

# An uncommon cause of recurrent falls in an elderly man

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Falls are common among the elderly population. Examinations for the cause of falls are usually mundane, but may be challenging, leading to surprising diagnoses. We report on a previously healthy elderly man who presented with repeated falls and rapidly progressive limitations in mobility, in addition to a stutter. Neuroimaging was particularly helpful for making the diagnosis in this patient.

## Introduction

Recurrent falls are a common syndrome among elderly people, and about 5% of these lead to serious injuries, with 1% resulting in hip fractures.<sup>1</sup> Examinations for recurrent falls usually consist of assessment of intrinsic and extrinsic risk factors. Extrinsic factors include environmental hazards such as poor lighting, slippery floors, or inappropriate use of walking aids. Intrinsic factors include gait and balance problems such as Parkinson's disease (PD), cognitive impairment, and sensory impairment (eg peripheral neuropathy or visual impairment). Among intrinsic factors, gait and balance are always assessed, but other neurodegenerative diseases other than PD could be missed if more subtle features are undetected. We report on an elderly man with recurrent falls who had more than just parkinsonism.

## Case report

A previous healthy 73-year-old man, who was still jogging in August 2008, was admitted to hospital after a fall in October 2008 and was noticed to have features of parkinsonism in his right arm. At a follow-up in February 2009, he had right arm weakness and stuttering, and reported recurrent falls since discharge. He had a shuffling gait on turning and his right arm showed hemi-rigidity. By then he could only walk for short distances outside, and needed to be escorted by his wife due to the high risk of falling. In September 2009, low-dose levodopa (125 mg 3 times daily) was started in view of the progressive poor gait initiation, bradykinesia, pill-rolling tremor, and rigidity. However, he stopped the treatment after 1 week due to increased rigidity. After another fall in December 2009, he underwent axial computed tomography (CT) of the brain, which showed neither skull fracture nor subdural haematoma.

### Key words

Accidental falls; Aged; Diagnostic imaging; Frontotemporal lobar degeneration; Primary progressive nonfluent aphasia

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At a review in the Geriatrics Clinic in January 2010, he was noted to have a masked face and tremorless rigidity of the arms, which was more prominent on the right side. His speech was slow, although coherent, and he had word-finding difficulties. Deep tendon reflexes were brisk, particularly in the right arm. Plantar reflex was equivocal on the right side, but normal on the left. He had difficulty relaxing his muscles on request, and a tendency to grasp objects placed in hands (grasp reflex). He showed marked gait apraxia, yet could perform cycling movements normally in the chair. He could only get up with assistance. Most of his activities of daily living (eg bathing and toileting) required assistance by his wife.

Careful review of the CT images taken in mid-December 2009 showed left frontal atrophy, especially in the posterior frontal region. There were no personality changes so far. In view of the language deficits and frontal-type gait apraxia, the tentative diagnosis was primary progressive aphasia (PPA) with gait apraxia. Brain perfusion scan was done and confirmed asymmetrical frontal lobe atrophy (Fig 1).

His Mini-Mental State Examination (MMSE)<sup>2</sup> score was 23 (above cut-off), showing intact delayed recall. The Montreal Cognitive Assessment (MoCA)<sup>3</sup> score was 14 (below cut-off), demonstrating deficits in execution, control, concentration, fluency, and delayed recall. Speech assessment indicated anomia-type aphasia. There was difficulty in expressive dysphasia with circumlocution, and a fluency problem resulting in slow

speech. Comprehension deficit was mild. There was little response to gait training, and resumption of levodopa (125 mg twice daily, then increased to 125 mg 3 times daily after 3 months). He was still unable to use his right hand for writing or feeding, and could only walk while holding onto furniture or supporting himself against the wall.

### Neuroimaging

Magnetic resonance imaging (MRI) of the brain was performed on a 3.0T MR scanner (Achieva TX; Philips Medical Systems, Best, The Netherlands). Axial T1-weighted image (T1WI), T2-weighted image, and fluid-attenuation inversion recovery were used to exclude any focal lesion or small-vessel disease of brain. Coronal T1WI was used to look for any cortical atrophy. The MR angiography time-of-flight technique was used to exclude major intracranial arterial stenosis. Diffusion tensor (DT) imaging was performed to demonstrate the connection fibre in the cerebral hemisphere.

Cerebral perfusion single-photon emission computed tomography (SPECT) using 25 mCi injection of <sup>99m</sup>technetium ethyl cysteinate dimer by dual-headed gamma camera (Infinia Hawkeye; General Electric, Milwaukee, US) equipped with a fan beam collimator was performed. Talairach analysis (3-dimensional reconstruction) and 3-planar slices were obtained.

