Idiopathic Parkinson’s Disease (IPD)
Objectives

• Definition
• Aetiology
• Pathology
• Clinical features
• Investigations
• Differential diagnoses
• Management
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Defining IPD

• Named after James Parkinson who published 'An Essay on the Shaking Palsy' in 1817, which established Parkinson’s as a recognised medical condition.

• He studied at the London Hospital Medical College, qualifying as a surgeon in 1784 when he was 29.
Defining IPD

-Degenerative, progressive disease affecting the basal ganglia.

Movement disorders:

1) Akinetic-rigid syndromes
   - Slowed movement.
   - Increased tone.
   -> IPD, drug-induced parkinsonism, multiple systems atrophy, progressive supranuclear palsy.

2) Dyskinesias
   - Added, uncontrollable movements.
   -> Essential tremor, chorea, myoclonus, tics.
Defining IPD

- Parkinsonism
- Progressive supranuclear palsy
- Lewy body dementia
- Vascular parkinsonism
- Drug-induced parkinsonism
- Multiple systems atrophy
Defining IPD

• Annual incidence- 0.2/1000.
• Prevalence- 1/500 (127 000 people in the UK).
• Tends to affect ≥50 years.
• 1/20 is under the age of 20 years.
• Incidence and prevalence increase with age.
• Equal sex incidence.
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Aetiology

• Unknown aetiology.

Several theories:

- Nicotine- IPD is less prevalent in smokers than lifelong abstainers.

- MPTP- caused severe parkinsonism in young drug abusers.

- Genetic factors- clustering of early-onset PD in some families.
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Pathology

Basal ganglia:

• Group of nuclei in the brain situated at the base of the forebrain (striatum, globus pallidus, substantia nigra [SN], nucleus accumbens, subthalamic nucleus).

• Associated with voluntary motor control, procedural learning, eye movements, cognitive and emotional functions.
Pathology

- Depletion of pigmented dopaminergic neurons in SN
- Reduced dopaminergic output from SN
- Inclusion bodies (Lewy bodies) develop in nigral cells
- Degeneration in other basal ganglia nuclei

Neurons in subthalamic nucleus become more active than usual in inhibiting activation of the cortex

Bradykinesia
Pathology
Objectives

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Clinical features

- Bradykinesia
- Postural instability
- Resting Tremor
- Rigidity
Clinical features

Resting Tremor

- Pill-rolling at rest
- Present: -At rest -When distracted
- Diminished: -On action
- Arms/legs/feet/jaw/tongue
Clinical features

- Rigidity
  - Cogwheel rigidity (upper limbs)
  - Lead pipe rigidity (legs)
  - Increased tone when opposite arm moves actively
  - Flexed posture
Clinical features

- Bradykinesia
  - Difficulty initiating movement
  - Reduced spontaneous blinking
  - Poor rapid fine movements (fingers)
  - Facial immobility (hypomimia)
Clinical features

- Loss of postural reflexes
- Difficulty making turns
- Retropulsion
Clinical features

Gait:

i) Stooping
ii) Slow to initiate walking
iii) Shortened stride
iv) Rapid small steps (shuffling)
v) Tendency to run (festinating)
vi) Reduced arm swing
vii) Impaired balance on turning

• Falls common in later stages.
• Parkinson’s gait
Clinical features

• Speech
- Monotone → tremulous, slurring dysarthria.
- Soft, rapid, indistinct.

• Cognitive
- Cognitive impairment in 1/3 of patients (loss of executive functions including planning/decision-making/controlling emotions).
- Depression.
Clinical features

• GI/others

- Constipation/heartburn/dribbling/dysphagia/weight loss.
- Greasy skin.
- Micrographia.
Clinical features

- Tremor
- Masklike facies
- Arms flexed at elbows and wrists
- Rigidity
- Hips and knees slightly flexed
- Stooped posture
- Short shuffling steps
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Investigations

• Clinical diagnosis.

• <50 years: Test for Wilson’s disease.

• CT head scan if:
  - Pyramidal/cerebellar/autonomic involvement.
  - Diagnosis is in doubt.
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Differential diagnoses

- Multiple systems atrophy
- Progressive supranuclear palsy
- Lewy body dementia
- Drug-induced parkinsonism
- Vascular parkinsonism
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<td>-Nocturnal wanderings +/- confusion</td>
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<td>Drug-induced parkinsonism</td>
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<td>-Taking dopamine antagonists/lithium</td>
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<td>-Lower limbs affected</td>
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<td></td>
<td>&gt;upper limbs</td>
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Management

Medical:

- Levodopa + peripheral decarboxylase inhibitor (E.g. Carbidopa, Benserazide).

  - Levodopa: Precursor of dopamine stimulates remaining neurons to produce more dopamine.

  - Decarboxylase inhibitor: Prevents peripheral decarboxylation to dopamine and peripheral SE’s.
Management

- Side effects of levodopa:
  - N&V
  - Confusion
  - Visual hallucinations
  - Delusions
  - Chorea

LT effects:
- Levodopa-induced involuntary movements.
- Gradually ineffective after several years.
- Episodes of immobility (freezing).

THEREFORE → drugs are avoided until **clinically necessary** (significant disability) because of delayed unwanted effects.
Management

- Other medical treatment options:
  - Dopamine receptor agonists (Bromocriptine/Cabergoline).
  - Amantadine.
  - Rivastigmine (cognitive changes).
  - Antioxidant compounds (Vitamins C & E- possible neuroprotective agents).
Management

• Surgical  
  - Stereotactic thalamotomy - temporary improvement of symptoms.

• Physiotherapy  
  - Reduces rigidity & corrects abnormal posture.

• Speech therapy  
  - Dysarthria/dysphonia.

• Neuropsychiatric  
  - SSRI’s for depression.
Management

Natural history:

• Slowly progressive (10-15 years).

• Bradykinesia & tremor worsen.

• Late deterioration despite Levodopa Rx occurs in 1/3-1/2 of patients after 3-5 years. This includes the ‘on-off’ phenomenon.

• Patient’s c/o limb & joint discomfort.
Management

• Prognosis

-Partly related to age of onset e.g. if symptoms start in middle life → disease likely to shorten lifespan (complications of immobility & tendency to fall).

Onset >70 years of age unlikely to shorten life/become severe.
Summary

- IPD is the most common cause of parkinsonism.
- Degenerative, progressive disease affecting the basal ganglia.
- Classical features include tremor, rigidity and bradykinesia.
- Mainstay of treatment is with levodopa & a PDI. Treatment is delayed until clinically necessary because of unwanted delayed effects of levodopa.
- Multi-disciplinary approach to management.
Thank you! 😊

Questions?