Parkinson's Australia

Parkinson's Disease Information Sheet 1.3
Diagnosis of Parkinson's

Parkinson's disease (Pd) is a progressive neurological condition which is characterized by both motor (movement) and non-motor symptoms. It is important to remember that presentation of symptoms is unique to each person with Parkinson's (PWP).

Pd was first described by Dr James Parkinson in 1817 in *An Essay on the Shaking Palsy*. He gave a detailed account of the symptoms he observed in six individuals in London. In addition to the cardinal signs listed below Dr Parkinson described the non-motor signs of Parkinson's which are also taken into account at the time of diagnosis in present time. His description remains a classic documentation of the condition which was later to bear his name.

In spite of medical advances in many areas the diagnosis of Pd is still based on the clinical presentation of the patient, the history provided and a neurological examination. Currently blood screening investigations are being researched and these may help with the diagnosis of a genetic form of Pd in the younger onset group.

Provisional medical diagnosis is based on assessment of 4 cardinal symptoms;

- Tremor
- Bradykinesia
- Muscle rigidity
- Postural instability

Tremor

It should be noted that although this is the most commonly identified symptom it is not present in all cases (30% of PWP will not experience tremor) and tremor is present in other conditions.

The classic Pd tremor is described as a "resting" tremor i.e. it is apparent when the limb is at rest. The Parkinson's tremor is regular and rhythmic and occurs at the rate of 4-6 times per second.

Initially tremor may affect a unilateral limb however with progression of the disease tremor may become bilateral. A classic tremor presentation in Pd involves the thumb and first finger and is known as "a pill rolling tremor".

Tremor may be exacerbated by stress.

Bradykinesia

Bradykinesia literally means "slow movement". The general effect is that it takes longer and requires more effort to complete daily tasks. Slowness and difficulty in initiating and executing automatic repetitive movements is commonly experienced. These actions include writing, fastening buttons, turning over in bed and walking. These difficulties are exacerbated when dual tasking is involved i.e. walking and talking.

Bradykinesia is also apparent in decreased eye blink and may lead to changes in swallowing and speech.

Muscle Rigidity

Muscle rigidity refers to the resistance felt in muscles when they are passively moved. This can be described as "lead pipe" rigidity when the resistance is consistent. "Cogwheel" rigidity is the term used when the resistance to passive movements has a regular jerking characteristic. Muscle rigidity as with all Pd symptoms first presents as unilateral however with progression becomes bilateral. Muscle rigidity accounts for the neck and shoulder pain sometimes experienced early in the disease process.

Postural Instability

Postural changes are evident as forward flexed or stooped posture. The ability to maintain posture and balance may be affected in Pd. This symptom may appear later and is often the cause of falls because the ability to correct one's balance is compromised. This may lead to unsteadiness when walking, turning or standing.

On examination, if three of the cardinal symptoms are present a provisional diagnosis of Pd may be made. A positive response to levodopa is often a further indicator of correct diagnosis.

Magnetic Resonance Imaging (MRI) and Computed Tomography (CT) are often carried out to rule out other diagnosis. The neurological changes within the basal ganglia are not routinely visible on these investigations.

In its classical presentation Pd may be easily diagnosed due to the typical posture, gait and facial changes however diagnosis of early disease is challenging because in excess of 70% of substantia nigra pars compacta cells may be lost before any clinical signs are apparent.

Autopsy based studies have shown that diagnostic accuracy is imperfect with up to 25% of cases indicating an erroneous diagnosis of Pd at the time of death.

There are currently no biological markers available that will lead to a definite diagnosis of Pd during life there have been some recent Australian advances. These are the initial development of a blood screening process to identify the genes implicated in Pd and another laboratory test to measure the levels of the protein alpha-synuclein in the blood. This protein is implicated in Pd as PWP have low levels while people without Pd have high levels. These laboratory tests are undergoing further development and trial.

In addition to Idiopathic Pd there are some conditions, known as Parkinson's Plus, which may initially mimic Pd. It is often with the progression of time and presentation of additional symptoms that these conditions may be diagnosed.

Parkinson's Australia strongly recommends that anyone facing a possible diagnosis of Pd be seen by a neurologist and/or a consultant specializing in Pd in order to determine as accurate a diagnosis as possible. Each state branch of Parkinson's Australia can supply a comprehensive list of such specialists.

For further information please contact your state organisation: FREECALL 1800 644 189

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