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Paroxysmal Exertion-induced Dyskinesia Effectively Treated with Methocarbamol

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Abstract

Paroxysmal dyskinesias/dystonias are rare movement disorders and can be classified into paroxysmal kinesigenic dyskinesia (PKD), paroxysmal non-kinesigenic dyskinesia (PNKD), and paroxysmal exertion-induced (PED) dyskinesia. While PKD responds to anticonvulsants, treatment options for PED are limited. We report a familial PED case that responded to methocarbamol and was able to return to baseline strenuous exercise.

Introduction

Paroxysmal dyskinesias (PDs) are characterized by episodes of sudden onset of dystonia, ballism, and choreoathetotic movements, without loss of consciousness. The first description of the paroxysmal movement came from Mount and Reback in 1940. They called it “familial paroxysmal choreoathetosis.”¹ The classification of PDs was introduced by Lance in 1977 based on the duration of attacks.² Demirkiran and Jankovic in 1995 described the current classification of PDs based on precipitating factors.³ They classified them into paroxysmal kinesigenic dyskinesia (PKD), paroxysmal non-kinesigenic dyskinesia (PNKD), and paroxysmal exertion-induced (PED) dyskinesia.

Lance reported the first case of PED in 1977.² PED episodes are precipitated by prolonged exercise or physical exertion. The usual age of onset for PED is 2 – 30 years. The typical duration of PED varies from five minutes to two hours, with a frequency between one per day to two per month.⁴ PEDs do not respond to a specific class of drugs. Some cases may respond to acetazolamide, antimuscarinics, and benzodiazepines.⁴ We are presenting a case of PED that responded very well to methocarbamol.

Case

A 23-year-old right-handed man was referred for the evaluation of exertion-induced paroxysmal dystonic episodes affecting the right leg and arm. He began to have these episodes at the age of 20 with progressive worsening. He noticed spasms and dystonic posturing in his right leg after doing his routine exercises and running. Over the years, episodes started happening with less exertion.

He needed to rest for 10 – 20 minutes to alleviate the symptoms and noticed a latency of a few hours before the subsequent episode.

His medical history included depression at the age of 19 that was treated with risperidone for one year. This caused orolingual tardive dyskinesia that disappeared one month after discontinuing the risperidone. His lab work was normal. Electroencephalograph (EEG), brain MRI, cervical spine, and thoracic spine were unremarkable except incidental finding of small (0.9x0.6 cm) benign pituitary cysts on brain MRI.

His family history was positive for paroxysmal exertional dystonia in his father. His brother and mother were in good health.

His neurological examination was normal at rest, but he developed mild dystonic eversion posturing of the right foot during heel walking.

He was initially managed with cyclobenzaprine, 10 mg three times per day, resulting in the reduction of symptoms for one year. He was next started on carbidopa-levodopa 25/100 mg 0.5 tabs three times per day; however, it produced significant nausea, dizziness, and dystonic posturing of his right hand, and therefore was referred to our Movement Disorders Center. We discontinued the levodopa and started methocarbamol 750 mg two to three times per day, with significant improvement in dystonic episodes. He was able to bike for 5 – 10 miles with methocarbamol.

We also saw his father, a 50-year-old right-handed man. At age 40, he started to notice paroxysmal dystonia in his hands after holding a tight grip for a long time (while hammering or using tools). Symptoms would begin with an abnormal sensation of tightness followed by a spasm in the forearms that would lead to paroxysmal episodes. At the time, his episodes were happening once per week and were lasting for 10 – 15 minutes. Neurological examination revealed increased latency with the opening hand after a firm grip. His lab work was unremarkable. Electromyography and nerve conduction studies of the upper extremities were unremarkable as well. He did not wish to take any medications for his paroxysmal episodes.

Patient consented to this case presentation.

Discussion

In recent years researchers have focused more on the genetics of paroxysmal dyskinesias. The most common gene associated with PED is SLC2A1, which encodes glucose transporter one (GLU-1) that regulates glucose transport across the blood-brain barrier.⁵ Other significant genetic conditions linked to PED are pyruvate dehydrogenase deficiency, GCH1, and ECHS1 mutation.⁵

The primary PEDs, where the energy metabolism pathways are involved, may respond to dietary modifications.⁵ Although the knowledge about PED is growing, we do not yet have a well-established symptomatic treatment of PED. While our patient decided against genetic testing, his PED episodes responded very well to methocarbamol.

Conclusion

In the presented case of PED, the patient had significant improvement of PED episodes with methocarbamol, and he was able to resume his usual physical activity. Methocarbamol is a centrally acting muscle relaxant, but its exact mechanism of action is not clearly understood. We believe that the role of methocarbamol in PED needs to be explored further as a potential symptomatic treatment to help patients regain their daily living functionality. ■

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