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Etiology, Treatment and Management of Parkinson's – A Review

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Abstract:
Aim: T
o review the etiology, treatment and management of parkinson's disease.
Objective:
To review the etiology, treatment, causes, treatment and management of parkinson's disease.
Background:
Parkinson's disease is also called as (paralysis Agitans). Agitans means shaking therefore the condition called as shaking palsy which is characterised by the rigidity, tremors and weakness of movements called hypokinesia. It mostly occurs in the late middle age. Therefore the concentration of the dopamine in this region ex: nigrostriatal system is reduced. Other parts of the brain also degenerate to some extent and contribute to non movement related symptoms. There are some of the treatment for the Parkinson's disease such as increase the dopamine in the brain either by replacing the dopamine or by mimicking the dopamine by inhibiting the breakdown .

INTRODUCTION:

Parkinson's disease is a degenerative disorder of the central nervous system which mainly effects the motor system. Motor symptoms may interfere with automated small hand movements [1]. Which causes impairment in tooth brushing ability. Which is considered as primary risk factors in parkinson's disease. In addition to non-motor symptoms such as dementia or apathy, altered motor behaviour and particularly motor fluctuations may influence the quality and frequency of daily oral hygiene care by these patients [2]. Non-motor symptoms occur in almost 90% of the patients which include apathy, depression, anxiety, sleep disturbances, fatigue and pain[3]. Advanced therapies aimed at smoothing out dopaminergic stimulation, such as levodopa-carbidopa intestinal gel infusion (LCIG), continuous subcutaneous apomorphine infusion or deep brain stimulation can be highly successful in treating motor fluctuations. In early stages of Parkinson's disease it can make motor function normal but duration gets slower as the disease progress. In the advanced stages fluctuations in motor function may cause major problem[4]. Primary Parkinson's disease has no known cause, although some atypical cases have a genetic origin. Tremor is the most common symptom. It is the most common; though around 30% of individuals with PD do not have tremor at disease onset, most develop it as the disease progresses.[5]. It affects the distal part of the limb. It also hypokinesia which means slowness of movement and is associated with difficulties along the whole course of the movement process, from planning to initiation and finally execution of a movement.[5].

ETIOLOGY:

There are many causes which may be environmental factors or it may be due to genetics. Coming to the environmental factors increased risk of Parkinson's disease is due to head injuries and pesticide exposure or farming[6,7]. These results indicate that chronic exposure to a common pesticide can reproduce the anatomical, neurochemical, behavioral and neuropathological features of Parkinson's disease. Heavy metals exposure has been proposed to be a risk factor, through possible accumulation in the substantia nigra. Coming to the genetics, Mutations in specific genes have been conclusively shown to cause PD. These genes code for alpha-synuclein (SNCA), parkin (PRKN), leucine-rich repeat kinase 2 (LRRK2 or dardarin), PTEN-induced putative kinase 1 (PINK1), DJ-1 and ATP13A2.[8,9]. People with these mutation develop Parkinson's disease. The role of the SNCA gene is important in PD because the alpha-synuclein protein is the main component of Lewy bodies.[9] Missense mutations of the gene (in which a single nucleotide is changed), and duplications and triplications of the locus containing it have been found in different groups with familial PD.[9]

TREATMENT:

The therapy is primarily based on the administration of levodopa associated with inhibitors of its peripheral degradation such as carbidopa or benserazide [10]. Moreover, for many years, dopamine agonists (DAs) have been incorporated into the therapeutic armamentarium of PD [10]. Levodopa is currently the most effective agent used in the treatmentof PD and has been the mainstay of therapy in recent decades. DAs are less effective than levodopa as a treatment regimen; however, they are associated with lower risks of dyskinesia and motor fluctuation. This has led to the wide-scale application of DAs in the early stages of PD in order to postpone the use of levodopa, or as an add-on treatment to reduce levodopa dosages [11,12]. Anti-oxidants like vitamin D and C had been proposed to prevent this disease but no positive results were seen.

MANAGEMENT:

Parkinson's disease cannot be cured but surgery and medication can provide relief to the Parkinson's disease. The main families of drugs useful for treating motor symptoms are levodopa (usually combined with a dopa decarboxylase inhibitor or COMT inhibitor which does not cross the blood–brain barrier), dopamine agonists and MAO-B inhibitors.[13]. Fluorodopa positron emission tomography (PET) measures levodopa uptake into dopamine nerve terminals, showing a decline of about 5% per year of striatal uptake. This diagnostic test reveals decreased dopaminergic nerve terminals in the striatum in both PD. and the Parkinson-plus syndromes but does not distinguish between them. A marked response to levodopa is helpful in the differential diagnosis, indicating presynaptic dopamine deficiency with intact postsynaptic dopamine receptors, features typical of PD. Some evidence indicates other drugs such as amantadine and anticholinergics may be useful as treatment of motor symptoms in early and late PD, but since the quality of evidence on efficacy is reduced, they are not first-choice treatments.[14] In addition to motor symptoms, PD is accompanied by a range of different symptoms. Different compounds are used to improve some of these problems.[15] Examples are the use of clozapine for psychosis, cholinesterase inhibitors for dementia, and modafinil for day somnolence.[15][16].

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