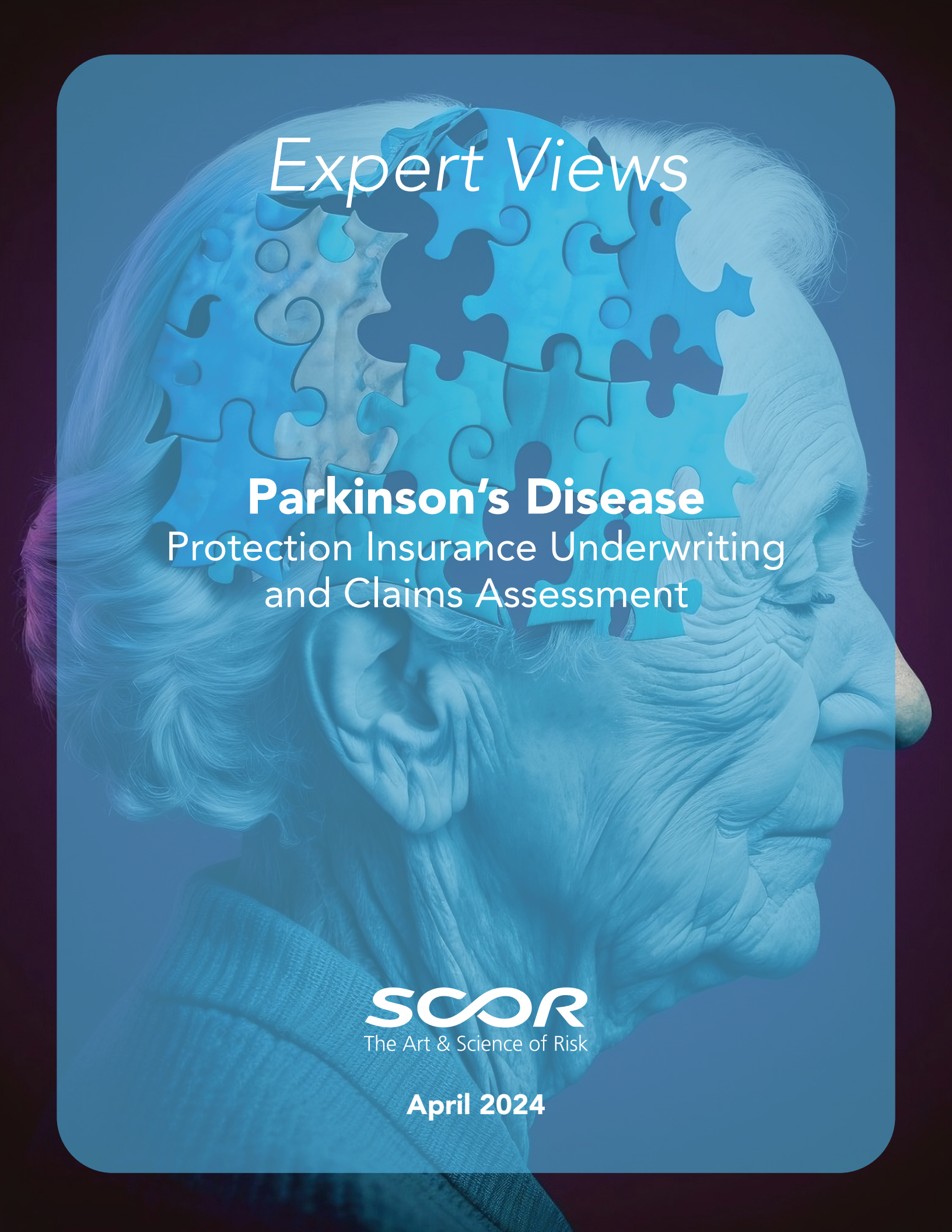

Expert Views

Parkinson's Disease

Protection Insurance Underwriting and Claims Assessment

SCOR
The Art & Science of Risk

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Contents

Introduction

Parkinson's disease, a complex and debilitating neurological disorder, has become increasingly prevalent in recent years, significantly impacting the lives of individuals and their families. As a progressive condition with no known cure, it brings a range of motor and non-motor symptoms that can lead to severe disability. The financial burden associated with Parkinson's disease, particularly for those diagnosed at younger ages, highlights the need for some form of insurance, whether it be Critical Illness (CI) or Income Protection (IP).

In this paper, we will delve into the multifaceted nature of Parkinson's disease, examining its clinical characteristics, progression, and the profound impact it has on the quality of life of those affected. We will also explore the importance of critical illness claims as a financial safety net for individuals confronting this chronic condition, shedding light on the need for awareness, understanding, and adequate support systems to ensure a better quality of life for those living with it.

Some, including healthcare professionals and people with the condition, call it Parkinson's disease, or PD for short. However, other organizations such as the charity Parkinson's UK call it Parkinson's as the word "disease" may sound negative. Therefore, we shall refer to it as Parkinson's throughout this paper.

By delving into the medical, social, and financial aspects of Parkinson's, this paper aims to provide a comprehensive overview of the challenges faced by individuals and their families.

Statistical Data

According to the World Health Organisation, the prevalence of Parkinson's has doubled in the past 25 years. Global estimates in 2019 showed over 8.5 million individuals with Parkinson's.[1] China has the highest population with Parkinson's in the world, estimated to have over half of the worldwide population with this disease.[2] In the US, around 90,000 people are diagnosed with Parkinson's every year, and the number is estimated to climb to 1.2 million by 2030.

Parkinson's is the fastest-growing neurological condition in the world, and currently there is no cure. Treatment is purely aimed at slowing down the disease and coping with the condition. This is largely a disease seen in older ages and a breakdown of the age bands in the UK case can be seen in Fig.1. However, younger ages can also become carriers.[3]

As Parkinson's is largely a condition seen in older ages, it does not feature so highly in claim statistics. Looking at SCOR's claims experience for Critical Illness claims in the last five years,

Fig. 1 – UK Incidence of Parkinson's by Age Band

Age (estimates throughout the UK for 2020)

Age estimates	Number estimates
50-59	9,000
60-69	28,300
70-79	62,400
80-89	43,600
80-89	43,600
90+	8,300

Parkinson's makes up just over 1% of claims seen. Due to the age profile of the majority of those diagnosed, they tend to be outside the insured population. While the population incidence is relatively high, claims experience is relatively low. However, Parkinson's is still a significant condition that underwriters and claims assessors need to understand.

What is Parkinson's?

Parkinson's is a chronic and progressive neurological disorder that affects the central nervous system. The condition can affect a person's movements, cognitive function, and ability to carry out daily activities. The disease occurs when the brain cells responsible for producing dopamine, a chemical messenger, begin to die off. Dopamine is a neurotransmitter that plays a crucial role in the regulation of movement, emotion, and motivation.

And there is "Parkinsonism," an umbrella term used to cover related conditions. Parkinsonism can be caused by a variety of conditions, including neurodegenerative disorders like Parkinson's, multiple system atrophy, progressive supranuclear palsy, corticobasal degeneration, and dementia with Lewy bodies. Parkinsonism can also be caused by blood vessel blockages in the brain (cerebrovascular disease, causing vascular Parkinsonism) or as a side effect of medications used to treat other conditions, notably antipsychotic medications. Rarely it can result from exposure to certain toxins, such as manganese, carbon monoxide, and pesticides.

