AN OLD GENTLEMAN WITH REPEATED FALL

Cheng Jen Ngai

Chairperson: Dr. CS Leung
DEFINITION OF FALL

- An event which results in a person coming to rest inadvertently on the ground or other lower level

other than a consequence of the following:
- sustaining a violent blow
- loss of consciousness
- sudden onset of paralysis, as in a stroke, or an epileptic seizure
FAL IS COMMON IN ELDERLY!

Local data:
- Prevalence rate of falls (>=1) in subjects > 70 years old = 18%
  *a retrospective cross sectional study of the community elderly (Ho SC et al 1996)*

- In another community study, 50% experience multiple falls every year, 20% who fall experience another fall within 6 months
CAUSES

Intrinsic factors:
- Gait and balance disorders
- Physiological -
  - decreased visual field and acuity, accommodation, adaptation to darkness
  - decreased auditory-vestibular function
  - decreased CNS function with slower reaction time, poorer position sense, impaired righting reflexes.
  - age related gait
- Pathological
  - Stroke
  - Parkinsonism
  - Normal pressure hydrocephalus
  - Cerebellar diseases
  - Apraxic gait in dementia
  - Peripheral neuropathy
  - Orthopedic problems
- Cognitive impairment
  - confusion
  - impaired judgment and insight
  - distraction
  - agitation
  - lack of awareness
- Medications

Extrinsic factors:
- Furniture: unstable or of inappropriate height
- Beds/ toilets: inappropriate height
- Uneven stairs and inadequate or no railing
- Steps and kerbs at entrance
- Cracked and uneven sidewalks
- Obstacles on the floor, eg: cords, wires, frayed carpets
- Inadequate lighting
- Slippery floors and bath tubs
- Electrical cords
- Improper shoes
ASSESSMENT AND MANAGEMENT

- *Multi-disciplinary multi-factorial risk factor assessment and intervention*
- Included a team with physicians, nurses, physiotherapist, occupational therapists, prosthetics and orthotics, podiatrist and social workers
- To delineate all the contributory intrinsic and extrinsic risk factors
- To formulate the management plan
- Has the strongest evidence in clinical studies that it will prevent further fall in old people
INTRODUCTION

- An elderly person who lives in the community who has experienced repeated fall
BACKGROUND INFORMATION

- Mr. Leung
- 77 years old
- Retired sailor
- Education level: primary school
- Lived with wife (65 years old) and a son at a flat of 3/F with no lift landing
- 2 other children lived apart
- Pre-morbid walked with stick
- ADLI (bathing, toileting independent, could go out by himself with a stick)
Past medical history

- Hypertension
- CA prostate with TURP in 2003

- Follow up at GOPD
- FU QEH surgical annually for the CA prostate
**Drug History**

- Atenolol 25mg daily
- Stemetil PRN

(Given by GOPD)

- No over-the-counter medication
- No traditional Chinese medicine
Admitted to CMC on 10/5/2012 for fall
Felt sudden non-rotational dizziness while walking alone at the street, associated with bilateral lower limb weakness and then fell on buttock
No preceding symptoms of palpitation, chest pain or headache
No LOC
Failed to sit up after fall
Mild bilateral elbow and chin abrasion after landing
No head injury
No hip pain
Called ambulance by passer-by
**Further History — Mobility Problem**

History from patient’s relatives:
- Patient had *mobility problems* for 2 years
- Blamed the *poor vision* this year affected patient’s walking
- Also complained on and off *non-rotational dizziness*
- Initially he could walk unaided, but required a stick since 9/2011
- Could not maintain the balance, patient described it as ‘左右摇摆,失去平衡’
- Repeated minor falls this year at home, usually on initiation of gait or turning
- There were no major injury or admission, exact number of falls could not be remembered
- Wife worried about the worsening mobility of patient, so spent more time accompanied him when going out
- As patient lived at the flat without lift service, patient didn’t tend to go out very often
FURTHER HISTORY –
COGNITIVE AND BEHAVIORAL PROBLEM

