

PARKINSON'S DISEASE AND PARKINSONISM

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OUTLINE

- Covering:-
 - Why this is an important area of Medical and Psychiatric care
 - The variety of Clinical Presentation
 - How to ask the right questions
 - How to do an appropriate examination
 - What investigations may be helpful
 - Routine and other management options
 - Adverse effects of Management
 - Special areas of interest such as drug induced parkinsonism, Lewy Body Disease, 'Vascular', PSP etc etc etc

CONTEXT

- Probability
 - Diseases causing parkinsonism
 - Drugs treating and contributing to parkinsonism
- Prevalence
 - PD (ICD-10= G20) = 2nd most prevalent Neurodegenerative disease after AD
 - 0.3% total population, 1% >60yr, 4% >80yrs
- Burden of disease and burden of treatment
- Available pharmaceutical treatments are numerous and readily available
- The Obvious cases are Easy!

CLINICAL PRESENTATION

Usual Features

- Remember the Triad (2 out of 4)
 - Tremor
 - Bradykinesia (slowing)
 - Rigidity
 - Postural instability
- Note, can and often will be accompanied by
 - Neuropsychiatric syndromes
 - Autonomic effects



Support Features

- Unilateral onset.
- Rest tremor present.
- Progressive disorder.
- Persistent asymmetry affecting the side of onset most.
- Excellent response (70-100%) to L-dopa.
- Severe L-dopa-induced chorea.
- L-dopa response for five years or more.
- Clinical course of ten years or more.
- Hyposmia.
- Visual hallucinations



TREMOR

- 70% will have tremor at presentation
- Typical tremor =
 - At Rest
 - Usually asymmetrical (a unilateral rest tremor in the foot is almost pathognomonic)
 - Relatively slow, regular, more coarse than Essential Tremor
 - Rarely constant
 - Progressive

BRADYKINESIA

- Subtle at first
- May start with fine motor skills (doing up buttons, tying shoelaces etc)
- 'psychological inertia'
- Affects rapid alternating movements
- Can be dependent on emotion ('SHOUT Fire" - **kinesia paradoxical**)
- Difficulty with initiation of and maintaining smooth movement (producing some curious yet classical phenomena and 'tricky treatments'!



RIGIDITY

- Muscle stiffness
- Can be almost cramp like and uncomfortable
- Often Axial (legs / neck / back)

5.1.1



VIDEO



PARKINSON'S DISEASE SUSPECT AN ALTERNATIVE DIAGNOSIS IF

- Rapidly progressive course
- Lack of dopaminergic response
- Early postural instability
- Cerebellar signs
- Early autonomic features
- Pyramidal signs
- Rapidly progressing or early dementia
- Supranuclear gaze palsy, slowed saccades
- Early falls
- Symmetry of motor manifestations
- Lack of tremor

UPDRS

- Research pedigree but a useful tool to organise examination findings.
- 4 Parts
 - I = Mentation, Behaviour and Mood (Enquiry)
 - II = Activities of Daily living (Enquiry)
 - III = Motor Examination (Observation)
 - IV = Complications of therapy (Observation)

MENTATION, BEHAVIOUR AND MOOD

- Intellectual impairment
- Thought disorder
 - Nil
 - Vivid dreams
 - Benign hallucinations, retained insight
 - Frequent, delusional, with reduced insight
 - Persistent, psychosis, unable to care for self
- Depression
- Motivation/initiative

ACTIVITIES OF DAILY LIVING

- Speech
- Salivation
- Swallowing
- Handwriting
- Cutting Food and Handling Utensils
- Dressing
- Hygiene
- Turning in Bed
- Falls
- Freezing
- Walking
- Tremor
- Sensory complaints

