



## ‘Falling off’ the dopamine wagon

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A 70 year-old man with Parkinson's disease (PD) of nearly 20 years' duration decided to see an acupuncturist for another opinion on the treatment of his PD. He was prescribed a series of herbal teas as well as 12 weeks of Chinese acupuncture. Following this treatment he felt better, not in terms of his motor symptoms, but because the teas relieved his chronic constipation. He was so delighted with the removal of this symptom that he questioned the need for PD medications, which amounted to total daily doses of 750 mg levodopa, 9 mg ropinirol, and 300 mg amantadine. Without consulting his neurologist, he abruptly decreased each of the prescribed doses by two-thirds. Shortly afterwards, he stumbled and fell on a tile floor, resulting in a sharp pain in the right groin radiating to the medial thigh. Radiographic evaluation of the right hip revealed an impacted right garden II femoral neck fracture, for which he underwent percutaneous pinning (Fig. 1).

Multiple factors contribute to the risk of falls for persons with parkinsonism. Hallmark symptoms of bradykinesia, rigidity, and postural instability may apply to all forms of parkinsonism. Neurogenic orthostatic hypotension is a manifestation of autonomic dysfunction that may lead to falls and is more common with parkinsonism due to synuclein pathology, such as PD or multiple system atrophy [1, 2]. Some persons with parkinsonism may have extraocular eye movement abnormalities, further increasing their risk of falling. This occurs most often in progressive supranuclear palsy and is thought to play a role in the 'early falls' often reported by patients at the time of diagnosis. Our patient had no evidence of orthostatic intolerance or eye movement abnormalities surrounding the time of his fall. He did however suffer from frequent freezing of gait (FOG) in addition to his tremor, bradykinesia, and rigidity, which led to his high intensity dopamine supplementation.

While persons with PD are at risk of falling, those with less mobility/core strength and declining bone density (which may follow reductions in dopaminergic therapy), are at increased

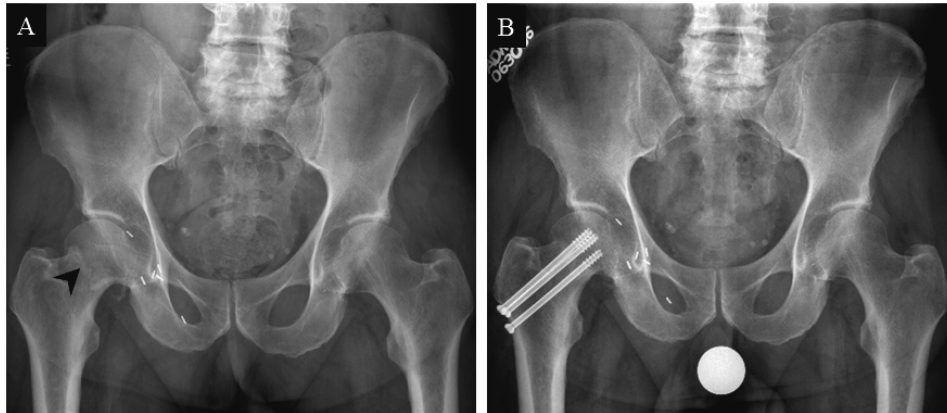
risk for not only falls, but also significant injury and mortality stemming from subsequent surgery and complications [3, 4]. Furthermore, there is an increased risk of fractures among patients with PD and dementia compared to those without dementia; however, this risk is not solely explained by the presence of dementia [5]. Vitamin D deficiency and perhaps bone loss may also contribute to this risk. It has even been proposed that low vitamin D levels may be related to the pathomechanism underlying PD [6]. Our patient had no cognitive impairment at the time of his fall, but was diagnosed with osteopenia shortly thereafter.

The cornerstone of treatment for PD is dopamine supplementation via levodopa, which improves motor symptoms of resting tremor, bradykinesia, and rigidity. Other symptoms associated with more advanced disease states, such as postural instability and FOG, are recalcitrant to dopaminergic therapies. However, there is secondary improvement in ambulation probably due to decreased bradykinesia and rigidity.

In advanced stages of PD, patients may begin to think that their medication has lost its effectiveness due to an accumulation of 'dopa-resistant' symptoms. It can be tempting for patients, with or without the guidance of their neurologist, to seek a simplified medication regimen and decrease dosage or frequency of dopaminergic drug therapy. In the case of our patient, this decision was catalyzed by the assumption that the improvement of non-motor symptoms indicated a concomitant improvement of motor symptoms. Our patient's experience vividly illustrates how abruptly altering dopaminergic medications can have disastrous consequences.

In addition to emphasising the critical role of dopamine supplementation in PD, this case teaches us an important lesson that non-motor symptoms of PD deserve attention. Such symptoms, including constipation, affect up to 90% of PD patients [7]. In his *Essay on the Shaking Palsy*, James Parkinson wrote: "The bowels, which had been all along torpid,

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**Figure 1.** Pelvic X-ray. **A.** Pelvic X-ray of a 70 year-old man showing right garden II femoral neck fracture (black arrowhead); **B.** Follow-up pelvic X-ray after surgical repair with pinning

now, in most cases, demand stimulating medicines of very considerable power” [8]. Treating such non-motor symptoms may not only improve quality of life, but avoid catastrophe.

Because PD is classically thought of as a disorder of movement, patients may be less likely to mention non-motor symptoms in the setting of a neurological follow up visit, unless explicitly questioned on the topic by their neurologist. Due to the complexity of PD, this can be a challenge to accomplish within the confines of a short office visit. However, its importance cannot be overstated because many non-motor symptoms such as mood disorders, cognitive impairment, and autonomic dysfunction have a negative impact on quality of life [9].

This case underscores the importance of a strong therapeutic alliance in which patients are comfortable discussing these issues and therapy options they may be considering. Our patient pursued a non-traditional approach with Eastern medicine, which he credited with improving his constipation, and that led him to reduce his dopaminergic medications. Discussing the option of supplementing rather than replacing his medications might have prevented his injury.

Fortunately, our patient's fall resulted in morbidity that was repairable. It is advisable to stress to patients and caregivers not to decrease dopaminergic therapy, even in advanced PD, unless serious adverse effects develop. In such instances, considering alternative forms of treatment (i.e. medications and surgery) may be appropriate.

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