The symptoms of Parkinsonism are similar to those of Parkinson's, but there are some differences. In Parkinson's, the symptoms typically start on one side of the body and then progress to the other side over time. In Parkinsonism, the symptoms may be more symmetrical and affect both sides of the body from the outset. Additionally, Parkinsonism may have more rapid progression, and typically patients do not respond to treatment for Parkinson's.

The symptoms of Parkinson's can vary from person to person and may develop slowly over time. The most common symptoms of Parkinson's are:

Tremors: Tremors are involuntary shaking or trembling movements, usually in the hands, arms, legs, or jaw. Tremors are often more noticeable when a person is at rest.

Bradykinesia: Bradykinesia is a slowing of movements and a decrease in the ability to initiate movement. This can cause difficulty with simple tasks such as getting out of a chair or writing.

Rigidity: Rigidity is stiffness or resistance to movement in the muscles. This can cause pain and make it difficult to move, resulting in slowed walking.

Postural instability: Postural instability is a loss of balance and coordination that can cause falls.

Other symptoms of Parkinson's may include:

- A stooped posture
- A shuffling gait
- Decreased arm swing
- Difficulty with fine motor skills, such as buttoning clothes
- Loss of facial expression
- Quiet speech
- Reduced sense of smell
- Sleep disturbances
- Depression and anxiety
- Cognitive changes, including memory loss and difficulty with multitasking

However, Parkinson's is not to be confused with Parkinsonism. Parkinsonism is an umbrella term used to describe a group of disorders that have similar symptoms to Parkinson's but are caused by different underlying conditions.

Dopamine

Dopamine also plays a role in regulating movement. The loss of dopamine-producing neurons in the substantia nigra is a hallmark of Parkinson's. Dopamine is a neurotransmitter, a chemical messenger in the brain that helps to regulate various functions, including movement, motivation, reward, and pleasure. It is produced in several areas of the brain, including the substantia

nigra, the ventral tegmental area, and the hypothalamus. In addition to its role in movement and reward, dopamine is also involved in several other functions in the brain, including attention, memory, and learning. It is believed to play a role in addiction, as drugs of abuse like cocaine and amphetamines can increase dopamine levels in the brain, leading to feelings of euphoria and reinforcing the addictive behavior.

Dopamine interacts with several receptors in the brain, including the D1, D2, D3, D4, and D5 receptors. These receptors are found in different regions of the brain and have different functions. Activation of the D1 receptors is associated with improved working memory and attention, while activation of the D2 receptors is associated with decreased activity in the basal ganglia, a group of structures in the brain involved in movement regulation. D5 receptors are believed to be involved in various cognitive functions, including learning and memory. Studies have suggested that D5 receptors in the hippocampus, a brain region critical for memory formation, play a role in spatial memory and certain aspects of learning. Imbalances in dopamine levels or dysfunction in the dopamine system can lead to several neurological and psychiatric disorders. In addition to Parkinson's, other disorders associated with dopamine dysfunction include schizophrenia, attention-deficit/hyperactivity disorder (ADHD), and addiction.

Risk Factors

The risk of developing Parkinson's tends to increase with age. But it's important to note that while age is a significant factor, Parkinson's can affect individuals of varying ages, and not everyone who is diagnosed with the condition falls within a specific age range. Genetic factors, environment, and other factors may also play a role in the development of Parkinson's.

The precise cause of Parkinson's is unknown, but research has shown that a combination of genetic and environmental factors plays a role in the development of the disorder. In some cases, Parkinson's appears to be hereditary, passed down through families. Mutations in several genes have been linked to the disease, including the SNCA, LRRK2, and GBA genes. However, in most cases, Parkinson's appears to be caused by a combination of genetic and environmental factors. Environmental factors that may contribute to the development of Parkinson's include exposure to toxins such as pesticides and solvents, head injuries, and viral infections. Research has also suggested that lifestyle factors such as diet and exercise may play a role in the development of the disease.

Several studies also have suggested that changes in the composition and function of the gut microbiome may be associated with Parkinson's. Additionally, there is evidence that the gut-brain axis may contribute to the progression of Parkinson's. Research on the role of specific gut bacteria in Parkinson's is still in its early stages. While there is evidence of an association between gut dysbiosis and Parkinson's, the precise mechanisms by which specific bacteria influence the disease require further investigation. Understanding the role of gut bacteria in Parkinson's may have implications for the development of future treatments. Interventions aimed at restoring a healthier gut microbiome, known as microbiota-targeted therapies, could be explored in the future as potential supplementary treatments for Parkinson's.

Diagnostic Investigations

Diagnosing Parkinson's can be challenging, as there is no single test that can confirm the diagnosis. Instead, doctors need to rely on a combination of a person's medical history, a physical examination, and various tests to make a diagnosis.

Patients with a typical presentation

To diagnose patients with a typical presentation of Parkinson's, as shown in the Early Warning Signs of Parkinson's Disease chart (Fig. 2), doctors will ask questions about the person's symptoms, medical history, and family history. They will also perform a physical examination to look for signs of tremors, rigidity, and bradykinesia. This will include a full neurological examination to assess a person's reflexes, coordination, and balance.

Following these physical tests, patients are likely to have imaging tests. Magnetic resonance imaging (MRI) and computed tomography (CT) scans may be used to rule out other conditions that can cause similar symptoms. If the physical exam and MRI findings further support the diagnosis of Parkinson's, then the doctors will prescribe a trial of dopaminergic medication.

Fig. 2 – Early Warning Signs of Parkinson's Disease

There are different types of dopaminergic medications available, and the specific medication chosen depends on the condition being treated. A drug called levodopa is commonly prescribed for patients with Parkinson's. Other conditions may require dopamine agonists or other drugs that modulate dopamine activity.

Once the medication is selected, the patient begins taking it according to a prescribed dosage and schedule. The initial dose is typically low and gradually increased over time, allowing the patient's body to adjust and minimise potential side effects.

During the trial, the patient's response to the medication is closely monitored. Regular follow-up appointments are scheduled to assess the patient's symptoms, track any changes or improvements, and monitor potential side effects.

Depending on the patient's response and any observed side effects, the doctors may adjust the dosage or make changes to the medication regimen. The goal is to find the optimal dose that provides maximum symptom relief while minimizing side effects.

The length of the trial varies depending on the condition being treated and the specific medication used. In some cases, a trial may last several weeks to allow sufficient time for the medication to take effect and for the patient's response to be evaluated. Doctors will determine the appropriate duration based on the individual circumstances. At the end of the trial, the doctor will assess the overall efficacy of the medication and its impact on the patient's symptoms. They will consider factors such as symptom improvement, tolerability, and any adverse effects. This will allow the doctors to decide whether to continue the medication, adjust the dosage, or explore alternative treatment options.

Patients with atypical presentation

An atypical presentation of Parkinson's refers to a set of symptoms and clinical features that deviate from the classic or typical manifestations of the disease. These atypical presentations can pose diagnostic challenges because they may mimic other neurological conditions or have unique features that make them distinct from typical Parkinson's.

