Ropinirole-Induced Anterocollis in Parkinson's Disease

Anterocollis or antecollis, also called dropped head, is characterized by disproportionate flexion of the head over the trunk and can result from either muscle weakness (i.e., myasthenia gravis and motor neuron disease) or muscle hyperactivity in cases of dystonia.[1]Anterocollis has been described in patients with Parkinson's disease (PD) or multiple system atrophy; however, it is still debated whether, in these cases, it is dystonic in nature or myopathic or both[2, 3] Anterocollis has also been described with use of antiparkinsonian medications, such as amantadine and certain dopamine agonists (DAs; cabergoline, pramipexole, or rotigotine).[2-7] We report here videotape evidence of anterocollis development secondary to ropinirole use, with full recovery after drug discontinuation.

Case Presentation

A 76-year-old man referred mood changes and apathy developing during the last 10 years. Soon after, he began to experience difficulty walking, needing to take shorter steps, and also referred clumsiness when using his hands. At the initial consultation, the patient referred symptoms consistent with rapid eye movement sleep behavior disorder, olfactory impairment, and chronic constipation as well as progressive immobilization. On the other hand, he denied urinary incontinence or syncope. After the diagnosis of PD was confirmed, he started levodopa four times a day. Symptoms progressed over a 10-year period with evident cognitive impairment in the last 2 years. The patient was treated with 675 mg of an l-dopa/carbidopa combination on a three times daily basis, along with 30 mg of duloxetine and 5 mg of donepezil daily, and 4 mg of extended-release ropinirole. On physical examination, 2 hours after his usual l-dopa dose, his International Parkinson and Movement Disorder Society/UPDRS Part III score was of 31 points. At the same time, his H & Y was 3 and Mini–Mental State Examination score was 27/30. The patient presented no “red flags” for atypical parkinsonism.

Last year, the patient developed a peptic ulcer, which required admission to the intensive care unit for 16 days. He developed transient delirium during his hospitalization and was treated with clozapine. Ropinirole, however, had to be withdrawn in order to resolve psychotic symptoms. One month later, clozapine was discontinued and ropinirole was progressively reintroduced with an initial dose of 8 mg and then increased to 12 mg. Two weeks after starting the 12-mg dose, he developed progressive anterocollis and slight camptocormia (see Video 1, left panel), to a degree in which his chin fell down and came into contact with his chest. Both active and passive mobilization of the neck was difficult. A simultaneous surface electromyography (EMG) was performed on the neck muscles, at rest (for 1 minute), during head rotation, tilt to either side, and during head flexion and extension. Both sternocleidomastoid and trapezius muscles showed signs of cocontraction. No signs of fasciculation or denervation were found in either muscle. Routine lab workup showed normal creatine kinase, aldolase, thyroid hormones, and acetylcholine receptor antibody values. Owing to the rapid symptom onset and the link to ropinirole increase, the drug was withdrawn. Four months after drug discontinuation, anterocollis completely resolved, although the cardinal symptoms of PD significantly deteriorated (see Video 1, right panel).

Discussion

Although postural disturbances are common in PD, mainly associated with the disease itself, they can also be caused or worsened by medication.[8, 9] Despite that DA-induced anterocollis has been previously reported, this is, to our knowledge, the first case associated with ropinirole use, confirming its dystonic nature. Still, 1 case of reversible Pisa syndrome associated with ropinirole in a PD patient has been previously described.[10] Pathophysiology of anterocollis in

PD is controversial, given that it is believed to be generated either by disproportionate neck muscle tone of both extensors and flexors, or through dystonia or myopathy of the extensor muscles.[2, 3, 7] In this case, surface EMG showed cocontraction of extensor and flexor neck muscles without evidence of myopathy, thus favoring a dystonic etiology. Risk factors related to anterocollis and similar dystonic features in PD besides DAs are age, disease duration, dementia, and female gender.[3, 8, 9] Clinicians treating PD patients should consider DAs as a potential source of anterocollis syndrome.