- Noticed *irritable mood* since 1/2012, easily became angry during conversation and quarreled with wife frequently
- Wife said patient sometimes didn’t act like the usual him, *got agitated* easily with *violent behavior*
- Once attempted to use the bare hand to hit his wife during conflicts
- Patient also feared that he was theft by others
- Self muttering occasionally, relatives could not understand the reason
- Easy forgetful, e.g:
  - asked questions repeatedly despite answers had been given
  - left water tap turned on/ lamp switched on unattended
  - Forgot to flush toilet after use
- No obvious disorientation in time, place, person
- No depressed mood noticed
**Further History – Cognitive and Behavioral Problem**

- Visited GOPD on 26/1/2012 and complained about the unusual behavior
- Referred to Occupational therapist for MMSE which was done on 2/3/2012: 19/30 (cut off: 22)
- Diagnosed as **dementia with BPSD** *(behavioral and psychological symptoms of dementia)* at GOPD and referred to Psychosomatic clinic
FURTHER HISTORY – SCREENING OF COMMON GERIATRIC PROBLEMS

- No incontinence of urine
- No swallowing problem
- No headache
- No chest pain
SUMMARY OF HISTORY

Mobility problem with repeated minor falls since about 5/2010

Agitated mood and unusual behavior since 1/2012

Stick walking since 9/2011

Admitted to medical for fall in 5/2012
**Physical Examination**

- **GCS:** 15/15
- Fully orientated, respond to questions and command, but *rather slow*
- Mood calm
- Supine BP:128/79, erect BP:137/75
- Body weight: 62kg, Body height: 1.7m, Body Mass Index: 21.6
- Vision: right: 20/40, left eye: 20/50
- Hearing: satisfactory
- Speech: clear
- Cardiovascular system NAD
- Abdominal system NAD
- Respiratory systems NAD
PHYSICAL EXAMINATION: CENTRAL NERVOUS SYSTEM

- Mask face +ve
- Bradykinesia +ve
- Minimal rigidity
- No resting tremor
- Power full
- Reflex normal
- Sensation normal
- No cerebellar sign
VIDEO

- A video showing patient’s gait
VIDEO

- A video showing patient’s ophthalmoplegia
FURTHER HISTORY – VISUAL PROBLEM

- Patient noticed *bilateral eyes blurred vision* since 5/2011
- He blamed the blur vision made walking down stair more difficult
- He complained about his blurred vision at GOPD and was referred to eye for assessment
The patient has ophthalmoplegia

Levels of lesion that can cause ophthalmoplegia:

1. The **orbit** of the eye, including mechanical restrictions of eye movement, as in Graves disease
2. The **ocular muscle**
3. The **neuromuscular junction**, as in myasthenia gravis
4. The **relevant cranial nerves**: ie: oculomotor, trochlear, and abducens palsies
5. **The brainstem nuclei of cranial nerves**, as in certain patterns of brainstem stroke
6. **White matter tracts connecting cranial nuclei**, as in internuclear ophthalmoplegia
7. **Dorsal midbrain structures**
8. Certain parts of the **cerebral cortex** (including the frontal eye fields), as in stroke
VIDEO

- A video showing patient vestibulo-ocular reflex
VESTIBULO-OCULAR REFLEX

- The *vestibulo-ocular reflex (VOR)* is a reflex eye movement that stabilizes images on the retina during head movement by producing an eye movement in the direction opposite to head movement, thus preserving the image on the center of the visual field.

