



Motor Symptoms of Parkinson's Disease – A Review Literature

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Citation: Agyekum HA. Motor symptoms of parkinson's disease -a review literature (2018) Neurophysio and Rehab 1: 38-41

Received: Dec 05, 2018

Accepted: Dec 14, 2018

Published: Dec 21, 2018

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Abstract

Parkinson disease is very common in the world, ranking second to Alzheimer's disease in terms of degenerative neurological disorders. The disease since 1817 has had so many researchers looking into it, bringing out symptoms, possible treatment options, causes of the disorder to mention but a few. There are a number of studies that have looked into the symptoms of the disorder, especially the non-motor symptoms of the disorder. This study looks into the motor symptoms associated with the disease. It brought out the causes, which the study from the literature review conducted asserted that there is no known cause for it, however, about 60 to 80 percent of persons with PD, have a degenerating number of dopamine, as such, this can be postulated to be the cause. The study also affirmed that there is no known treatment for the symptoms, doctors and medical practitioners would therefore have to try different forms of treatment till the right one for the patient is arrived at.

Keywords: Parkinson's disease, Alzheimer's disease, Dopamine, Substantia nigra, Tremor, Rigidity, Akinesia and Bradykinesia

Introduction

Parkinson's disease is the second most common degenerative neurological disorder after Alzheimer's disease [1]. The disease has been described by James Parkinson in 1817 as a chronic neurodegenerative disease characterized by the loss of dopaminergic neurons in the substantia nigra which leads to decreased levels of dopamine in the striatum and disrupted motor control. Since its description in 1817, there have been a lot of researches being done to understand the disorder, with some researches being conducted on its prevalence in Sub-Saharan Africa. This proves that the disease is a worldwide canker which needs much attention and awareness creation.

It is estimated that the disease affects over 1% of the population over the age of 60, which in the UK equates to over 127,000 individuals (or 500,000 individuals in the USA), while in individuals over the age of 85 this prevalence reaches 5%, highlighting the impact that advancing age has on the risk of developing this condition [2-4]. Williams and colleagues [5] reported in their article on Parkinson's disease in sub-Saharan Africa that the disease has a prevalence ranging from 7/100,000 in Ethiopia to 67/100,000 in Nigeria, with the most recent community-based study reporting a mean age at onset of 69.4 years. Since no exact test exists for the diagnosis of PD, the exact number of people with this disorder cannot be determined, with some being misdiagnosed because of the disease's similarity to other diseases [6].

Studies have acknowledged that there is a higher chance of PD occurring more in men than in women [7], with statistics of it occurring in about 50% more men than women [6]. There is no exact reason for the differences however some researchers have suggested explanations that, the protective effect of estrogen in women, the higher rate of minor head trauma, and exposure to occupational toxins in men, and

genetic susceptibility genes on the sex chromosomes could account for these differences [7]. Gillies and colleagues [8] are however of the view that the difference is determined, largely, by biological sex differences in the NSDA system which, in turn, arise from hormonal, genetic and environmental influences. Over the years, researches have concluded that the disorder is not inherited, that is it does not pass down through family line. Though some cases of PD occur in families, 90% of cases are sporadic, meaning they occur without an, as yet, identified inherited genetic predisposition [2], simply put, it does not happen because one of the family member has it.

Recent researches that have been done on PD attempt to look at the causation factors of PD. The purpose of this paper is to review the large body of literature on Parkinson's disease, looking at what has been done so far. It is estimated that the number of people with PD in 2005 was approximately between 4.1million and 4.6 million and that will more than double by 2030 to between 8.7 million and 9.3 million [9]. Towards this regard, the paper aims at bringing out a comprehensive report on what has been done, with particular attention to the symptoms of the disease, examining the early stage symptoms and what people should expect.