Magnetic resonance imaging showed cortical atrophy of the left Wernicke and Broca areas (Fig 1a). Corresponding DT imaging (Fig 1b) showed markedly decreased conduction fibres (arcuate fasciculus) between the left Wernicke and Broca areas compared with the contralateral side, which was consistent with the recent findings of the left perisylvian parietotemporal areas subserving a generic and critical role in the circle of parity between perception and production of both words and gestures.<sup>4</sup> Talairach analysis of the patient's brain perfusion compared with the computer database showed moderate-to-severe hypoperfusion in the left Wernicke and Broca areas (Fig 2), which was particularly severe in the left Broca area and was consistent with the findings in the literature.<sup>5,6</sup> Single-photon emission computed tomography of the basal ganglia showed asymmetrical hypoperfusion in the basal ganglia, mild hypoperfusion in the right hemisphere, but normal perfusion in the left hemisphere. Axial SPECT of the brain showed asymmetrical hypoperfusion (L>R). There was a mismatch of hypoperfusion between the basal ganglia and the cerebral cortex.

### Discussion

Falls are a common syndrome among elderly people, and occur in one-third of the community-dwelling

## 一名老人反覆跌倒的罕見病因

跌倒是老人常見的問題。找出患者跌倒原因的常規程序，有時或因突破性的發現而變得具挑戰性。本文報告一名健康大致正常的老年患者因反覆跌倒而求診，他的活動能力在短時間內漸漸出現障礙，並有口吃問題。在這宗病例中，神經影像學尤其有助確診。

older population, with an even higher incidence in the institutionalised older population. Parkinson's disease is commonly associated with falls, but frontal gait disorder can mimic PD. The frontal lobe is an important area in gait pattern preservation and frontotemporal lobar degeneration (FTLD) leads to a variety of conditions, including the frontal variant of frontotemporal dementia (fvFTD) with prominent

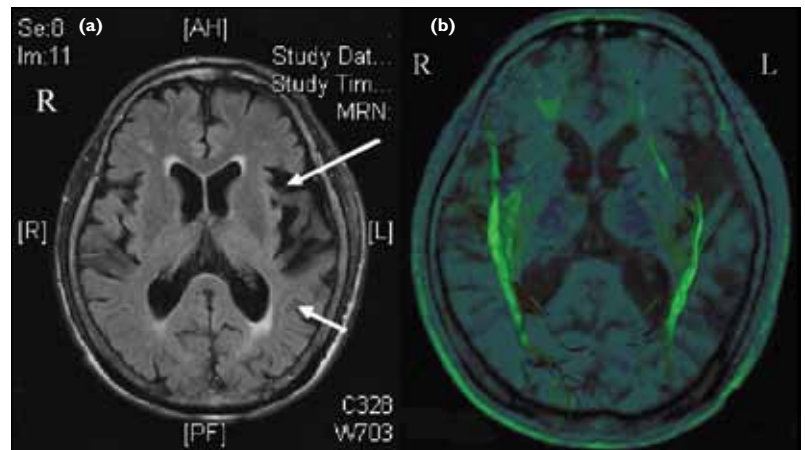


FIG 1. (a) Fluid-attenuation inversion recovery magnetic resonance imaging of the brain shows cortical atrophy in the left Broca and Wernicke areas (long and short arrows, respectively). (b) Fibre tractography by diffusion tensor imaging shows that the left-sided fibre tract connecting the Broca and Wernicke areas is significantly reduced compared with the relatively normal right-sided fibres

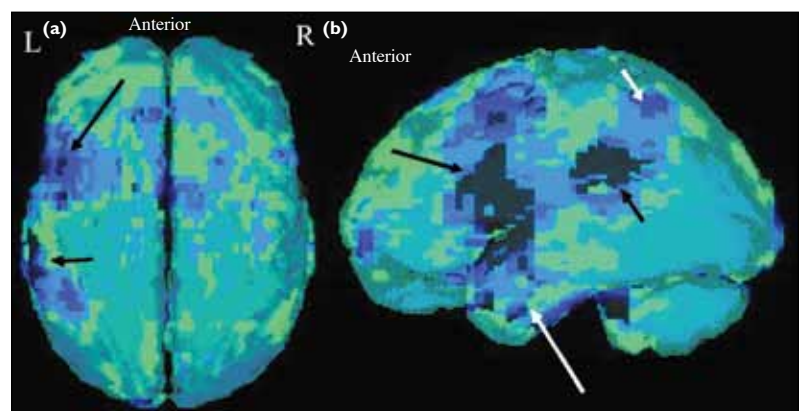


FIG 2. (a) Vertex view of Talairach analysis of cerebral perfusion by injection of <sup>99m</sup>technetium ethyl cysteinate dimer. Broca and Wernicke areas in the left cerebrum showed moderate-to-severe hypoperfusion (long and short arrows, respectively). (b) Left lateral view of cerebral perfusion. There is moderate hypoperfusion in the left temporal lobe and left parietal lobe (white long and short arrows, respectively) in addition to the Broca and Wernicke areas (black long and short arrows, respectively)