MOTOR EXAMINATION

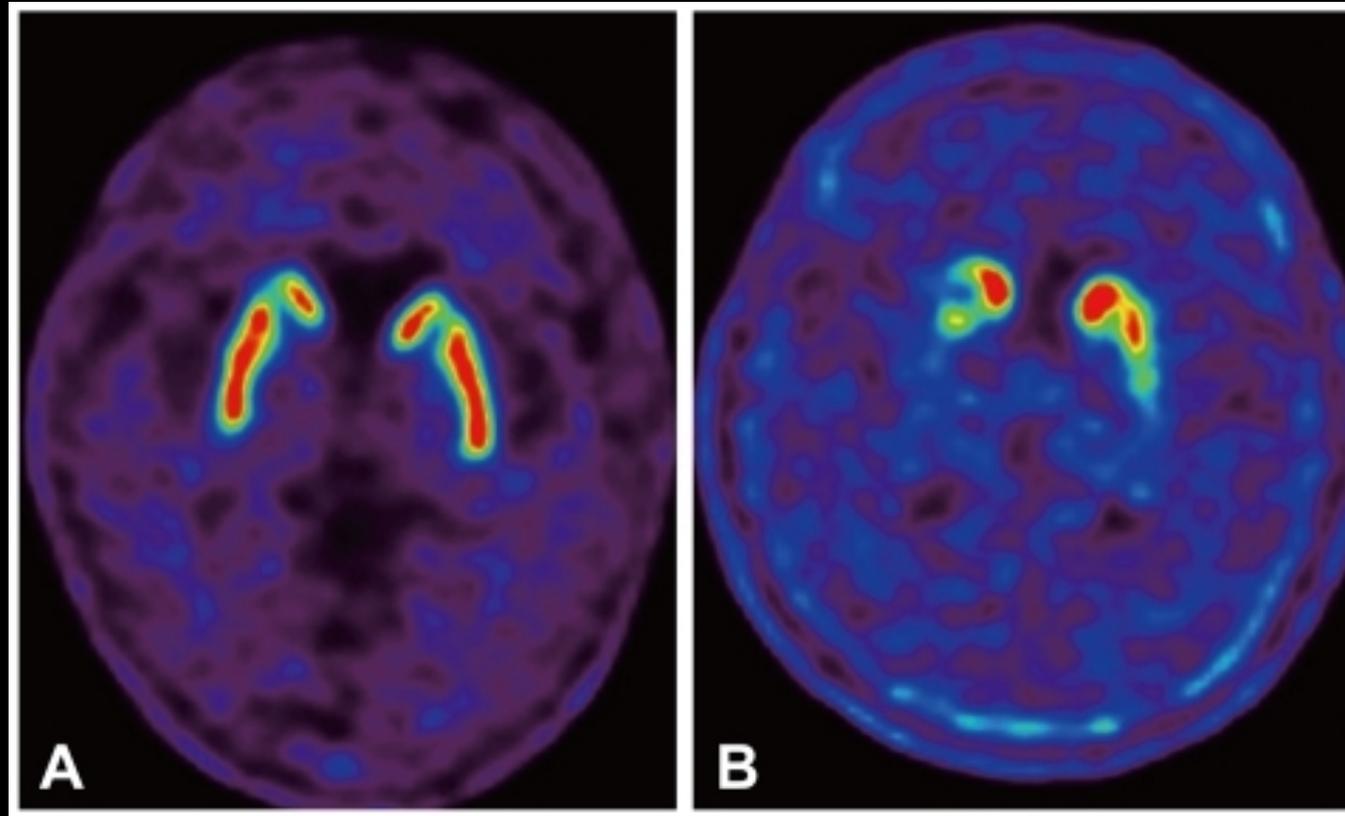
- Speech
- Facial Expression
- Tremor at rest
- Action or postural tremor
- Rigidity
- Finger Taps
- Hand movements
- Rapid alternating movements
Hands
- Leg Agility
- Arising from Chair
- Posture
- Gait
- Postural stability
- Body Bradykinesia

17.1



INVESTIGATIONS

- CT or MRI brain scan:
 - For patients who fail to respond to therapeutic doses of L-dopa (at least 600 mg/day) administered for 12 weeks.
 - MRI scanning is needed to exclude rare secondary causes (eg, supratentorial tumours and normal pressure hydrocephalus) and extensive subcortical vascular pathology.^[1]
 - Functional MRI and CT imaging are useful research tools. Blood flow changes monitored by these methods and correlated with functional disability are providing useful clues as to the structural abnormalities which cause Parkinsonism and PD.^{[10][7]}
- PET scanning with fluorodopa can localise dopamine deficiency in the basal ganglia, while autonomic tests and sphincter electromyography may support a diagnosis of multiple system atrophy.
- Genetic testing may be required - eg, Huntington's gene. Fewer than 5% of all PD cases are caused by known single-gene mutations.^[7]
- Olfactory testing to help differentiate PD from other Parkinsonian disorders.^[7]
- Further investigations for young-onset or atypical disease may include measurement of ceruloplasmin levels (Wilson's disease) and syphilis serology.



I-FP-CIT PET imaging in two DIP patients. DAT uptake was normal and symmetric in the bilateral striatum in a pure DIP patient (A), whereas it decreased severely in the right striatum in a patient who was diagnosed with PD unmasked by DRBAs (B). DAT: dopamine transporter, DIP: drug-induced parkinsonism, DRBA: dopamine receptor blocking agents, PD: Parkinson's disease

COMPLICATIONS OF THERAPY

- Confusion, hallucinations, delusions, agitation, psychosis.
 - Nausea, dizziness, headache, orthostatic hypotension
 - Motor fluctuations
- Dopamine dysregulation syndrome – a cyclical mood disorder with hypomania and possible manic psychosis
- Impulse control disorders including hypersexuality and pathologic gambling may occur
- Patients on dopamine agonists may develop impulse control problems
- The adverse effects of dopamine agonist are similar to levodopa. Peripheral edema is more common with agonists
- Elderly and demented patients are more susceptible to psychiatric side effects
- “sleep attacks”

ROUTINE MANAGEMENT

- Dopaminergic agents as in Sinamet / Madopar in various preparations
- Primary Dopa agonists
 - Ropinerole
 - Pramipexole
 - Pergolide
 - Bromocryptine
- Anticholinergics
- Adjuvant agents
 - COMPT inhibitors



ADVERSE EVENTS AND LATE DISEASE PHENOMENON

PARKINSONISM

- 'Secondary' parkinsonism, ICD-10 = G21
 - Drug induced
 - Trauma
 - Vascular
 - NPH
- Other conditions PD Plus
 - Lewy Body Disease
 - Progressive Supranuclear Palsy - SRO
 - Multisystem Atrophy

DRUG INDUCED PARKINSONISM

- Suggestive features:-
 - Use of potentially causative medications prior to onset
 - 7% of people with parkinsonism have a h/y of relevant drug exposure
 - 50% occur <1 month
- Acute / rapid onset of levodopa unresponsive motor pD
- Symmetrical
- Improves after medication discontinuation / change
- Presence of Tardive Dyskinesia
- Normal DaT binding on PET
- Reduced raclopride binding on PET

MANAGEMENT OF PARKINSONISM

- Stop the offending agent?
 - Usually effective ? but potentially risky!
- Swap to less 'toxic medications'
- Anticholinergics including trihexyphenidyl, benztropine, amantadine, and levodopa have been empirically tested for their ability to relieve symptoms of DIP, but this has produced no clear evidence of their effects in DIP patients