Pathophysiology of PD

The pathophysiology of PD involves the progressive loss of dopamine-containing neurons of the pars compacta of the substantia nigra leading to denervation of the nigrostriatal tract and significant reduction of dopamine at the striatal level [10]. The consequence of this denervation process is an imbalance in the striato-pallidal and pallido-thalamic output pathways, which is responsible for the major motor deficits

[11]. According to Hamani and Lozano [12] the number of dopaminergic neurons in the substantia nigra varies from species to species, with humans having approximately 220,000 dopaminergic neurons in the substantia nigra of each hemisphere [13]. When more than 50 percent of these cells are lost, patients start to develop the signs and symptoms of the disease—tremor, rigidity, akinesia and bradykinesia (poverty and slowness of movement), as well as postural and gait abnormalities [12]. The model proposes that dopamine deficiency produces dysfunction in the striatum, leading to decreased activity in the direct pathway, from GABAergic striatal neurons to the internal segment of the Globus Pallidus (GPI) and substantia nigra pars reticulata (SNpr) and an increased drive through the indirect pathway, involving particularly the external segment of the Globus Pallidus (GPe) and Sub-Thalamic Nucleus (STN). This results in a disruption of the activity in basal ganglia output structures (GPI and SNpr), which in turn disrupts the activity in brain stem motor areas, including the pedunculopontine nucleus and the thalamocortical motor system. This disruption is thought to be responsible for the difficulty in initiation of movements and the poverty of motion that are characteristic of PD. They concluded by bringing out the shortfalls of the model which is its inability to adequately explain some of the other cardinal features of Parkinson's disease, such as tremor or rigidity. Further, the model does not take into account that dopamine exerts its effects not only in the striatum but also throughout basal ganglia nuclei and at cortical levels.

Genetic predisposing factors in combination with environmental factors are thought to be responsible for the cellular changes leading to progressive neuronal degeneration in which mitochondrial dysfunction, oxidative mechanisms and failure of the protein degradation machinery at the cellular level are probably involved [14]. The presence of Lewy bodies (cytoplasmic proteinaceous inclusions) in surviving dopaminergic neurons is the pathological hallmark of PD [10] (Figures 1 and 2).

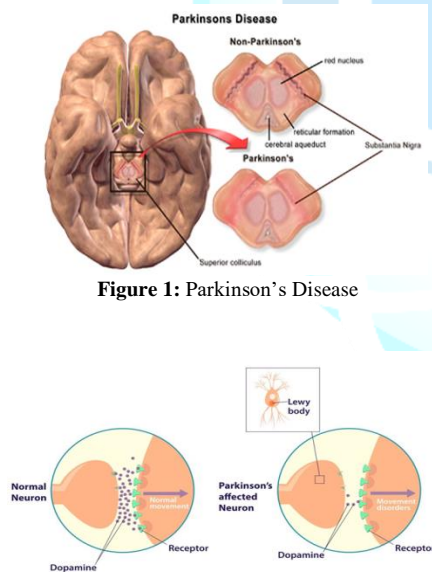


Figure 1: Parkinson's Disease

Figure 2: Dopamine levels in a normal and a Parkinson's affected neuron

Motor Symptoms

Researches that have been done to look at the symptoms associated with Parkinson's Disease (PD) have shown that it is a degenerative neurological disorder, associated with primary and secondary motor

symptoms [15]. Although it has been assumed that the various motor symptoms in PD are caused solely by striatal dopamine depletion [16,17], many studies have shown that additional neural structures and neurochemical systems are also responsible for the occurrence of motor symptoms in PD; these include prefrontal cortical areas and the cerebellum, as well as serotonergic, glutamatergic, and cholinergic systems [18,19]. For the purpose of the study, motor symptoms of PD that will be considered will be akinesia, bradykinesia, tremor and rigidity.

Akinesia

Akinesia is a term for the loss of ability to move your muscles voluntarily. In describing this symptom of PD, Kinnier Wilson had this to say "it seems as if the patient does not care to continue the task or put an adequate amount of effort into a particular movement" [20]. Even though a major symptom of PD, akinesia can also appear as a symptom for other conditions too. According to a study in the Journal of Neurology, Neurosurgery & Psychiatry, 47 percent of more than 6,600 people with Parkinson's disease who responded to a questionnaire reported akinesia or freezing as a symptom.