Here are a few examples of atypical presentations of Parkinson's:

Parkinson-Plus syndromes: This group of disorders exhibits Parkinson's-like symptoms but have additional features that distinguish them from typical Parkinson's. Examples include multiple system atrophy (MSA), progressive supranuclear palsy (PSP), and corticobasal degeneration (CBD), and dementia with Lewy bodies (DLB). These conditions often involve more rapid progression, early postural instability, autonomic dysfunction, (improper function of the autonomic nervous system), and additional neurological signs. These syndromes are collectively known as "Parkinson-plus syndromes," because they present with not only parkinsonian motor symptoms but also a broader range of symptoms that affect various parts of the nervous system. Each syndrome has its distinct clinical features, pathology, and progression, and they are generally more challenging to diagnose and manage compared to idiopathic Parkinson's.

Young-onset Parkinson's: While Parkinson's is commonly associated with older age, it can also occur in younger individuals. Young-onset Parkinson's refers to the development of the condition before the age of 50. It may present with different features compared to late-onset Parkinson's, including a higher prevalence of dystonia (involuntary muscle spasms and contractions), rapid progression, and a higher likelihood of genetic factors.

Drug-induced parkinsonism: Certain medicines, such as antipsychotics, antiemetics, and calcium channel blockers, can induce parkinsonian symptoms as a side effect. The presentation may be like typical Parkinson's but is reversible once the offending medication is discontinued.

Tremor-dominant Parkinson's: While tremor is a common feature of Parkinson's, some individuals may primarily present with a predominant tremor and minimal bradykinesia or rigidity. This tremor-dominant variant can be mistaken for essential tremor or other tremor disorders.

These are just a few examples of atypical presentations of Parkinson's. Diagnosing Parkinson's and its variants requires a comprehensive evaluation by a healthcare professional with expertise in movement disorders. To help distinguish Parkinson's from those listed above, additional tests can include the following:

Dopamine transporter scan (DaT scan)

A DaT scan is a type of brain imaging procedure used to assess the function of dopamine transporters in the brain. As described earlier, dopamine is a neurotransmitter that plays a crucial role in regulating movement and emotions. In Parkinson's, there is a gradual loss of dopamine-producing cells in a region of the brain called the substantia nigra. This loss of dopamine leads to the characteristic motor symptoms of the disease, such as tremors, rigidity, and difficulty with coordination.

A DaT scan involves injecting a radioactive tracer called a radiopharmaceutical into the bloodstream. This tracer binds to dopamine transporters in the brain, which are responsible for reabsorbing dopamine after its release. By using a special camera, the distribution and density of dopamine transporters can be visualized and measured. Areas with reduced dopamine transporter activity suggest a loss of dopamine-producing cells, indicating Parkinson's or other parkinsonian syndromes. The DaT scan is a valuable diagnostic tool as it helps differentiate Parkinson's from other conditions that may have similar symptoms. It can also assist in the early detection of Parkinson's before significant dopamine cell loss has occurred.

Genetic testing

Some genetic mutations have been associated with atypical parkinsonian syndromes. Genetic testing may be considered in certain cases, especially if there is a family history of Parkinson's or other neurological conditions. Genetic tests commonly used to diagnose Parkinson's include:

SNCA gene testing: Mutations in the SNCA gene have been associated with an increased risk of developing Parkinson's. Testing for mutations in this gene can help confirm a genetic cause in some cases.

LRRK2 gene testing: Mutations in the LRRK2 gene are another known genetic cause of Parkinson's. Testing for LRRK2 gene mutations may be performed, especially in cases with a family history of the disease or early-onset Parkinson's.

PINK1 and Parkin gene testing: Mutations in the PINK1 and Parkin genes are associated with a rare, early-onset form of Parkinson's. Genetic testing for mutations in these genes may be considered in cases with early-onset symptoms.

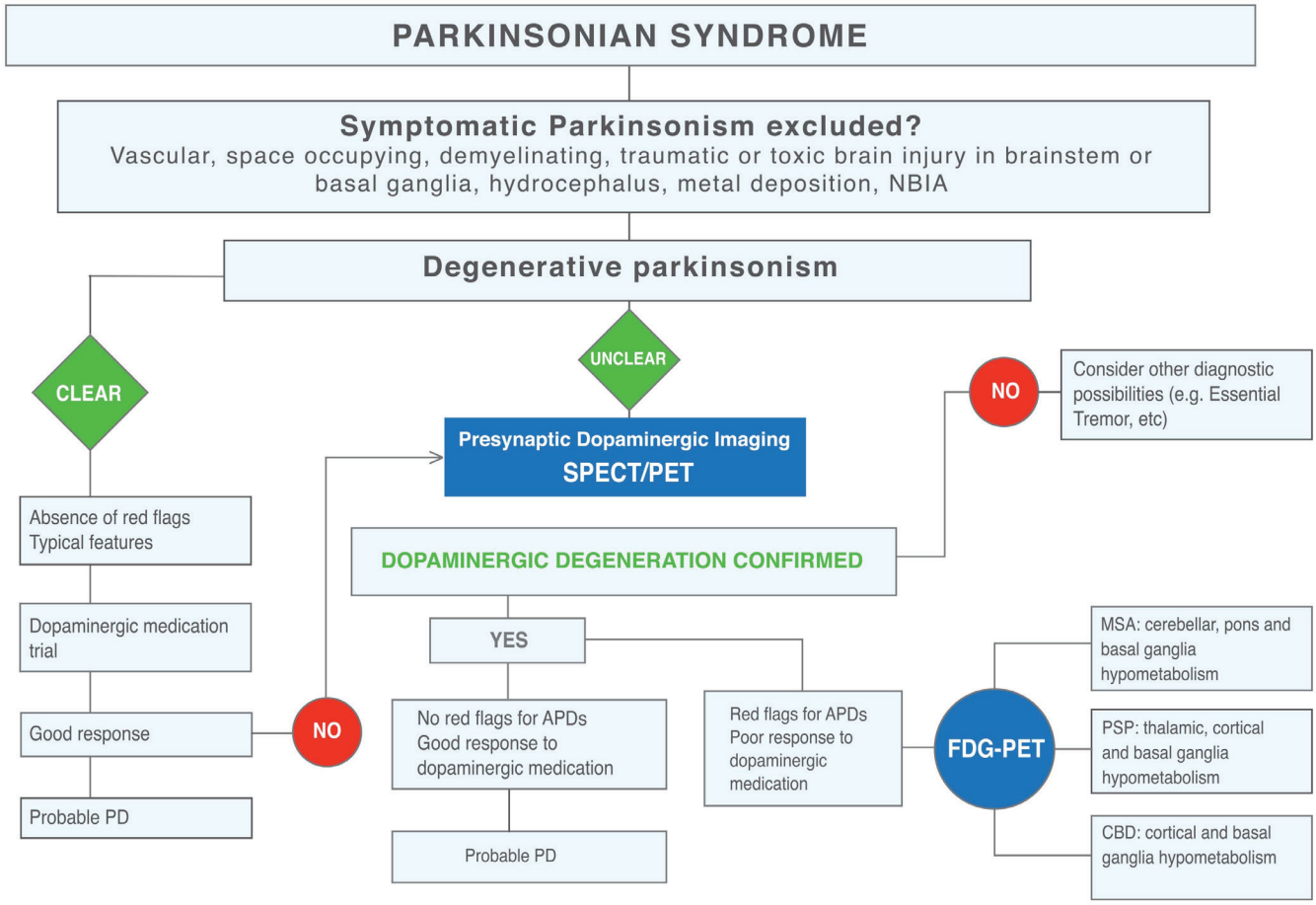
GBA gene testing: Mutations in the GBA gene have been linked to an increased risk of developing Parkinson's and are more commonly associated with a condition known as Gaucher's disease. Testing for GBA gene mutations may be conducted, particularly in cases with a family history of Parkinson's or certain clinical features.