- The vertical VOR can be activated by manually flexing and extending the neck while the patient views a distant target. If the extent of the vertical eye movement limitation is improved with this maneuver, then the lesion is *supranuclear* in origin.
VESTIBULO-OCULAR REFLEX
Bell phenomenon

- The *Bell phenomenon* consists of upward eye deviation behind closed lids. This can be assessed clinically by holding the eyelid partially open and instructing the patient to try forcefully closing the eye.
- The upward movement of the eye is present in the majority of the population, and is a defensive mechanism.
- If the extent of the vertical eye movement limitation is improved with this maneuver, then the lesion is *supranuclear* in origin.
SUMMARY OF PROBLEMS

- Repeated fall with poor balance
- Parkisonism feature of mask face and bradykinesia
- Cognitive impairment with irritable mood
- Ophthalmoplegia with positive vestibulo-ocular reflex, suggestive of supra-nuclear lesion
DIFFERENTIAL DIAGNOSIS

1. Parkinson’s disease
2. Parkinsonism plus disease
   eg: Progressive supranuclear palsy
3. Lewy body dementia
4. Myasthenia gravis
5. Multi-infarct dementia
6. Normal pressure hydrocephalus
# Investigation

<table>
<thead>
<tr>
<th></th>
<th>Result</th>
<th>Reference range</th>
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<tbody>
<tr>
<td>Haemoglobin</td>
<td>12.2</td>
<td>13.4-17.1</td>
</tr>
<tr>
<td>ALT</td>
<td>88</td>
<td>35-128</td>
</tr>
<tr>
<td>Creatinine</td>
<td>91</td>
<td>55-103</td>
</tr>
<tr>
<td>Calcium</td>
<td>2.21</td>
<td>2.07-2.37</td>
</tr>
<tr>
<td>Inorganic phosphate</td>
<td>1.07</td>
<td>0.83-1.43</td>
</tr>
<tr>
<td>Vitamin B12</td>
<td>290</td>
<td>133-675</td>
</tr>
<tr>
<td>Folate</td>
<td>18.9</td>
<td>&gt;6.8</td>
</tr>
<tr>
<td>TSH</td>
<td>1.5</td>
<td>0.17-4.37</td>
</tr>
<tr>
<td>Anti-AchR</td>
<td>0.33</td>
<td>&lt;0.45</td>
</tr>
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</table>
• CT brain: no focal lesion, no dilated ventricles
• CT orbit: no abnormality detected
MRI

Mainly for detecting any brainstem lesion:

- Mild periventricular T2 hyperintensity seen, scattered non-specific deep white hyperintense foci seen
- AP diameter of midbrain is about 17mm
- AP diameter of pon 23mm
MOBILITY AND FUNCTIONAL ASSESSMENT

- **Physiotherapist:**
  Walk with stick under supervision
  EMS:9/20
  BBS:26/56

- **Occupational Therapist:**
  BI:75/100
  ADL up to toileting
  MMSE:20/30 (cut off 22/30)
### MMSE:

**Medical History & Diagnosis:**

<table>
<thead>
<tr>
<th>Date:</th>
<th>Therapist:</th>
</tr>
</thead>
<tbody>
<tr>
<td>15.5.</td>
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</tr>
</tbody>
</table>

**MMSE**

1. **Orientation**
   - Time: year / season / month / date / day of week
     - 0 - 5
   - Place: district / region / hospital / block / ward or floor
     - 0 - 5

2. **Registration**
   - Name: apple, train, newspaper
     - 0 - 3

3. **Attention & Calculation**
   - Serial seven (93, 86, 79, 72, 65) or
     - 0 - 5
   - Digit-span backward “42731”
     - 1

4. **Language**
   - Name a watch and a pencil
     - 0 - 2
   - Repeat a sentence
     - 0 - 1
   - Follow 3-stage command
     - 0 - 3
   - Follow written command
     - 0 - 1
   - Say or write a sentence
     - 0 - 1
   - Copy intersecting pentagons
     - 0 - 1

5. **Recall**
   - Recall apple, train, newspaper
     - 0 - 3

<table>
<thead>
<tr>
<th>Total Score</th>
<th>30</th>
<th>10</th>
<th>/30</th>
<th>/30</th>
<th>/30</th>
</tr>
</thead>
</table>

**Cut-off Score for Dementia**

- 22 < education ≥ 24 months
- ≤ 20: 6 months < education < 24 months ≤ 18: education ≤ 6 months

**Comments:**

- Age:

- Residual address:

Last Revision Date: Jun/2010
Likely Diagnosis:

Progressive supranuclear palsy (PSP)
What is progressive supranuclear palsy?

