Parkinson's Disease (PD)

Fact Sheet

What is Parkinson's disease?

Parkinson’s is a chronic, progressive neurodegenerative disorder that affects the motor system and has variable non-motor components including cognitive and autonomic changes.¹

The symptoms of Parkinson’s disease are mainly caused by loss of dopamine-producing cells in the brain but exactly why dopamine-producing cells become lost is unclear. The Parkinson’s has different symptoms, but the most common are tremor, muscle rigidity and slowness of movement.

Currently there is no treatment available to slow down or reverse the disease. The goal of the treatment is to reduce symptoms with as few side effects as possible.

Key facts

- Parkinson's Disease (PD) is a chronic progressive disorder of the central nervous system with approximately 6.2 million people affected worldwide.² It is the second most common neurodegenerative disorder next to Alzheimer's disease.

- As the incidence of Parkinson's rises significantly with age, and people are living longer, the prevalence of Parkinson's is set to rise dramatically in the future and there may be nearly 13 million people with Parkinson's by 2040.³

- Although it is a disease more common in older age groups, one should keep in mind that about 10% of patients are affected at an age below 50 years.
History

The disease is named after James Parkinson (1755 – 1824), the son of an apothecary and surgeon who was born on 11 April, 1755 in London, United Kingdom. This is the reason why we celebrate World Parkinson's Day on 11 April. James Parkinson studied at the London Hospital Medical College, qualifying as a surgeon in 1784 when he was 29. He is most famous for publishing “An Essay on the Shaking Palsy” in 1817, which provided the first clear clinical description for the disorder. Six decades later, Jean-Martin Charcot, a French neurologist and professor of anatomical pathology, was the first to suggest the use of the term “Parkinson's disease”.

Known causes of Parkinson's disease

Parkinson's is a disease of the central nervous system caused by loss of dopamine-producing cells in the brain. However, exactly why dopamine-producing cells become lost is unclear. Research suggests that a combination of genetic and environmental factors may be responsible. How these two factors interact varies from person to person. It is also unclear why some people develop the disease but not others. Accordingly, a better understanding of the functional interactions among these factors that lead to disease onset is needed in order to identify and set up appropriate pharmacological treatment options.

Loss of dopamine

Parkinson's symptoms are triggered by a decrease in the levels of the messenger dopamine, which allows messages to be sent to the parts of the brain that co-ordinate movement, due to the death of dopamine-producing nerve cells in the substantia nigra. With the loss of dopamine-producing nerve cells, these parts of the brain are unable to function normally, causing the symptoms of Parkinson's to appear. Typically, if first symptoms occur, a loss of over 70% of the neuronal cells in the substantia nigra has already become obvious.

Genetic origin

It is rare for Parkinson's to be passed from parent to child. However, in recent years a number of cases have been identified in which Parkinson's seems to be at least partly genetic. In these cases, a mutated gene appears to have passed from one generation to the next and Parkinson's has developed in a number of people in the same family.

Environmental causes

Some evidence suggests that environmental factors, including toxic chemicals, viruses, bacteria and heavy metals, may cause dopamine-producing neurons to die, leading to the development of Parkinson's. In particular, there has been much speculation about a link between the use of herbicides and pesticides and the development of Parkinson's.
Diagnosis

Parkinson's is not always easy to diagnose because there is no specific test for the condition. Symptoms vary from person to person and a number of other illnesses have similar symptoms, which means misdiagnoses can occur.

Various types of exams can be carried out to examine the anatomy and functioning of the brain and other parts of the nervous system. These can help to distinguish Parkinson’s from other conditions with similar symptoms:

**CT (Computerised Tomography) scan**
This technique involves X-rays being passed through the body from different angles to build up cross-section pictures of the brain. It may help to rule out vascular disease and tumours as the cause of Parkinson’s-like symptoms.

