected by third-party payers.

Anticholinergics

Acetylcholine's effects at muscarinic receptors postsynaptic to striatal interneurons are countered by anticholinergic drugs like trihexyphenidyl and bztropine. They have no impact on bradykinesia and are mostly used to lessen tremor. Acetylcholine antagonists have a number of negative side effects, including memory loss, hallucinations, dry mouth, diarrhoea, and urine retention.

Adenosine receptor antagonists

As a supplement to levodopa in PD patients having off episodes, an adenosine A2 receptor antagonist. The medication, which comes in 20 mg and 40 mg tablets, offers a negligible benefit to patients who have motor irregularities brought on by levodopa. Although it has been observed to aggravate or cause dyskinesia, vertigo, hallucinations, etc., it is generally well managed.

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