It's important to note that genetic testing for Parkinson's is typically performed in specialised centres with expertise in movement disorders or genetic testing. The results of genetic testing are generally used in conjunction with clinical findings to assist in diagnosis and guide appropriate management strategies. Genetic testing may not be necessary for all individuals with Parkinson's.

Blood tests: Blood tests may be performed to evaluate for other conditions that can cause parkinsonian symptoms, such as metabolic disorders or thyroid dysfunction.

Diagnostic Criteria for Parkinson's

According to the Movement Disorder Society, the clinical diagnostic criteria for Parkinson's are based on the following[4]:



Once the diagnosis of Parkinson's has been established, the Hoehn and Yahr scale is a widely used rating system that assesses the severity and progression of symptoms[5]. It was developed by Drs. Melvin Yahr and Margaret Hoehn in 1967 and has since become a standard tool in clinical research and practice for evaluating Parkinson's.

The scale categorises Parkinson's into five stages based on the severity of motor symptoms and their impact on daily activities. These stages provide a general framework for understanding the progression of the disease but are not exhaustive in capturing all aspects of Parkinson's symptoms or non-motor features.

Here is a summary of the Hoehn and Yahr scale stages:

Stage 1: This stage represents the mildest form of Parkinson's. Symptoms are usually unilateral, affecting only one side of the body, and mild tremors or movement impairments may be present. However, these symptoms do not significantly interfere with daily activities.

Stage 2: Symptoms in this stage become bilateral, affecting both sides of the body. Balance issues and posture abnormalities may start to manifest, but individuals can still maintain independence with daily tasks.

Stage 3: This stage marks a moderate progression of the disease. Balance and coordination difficulties increase, and the individual may experience impaired reflexes and slower movements. Despite these challenges, individuals can still manage daily activities with some assistance.

Stage 4: Parkinson's symptoms become severe at this stage. The individual requires assistance to perform daily tasks and experiences significant limitations in movements. Walking may be possible but requires assistance, and rigidity and bradykinesia (slowness of movement) become prominent.

Stage 5: This final stage represents the most advanced and debilitating form of Parkinson's. The individual is typically wheelchair-bound or bedridden, with severe motor impairments. They may also experience hallucinations and delusions, and 24-hour care is usually required.

It is important to note that the Hoehn and Yahr scale primarily focuses on motor symptoms and may not fully capture non-motor symptoms such as cognitive impairment, mood changes, and autonomic dysfunction, which can also significantly impact a person's quality of life in Parkinson's.

Modern developments in diagnosing and prognosticating Parkinson's

There are many avenues being explored and researched in the detection and treatment of those diagnosed with Parkinson's, from fluid and tissue markers to new scans, e.g., retinal scans and the use of digital technologies to measure gait, eye movements, sleep etc. that can give added insight to signs that cannot easily be detected with basic clinical assessment. There are too many to go into detail in this publication; however, we have explored a number of these in previous SCORacle articles, which can be found here.

SCOR's Neurology Chief Medical Officer Professor Dennis Chan comments about how technology has the potential to revolutionise healthcare for people with conditions such as Parkinson's:

"The advent of commercially available wearable technologies, such as smartphones, smartwatches, and fitness trackers, provide a hitherto unavailable opportunity to capture digital measures of movement and behavior that are likely in time to supplement the traditional clinical assessment and potentially even replace legacy approaches. Such tech-based approaches have several significant advantages over current clinical practice. First, the provision of quantitative digital rather than analogue readouts facilitates diagnosis, disease tracking and response to therapeutic intervention while the acquisition of datasets encompassing different aspects of function is ideally suited to the implementation of machine learning algorithms to extract additional diagnostic features invisible to traditional analysis methods. Second, such approaches can capture a wider range of behaviors than is possible within the time- and space-limited restrictions of a clinic appointment and will deliver real life measures that are more ecologically valid than current assessments of mobility than are rooted in historical neurological practice. Finally, the option of remote sensing at home may potentially be more convenient for patients than visits to hospitals or clinics that may be geographically distant and hard to access."

However, Professor Chan also warns that these technological breakthroughs must be approached with caution.

"While digital approaches may overcome many of the limitations of current practice, any future implementation in routine clinical practice will need to address several key issues. As with any tech-based approach, care will need to be taken to mitigate against the risk of obsolescence associated with future hardware and software upgrades. Public-patient acceptability will need to be gauged, with a particular focus on issues of privacy preservation and management of personal data as well as on societal issues such as concerns about two-tier systems favoring individuals and communities with greater tech awareness and usage. All future digital tools will need careful evaluation in terms of identification of use cases, validation to assess fitness for purpose for each use case, and setup of appropriate regulatory and governance frameworks. In brief, while digital approaches may in time be transformative for PD diagnosis and management, their clinical deployment will require major infrastructural change and risk management."

Treatment and Prevention

While there is currently no cure for Parkinson's, there are several treatment options available to manage symptoms and improve the quality of life for those diagnosed with the condition.

Medication

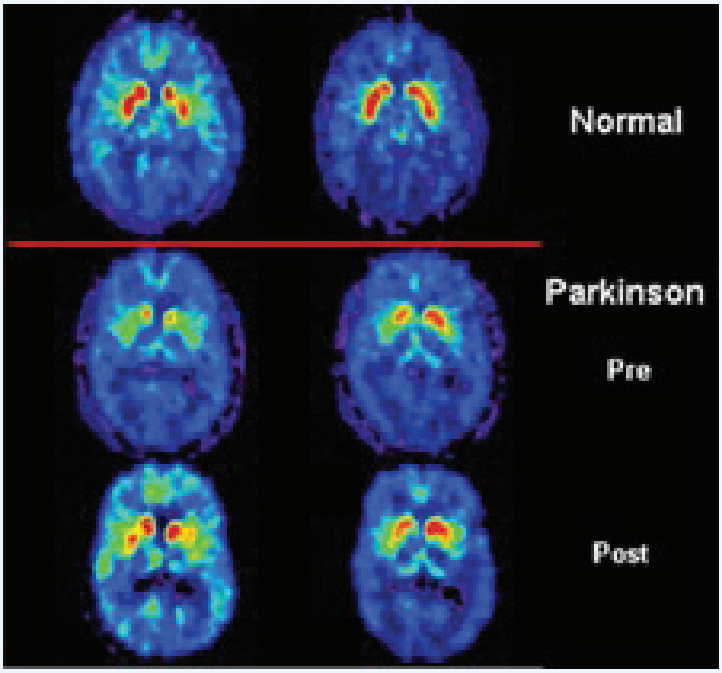
Medication is a cornerstone of Parkinson's management, aimed at alleviating the motor symptoms associated with the condition. There are several classes of drugs commonly prescribed for Parkinson's patients:

Levodopa: Levodopa is the most effective medication for managing the motor symptoms of Parkinson's. It is converted into dopamine, a neurotransmitter that is deficient in the brain of Parkinson's patients. Often combined with carbidopa (to enhance its absorption and reduce side effects), it helps improve muscle control, reduce rigidity, and alleviate bradykinesia.