behavioural symptoms,<sup>7</sup> and two language-dominant dementias: semantic dementia or fluent progressive aphasia, and PPA or progressive non-fluent aphasia. Abnormal speech associated with falls with gait apraxia should alert clinicians to the possibility of PPA, a rarer form of FTLD.<sup>7,8</sup> As cognitive impairment in FTLD could either be exaggerated (due to language impairment)<sup>7,9</sup> or be missed by the MMSE (due to the deficiency of the MMSE in testing frontal lobe functions), the MoCA is a more appropriate screening test if frontal lobe deficits are suspected.<sup>10</sup> Language should also be assessed.

The difficulty of clinical diagnosis for this patient lies in the lack of behavioural features typical of the more common fvFTD, such as impulsiveness, disinhibition, aggression, or apathy. Instead he demonstrated progressive non-fluent speech difficulties compatible with PPA, a rarer form of frontotemporal dementia that affects expressive language, with relatively well-preserved cognition<sup>11</sup> and usually no behavioural symptoms. Other features of rigidity and positive primitive reflexes supported the diagnosis.<sup>12</sup> However, another differential diagnosis could be corticobasal syndrome (CBS), another rare form of focal brain degeneration, which usually presents with motor symptoms such as hemirigidity, falls, and limb apraxia, and language fluency problems may develop later.<sup>13</sup> As it was not possible to define clearly whether the speech problem or the gait problem had presented first, clinical diagnosis was difficult. There have been reports of PPA progressing into CBS, or an overlapping of the disorders, hence follow-up imaging may be helpful to delineate between the two conditions.<sup>14,15</sup> As a minority of these patients also developed motor neuron diseases, monitoring for muscle wasting, fasciculation, and swallowing problems are warranted at follow-up. Primary progressive aphasia, fvFTD, and CBS may present the different ends of a spectrum of disease called pick complex,<sup>16</sup> with variable behavioural, language, and motor symptoms at presentation, reflecting differential focal degeneration at disease onset.<sup>9</sup> Genetic studies have also shown that CBS and PPA might both be linked to mutations on chromosome 17.<sup>13</sup>

Previous genetic studies of familial PPA linked the disorder to a defect in chromosome 17. Recent genetic studies have identified mutations of the progranulin gene on this chromosome in familial PPA.<sup>17</sup> Whether the same mutations are responsible for sporadic cases of PPA is uncertain. However, apolipoprotein E ε4 haplotype was also found to be associated with PPA and was present in 20% of patients with PPA in one series.<sup>18</sup>

At present, no treatment is available for PPA or CBS. Results with cholinesterase inhibitors and an N-methyl D-aspartate receptor antagonist have been disappointing.<sup>16</sup> As the language impairment invariably progresses, it is important to teach the patient other means of communication. For this patient, visual impairment and right arm rigidity inhibited him from using writing to communicate. Education of the patient and the caregiver becomes important as patience is needed for communication. Fall prevention and mobility aids are also important due to high fall risk.

In FTLD, neuroimaging may serve as a biomarker to distinguish fvFTD or PPA from other more generalised dementias, and is also needed to rule out focal structural lesions (tumours, abscesses, or strokes) in the frontotemporal lobes. In this patient, imaging was helpful to distinguish PPA from the more common subcortical vascular dementia, which may also present with slowness of speech, apraxic gait, and brisk reflexes. Frontal asymmetry with widening of the Sylvian fissure may be masked by generalised widening of sulci in older individuals, but MRI can demonstrate the marked asymmetrical atrophy of the dominant (left) frontotemporal lobe, while functional imaging using cerebral perfusion SPECT and DT imaging can demonstrate the hypometabolism of the related areas and the reduction in conduction fibres in the language areas, respectively. Follow-up MRI and SPECT may be able to discern between PPA and CBS.

## Conclusion

Parkinsonism associated with falls may mask frontal gait disorders. Language deficits, especially when associated with physical signs such as rigidity, apraxia, or falls, should prompt investigation for frontotemporal degeneration disorders, even without typical behavioural symptoms. Structural and functional neuroimaging are helpful to rule out focal lesions and demonstrate focal atrophy in focal degenerative dementias.