**MRI (Magnetic Resonance Imaging) scan**
This scan uses magnetic charges rather than X-rays to form images of the brain or other parts of the body. It may help to distinguish Parkinson’s from Parkinson’s-like conditions such as Progressive supranuclear palsy (PSP) and Multiple System Atrophy (MSA).

**DaTSCAN™-SPECT scan**
This scan is used to identify loss of the dopamine-producing cells in the brain, which leads to Parkinson’s. It can be used to distinguish Parkinsonian tremor from other types of tremor, such as essential tremor.

**PET (Positron Emission Tomography) scan**
This imaging technique can be used to help diagnose Parkinson’s, but as it is more expensive and not as readily available as SPECT, it is mainly used in research.

Symptoms

Symptoms start to appear when the brain can’t make enough dopamine to control movement properly. They generally develop slowly over years and the progression of symptoms is often a bit different from one patient to another. Parkinson’s symptoms can be divided as follows:

**Motor symptoms** – symptoms involving movement, such as tremor, slowness of movement (bradykinesia), gait and balance issues, muscular stiffness (rigidity)

**Non-motor symptoms** – symptoms not related to movement, such as fatigue, urinary, sleep and gastrointestinal issues, pain

In addition, doctors also divide symptoms into primary and secondary symptoms.

- **Primary symptoms** are the most noticeable or important symptoms. The three primary symptoms of Parkinson’s are all motor symptoms: tremor, rigidity or stiffness and slowness of movement (bradykinesia). Balance and posture are also affected as Parkinson’s progresses, so postural imbalance is sometimes seen as the fourth primary symptom.

- **Secondary symptoms** are less obvious symptoms which still have an impact on quality of life. These can be either motor or non-motor (pain and discomfort in an arm or leg, anxiety, depression, slowness of thinking or cognitive impairment, memory problems, tiredness, disturbed sleep, constipation, sexual, swallowing problems, dysautonomia).
Currently there is no treatment available to slow down or reverse the disease. The goal of the treatment is to reduce symptoms with as few side effects as possible. Despite the disease having low impact on life expectancy, patients with Parkinson’s disease experience progressive disability and reduced quality of life at all stages of the disease and at all ages. Several studies indicate that quality of life is affected not only by the motor symptoms of Parkinson’s, but also by the non-motor symptoms such as depression and cognitive state.⁴

Even though there is no cure for Parkinson’s, a number of treatments are available and effective in improving the symptoms. Treatments consist of medication, surgery, and physical therapy. The medications that are most commonly used work by replacing or mimicking the effects of dopamine, with the aim of restoring the deficiency of dopamine in the brain and re-establishing normal function. The identification of the right medications for long-term treatment of Parkinson’s symptoms in each patient remains a great effort and a challenge. Invasive treatment for patients with advanced Parkinson’s disease are: apomorphine pump, Duodopa, and deep brain stimulation. Indications for these treatments need to be discussed with the treating neurologists.

Levodopa (L-Dopa) is the precursor to dopamine. Most commonly, Levodopa is used as a dopamine replacement agent for the treatment of Parkinson disease. It is most effectively used to control the majority of motor and non-motor symptoms of PD.⁵

Dopamine agonists can be used alone or in combination with levodopa. These medications are not as strong as levodopa-type medications that are used for Parkinson’s disease, but they don’t have the more severe uncontrolled movement related side effects, called dyskinesia, associated with long-term use of levodopa.⁶ However, they can induce addictive behavior (for example gambling) that need to be monitored.

Anticholinergics have a mild antiparkinsonian effect and are most useful in young patients with tremor and muscle spasms (dystonia).

Selegiline and COMT-inhibitors can help to improve the efficacy of L-Dopa.

Amandatine has a mild antiparkinsonian effect and can also reduce involuntary movements provoked by L-Dopa.

Further information

European Parkinson’s Disease Association
www.epda.eu.com

Parkinson’s UK
www.parkinsons.org.uk

REFERENCES
1. http://www.nrronline.org/article.asp?issn=1673-5374;year=2018;volume=13;issue=8;spage=1342;epage=1345;aulast=Lee

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