Dopamine agonists: These drugs mimic the effects of dopamine in the brain and can be used alongside or as an alternative to levodopa. They can help improve motor symptoms and reduce the "off" periods (times when medication is less effective).

MAO-B inhibitors: Monoamine oxidase-B (MAO-B) inhibitors help prevent the breakdown of dopamine in the brain, thereby increasing its availability. This class of drugs can extend the duration of symptom relief provided by levodopa.

Figure 3 – Impact of Levodopa



In the top panel a normal scan, in the middle panel abnormalities in the putamen (red uptake in the figure) in a patient with Parkinson's, and in the lower panel a return to an almost normal scan following the introduction of levodopa.

Anticholinergic drugs: These medications can help control some of the tremors and muscle stiffness associated with Parkinson's.

Amantadine: This drug is often used to treat levodopa-induced dyskinesia (involuntary movements) and may have some effect in reducing other Parkinson's symptoms.

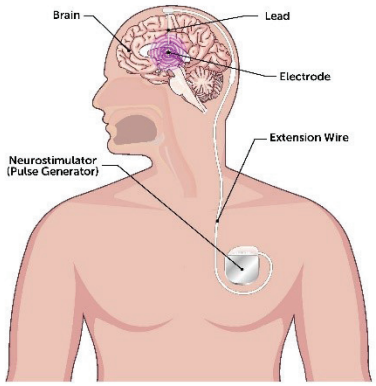
It's important to note that medication management in Parkinson's is highly individualised. The type and dosage of medications prescribed will vary from person to person, depending on their specific symptoms, the stage of their disease, and their overall health.

Surgery

Whilst medication is the primary source of treatment in the early phases of Parkinson's, in some cases, when medication alone is not sufficient to control symptoms or when individuals experience medication-related complications, surgical interventions may be considered. The two most common surgical options for Parkinson's are:

Deep Brain Stimulation (DBS): DBS involves the implantation of electrodes into specific regions of the brain. These electrodes are connected to a pacemaker-like device that delivers electrical impulses, modulating abnormal brain activity and alleviating motor symptoms. DBS can provide significant improvements in tremors, rigidity, and bradykinesia.

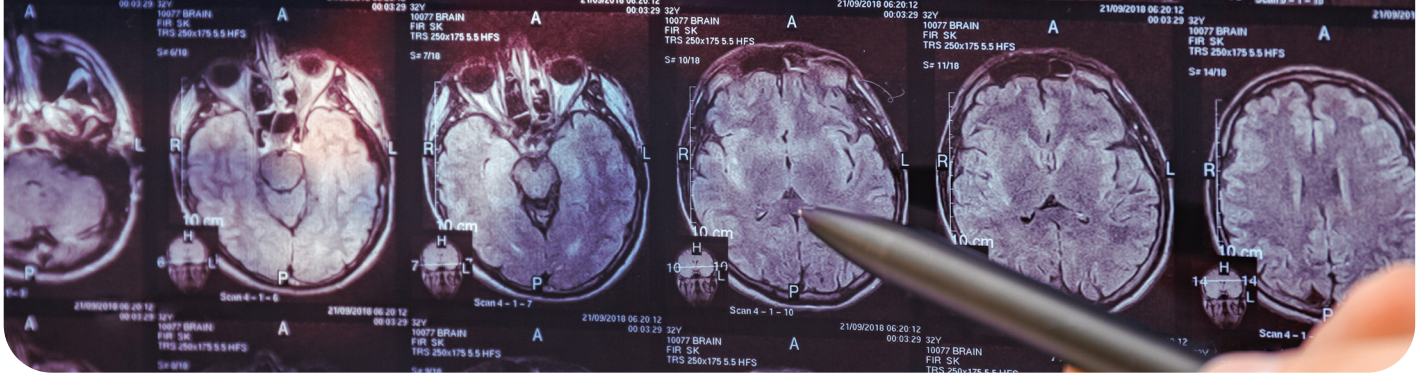
Deep Brain Stimulation (DBS)



Lesion surgery: In rare cases, surgery to create controlled lesions in the brain may be considered. This approach is less common and typically reserved for individuals who are not candidates for DBS.

Non-pharmacological interventions

While medication and surgery play critical roles in managing Parkinson's, non-pharmacological interventions are also essential for improving the overall quality of life for individuals with the condition. These interventions can be quite varied and again will depend on the individual circumstances of the patient and can include physiotherapy, occupational therapy, speech and swallowing therapy, supportive care, and exercise.



Prognosis

While Parkinson's is a complex condition with a significant impact on a patient's quality of life, the prognosis for individuals with the disease can vary significantly.

Motor symptoms in Parkinson's often start mildly but progress over time. Tremors, rigidity, bradykinesia (slowness of movement), and postural instability are hallmark features of the disease. While medications and various therapies can help manage these symptoms, they typically worsen as the disease advances.

Non-motor symptoms of Parkinson's can significantly impact an individual's quality of life as well. These include depression, anxiety, sleep disturbances, cognitive impairment, and autonomic dysfunction (occurs when the autonomic nervous system, which controls functions responsible for well-being and maintaining balance, does not regulate properly). The progression of these non-motor symptoms varies, with some individuals experiencing them early in the disease course, while others may develop them later.

Medication is essential in treating and coping with Parkinson's. However, they aren't without their side effects, and as the disease progresses, individuals may experience medication-related complications, such as motor fluctuations and dyskinesias (involuntary movements). These side effects can be challenging to manage and may require adjustments to treatment regimens.

A subset of individuals with Parkinson's may develop more severe cognitive impairment, which can progress to dementia. This condition, known as Parkinson's disease dementia (PDD) can significantly affect an individual's independence and daily functioning.

While Parkinson's is not typically considered a life-threatening condition itself, it can increase the risk of mortality. People with Parkinson's are more prone to complications such as falls, aspiration pneumonia, and infections due to impaired mobility and swallowing difficulties. Additionally, comorbid conditions often associated with Parkinson's, such as cardiovascular disease and diabetes, can contribute to increased mortality risk.

The prognosis for Parkinson's is highly variable among individuals. Some people may experience a relatively stable disease course for many years, while others may progress more rapidly. Many factors influence the rate of progression, including age at onset, genetic factors, and the presence of comorbidities.

Many individuals with Parkinson's have multiple chronic conditions, and the interplay between these conditions can affect both morbidity and mortality. Timely management of these comorbidities is crucial to improve overall prognosis.

