CASE REPORTS

Amantadine-induced myoclonus in a patient with progressive supranuclear palsy

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Abstract

Progressive supranuclear palsy (PSP) is a tauopathy that generally results in a hypokinetic disorder. Treatment is largely symptomatic, with some small studies indicating a benefit with dopaminergic therapy. Myoclonus is a hyperkinetic disorder that can be seen as part of later stage Parkinson's disease and in multiple system atrophy, but is rarely seen in PSP. Here we report a case of myoclonus precipitated by amantadine in a patient with PSP.

Keywords: progressive supranuclear palsy, amantadine, myoclonus, elderly

Case report

Progressive supranuclear palsy (PSP) is a tauopathy that results in a hypokinetic movement disorder. Other tauopathies that may be seen by a geriatrician include Alzheimer's dementia, frontotemporal dementia and corticobasal degeneration. Symptoms of bradykinesia, axial rigidity, postural instability and falls, supranuclear palsy, dysarthria and higher cortical dysfunction predominate in PSP [1], and while dystonia is seen in a proportion of patients, other hyperkinetic features point away from this diagnosis. Treatment of this disorder remains unsatisfactory, with some patients reporting a modest benefit in parkinsonian symptoms from the dopaminergic therapy. Amantadine has been reported in small retrospective studies to be helpful in a proportion of patients, although its use is limited by frequent adverse events [2]. We report a case of myoclonus precipitated by amantadine in a PSP patient.

A 74-year-old gentleman was referred to our movement disorders service with a 3-year history of impaired dexterity, balance disturbance and a tendency to fall backwards. His family reported some dysarthria and mild cognitive slowing. On examination, he had evidence of motor perseveration, a broad-based short-stepped gait and an increase in axial tone. There were macrosaccadic square wave jerks (large amplitude inappropriate saccades that take the eyes off a target, followed by a corrective saccade) and a supranuclear vertical gaze palsy. Mild bradykinesia was present in both upper limbs. Neuropsychological testing was in keeping with a diagnosis of PSP, with impairments in frontal lobe functioning and marked apathy. The diagnosis was supported by an abnormal DaTSCAN, with significantly reduced tracer uptake bilaterally. Amantadine was commenced at a dose of 100 mg daily, to increase after 3 weeks to 100 mg b.i.d. Five days after increasing the dose to twice daily, the patient developed brief mild shock-like involuntary movements of his right upper limb. Over the course of a week, the myoclonic movements progressed to affect both the upper and lower limbs, at which time he was admitted to hospital. He was conscious throughout these episodes, with no other hyperkinetic features and no deterioration in his PSP. Laboratory investigations were unremarkable, and he was discharged home after an overnight stay. Neurophysiological studies were not performed. The amantadine was stopped, and 5 days later his myoclonus had completely subsided with no subsequent recurrence. He was later commenced on levodopa with no ill effects.

Myoclonus can be described as a sudden brief jerk caused by involuntary muscular contraction or inhibition [3]. The majority of causes of myoclonus are secondary to other disorders, and it is well recognised that myoclonus may be precipitated by a variety of medications [3]. In a French pharmacovigilance database study of drug-induced myoclonus, the most frequently involved drugs were anti-infectious agents, antidepressants, anxiolytics and...
opiates, with antiparkinsonian treatments implicated in a minority of cases [4]. Although myoclonus can occur in patients with Parkinson’s disease, multiple system atrophy, corticobasal degeneration and Lewy body dementia [5], it is rarely seen in PSP [6]. There have been two previous case reports of myoclonus in patients with Parkinson’s disease on amantadine [7, 8], but to our knowledge only one other case has been reported in a case of probable PSP on amantadine [9], and this was cranial myoclonus, which contrasts with the generalised myoclonus reported here. The mechanism of action of amantadine is poorly understood, although its actions may be secondary to antiguaminergic properties and the antagonistic effect on NMDA receptors, increasing levels of dopamine release. The drug may precipitate myoclonus in individuals at risk, possibly by altering the degree of dopamine, serotonin or glutamate release. This case illustrates the importance of always asking the question ‘Could this phenomenon be drug induced?’

Key points
- Myoclonus is a hyperkinetic movement disorder that can be described as a brief shock-like movement.
- Myoclonus can be seen in some parkinsonian disorders but is rare in progressive supranuclear palsy (PSP).
- Neuropsychiatric medications can precipitate myoclonus, and here we report amantadine-induced myoclonus in a patient with PSP.

Conflicts of interest
None declared.

References

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An unusual case of anaemia in an octogenarian

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Abstract

Older patients referred for further investigation of anaemia are common in a geriatric medicine clinic and it is important to consider a wide range of underlying diagnoses. We present an unusual case of anaemia in an octogenarian in whom a diagnosis of visceral leishmaniasis was made. This is a rare and unusual diagnosis in this cohort of patients, especially within the UK; however, it is important to identify it, given its high mortality if left untreated. Our case, presumably contracted