

Case 4 : Parkinson disease



## Learning issues:

- 1. Structures and functions of basal ganglia
- 2. Role of basal ganglia in controlling fine movements
- 3. Neurotransmitters (particularly dopamine) responsible the normal function of basal ganglia and control of fine movements
- 4. Pathology of Parkinson disease
- 5. Interpretation of patient's clinical symptoms and signs
- 6. Biochemical and molecular mechanisms underlying the development of Parkinson disease
- 7. Pharmacology of drugs used in treatment of Parkinson disease





Extra information

### Key information and Presenting problems:

- Male, 65 years old, retired security officer
- tremor of his right arm and hand
- stiffness in his shoulder region (rigidity)
- slow in his movements (bradykinesia) for last 6 months



#### **History:**

- It increase when people comment about his tremor and when he is stressful situation.
- No pain on rest or with moving his shoulder
- Difficult to turn his body in bed or move from side to side
- He feels unsteady
- He walks in short steps
- Disturbance in his sleep for the last 4-5 years
- He was admitted to hospital when he was 12 years old for appendectomy
- He has no history of any medical problems in his family.
- NO Smoking
- NO Medication and Allergy



- Cogwheel resistance: <a href="http://youtu.be/8xxe2WWWoY">http://youtu.be/8xxe2WWWoY</a>
- Parkinsonian gait: <a href="http://youtu.be/j86omOwx0Hk">http://youtu.be/j86omOwx0Hk</a>

Disease of muscle

Myopathy



#### **Clinical examination**

-Decreased facial expression (masked face)

-speaks in low tone voice

-he rests his right arm on his body while sitting ,his right arm and hand show <u>tremor</u>.
-No evidence of <u>hypothyroidism</u>.

#### Motor Power

**Motor power examination** 

Gait He stands in a slow movement and his body

is leaning forward .he walks in slow and

short steps (festinating gait)

cogwheel like resistance (initial resistance

followed by a release of movement)

On flexion of his right elbow There is

#### Sensory system examination

**Normal** 

#### Musculoskeletal system

**Normal** 

No evidence of myopathy

#### vital signs

#### **Cranial nerves**

all are Normal

#### CVS and Respiratory Examinations

Normal

#### • Investigation:

- o investigation are <u>not</u> needed for diagnosis of Parkinson disease
- It is diagnosed through: <u>History + Clinical</u> examinations

#### Management:

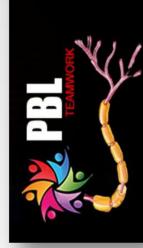
- Levodopa coupled with a peripheral decarboxylase inhibitor (PDI) tablets.
- The doctor advises him to revisit in five weeks

#### Prognosis:

- ✓ Five weeks later ,he feels much better. His arm and hand tremor are much less than used to be .
- ✓ Over the next 5-6 months Mr Saad shows some improvement . He much less tremor and his walking is more stable. He is able to do work that needs fine movements such as unbutton his shirt or tie his shoe laces .

#### Diagnosis:

✓ Parkinson disease



## Explanation of the treatment:

LEVODOPA (L-DOPA)

Peripheral decarboxylase inhibitor (PDI)

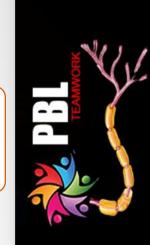
The precursor to Dopamine

It will be decarboxylated to DOPAMINE in the brain and periphery.

N.B: the doctor didn't give Dopamine directly, because Dopamine doesn't cross <u>blood</u> brain barrier  Without PDI: Since L-DOPA will be decarboxylated to DOPAMINE

DOMPAMINE in the blood causes side effects (nausea, Vomiting)

 Therefore, PDI is given to reduce the incidence of nausea and vomiting and inhibit peripheral decarboxylation.



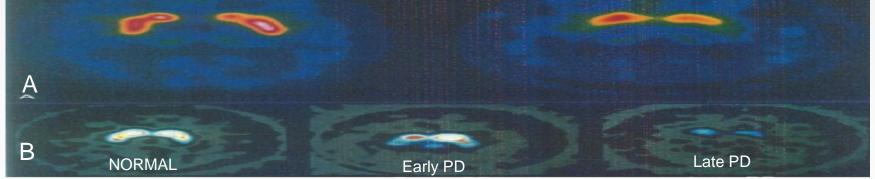
#### ❖ Progress 1:

The doctor asks Saad if he wants to be part of the doctor's RESEARCH. He explains to him
the value of the research for possible new discovers that could help in treating patients
with PARKINSON disease. Saad agrees to join the research project.

#### ❖ Progress 2:

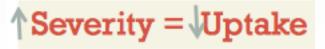
- Research objects could help in:
- ✓ Understand the progression of the disease
- ✓ Mechanism of the medication
- √ Role of therapy in delaying disease progression
- The images show a <u>reduction</u> in the uptake of 18F DOPA, and beta-CIT spect uptake by the putamen of patients wit parkinson disease (PD) compared to normal. Saad's images confirm that he has an early PD. N.B: these investigations only for the research requirements

NORMAL PD patient



**A:** 18F DOPA PET uptake in the Putamen is reduced in patients with PD compared with normal controls.

**B:** Reduction in Beta- CIT spect uptake in the Putamen correlate with the severity of PD



#### **PARKINSON DISEASE:**

- ☐ Is a degenerative disorder of the CNS. The motor symptoms of Parkinson's disease result from the death of dopaminergic neurons of the substantia nigra or their projection to the striatum.
- □ the cause of this cell death is unknown (idiopathic), but several factors appear to play role, including specific genetic mutations and/or environmental triggers (Drugs affecting neuron e.g. Dopamine antagonists)
- **□** Symptoms:
- √ Masked face
- √ Bradykinesia
- ✓ Festinating gait
- ✓ Tremor
- ✓ Low tone voice
- ✓ Rigidity
- ✓ Cogwheel resistance



# Juestions

Q6

Q7

Q8



Can the doctor use Dopamine instead of Levodopa?

Q2

Q3

Q4

**Q5** What is the scientific basis behind these investigations were in the research?

No, because Dopamine can not cross BBB.

Assessment of the functional status of the different parts of the basal ganglia

Research objects could help in:

If levodopa is given without PDI, what will happen?

Levodopa will decarboxylate to Dopamine, causing nausea and vomiting. And small amounts of Levodopa will reach the brain.

✓ Understand the progression of the disease

✓ Mechanism of the medication

✓ Role of therapy in delaying disease progression

What is the location lesion of

What is the appropriate diagnosis

method for Parkinson disease?

**History + Clinical examination.** 

parkinson disease?

Substantia nigra.

What is the type of rigidity in the elbow?

Describe the festinating (parkisonian) gait? he walks in slow and short steps + leaning

Cogwheel resistance

forward



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