An elderly woman with calcification of the basal ganglia who presented with shuffling gait: a review with a case report

CASE REPORT

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INTRODUCTION

Elderly patients with an unsteady shuffling gait or signs that mimic Parkinson's disease (PD) are often referred to geriatricians for confirmation or exclusion of PD. Nonetheless these same signs and symptoms occur with many other conditions. We report on a patient who was referred by her general practitioner for investigation of possible PD because of an unsteady shuffling gait.

CASE REPORT

In September 2001, an 84-year-old woman presented with unsteady gait that had worsened over a number of years and a history of falling. Her family had observed a gradual decline in cognitive function, particularly her memory. The patient had no other significant medical history and took no regular medication. She had recently stopped taking calcium supplements.

Computed tomography (CT) scan of her brain revealed heavy calcification in the basal ganglia and dentate nuclei (**Figures 1-3**). There was also mild ventricular dilatation and cerebellar atrophy. Her full blood count was normal and B12 was marginally low at 150 pmol/L (normal range, 180-660 pmol/L). Biochemistry—including electrolytes, urea, creatinine, and calcium—was within normal limits, as were thyroid stimulating hormone and rapid plasma reagin levels. Her score on the mini-mental state examination was 21/30.[1] Sitting and standing blood pressure was 130/80 mm Hg and central nervous system examination revealed no pyramidal weakness but brisk bilateral reflexes. There were no sensory losses or cerebellar signs. When asked to walk, she mobilised with small shuffling steps and her gait was not wide. There was no rigidity or bradykinesia.

DISCUSSION

The patient exhibited a festinating gait that involves a symmetric, rapid shuffling of the feet, and is usually associated with PD.[2] Nevertheless she had no rigidity or bradykinesia or anything suggestive of parkinsonism other than a shuffling gait.

As she had no history of taking antipsychotic drugs, heavy calcification in the basal ganglia was determined to be the most likely cause of her shuffling gait. A literature search of the clinical presentations of basal ganglia calcification revealed that there are diverse presentations, the most common including seizures, mental deterioration, and disorders of cerebellar or extra-pyramidal function.[3] Movement disorders, chorea, or parkinsonism are present in 20 to 30% of patients with basal ganglia calcification, while some patients are asymptomatic.[3]

Another study[4] suggested that movement disorders are the most common manifestation of bilateral striopallidodentate calcinosis (basal ganglia calcification), with parkinsonism the most common of the various movement disorders. Klawans et al[5] noted that basal ganglia calcification should be sought in all cases of levodopa-resistant parkinsonism, because treatment of hypoparathyroidism, if present, can potentially halt progression of the disease.