- Steele-Richardson-Olszewski syndrome
- Also recognized as atypical parkinsonian syndrome (or Parkinson-plus disorder)
- A sporadic *neurodegenerative* disease that diagnosed clinically
- Characteristics include progressive supranuclear ophthalmoplegia, gait disorder and postural instability, dysarthria, dysphagia, rigidity, and frontal cognitive disturbance
- *Supranuclear* in this context refers to a lesion that is situated *above the ocular motor nuclei*, thus sparing the ocular motor nuclei, nerve fascicles, and neuromuscular juntional and extraocular muscles.
EPIDEMIOLOGY

- PSP is uncommon but not rare!
- Most common degenerative forms of atypical parkinsonism
- The prevalence in the United Kingdom is 6.4 per 100,000
- The mean age of onset for PSP is approximately 62 years. Virtually no cases of PSP have been reported in patients younger than age 40
- The median interval between onset and diagnosis is 3 years (range, 0.5-9 years)

Nath U, Ben-Shlomo Y, Thomson RG, et al. The prevalence of progressive supranuclear palsy (Steele-Richardson-Olszewski syndrome) in the UK. Brain 2001; 124:1438
PATHOLOGY

- Macroscopically, there is atrophy of midbrain, pallidum, thalamus, subthalamic nucleus, as well as mild symmetric frontal atrophy,
- Microscopically, there is accumulation of a specific tau protein isoform (4R-tau) that appear in neurons as flame-shaped neurofibrillary tangles and in glial cells as tufted astrocytes within the globus pallidus, subthalmic nucleus, substantia nigra, and brainstem.
**Tau Protein**

- Tau is a protein that is involved in axonal transport and stabilization of neuronal microtubules.
- It is thought that abnormal phosphorylation of tau interferes with microtubule function, impairs axonal transport, and leads to tau aggregation into neurofibrillary tangles.
- The exact triggers for the conversion from normal tau to the aggregate form are not completely understood.
- Abnormalities of this protein have also been noted in several neurodegenerative diseases, eg: *Alzheimer’s disease, frontotemporal dementia, post-encephalitic parkinsonism*. 
The areas most consistently involved include:

- Globus pallidus, substantia nigra, subthalamic nucleus
- Midbrain tectum and pontine nuclei
- Locus ceruleus, periaqueductal grey matter
- Caudate, putamen and cerebral cortex
- Occasionally, the Purkinje cells of the cerebellum
- Dilated third and fourth ventricles (in patients with long standing disease)
**Pathology**

- The anatomical sites involved with PSP correlate with the clinical signs and symptoms

<table>
<thead>
<tr>
<th>Site of lesion</th>
<th>Clinical features</th>
</tr>
</thead>
<tbody>
<tr>
<td>cerebral cortex (particularly the frontal lobe)</td>
<td>cognitive and behavioural changes</td>
</tr>
<tr>
<td>nigrostriatopallidal area</td>
<td>rigidity, bradykinesia and postural instability</td>
</tr>
<tr>
<td>cholinergic pontomesencephalic area</td>
<td>gaze palsy, sleep disturbances and axial motor abnormalities</td>
</tr>
<tr>
<td>lower brainstem</td>
<td>dysarthria and dysphagia</td>
</tr>
</tbody>
</table>
PATHOLOGY

- Neurochemical studies indicate that the degenerative process in PSP involves several neurotransmitter systems at multiple different sites.
- PSP is not a primary "neurotransmitter" disease, but a disorder in which multiple subpopulations of neurons degenerate with neurotransmitter availability diminished as a secondary effect.

