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Review on Parkinson's Disease and Medicinal Plant for Treatment

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ABSTRACT

With over 10 million cases worldwide, Parkinson's disease (PD) is the most common chronic neurodegenerative motor condition. The progressive loss of dopaminergic neurons in the brain's substantia nigra region is a characteristic aspect of Parkinson's disease (PD). According to recent studies, there may be increased interest in using natural products—particularly plants—to treat Parkinson's disease. Even if they are useful in treating symptoms, conventional pharmaceutical treatments frequently have drawbacks and restrictions. Growing interest has been seen in the potential of herbal remedies as a supplemental or alternative treatment for Parkinson's disease in recent years.

Keywords: Parkinson's disease, Neurological disorder, Medicinal plant.

Introduction: -

The second most common neurodegenerative disease in the world is Parkinson's disease. Over 6 million individuals. One of the main causes of neurological disorders is Parkinson's disease, and in the past 30 years, this number has climbed 2.5 times [1,2]. Most cases of Parkinson's Diseases are idiopathic, but genetic and environmental contributions are known. Exposure to pesticides, herbicides, and heavy metals has been linked to an increased risk of Parkinson's disease in some. Epidemiological studies, while smoking and caffeine use are associated with reduced risks [3]. The majority of PD patients appear clinically Tremors, stiffness, and slowness of movement are some of the symptoms. Autonomic dysfunction, discomfort, difficulty walking, and later stages of cognitive decline [4,5,6]. PD usually has symptoms and signs Observation of long-term response to dopamine Drug trials (levodopa or dopamine agonists) are more common Used for diagnosis. There is no significant information about magnetic resonance imaging. Imaging studies or computed tomography available [7]. Genetic markers for PD are being reviewed Diagnosis. A wide range of experiments are focused on beta-amyloid cerebrospinal fluid protein levels, tau and alpha-synuclein [8]. Newly available treatments applications in the nervous system and neurodegenerative diseases Sympatomatic relief, not permanent relief [9].

Epidemilogy: -

Parkinson's disease is an age-related disease. The incidence and prevalence increase with age. However, there is a misconception that Parkinson's disease older people are particularly affected by layoffs. The age of genesis is about 25% of 65 years, the victims 5-10% under the age of 50 years [10]. Onset Parkinson's disease introduced in relation to disabled people. Age at which it appears, under 40 years of age (may include under 50 years old). The disease occurs throughout the world, there are no significant epidemiological differences Rapid growth in high-income countries like Europe. The number of deaths and disabilities from Parkinson's disease has risen globally during the past 20 years. [11,12]. Parkinson's disease affects both men and women, but it affects women more severely. They might be better than males in some ways, such as their case Disease rates are low, particularly in the 50–59 age group. [13]. Number for disability are highest in men. however, Women have another disadvantage: they are There is a high risk of dyskinesia and changes in motor and non-motor responses that may occur due to their usually lower bodyweight.

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In addition, women more frequently Complaints about urinate and depression [14,15,16]. men are more likely to develop heart failure [17].

Etiology: -

Relative contributions of genes and environment/life. The pathogenesis of PD is discussed. in middle age the age of 60 years is the biggest risk factor For PD [18,19]. Men appear to experience this frequency more frequently than women (mean 1.3 to 2.0), however this could be due to variations in the prevalence of lifestyle factors like smoking and postmenopausal hormone use. and the intake of caffeine [20]. As with other neurodegenerative diseases, biological Dysfunctions such as telomere dysfunction, genomic instability, epigenetic changes, the ubiquitin-proteasome system and autophagy, and mitochondrial defects can be explained. aids in nerve cell death [21,22].

Pathogenesis: -

Dopaminergic activity is produced by Neuron of the extrapyramidal region of the midbrain. In addition, the center, periphery, α-synuclein protein of the autonomic nervous system Known as Lewy bodies, the cause of P.D. Main symptoms of Parkinson's disease remains unknown. However, most researchers mention Combination of genetic and environmental variables [23]. Brock hypothesis suggests that early pathological changes occur at Medulla oblongata and olfactory bulb (break stages 1 and 2) before passing rostral to the substantia nigra and midbrain (break stages 3 and 4), after which clinical symptoms appear likely to be; In the final stage, cortical areas are finally affected (break stages 5 and 6)[23]. In parts 3 and 4, pathology develops in the substantia nigra pars compacta and other midbrain and forebrain structures. Disease areas are associated with the common symptoms of Parkinson's disease behavior. In advanced Parkinson's disease, the pathology goes to the cortex and the beginning of cognitive and cognitive impairment [24]. Protein aggregation linked to Parkinson's disease Dopamine- producing cells die. therapeutic supplements Dopamine is the mainstay of treatment for Parkinson's disease. But other neurotransmitter systems don't work either Serotonin, acetylcholine and Parkinson's disease Norepinephrine systems [25,26,27,28,29,].