Parkinson's is a complex and progressive disorder that presents a diverse range of symptoms and outcomes. To improve prognosis, a multidisciplinary approach that includes medication, surgery, rehabilitation, and support for both motor and non-motor symptoms is essential. Additionally, managing comorbid conditions and promoting a healthy lifestyle can help individuals with Parkinson's maintain the best possible quality of life while facing the challenges posed by the disease.

Parkinson-plus Syndromes

Parkinson-plus syndromes, also referred to as atypical parkinsonism or Parkinsonism-plus syndromes, are a group of neurodegenerative disorders that share some clinical similarities with Parkinson's but are distinguished by their atypical features and more aggressive disease progression. Unlike Parkinson's, which is primarily characterised by dopaminergic neuron degeneration and responsive to levodopa therapy, Parkinson-plus syndromes involve various types of neurodegeneration and typically respond poorly or only partially to dopaminergic medication.

Progressive supranuclear palsy (PSP)

PSP is a rare and progressive neurodegenerative disorder that primarily affects movement and balance. It has clinical similarities with Parkinson's and its more atypical features. PSP is characterized by the degeneration of specific brain regions, leading to a range of motor and non-motor symptoms.

Motor symptoms: The most prominent feature of PSP is the presence of various motor symptoms. These include:

- Supranuclear gaze palsy: PSP is known for its hallmark feature, which is difficulty in moving the eyes voluntarily. This leads to a distinctive and notable gaze impairment, particularly affecting vertical eye movements. This gaze palsy can result in frequent falls, as patients may struggle to look down when walking or may fall backward.
- Postural instability: Patients with PSP often experience severe postural instability, leading to frequent falls. Unlike Parkinson's, where balance issues tend to develop later in the disease course, postural instability is an early and pronounced symptom in PSP.
- Rigidity and bradykinesia: PSP patients typically experience muscle stiffness (rigidity) and slowness of movement (bradykinesia), resembling some aspects of Parkinson's.
- Speech and swallowing difficulties: Problems with speech and swallowing are common and can be challenging for both patients and caregivers.

Cognitive and non-motor symptoms: Many individuals with PSP develop cognitive problems, including difficulties with memory, executive functions, and reasoning. However, these cognitive changes are generally less pronounced than in some other neurodegenerative conditions like Alzheimer's disease. Patients may also experience mood disturbances such as depression and irritability.

Diagnosing PSP can be challenging due to its rarity and the overlap of some symptoms with other neurodegenerative conditions. Clinical evaluation by a neurologist with expertise in movement disorders is crucial. Brain imaging such as MRI or PET scans can help identify specific structural changes in the brain that are characteristic of PSP. A confirmed diagnosis often requires post-mortem examination of the brain tissue to identify the accumulation of abnormal tau protein aggregates, which is a hallmark of PSP.

Like Parkinson's, there is no cure for PSP, and current treatments aim at managing its symptoms and improving the patient's quality of life. Medications, physiotherapy, and speech therapy can help alleviate some of the motor and non-motor symptoms. Caregiver support and safety measures are essential due to the high risk of falls and postural instability. In general, life expectancy for individuals with PSP is estimated to be around five to 10 years from the onset of symptoms, although there is a considerable range of variability.

Multiple system atrophy (MSA):

MSA is a rare and progressive neurodegenerative disorder that affects both the autonomic nervous system (responsible for regulating involuntary bodily functions) and the motor system (responsible for controlling movement). MSA

usually occurs in adults between the ages of 50-60 years but can be seen in both younger and older adults. There is no evidence that suggests the condition is hereditary. MSA is typically classified into two main subtypes:

MSA with predominant parkinsonism (MSA-P): This subtype exhibits prominent motor symptoms resembling those seen in Parkinson's, such as bradykinesia, rigidity, and postural instability.

MSA with predominant cerebellar ataxia (MSA-C): This subtype is characterised by a loss of coordination, speech difficulties, and other cerebellar symptoms, often leading to an unsteady and staggering gait.

Both subtypes of MSA can exhibit motor symptoms, but the specific presentation may differ. Common motor symptoms include muscle rigidity, bradykinesia, and tremors in MSA-P or ataxia, dysarthria (difficulty with speech), and incoordination in MSA-C.

MSA is known for its prominent autonomic nervous system involvement, leading to various symptoms like orthostatic hypotension (a drop in blood pressure upon standing), urinary dysfunction, constipation, sexual dysfunction, and sweating abnormalities. Many individuals with MSA experience non-motor symptoms, including sleep disturbances, respiratory problems, cognitive changes, and mood disorders.

Dysarthria and dysphagia (difficulty swallowing) are common in MSA and can significantly impact daily life. MSA is characterised by the accumulation of abnormal protein deposits in the brain, primarily alpha-synuclein. These protein aggregates, known as glial cytoplasmic inclusions, are found in various regions of the brain and contribute to neurodegeneration. The specific distribution of these protein deposits in MSA is different from that in Parkinson's, which is why the clinical features of MSA differ significantly.

Diagnosing MSA can be challenging due to its rarity and the overlap of some symptoms with other neurodegenerative conditions. Clinical evaluation, including a detailed medical history and neurological examination, is essential for diagnosis. Brain imaging such as MRI can reveal characteristic changes in the brain and help support the diagnosis. A definitive diagnosis often requires post-mortem examination of brain tissue to identify the presence of glial cytoplasmic inclusions.

Treatment and prognosis

There is currently no cure for MSA, and treatment focuses on managing the individual's symptoms and improving their quality of life. Medications and therapies may be used to address specific symptoms, such as orthostatic hypotension, movement difficulties, and autonomic dysfunction. Supportive care and therapy, including physical and speech therapy, are essential to help manage symptoms and improve functioning.

It's important to note that MSA is a diverse condition, and individuals can experience different clinical courses. The two main subtypes, MSA-P and MSA-C, may have slightly different clinical features and progression rates.

In general, MSA is considered a more aggressive and rapidly progressive disorder compared to some other neurodegenerative conditions. Life expectancy for individuals with MSA is often shorter compared to the general population, but the exact duration can vary widely. On average, life expectancy after the diagnosis of MSA is estimated to be around six to 10 years, but individual experience varies.

Several factors can influence life expectancy in MSA:

- Age of onset: Individuals with MSA who develop symptoms at a younger age may experience a more extended disease course than those with late-onset symptoms.
- Comorbid conditions: The presence of other medical conditions can affect life expectancy. Individuals with MSA may be at an increased risk of complications such as aspiration

pneumonia, falls, and cardiovascular issues.

- Rate of disease progression: The speed at which the disease progresses can vary among individuals. Some may have a more aggressive course, while others may progress more slowly.
- Response to treatment: Currently, there is no cure for MSA, and treatment focuses on managing the individual's symptoms and improving their quality of life. The effectiveness of treatments and the individual's response to them can influence their life expectancy.

Corticobasal syndrome (CBS):

CBS is a rare and progressive neurological disorder characterised by a combination of motor and cognitive symptoms. It is considered one of the atypical parkinsonian syndromes and is often classified as a variant of corticobasal degeneration (CBD), a rare neurodegenerative condition. CBS can be challenging to diagnose and manage due to its complexity and the diversity of its clinical features.