<table>
<thead>
<tr>
<th>Sites of degeneration</th>
<th>Neurotransmitter involved</th>
<th>Clinical features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nigrostriatal pathway</td>
<td>Dopamine</td>
<td>Rigidity</td>
</tr>
<tr>
<td>Caudate, putamen, nucleus accumbens and frontal cortex</td>
<td>choline acetyltransferase (CAT) activity</td>
<td>Cognitive impairment</td>
</tr>
<tr>
<td>basal ganglia (striatum and globus pallidus interna and externa)</td>
<td>γ-Aminobutyric acid (GABA)</td>
<td>Rigidity</td>
</tr>
</tbody>
</table>
The cause of PSP remains unknown
Most cases appear to be sporadic
Both environmental and genetic influences have been postulated
The role of heredity in the pathophysiology of PSP remains elusive

A genome-wide association study reported an increased risk of PSP for two independent variants of microtubule-associated protein tau gene (MAPT). In addition, the study found an increased risk for several additional genes (STX6, EIF2AK3, and MOBP)
The significance of which is unclear

SYMPTOMS

The cardinal manifestations of PSP:
1. Disturbance of gait resulting in falls.
2. Supranuclear ophthalmoparesis or plegia
3. Dysarthria
4. Dysphagia
5. Pseudobulbar palsy
6. Frontal cognitive abnormalities
7. Sleep disturbances
**History**

- The onset of PSP is insidious and usually includes a prolonged phase of vague fatigue, headaches, arthralgias, dizziness, and depression.
- Patients also experience *subtle personality changes, memory problems*.
- The initial symptoms can often involve *unexplained imbalance or falls*.
- Patients have *stiff and broad-based gait*, usually fall backward.
- Over time, dysarthria, dysphagia, and visual symptoms appear, followed by a wider spectrum of symptoms, including sleep disturbance with insomnia, clumsiness, impaired handwriting, and oscillopsia.
- *Vertical gaze palsy is the most distinctive single clinical feature*.
- Family members are often a more accurate source of such information than the patient is.
In a neuropathologic study, the most common symptoms at disease onset were

1. postural instability and falls (63%)
2. dysarthria (35%)
3. bradykinesia (13%);
4. visual disturbances such as diplopia, blurred vision, burning eyes, and light sensitivity (13%)

**Compare Parkinson’s Disease and Progressive Supranuclear Palsy**

**Similarities:**
- Both cause *stiffness, movement difficulties, and clumsiness*
- Both diseases share features like: *onset in late middle age, bradykinesia and rigidity*

**Differences:**
- Rigidity tends to be much more pronounced in the neck (*Axial*) than the limbs (*Peripheral*) for PSP
- People with PSP usually *stand straight* or occasionally even *tilt their heads backward* (and tend to fall backward), while those with Parkinson's disease usually *bend forward*
- The *early appearance of gait and balance dysfunction* in PSP, in contrast to Parkinson disease, in which imbalance tends to occur late in the disease
- Problems with *speech and swallowing* are much more common and severe in PSP than in Parkinson's disease, and tend to show up earlier in the course of the disease
- *Eye movements* are abnormal in PSP but close to normal in Parkinson's disease.
- *Tremor* is rare in PSP
- People with PSP respond poorly and only transiently levodopa
PHYSICAL EXAMINATION: MOTOR

masked face and a startled expression
Cognitive and Behavioral Changes

- **Cognitive dysfunction and personality change** are common in patients with PSP, generally milder in degree than those seen in patients with primary dementing illnesses such as Alzheimer disease.

- The neuropsychological profile of PSP primarily involves *frontal lobe dysfunction*. The patients manifest impaired abstract thought, decreased verbal fluency, motor perseveration, and frontal behavioral disturbances.

- The **presence of early and severe executive deficits** is a common finding in PSP. Executive dysfunction may be the presenting symptom of PSP in some patients and is characteristic of the later stages of the disease.

- **Behavioral abnormalities** are also common in patients with PSP. The most common behavioral symptoms were *apathy, disinhibition, dysphoria, and anxiety*. *Depression* is also common.
INVESTIGATIONS

- The clinical diagnosis of PSP depends primarily on history and physical examination
- Workup is directed at eliminating other diagnoses
Although CT scans sometimes disclose pathological changes including generalized or brainstem atrophy in patients with PSP, current data suggest that this diagnostic tool is of limited use in routine clinical practice.