Diagnosis: -

The history and physical examination are the main factors used in the diagnosis of Parkinson's disease. Both motor and nonmotor symptoms should be evaluated in the clinical history. The likelihood of diagnosing Parkinson's disease is increased in families when primary Parkinson's disease runs in the family [30]. It takes clinical criteria to diagnose Parkinson's disease (PD). Parkinson's disease is characterized by bradykinesia and either stiffness, shock, or both types of relaxation [31]. Individuals with Parkinson’s disease should be treated (i.e. genuine clinical presentation but not clinical evidence).

Two out of the four requirements may be your minimum: (1) resting on its own, (2) responding well to dopaminergic medication (such as caribidopalidopa), (3) dyskinesia brought on by levodopa, or (4) Using the mind to view the mind. In myocardial infarction, iodine-123-meta-iodobenzylguanidine is reduced. The art of calligraphy [31]. Dyskinesia involuntary dance-like choreoathetoid movements occur with dopaminergic therapy. Dyskinesia typically takes years to manifest. When symptoms of Parkinson's disease first develop, its limited effects are helpful for diagnosing the condition. [32]. In some settings Parkinson's disease cannot be confirmed if it can be confirmed with medication Responsible for the patient's signs and symptoms or if additional the results suggest an alternative diagnosis [31]. dopamine transporter light emission DaT SPECT detects presynaptic dopamine Neurologic disorders in Parkinson's disease After developing devastating neurological symptoms of Parkinson’s disease Radioactive labels targeting dopamine transporters in the basal ganglia. DaT SPECT is very accurate (98% to 100%). sensitivity and specificity) to detect loss of striated nevus cells People with Parkinson's disease [33]. Magnetic resonance imaging (MRI) is usually not helpful Parkinson’s disease study. Specific findings on MRI (eg, abnormal parkinsonism index on magnetic resonance imaging in progressive supranuclear palsy) help distinguish Parkinson’s disease from other parkinsonian diseases; Advanced technology is the future Diagnostic and prognostic potential [34,35]. 5 MRI findings may suggest extensive cerebrovascular disease or gaps in the basal ganglia a possible vascular contribution. Primarily used outside the United States United States, iodine-123- meta-iodobenzylguanidine myocardial scintigraphy helps in the evaluation of sympathetic nerve relaxation, which occur frequently as part of parkinsonism [36].

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Table 1. Neurotransmitters and Pharmacologic Agents Relating to Parkinson Disease Symptoms

Neurotransmitters and Drugs Influencing the Neurotransmitter

Symptom or Sign	Dopamine	Serotonin	Norepinephrine	Acetylcholine	Other
Motor impairment (eg, bradykinesia, rigidity, tremor, gait disturbance)	Levodopa preparations, dopamine agonists (eg, pramipexole, ropinirole), monoamine oxidase-B inhibitors (eg, rasagiline, selegiline), catechol-O-methyl transferase inhibitors (eg, entacapone)			Anticholinergic agents for tremor (eg, trihexyphenidyl) a; cholinesterase inhibitors for gait (eg, rivastigmine)a,b	Amantadinec
Cognitve impairment	Monoamine oxidase-B inhibitorsa,b			Cholinesterase inhibitors	
Psychosis	Quetiapine, clozapinea	Pimavanserin		Cholinesterase inhibitorsa,b	
Depression, anxiety	Dopamine agonistsa	Selective serotonin reuptake inhibitors, selective serotonin and norepinephrin e reuptake inhibitors, tricyclic antidepressant s	Selective serotonin and norepinephrine reuptake inhibitors, tricyclic antidepressants	Tricyclic antidepressants	

a Indicates US Food and Drug Administration approved for another use but off-label use for the sign or symptom in this row.
b Studied for this use with insufficient evidence to date to support routine use
c Amantadine may affect multiple neurotransmitter systems including dopamine and glutamate.