Akinesia is characterized by abnormal stiffness due to increased tone of both the agonist and antagonist muscles (lead pipe) or cogwheel rigidity due to superimposed or underlying tumor [21]. Akinesia can also be referred to as freezing. Some of the symptoms associated with akinesia include difficulty when a person starts out to walk somewhere, muscle rigidity, usually beginning in the neck and legs, muscles in the face can also become rigid, similar to a mask and there is also sudden inability to move the feet properly, especially when turning or approaching a destination [22]. Usually Parkinson associated akinesia is brought about as a result of a reduction in dopamine in the brain, causing a difficulty in the ability to move body parts [22].

Treatments for Parkinson's disease-related akinesia can be more complicated as no one treatment can be used or is known to cure this symptom. Doctors will often prescribe medications that increase the amount of the neurotransmitter dopamine in the body or the activity it causes. Getting regular exercise can help you reduce the pain and discomfort that can happen with akinesia and other motor function conditions that may result from PD. It will be advised for a patient with this symptom to talk to a doctor or a physical therapist about developing an exercise plan that's comfortable and safe depending on the symptoms and the progression of akinesia. Exercise has been shown to delay functional decline in PD.

Bradykinesia

Bradykinesia has its origination from Greek words 'slow' and 'movement'. The term which describes slowness in carrying out, rather than initiating, movements was first used by Dr James Parkinson in 1817. Bradykinesia is one of the early signs of a movement disorder such as Parkinson's or Parkinsonism. A doctor or neurologist who observes bradykinesia may well suspect that the person has Parkinson's. The exact cause of the disorder is unknown [23], however, there are evidence of it being caused by reduced levels of dopamine in the brain and is often first noticed by family and friends. Movement is particularly impaired when novel movements are attempted [24], or when several movements are combined [25,26]. Whereas in akinesia there is the inability to move body parts or a difficulty in movement, in bradykinesia there is slowness in movement.

Theoretically, bradykinesia can be categorized into the verbal-conceptual models and the mathematical and computational models [23]. The verbal-conceptual models describe the brain areas, pathways and interactions thought to lead to parkinsonian bradykinesia whereas the mathematical and computational models describe the interactions between the various brain areas involved in movement control and execution that are relevant in parkinsonian bradykinesia [23].



Bradykinesia cannot be cured; however, the symptoms can be managed somewhat with medications. The most helpful medications for bradykinesia are those that increase dopamine levels. Doctors can try various medicines until the right one for the patient is found. The challenge here will be finding the right medication for the patient due to the medications becoming less effective over time. There are therefore other treatment plans that doctors can plan for their patients. There is the surgical procedure known as deep brain stimulation that may be used for certain people living with Parkinson's. This treatment involves surgically placing electrodes into specific locations in your brain. Once the electrodes are in place, the patient will have electrical impulses sent to stimulate the brain at the electrode sites. This treatment is normally reserved for those with severe Parkinson who do not respond well to traditional medications. The patient can also adopt lifestyle remedies for symptom management. Usually, whether you are taking medications or not, there are lifestyle changes and steps a patient with bradykinesia associated with Parkinson can add at home as part of their treatment plan. However, they should be sure to talk to their doctor or medical practitioner before adding anything to the treatment plan. Some of these lifestyle or home treatments may include eating a healthy diet usually high in fiber, doing physical therapy with exercises that increase strength, balance, and flexibility like walking and swimming, and taking the steps needed to help avoid falling [27].

Tremor

A tremor is an involuntary quivering movement or shake. It is characterized clinically by involuntary, rhythmic and alternating movements of one or more body parts [28]. A consensus statement of the Movement Disorder Society has classified three tremor syndromes associated with Parkinson's disease [29] to include resting, postural, and kinetic. Tremor is very common in Parkinson's, affects about 80% of people with Parkinson's [30]. The tremor that occurs in Parkinson's disease is different than almost all other tremors because it is a "resting tremor," present primarily at rest [30]. It goes away with movement, but often returns when the limb (usually a hand or the fingers) is held in one position, as in holding a spoon or fork to the mouth, which is why those with Parkinson's are known to spill things. Parkinson's disease tremor may affect almost any part of the body, but most commonly involves the fingers, followed next most commonly by the hands, jaw, and feet in that order [30].