MRI

- Routine and volumetric MRI imaging may show *midbrain atrophy*, a finding that may help in differentiating patients with PSP from healthy controls and those with Parkinson’s Disease.
- The midbrain atrophy resembles *hummingbird silhouette*.
- A study designed to provide a quantitative assessment of atrophy by measuring midbrain diameter or area found that a *diameter of <17 mm on axial MRIs* differentiates patients with PSP from healthy controls.
- However, the utility of the measurement for the diagnosis of PSP in clinical practice remains to be established.


**HUMMINGBIRD SIGN**

- T2-weighted MRI image of the brain showing the *selective atrophy of midbrain* with preservation of pons (divided by the black line)
- The atrophy of the midbrain tegmentum results in the concavity forming the silhouette of the head of the Hummingbird. This feature is called the hummingbird sign
- The hummingbird sign is reported to have a sensitivity of nearly 100% in diagnosis of PSP

*BMJ Case Reports 2012; doi:10.1136/bcr-2012-006263*
MRI of our patient

MRI with typical atrophy of midbrain
TAU PROTEIN IN CEREBROSPINAL FLUID

- Borroni et al proposed **evaluation of tau forms in cerebrospinal fluid (CSF)** as a biomarker for PSP
- CSF contains both extended (55 kd) and truncated (33 kd) tau forms, and the truncated-to-extended ratio is **significantly lower in PSP** than in other neurodegenerative disorders. In the study, the ratio was 0.504 ± 0.284 in PSP patients, 0.899-1.215 in patients with other neurodegenerative conditions, and 0.989 ± 0.343 in controls
- Further study of such biomarker candidates is required before these become incorporated into diagnostic algorithms or criteria

Basic features
- gradually progressive disorder
- onset age >40 yr
- no evidence of other diseases that could explain the clinical features as indicated by exclusion criteria

Diagnosis of clinically possible PSP
- vertical supranuclear palsy OR slowing of vertical saccades
- postural instability with falls within a year of disease onset

Diagnosis of clinically probable PSP
- vertical supranuclear palsy
- prominent postural instability with falls within a year of disease onset

Diagnosis of definite PSP
- Clinically probable or possible PSP and histopathologic evidence of typical PSP

Supportive features
- symmetrical akinesia or rigidity, proximal more than distal
- abnormal neck posture, especially retrocollis
- poor or absent response of parkinsonism to levodopa
- early dysphagia and dysarthria
- early onset of cognitive impairment including two or more of the following: apathy, impairment in abstract thought, decreased verbal fluency, utilization or imitation behavior, or frontal release signs

Exclusion criteria
- recent history of encephalitis
- alien limb syndrome
- cortical sensory deficits
- focal frontal or temporoparietal atrophy
- hallucinations or delusions unrelated to dopaminergic therapy
- cortical dementia of Alzheimer type
- prominent, early cerebellar symptoms
- unexplained dysautonomia
- neuroradiological evidence of relevant structural abnormality
- Whipple’s disease, confirmed by polymerase
The story doesn’t end here after making the diagnosis……….

- The expected long term problems:
  1. Gait and mobility problem, with fall risk
  2. Visual problem
  3. Swallowing and speech problem, with aspiration risk
  4. Cognitive and behavioral problem
  5. Mood problem
  6. Long time care problem and caregivers’ burden
  7. Palliative care and advance directive

- The management list could be endless!!
CONCERNS OF WIFE

1. Mobility issue:
- Wife being the main caregiver
- Home without lift service
- Fear for further fall

2. Cognitive and behavioral issue
- Regard the behavioral change unusual
- Fear for the violent act of patient
**Management**

- There are *no treatments* that alter the natural history of disease in PSP and no drugs that provide significant symptomatic benefits.
- Only a few patients respond to dopaminergic or anticholinergic drugs, and responses often are short-lived and incomplete.
- Treatment focuses on relieving symptoms while trying to ensure that patients have the best possible quality of life.
- A *multidisciplinary approach* is essential!!
MANAGEMENT PLAN