As the name suggests, CBS primarily presents with motor abnormalities that affect one side of the body more than the other (asymmetric). These motor symptoms include:

- Bradykinesia
- Rigidity
- Myoclonus (sudden, involuntary muscle jerks)
- Apraxia (difficulty performing purposeful and coordinated movements)
- Dystonia (iInvoluntary muscle contractions that lead to abnormal postures)
- Cognitive and behavioral symptoms

In addition to motor deficits, CBS often includes cognitive and behavioral changes, which may include:

- Cognitive impairment: Individuals may experience difficulties with memory, executive functions, and language.
- Alien limb phenomenon: This is a strange sensation where a limb appears to move on its own or against the person's intention.
- Behavioral changes: Mood disturbances, including apathy, depression, and impulsivity, may be present.

The asymmetric nature of the motor symptoms, combined with the cognitive and behavioral changes, can be a hallmark feature of CBS.

The underlying cause of CBS is not fully understood, but it is related to the accumulation of abnormal tau protein in the brain. This tau protein aggregation contributes to neurodegeneration and the characteristic clinical features of the condition. Diagnosing CBS can be challenging due to its rarity and the variability of its clinical presentation. It often requires a thorough clinical evaluation by a neurologist with expertise in movement disorders.

Brain imaging, such as MRI or PET scans, can reveal specific patterns of brain atrophy that are consistent with CBS. Like many of these conditions, a definitive diagnosis can only be confirmed by post-mortem examination of the brain tissue to confirm the presence of tau protein aggregates. Therefore, the clinical features are very important for medics to label the diagnosis.

There is no cure for CBS, and treatment primarily focuses on managing the individual's symptoms and improving their quality of life. Medications and therapies may be used to address specific symptoms, including motor symptoms and cognitive or behavioral changes. Physiotherapy and occupational therapy can help individuals manage their motor deficits and adapt to daily activities. Like MSA, several factors can influence life expectancy in CBS, such as clinical presentation, age at onset, comorbidities, and response to therapies. So, whilst it can be variable, people with CBS tend to live between six to eight years.

Dementia with Lewy bodies (DLB):

DLB is a complex and progressive neurodegenerative disorder that falls under the umbrella of Lewy body diseases, which also include Parkinson's and Parkinson's dementia. DLB is characterised by the presence of abnormal protein deposits, known as Lewy bodies, in the brain. These Lewy bodies are composed of alpha-synuclein protein and are associated with cognitive, motor, and psychiatric symptoms. DLB is the second most common degenerative dementia after Alzheimer's disease. Key features of DLB include:

- Cognitive impairment: DLB is primarily characterised by cognitive decline, which often includes memory problems, executive dysfunction, and visuospatial impairments.
- Visual hallucinations: Visual hallucinations are a common early feature of DLB, often involving seeing people, animals, or objects that are not present. These hallucinations can be distressing for the individual.
- Motor symptoms: Individuals with DLB may experience motor symptoms similar to those seen in Parkinson's, such as bradykinesia, rigidity, and tremors.
- Fluctuating alertness: Fluctuations in alertness and attention are a hallmark feature of DLB. Individuals may have periods of lucidity followed by confusion or drowsiness.
- REM Sleep Behavior Disorder (RBD): RBD is characterized by acting out one's dreams during rapid eye movement (REM) sleep, often leading to physical movements, vocalisations, and potential injuries.
- Autonomic dysfunction: DLB can also affect the autonomic nervous system, leading to symptoms such as orthostatic hypotension (low blood pressure upon standing), urinary incontinence, and constipation.

Much like PSP, MSP and CBS, life expectancy for individuals with DLB can vary, and it is influenced by several of the same factors, such as age of onset, comorbid conditions and rate of progression and response to treatment. There is no cure for DLB, and symptomatic treatments, including medications and supportive care, may help manage some of the symptoms and improve the individual's quality of life. The average life expectancy of DLB is five to eight years after the initial diagnosis. However, there have been instances with some people living up to 20 years after their diagnosis.

Critical Illness Definitions for Parkinson's

Critical Illness definitions for Parkinson's are not straightforward nor permanent. In the UK, for example, Parkinson's has been included in the ABI model wording since it was first introduced in the 1999 Statement of Best Practice for Critical Illness and most recently, ABI Guide to Minimum Standards. The model wordings have evolved since then, and below is the latest definition:

2014 and 2018 - Parkinson's disease [before age x] - *resulting in permanent symptoms.* A definite diagnosis of Parkinson's disease [before age x] by a Consultant Neurologist. There must be permanent clinical impairment of motor function with associated tremor and muscle rigidity.

For the above definition the following are not covered:
- Parkinsonian syndromes/Parkinsonism

Whilst the ABI definitions required a minimum age to be covered for Parkinson's, many insurers do not age-bar their definitions. For those that did select an age, this was typically age 55.

In this definition, Parkinsonian syndromes were excluded under the Parkinson's definition. However, they are now an insured condition in their own right, and whilst the ABI do not offer a model wording in the Guide to Minimum Standards, a typical definition in the market is as seen in the callout to the right.

This definition also includes Parkinsonism-dementia-amyotrophic lateral sclerosis complex (PD-ALS). This is not typically considered a Parkinson's-plus syndrome. PD-ALS is a relatively rare neurodegenerative disorder characterised by a combination of symptoms from Parkinson's (parkinsonism), dementia, and amyotrophic lateral sclerosis (ALS). This complex condition is sometimes referred to as PDC (Parkinsonism-dementia complex).

Parkinson plus syndrome - resulting in permanent symptoms

A definite diagnosis by a consultant neurologist or consultant geriatrician of one of the following Parkinson plus syndromes:

- Corticobasal ganglionic degeneration
- Diffuse Lewy body disease
- Multiple system atrophy
- Parkinsonism-dementia-amyotrophic lateral sclerosis complex
- Progresive suparnuclear palsy.

There must also be permanent clinical impairment of at least one of the following:

- motor function; or
- eye movement disorder; or
- postural instability; or
- dementia.

It is essential to understand that PD-ALS is a distinct and unique syndrome with its own set of clinical features, rather than being categorized as a Parkinson's-plus syndrome. The underlying causes and mechanisms of these conditions may also differ significantly.

As Parkinson's is a long-term, chronic condition, the impact it can have on an individual is significant, particularly if they are diagnosed in younger ages. Therefore, in recent years in the UK, some insurers offer an uplift in their policies that will increase the amount payable for a successful claim. Typically, for those diagnosed under the age of 55, if this is a particular feature of their product, an insurer will pay out an additional amount of 100% of the sum assured, up to a maximum of £200,000.

Underwriting Assessment

Regretfully, offering terms to an applicant for CI or IP cover who already has Parkinson's is not possible. However, there are instances where life cover is obtainable.

Parkinson's is a long-term chronic condition, and so there are certain factors where cover can be offered. However, this is likely to be subject to a significant rating. At the time of writing this paper, SCOR's underwriting manuals in the UK, for example, had the rating profile shown as Table 1 when considering terms for a life policy for an applicant with Parkinson's. Other areas, such as the US, as shown in Table 2, slightly differ from the UK case. The rating profiles between the two markets are very similar. However, the subtle differences are primarily down to the age groupings and how these are broken down.