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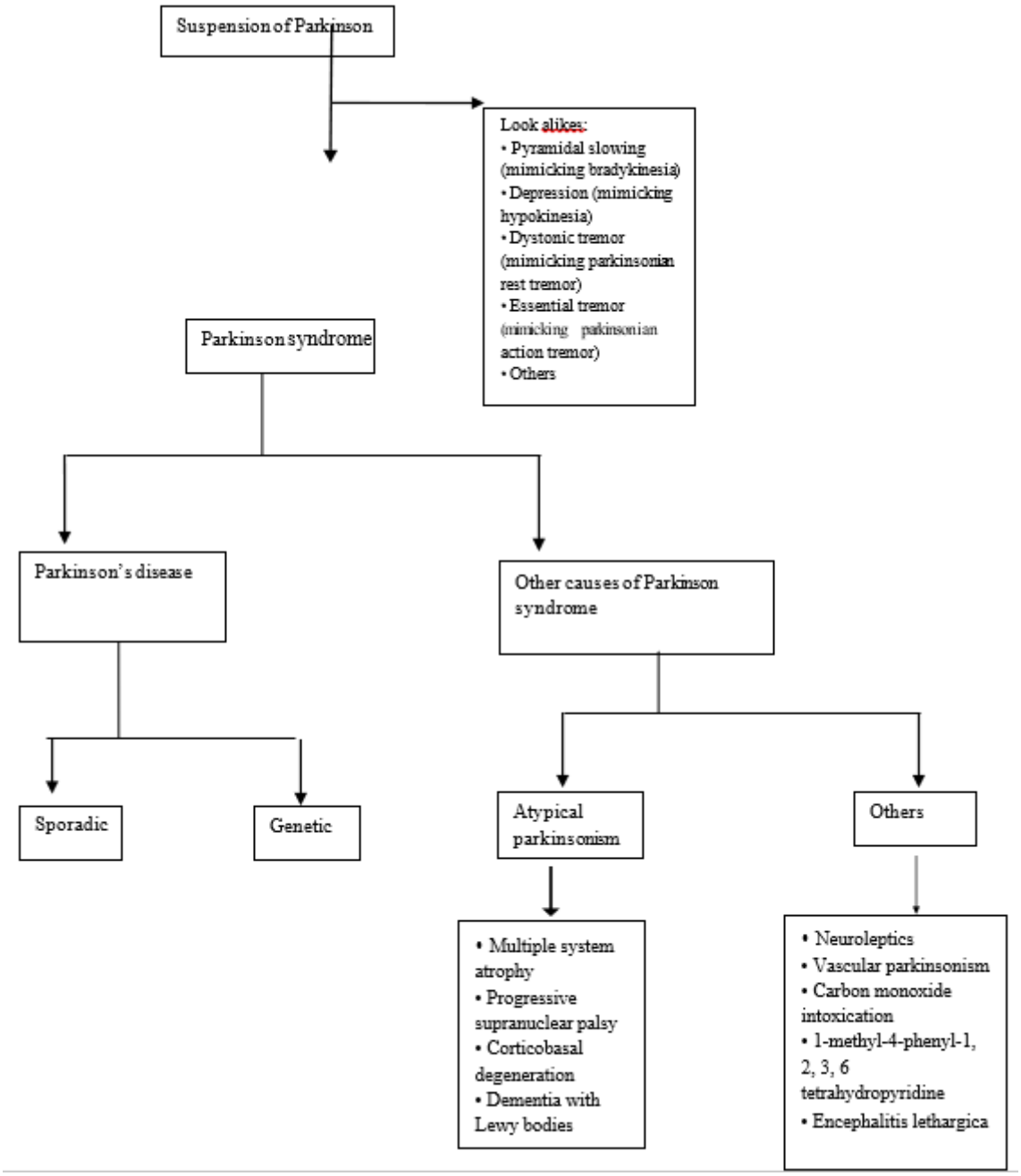

Figure 1: The clinical diagnostic process of PD

Treatment of Parkinson's Disease: -

PD is a complex neurodegenerative disease with multiple motor and non-motor features that requires an individualized therapeutic approach. Delivery of clinical trials is planned Evidence-based data should refer to known populations patients and controls and should use the most objective, reliable, and validated tools to assess the effects of a therapeutic intervention. Although there are different clinical assessment scales and other items were used to measure responses UPDRS is increasingly used online for a variety of treatments The primary outcome measure in any clinical trial [37]. For medical, surgical treatment options PD patients are exposed at different stages of their disease Next. In addition to traditional treatments, practice It also highlights available evidence and emerging issues and experimental PD treatments [38].