Management

Pharmacological treatment

- levodopa
- Amantidene
- zolpiderm

Non-pharmacological treatment

- Management of mobility problem
- Management of visual problem
- Management of swallowing problem
PHARMACOLOGICAL TREATMENT: LEVODOPA

- Generally produces **NO dramatic symptomatic improvement** in patients with PSP, in sharp contrast with its effect in patients with idiopathic Parkinson disease.
- The main role of levodopa in patients with suspected PSP is diagnostic; a poor or unsustained response to levodopa therapy is generally observed in patients with PSP and can help to distinguish PSP from idiopathic Parkinson disease.
- Levodopa therapy may provide some degree of **transient benefit** as suggested by several small retrospective reports.
- The improvement was **not sustained** and resulted in adverse effects in more than half of the patients.
- A common side effect of levodopa in patients with PSP is visual hallucinations, other side effects include dyskinesia, oromandibular dystonia, and apraxia of eyelid opening.


PHARMACOLOGICAL TREATMENT: ZOLPIDERM

- Zolpiderm is a gamma aminobutryic acid (GABA)ergic agonist of the benzodiazepine type 1 receptor
- Zolpiderm immediate release formulation has been reported to show short-term improvements in motor function and voluntary saccadic eye movements in patients with PSP over 6 months, but the benefits were not sustained
- Further studies are needed to determine the potential roles of GABA neurotransmission in PSP

**Pharmacological treatment: Amantadine**

- Amantadine can provide *a transient therapeutic benefit* in a minority of cases and can also help drooling and dyskinesia.
- The response is limited and only reported in small studies.
- Improvement is likely related to NMDA antagonistic properties of Amantadine.

NON-PHARMACOLOGICAL MANAGEMENT: LONG TERM MONITORING

- To help the patient and family adjust to the changing needs incurred by PSP
- Close follow-up care should be provided, with particular attention to patient’s problems and needs
- Caregiver support is important
- Patient’s mood should be concerned
MANAGING MOBILITY PROBLEM

- Regular exercise can help to strengthen the muscles, improve posture and prevent stiffening of the joints.

- *Physical therapy and rehabilitation* involvement may help maximize ambulation safety and facilitate instruction in the use of a walker, wheelchair, or other aids.

- Preliminary evidence suggests that the combination of *balance training* reduces the fall risk of patient.

- Occupational therapist provide advice about home safety and fall prevention strategies.
MANAGING EYE PROBLEM

- Chronic conjunctivitis is common in individuals with PSP because of the reduced blink rate in these patients. It can be treated with applications of methylcellulose to lubricate eyes and reduce irritation.

- *Botulinum toxin* (BTA) can be used to help relax the muscles of the eyelids.

- Glasses that contain specially designed lenses can help some people who are having problems looking down. Wearing wraparound, dark glasses can help those who are vulnerable to bright light.

- *Eye movement and visual awareness exercises* can be beneficial for enhancing suppression of fixation and gaze shift in patients with PSP.
MANAGING SWALLOWING PROBLEM

- When swallowing starts to become affected, consultation with a speech therapist and dietitian may help in modifying the diet so that food and liquids are easier to swallow while ensuring a healthy, balanced diet.
- Physical techniques could be used to make swallowing easier. For example, some people find that moving their chin forward while swallowing helps to prevent any food from entering their airways.
- Nasogastric tube and percutaneous endoscopic gastrostomy may be recommended in severe cases of dysphagia.
Progress of our patient

- Low dose 0.5 tab BD sinemet 25/100 started after admission
- Patient had subjective improvement in mobility! (?? Related to drug effect, placebo effect or training effect)
- Transferred to rehab ward for further training
- **Balance training** and **fall prevention strategy** were emphasized
- EMS: 9 → 11/20
- BBS: 26 → 32/56
- BI: 75 → 82/100, ADL up to toileting
DISCHARGE PLAN

