NEUROIMAGING in Parkinsonian Syndromes
(Focus on Structural Techniques: CT and MRI)

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• Primary Parkinsonism
  – Idiopathic Parkinson’s Disease
  – Atypical Parkinsonian Syndromes:
    • Multiple System Atrophy
    • Progressive Supranuclear Palsy
    • Cortico-Basal Degeneration

• Secondary Parkinsonism
  – Brain Tumors
  – Cerebrovascular disease
  – Other Etiologies
PRIMARY PARKINSONISM
Parkinson’s Disease

- Structural Neuroimaging (brain CT scan, MRI) in Parkinson’s disease is NORMAL.
- Neuroimaging helps differentiating idiopathic PD from other primary & secondary Parkinsonian syndromes.
- Only High-Field MRI (7 Tesla) may detect subtle abnormalities (research purposes)
Primary Atypical Parkinsonism (1a)
Multiple System Atrophy - Parkinsonian type (MSA-P)

MAIN CLINICAL FEATURES
- Poor response to Levodopa
- Rapidly Progressive; Symmetric
- Autonomic System Dysfunction
  - Genito-Urinary Dysfunction
    (incontinence, erectile dysfunction in males)
  - Orthostatic Hypotension
- Dystonia (mainly facial)
- Dysphagia; Dysarthria

MRI findings (T2-w images)
1) Hypointense signal in posterior Putamen
   (Reduced signal looks ‘Dark’; due to iron deposition)
2) External Hyperintense rim
   (Increased signal looks ‘White’; due to gliosis i.e. cerebral scar)
Primary Atypical Parkinsonism (1b)

Multiple System Atrophy - Cerebellar type (*MSA-C*)

**MAIN CLINICAL FEATURES**

- Poor response to Levodopa
- Rapidly Progressive; Symmetric
- Signs of Cerebellar Dysfunction
  *(ataxia, dysarthria, kinetic tremor, etc.)*
- Autonomic System Dysfunction
  - Genito-Urinary
  - Orthostatic Hypotension
  - Dysphagia

**MRI findings** *(T2-w images)*

1) *Hypointense signal in posterior Putamen + Hyperintense rim*

2) *Atrophy of Pons, Cerebellum, MCP +*

3) *Pons ‘Cross’ sign*
REMINDER:
Putaminal Hypointensity without external hyperintense rim may occur in healthy elderly subjects (75 - 80+)

van Es et al., Neurobiology of Aging, 2008
Primary Atypical Parkinsonism (2)
Progressive Supranuclear Palsy (**PSP**)  

**MAIN CLINICAL FEATURES**
- Early unbalance with frequent Falls  
- Vertical gaze palsy  
- Frontal-lobe Dementia (apathy, perseverence, disinhibition)  
- Poor response to Levodopa  
- Rapidly Progressive; Symmetric  
- Axial involvement (marked neck rigidity)  
- early Dysarthria and Dysphagia  

**MRI findings**  
*Best images in sagittal view*
1) Midbrain Atrophy  
(so-called ‘colibri’ or ‘penguin’ sign)
2) Frontal lobe cortical atrophy  
(including cingulate cortex and corpus callosum)
Primary Atypical Parkinsonism (2)  
Cortico-Basal Degeneration (CBD)

MAIN CLINICAL FEATURES
- Markedly Asymmetric
- Limbs Apraxia (inability to perform complex movements)
- Asymmetric Dystonic posture (mainly affected hand)
- Poor response to Levodopa
- Rapidly Progressive
- Early Dysarthria and Dysphagia
- Overlaps with PSP in advanced stages: frontal-lobe dementia, vertical gaze palsy, unbalance with falls.

MRI findings
- Markedly Asymmetric Fronto-Parietal Cortical Atrophy
SECONDARY PARKINSONISM
Secondary Parkinsonisms
Brain Tumors

Brain CT
Brain MRI T1-weighted
Secondary Parkinsonisms
Vascular disease

A) Diffuse white matter damage by chronic small vessels disease (hypertension, diabetes). Very common.

CLINICAL
- Insidious onset
- Symmetrical, lower-body: gait disturbance with freezing, broad-based, falls
- Frontal-lobe cognitive dysfunction
- Poor response to Levodopa

B) Acute damage to Substantia Nigra or Striatum. Uncommon.

CLINICAL
- Subacute onset
- Unilateral parkinsonism
- Usually with dystonia, pyramidal signs.
- Response to Levodopa (limited to SN lesion)
Secondary Parkinsonisms
Chronic Subdural Haematoma

Chronic subdural haematomas presenting with Parkinsonian signs.
Peppard RF, Byrne E, Nye D.

Chronic subdural haematomas and Parkinsonian syndromes.
Wiest RG, Burgunder JM, Krauss JK.
Department of Neurology, Inselspital, University of Berne, Switzerland.

Parkinsonism secondary to subdural haematoma.
Gelabert-Gonzalez M, Serramito-Garcia R, Aran-Echabe E.
Department of Surgery, School of Medicine, University of Santiago de Compostela, San Francisco 1, 15705, Santiago de Compostela, Spain. miguel.gelabert@usc.es
Secondary Parkinsonisms
Normal Pressure Hydrocephalus

CLINICAL TRIAD:
Insidious development of
- Gait disturbance (apraxic, short-step, freezing)
- Urinary Incontinence
- Frontal-lobe cognitive dysfunction

Oftern misdiagnosed as Alzheimer’s or Parkinson’s!

Important: it is reversible after shunt placement!

Imaging findings
1) Abnormal Enlargement of Ventrices, with frontal horns rounding ($\geq 30\%$ of skull diameter)
2) Normal (or flat at tened) Cortical sulci
3) Brain MRI: periventricular hyperintensities
4) No visible evidence of obstruction to CSF flow
Secondary Parkinsonisms
Basal Ganglia Calcinosi (Fahr’s Disease)
Secondary Parkinsonisms
Exposure to Manganese

ACUTE EXPOSURE to HIGH CONCENTRATIONS
MANGANISM:
- severe PKS unresponsive to levodopa
- psychiatric and behavioral disturbances
- Dystonic gait (‘cock-walk’) with falls

SOURCES:
- Occupational: welding; mining; smelting
- Drugs of abuse (Ephedrone)

IMAGING:
- Globus Pallidus hyperintensity at T1-w MRI

CHRONIC EXPOSURE to LOW CONCENTRATIONS
- Risk factor of Parkinson’s disease
- Damage also of substantia nigra and striatum
- Responsive to Levodopa
Secondary PKS in sub-Saharan Africa: What is the role of Pesticides?

Chronic Exposure to PARAQUAT and MANCOZEB represents an environmental risk-factor for Parkinson’s disease.

CONCLUSIONS

• Neuroimaging may help clinician in the differential diagnosis of patients with Parkinsonism.

• It is supportive but *never* substitutes a good clinical assessment (history and examination).

• Brain Computed Tomography should be performed in all patients to exclude secondary causes.

• Brain Magnetic Resonance Imaging may help in early diagnosis of Primary Atypical PKS (MSA, PSP, CBD).
THANKS for the attention

QUESTIONS??