Table 1: Parkinson's rating profile (UK)

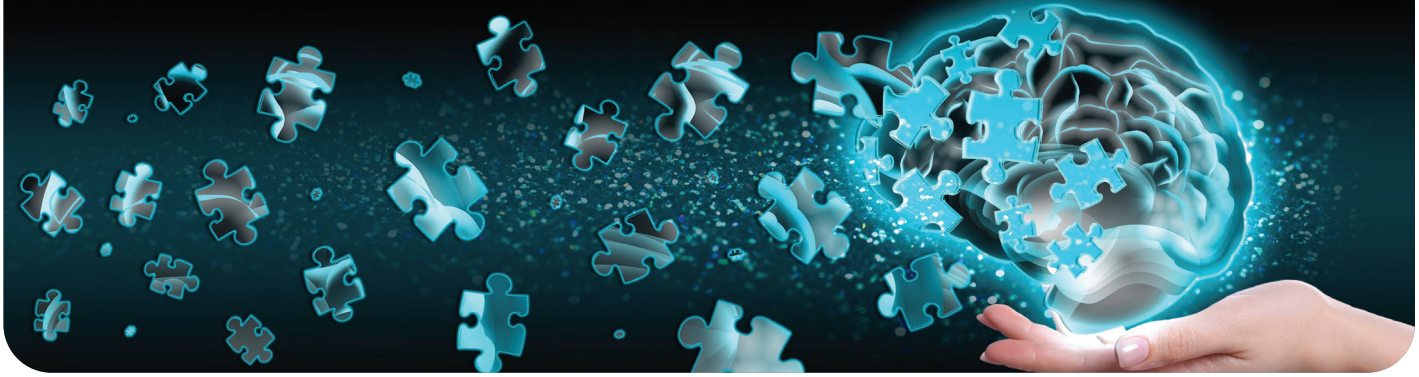
No underlying disease, idiopathic Morbus Parkinson		
Rapidly progressive	○	Regret no offer
Unstable	○	Postpone
Stable		
Mild, stage I or II on Hoehn and Yahr scale ?		
Age < 55	○	+100
Age ≥ 55	○	+50
Moderate, stage III or IV on Hoehn and Yahr scale ?		
Age < 55	○	+200
Age ≥ 55	○	+100
Severe, stage V on Hoehn and Yahr scale ?		
Age < 55	○	+300
Age ≥ 55	○	+200
Secondary, e.g. due to drugs, atherosclerosis, multisystem disorders, encephalitis, trauma	○	Regret no offer

Table 2: Parkinson's rating profile (USA)

	LIFE	WP	ADB
Idiopathic Parkinson's Disease			
Clinical stage (based on Hoehn-Yarr scale)			
Stage I			
Applicant under age 50	+100	Decline	Decline
Applicant age 50 - 59	+75	Decline	2x
Applicant age 60 or over	+50	Decline	+0
Stage II			
Applicant under age 50	+150	Decline	Decline
Applicant age 50 - 59	+100	Decline	Decline
Applicant age 60 or over	+50	Decline	+0
Stage III			
Applicant under age 50	+200	Decline	Decline
Applicant age 50 - 59	+150	Decline	Decline
Applicant age 60 or over	+100	Decline	Decline
Stage IV or Stage V	Decline	Decline	Decline
Secondary Parkinsonism	RMD; usually RFC and add ratings for residual symptoms above	RMD; usually RFC and add ratings for residual symptoms above	RMD; usually RFC and add ratings for residual symptoms above
Family history of Parkinson's Disease only	Rate as Family history of Parkinson's disease	Rate as Family history of Parkinson's disease	Rate as Family history of Parkinson's disease
Additional rating factors			
History of mild / moderate depression	Add rating for Depression	Decline	Add rating for Depression
History of or presence of severe depression or dementia	RMD; usually Decline	Decline	Decline

The face amount, term, and age of the applicant will need to be considered. However, the Hoehn and Yahr scale is an important factor when considering whether terms are appropriate.

Understandably, the milder the symptoms, the better the terms that can be offered. It is important to remember that whilst Parkinson's can be mild in the early phases, it is a long-term, progressive condition that can also cause a number of comorbidities which in turn may have an impact on mortality. Therefore, careful consideration to all the factors mentioned are key. A CMO opinion is also advised in many circumstances.



Claims Philosophy

The three fundamental features of Parkinson's are bradykinesia, rigidity of movement, and tremor at rest. A definite diagnosis of Parkinson's can only really be made at autopsy as there are no specific tests that will confirm the diagnosis during the claimant's lifetime. Although a DaTSCAN will identify claimants who have a loss of dopamine, which leads to tremor, slowness of movement, muscle stiffness, and balance problems, this will not confirm the diagnosis of Parkinson's in isolation. Where there is any doubt over the diagnosis, it may be useful to request a copy of the DaTSCAN.

Our understanding is that a diagnosis of Parkinson's is based on clinical evidence of bradykinesia, rigidity of movement, and / or tremor at rest, and by ruling out any other causes of the claimant's symptoms. Postural instability is often included as a fourth or supporting feature. Therefore, SCOR will consider that the 'definite diagnosis' aspect of the definition has been met where the diagnosis is confirmed by a consultant neurologist and has been based on these criteria. However, if a DaTSCAN has been performed and has not identified a loss of dopamine, the basis of the diagnosis should be questioned with the consultant.

With regard to the requirements for permanent clinical impairment of motor function, the 2006 and 2011 ABI definitions both require three areas of motor function impairment to be fulfilled.
The 2014 and 2018 definitions remove the requirement for postural instability as this is often a late development within the disease process, and it was felt to be too arduous to insist that claims were not assessed until this was a permanent feature of the disease. But the requirement of the other two areas of impairment of motor function remain. However, providing the diagnosis was made in line with the above guidelines, we will consider a claim based on clinical evidence of bradykinesia and just one of the other permanent clinical impairments of motor function.

As stated previously, Parkinson's is a clinical diagnosis, and there are no tests to confirm it. As Parkinsonian syndrome can be indicative of other disorders (e.g., head injury, encephalitis, carbon monoxide poisoning, or cerebral arteriosclerosis), we would want to ensure that alternative diagnoses have been ruled out. The syndrome is purely the collection of symptoms that can be found in a patient with Parkinson's. The intention of the 2011, 2014, and 2018 definitions is simply to clarify the situation regarding Parkinsonian syndrome.

Summary

Parkinson's, affecting millions of people worldwide living with day-to-day struggles, can also pose underwriting and claim assessment challenges to insurers, due to its complex and long-term nature, as described in this article. It is imperative that underwriting and claims professionals have a deep and fundamental understanding of the disease, stay current with the updated information, and have access to credible advisors with first-class expertise.

This paper is not designed to determine what underwriting outcome should be reached or whether a claim is valid or not. It is to be used as a guide to help with the assessment process and where to seek evidence that may help an underwriter or claims assessor come to a decision. It is important to remember that the guidance in the paper is accurate at the time of distribution.

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This article was written by:

Paul Blyth
Head of Underwriting and
Claims Proposition
SCOR UK and Ireland

Please feel free to visit us at scor.com

SCOR SE
5 avenue Kléber - 75795 PARIS Cedex 16
France
scorglobalpc@scor.com

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