- Patient wished to go back home, rather than nursing home care
- Refuse training at GDH as patient didn’t want to travel frequently to the hospital for training
- Plan close follow up and OPD for fall prevention

** Considerations:
- Wife was the only main caregiver
- Patient lived at the apartment of 3/F without lift
REVIEW IN 4 WEEKS AFTER DISCHARGE

- Mobility static, mainly walked with stick, no further improvement
- Had several falls after discharge at home and on the street
- Sustained mild injury to the elbow and forearm
- Tried increase Sinemet to one tab tds on 20/6/2012
Comments by psychiatrist:
- Progressive global cognitive decline
- Progressive decline in self-care ability
- Self-muttering, with vague paranoid idea and confabulation of information on & off

Dx: senile dementia

Given:
1. Donepezil 5mg Nocte
2. Risperidone 0.5mg daily
Review in 12/2012

- Deterioration of mobility, patient claimed it is difficult for him to walk
- Repeated minor falls
  (?? Due to the risperidone or natural progression of disease)
- Put on wheelchair
- Moved to Old Age Home in 11/2012!
  Wife said it is mainly due to worsening mobility

- Swallowing maintained
- Still communicable
- Poor gait initiation, but after start, can walk with assistance

- letter to psychiatry colleague for early FU
- refer YMT GDH
- suggest off risperidone to see effect
USE OF RISPERIDONE IN PSP

- Psychotic symptoms in PSP may respond to antipsychotic drugs, but caution should be exercised as these patients are prone to the extrapyramidal side effects.

- There is no literature on the use of atypical antipsychotics which may have less propensity to produce extrapyramidal side effects.

SUMMARY OF HISTORY

Mobility problem with repeated minor falls since about 5/2010

Agitated mood and unusual behavior since 1/2012

Sinemet started with mild improvement of mobility

Moved to old age home in 11/2012

Stick walking since 9/2011

Admitted to medical for fall in 5/2012

Risperidone started by psychiatrist in 7/2012
Prognosis

- Disease progression in PSP usually occurs fairly rapidly and relentlessly.
- The prognosis of PSP remains poor.
- Most patients become dependent for care within three or four years from presentation.
- Life span was shortened, often leading to death within ten years after symptom onset.
- Mean survival ranges from 5.9 to 9.7 years according to the different series.
LATEST REVIEW IN 1/2013

- Walked with frame at OAH, fair motivation in mobility
- Repeated minor falls, about 2-3 times a month
- Sustained fracture right 4th proximal phalange, put on POP
- Speech static
- Swallowing problem began, needed to take food slowly
- Occasional functional incontinence, being put on napkin at OAH
- Ophthalmoplegia persisted
- Mood stable

- Went for training at YMT GDH, wife was satisfied with the progress
- Refer to the speech for further swallowing assessment
SUMMARY

- PSP is uncommon but not rare
- Characteristics include supranuclear ophthalmoplegia, gait disorder and postural instability, dysarthria, dysphagia, rigidity, and frontal cognitive disturbance
- The diagnosis of PSP depends primarily on history and physical examination
- There are no treatments that alter the natural history of disease in PSP and no drugs that provide significant symptomatic benefits
- Levodopa generally produces NO dramatic symptomatic improvement in patients with PSP
- Treatment focuses on relieving symptoms while trying to ensure that patients have the best possible quality of life
AN INTERVIEW TO A PATIENT WITH PSP

PSP: A brain disease with no known cure
BY SARAH SPAETH – POSTED ON 28 MARCH 2012
POSTED IN: EDITORS PICK, PEOPLE, SOCIETY

My name is Chan Kwok Kwong. I am 52 year old.
AN INTERVIEW WITH A PATIENT WITH PSP

• Emotional I am a lot more moody and temperamental......’
• ‘I get upset easily......’
• ‘Of course I get sad! There is no explanation, there is no cure......’
• ‘When one is at his lowest, there will definitely be complaining and frustrating life......’

- Support to the patient and the family is essential!
Thank you!