Parkinson's disease pharmacological intervention: From chemicals to therapeutics.

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Introduction

A discovery of Parkinson's disease is caused by a lack of dopamine led to the development of logical medicines to address this shortfall. After a little initial scepticism, the dopamine precursor levodopa has proven to be an effective Parkinson's disease medication. Prior to the introduction of levodopa medication, surgical procedures to alleviate tremor and rigidity in Parkinson's patients were used. Meyers pioneered surgical lesioning treatments that addressed disease rather than hemiparesis, which was a side effect of previous surgeries. Parkinson disease (PD) is a nervous system ailment that affects people throughout adult life and causes tremor, slowness of movement, gait instability, and rigidity. Even though, these treatments only provide temporary but significant relief from early symptoms and do not stop the condition from progressing. First step towards preventing cell death and curing or slowing PD is to understand why and how vulnerable cells in motor and nonmotor regions of the brain die [1,2].

Diagnosis

Despite massive breakthroughs in our understanding of disease mechanisms, the diagnosis of Parkinson's disease is usually decided solely on the basis of the patient's history and physical examination. Nonmotor problems such as insomnia, sadness, weariness, constipation, and panic are common among the related symptoms. Even if the neurological exam is normal, the early PD patient may complain of stiffness, slowness, tremor, and instability [3].

Pathogenesis

Nerve cells loss is seen in PD patients' brains, particularly in the dopamine-rich, pigmented neurons of the SN, as well as the presence of Lewy bodies and Lewy neurites in many brain locations. Considering this extensive pathology, much of the study into the pathophysiology of Parkinson's disease has concentrated on the dopaminergic SN cell loss and Lewy aggregates. The previous focus in dopaminergic deficiencies was tied to the common motor signs of Parkinson's disease for which patients seek treatment [4].

The significance of oxidative stress

Autosomal-recessive PD is caused by mutations in the DJ-1 gene, which are comparable to parkin mutations. Patients respond to levodopa with asymmetric onset of symptoms, delayed development, and varying severity.

Symptomatic therapy

Present PD medication and surgical treatments are usually a symptom and have little effect on disease progression. Levodopa combined with a peripheral decarboxylase inhibitor is still the most effective medicinal treatment. Surgical DBS is likely the most important advancement in the treatment of symptomatic Parkinson's disease. Without considering advancements in the treatment of non-motor sequelae, a review of symptomatic PD therapy would be incomplete. Considering the positive reaction to medicinal and surgical treatments for motor complaints [5].

Conclusion

Whereas ground-breaking results that identified Parkinson's disease as a disease of dopamine deficiency led to the development of rational symptomatic therapies like levodopa and dopamine agonists. Proteasomal and mitochondrial dysfunction, oxidative stress, protein misfolding, and abnormal phosphorylation all have a part in pathogenesis of PD, according to the discovery of monogenetic variants of a disease.

References

1. Nutt JG, Wooten, GF. Clinical practice. Diagnosis and initial management of Parkinson’s disease. N Engl J Med. 2005;353:1021-27.
2. Elbaz, A, Bower JH, Maraganore DM, et al. Risk tables for parkinsonism and Parkinson’s disease. J Clin Epidemiol. 2002;55:25-31.
3. Poirier, LJ, Sourkes, TL. Influence of the substantia nigra on the catecholamine content of the striatum. Brain. 1965;88:181-192.
4. Carlsson A, Lindqvist M, Magnusson T. 3,4-Dihydroxyphenylalanine and 5hydroxytryptophan as reserpine antagonists. Nature. 1957;180:1200.
5. Cotzias, GC, Papavasiliou, PS, Gellene R. Modification of Parkinsonism--chronic treatment with L-dopa. N Engl J Med. 1969;280:337-345.

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