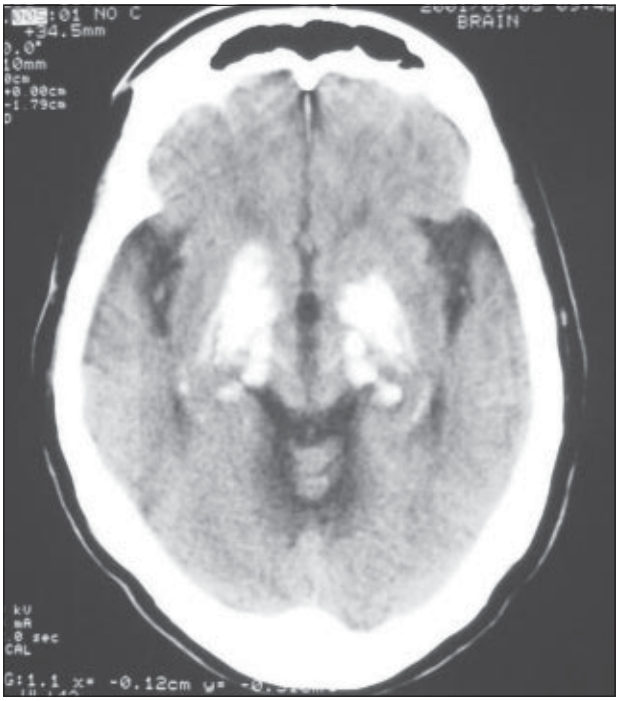

Figure 1. Calcification of the basal ganglia

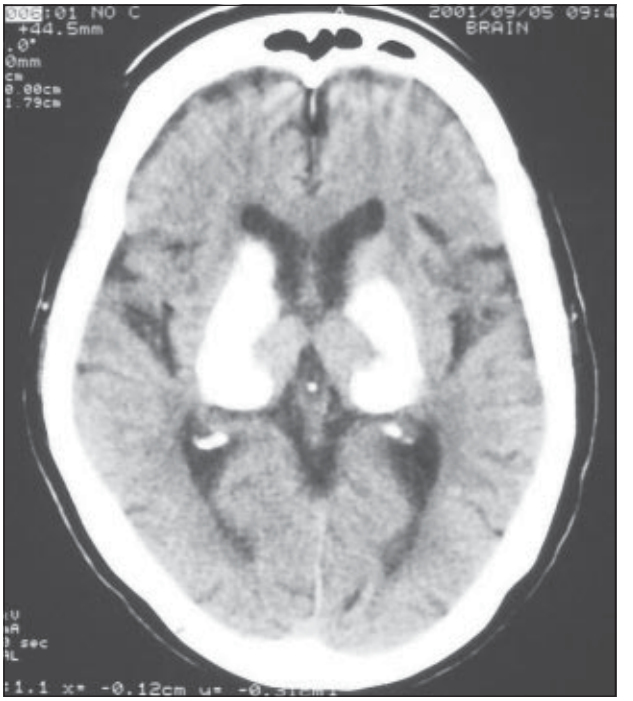

Figure 2. Extensive calcification of the basal ganglia

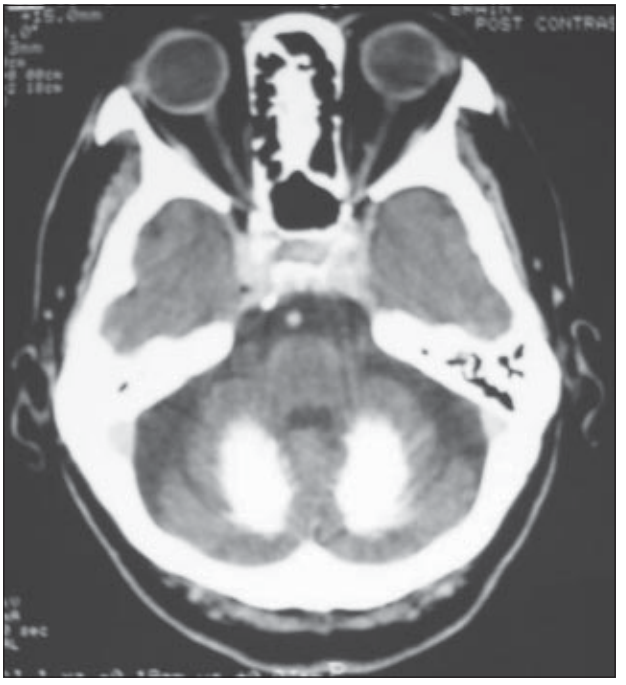

Figure 3. Calcification of the cerebellum

Box
Causes of symmetrical calcification of the basal ganglia[6]

Physiological
Hypoparathyroidism (various casuses including pseudohypoparathyroidism)
Hyperparathyroidism
Familial idiopathic cerebral calcification (Fahr's syndrome)
Birth anoxia
Carbon monoxide intoxication
Lead poisoning
Tuberous sclerosis
Cockayne's syndrome
Postinfectious
Acquired immunodeficiency syndrome (especially in children)
Radiation therapy
Methotrexate therapy
Kearns-Sayre syndrome and other mitochondrial diseases
Familial encephalopathies
Down syndrome

Nonetheless, the calcium level was within normal limits in our patient.

Cerebral deposition of calcium occurs in many pathological brain processes. Calcification, specifically of the basal ganglia, is associated with hypoparathyroidism of any type and, rarely, with other metabolic diseases; as a familial trait; and sporadically, without abnormal calcium metabolism (Box). The emergence of CT has led to the finding that sporadic calcification is the most common form, present in up to 1.5% of all brain scans.[6]

Most individuals have no corresponding clinical

signs and although symptomatic patients generally have more extensive calcification, no correlation has been found between the extent of CT or pathological changes and symptoms.[6] The literature has also reported familial cases of calcification.[7,8] Nonetheless, our patient had no such family history.

CONCLUSION

This case illustrates that basal ganglia calcification is a potential, albeit uncommon, cause of parkinsonian signs and highlights the need to be vigilant for rarer causes of parkinsonism. Although some authors have questioned the significance of calcification in those few patients who are symptomatic, others have advised the necessity of looking for it. It has been suggested that striatal disease from calcification can cause parkinsonism that tends to be less responsive or non-responsive to levodopa.

References

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