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Table 2: Treatment of PD

	Class	Drug	usage	Side effects
First line treatment	Dopamine agonist	Carbidopa/levodopa	Monotherapy to treat bradykinesia, postural instability	Headache depression, dizziness
Second line treatment	Dopamine agonist	Pramipexole,ropinirole	Monotherapy or adjunct to levodopa to treat bradykinesia	Hypotension dizziness abnormal dream nausea
		bromocriptine	Due to adverse effects and monitoring (baseline and annual ESR,renal function and chestn required this drug is indicated only if the patient has failed all other pharmacologic therapy	Dizziness nausea low blood sugar pulmonary fibrosis
	MAO B inhibitors	Selegiline	Off-label use as monotherapy	Headache, dizziness nausea
		Rasagiline	Monotherapy or adjunct to carbidopa/levodopa to treat bradykinesia, postural instability	Hypotension headache dizziness rash nausea
		safinamide	Apporoved march 2017 as adjunctive therapy to reduce off time	Hypotension, falls increased ALT and AST, nausea
Third line treatment	Antiviral	amantadine	Monotherapy or adjunctive therapy to treat dyskinesia. should not be drug of first choice	Hypotension syncope, peripheral edema.

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Table 3. Plant remedies in management of Parkinson's disease: -

Sr. No	Plant Name	Family	Plant Part	Ref
1	*Acanthopanax senticosus*	Araliceae	root & rhizome	39
2	*Chrysanthemum indicum*	*Asterceae*	Whole plant	40
3	*Withania*	*Solanaceae*	Root	41
4	*Trifolium*	*Fabaceae*	Whole plant	42
5	*Tripterygium*	*Celastraceae*	Root& bark	43
6	*Nardostachys*	Valirenaceae	Root	44
7	*Mucuna*	Fabaceae	Seed	45
8	*Bacopa*	Plantaginaceae	Whole plant	46
9	*Gynostemma*	Cucurbitaceae	Leaves	47
10	*Clausena*	Rutaceae	Leaves	48
11	*Cynodon*	Poaceae	Plant extract	49
12	*Centella*	Apiaceae	Whole plant	50
13	*Ocimum*	*Lamiaceae*	Whole plant	51
14	*Plumbago*	Plumbaginacea	Whole plant	52
15	*Hypericum*	Guttiferae	Whole plant	53
16	*Alpinia*	Zingiberaceae	Kernel extract	54
17	*Cassia Tora*	Fabaceae	Seed	55
18	*Polygogum cuspidatum*	Polygonaceae	Rizome	56
19	*Gastrodia elata*	Orchidaceae	Whole plant	57
20	*Gynostemma pentaphyllum*	Cururbitaceae	Whole plant	58
21	*Ginkgo biloba*	Ginkgoaceae	Whole plant	59
22	*Panax ginseng*	Araliaceae	whole plant	60
23	*Bacopa monnieri*	Plantaginaceae	Leaves &stems	61
24	*Mucuna pruriens*	Leguminosae	Seeds	62
25	*Withania somnifera*	Solanaceae	roots	63
26	*Curcuma longa*	Zingiberaceae	Rhizomes	64
27	*Gingko Biloba*	Ginkgoaceae	leaves	65
28	*Camellia sinensis*	Theaceae	Leaves	66
29	*Pinellia ternate*	Araceae	rhizome	67
30	*Rehmanniae Radix*	Orobanchaceae	Roots	68

Summary: -

The main feature of Parkinson's disease, a neurodegenerative condition affecting the central nervous system, is the progressive loss of dopamine-producing brain neurons. Many other motor symptoms, such as tremors, rigidity, bradykinesia (slow movement), and postural instability, are brought on by this dopamine deficiency. Non-motor symptoms can also manifest, including anxiety, depression, and cognitive decline. Although the precise etiology of Parkinson's disease is unknown, a number of genetic, environmental, and lifestyle factors are thought to have a role in the condition's development. Parkinson's disease is a complicated neurological disorder that can be treated in a number of ways, such as with drugs and surgery. Further research is necessary to determine the safety and effectiveness of medicinal plants for Parkinson's patients, even if they may have potential as supplemental therapy.

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