The resting tremor is a tremor that occurs during voluntary movements [31]. Resting tremor occurs when the muscle is relaxed, such as when the hands are resting on the lap. With this, a person's hands, arms, or legs may shake even when they are at rest [32]. Often, the tremor only affects the hand or fingers. This type of tremor is also called a "pill-rolling" tremor because the circular finger and hand movements resemble rolling of small objects or pills in the hand [33]. Parkinsonian tremor is caused by damage to structures within the brain that control movement [32]. This resting tremor, which can occur as an isolated symptom or be seen in other disorders, is often a precursor to Parkinson's disease. The symptoms of this tremor can be evidently increased by stress or emotions. Approximately 70% of PD patients experienced tremor during the course of the disease [34]. There is no cure for tremor; however, the medication for the treatment of Parkinson's disease can be used to control this symptom.

Rigidity

Rigidity has been defined by Xia and Rymer [35] as an increased resistance of a joint to passive movement. The resistance is essentially constant throughout the range of movement [35]. Rigidity is associated with feeling of stiffness experienced by the patient, and clinicians may assess rigidity by examining the resistance of a muscle against passive stretching [36]. One of the major manifestations of Parkinson's disease is rigidity. The only symptom unequivocally produced by rigidity is a feeling of stiffness [36]. This term refers to the phenomenon of increased resistance when stretching a muscle passively. Although some features of rigidity have been characterized, the detailed

physiology is still unknown [37], it is believed that it is associated with the reduction in dopamine, a neurotransmitter (chemical messenger) that relays the message to particular areas of the brain to produce smooth, purposeful movement [38]. It has been estimated that about 90 to 99 percent of people with Parkinson's experience rigidity. Rigidity is often associated with slowness of movement (bradykinesia). It is often preceded by aching, stiffness or a feeling of weakness in the muscles [39]. Everyone's experience is different but often rigidity starts in one arm, spreads to the leg on that side and then to the trunk and other side of the body. Rigidity will progress faster in some people than in others [39].

Though there is no definite cure for rigidity, a number of treatments could be applied till the best cure is achieved. Treatment is tailored to the individual patient and may include medications, surgery (deep brain stimulation), and complementary or alternative medicine [38]. A physiotherapist can advise on exercises to maintain or improve both mobility and the range of movement in muscles and joints. He or she can also suggest strategies to perform daily activities in a more effective way, for example how to roll in bed or get up from a chair. An occupational therapist or physiotherapist will be consulted to advice on devices and aids to help you in your everyday life [39]. Choice of therapy should be customized to the individual patient with an understanding of the risks and benefits of each class of medication [40].

Conclusion

The motor symptoms of PD are the very first symptoms you will notice in a patient with the disease. Usually, it is the motor symptoms that doctors and other medical practitioners use as a basis for diagnosing the disease since there is no definite test. This article looked at literature on the motor symptoms of the disease, how you can identify them and the steps you can take towards the treatment. From the researches reviewed it was identified that there is usually no major known cause for these symptoms, but usually when the motor symptoms of PD appear, 60 to 80 percent of the neurons that generate dopamine have been destroyed [6]. It can therefore be concluded that these symptoms are brought about from the reduction of dopamine. Even in the treatment of these symptoms, medicines that increase the level of dopamine in the system are recommended. It was also realized that there was no definite cure for these symptoms just as the disease itself; however, there are treatments that can help reduce the problems associated with these symptoms. There has not been much work done on the reason for the delay in finding a cure for the disease, however, Dr. Soania Mathur [41] in an article has highlighted some reasons for this, which includes the brain being too complicated coupled with a complicated disease making it difficult to find a cure. It can be concluded that drugs that are being prescribed for Parkinson's can be used to treat these symptoms. There are also other forms of treatments like medical surgery, exercises and change of lifestyles that can help with these symptoms. Due to the unknown cure for the symptoms, doctors would have to try on different treatment plans to arrive at the best solution for the symptoms.

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