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## References

- Masud T, Morris RO. Epidemiology of falls. *Age Ageing* 2001;30 Suppl 4:3S-7S.
- Chiu HF, Lee HC, Chung WS, Kwong PK. Reliability and validity of the Cantonese version of Mini Mental state examination — a preliminary study. *J Hong Kong Coll Psychiatry* 1994;4:25-8.
- Wong A, Xiong YY, Kwan PW, et al. The validity, reliability and clinical utility of the Hong Kong Montreal Cognitive Assessment (HK-MoCA) in patients with cerebral small vessel disease. *Dement Geriatr Cogn Disord* 2009;28:81-7.
- Nelissen N, Pazzaglia M, Vandenbulcke M, et al. Gesture discrimination in primary progressive aphasia: the intersection between gesture and language processing pathways. *J Neurosci* 2010;30:6334-41.
- Soriani-Lefèvre MH, Hannequin D, Bakchine S, et al. Evidence of bilateral temporal lobe involvement in primary progressive aphasia: a SPECT study. *J Nucl Med* 2003;44:1013-22.
- Abe K, Ukita H, Yanagihara T. Imaging in primary progressive aphasia. *Neuroradiology* 1997;39:556-9.
- Hodges JR. Frontotemporal dementia (Pick's disease): clinical features and assessment. *Neurology* 2001;56(11 Suppl 4):6S-10S.
- Cairns NJ, Bigio EH, Mackenzie IR, et al; Consortium for Frontotemporal Lobar Degeneration. Neuropathologic diagnostic and nosologic criteria for frontotemporal lobar degeneration: consensus of the Consortium for Frontotemporal Lobar Degeneration. *Acta Neuropathol* 2007;114:5-22.
- Mesulam MM, Grossman M, Hillis A, Kertesz A, Weintraub S. Core and halo of primary progressive aphasia and semantic dementia. *Ann Neurol* 2003;54 Suppl 5:11S-14S.
- Cummings JL, Miller BL. Conceptual and clinical aspects of the frontal lobe. In: Miller BL, Cummings JL, editors. *The human frontal lobes: functions and disorders*. New York: The Guilford Press; 2007: 12-24.
- Mesulam MM. Slowly progressive aphasia without generalized dementia. *Ann Neurol* 1982;11:592-8.
- Neary D, Snowden JS, Gustafson L, et al. Frontotemporal lobar degeneration: a consensus on clinical diagnostic criteria. *Neurology* 1998;51:1546-54.
- Boeve BF, Lang AE, Litvan I. Corticobasal degeneration and its relationship to progressive supranuclear palsy and frontotemporal dementia. *Annals Neurol* 2003;54 Suppl 5:15S-19S.
- Kertesz A, Davidson W, Munoz DG. Clinical and pathological overlap between frontotemporal dementia, primary progressive aphasia and corticobasal degeneration: the Pick complex. *Dement Geriatr Cogn Disord* 1999;10 Suppl 1:46S-49S.
- Gorno-Tempini ML, Murray RC, Rankin KP, Weiner MW, and Miller BL. Clinical, cognitive and anatomical evolution from nonfluent progressive aphasia to corticobasal syndrome: a case report. *Neurocase* 2004;10:426-36.
- Mesulam MM. Primary progressive aphasia: a 25-year retrospective. *Alzheimer Dis Assoc Disord* 2007;21:8S-11S.
- Mesulam M, Johnson N, Krefft TA, et al. Progranulin mutations in primary progressive aphasia: the PPA1 and PPA3 families. *Arch Neurol* 2007;64:43-7.
- Gorno-Tempini ML, Dronkers NF, Rankin KP, et al. Cognition and anatomy in three variants of primary progressive aphasia. *Ann Neurol* 2004;55:335-46.

## Answers to CME Programme

### *Hong Kong Medical Journal* June 2011 issue

Hong Kong Med J 2011;17:174-9

#### I. Implications of using World Health Organization growth reference (2007) for identifying growth problems in Hong Kong children aged 6 to 18 years

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|---|---|
| A | 2 |
| B | 1 |
| C | 4 |

Hong Kong Med J 2011;17:189-94

#### II. A synopsis of current haemophilia care in Hong Kong

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|---|---------|----------|---------|----------|----------|
| A | 1. True | 2. True  | 3. True | 4. True  | 5. False |
| B | 1. True | 2. True  | 3. True | 4. False | 5. False |
| C | 1. True | 2. False | 3. True | 4. True  | 5. False |