EXACERBATION OF IDIOPATHIC PAROXYSMAL KINESIGENIC DYSKINESIA IN REMISSION STATE CAUSED BY SECONDARY HYPOPARATHYROIDISM AFTER A THYROIDECTOMY


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Introduction: Paroxysmal kinesigenic dyskinesia (PKD) is a rare movement disorder characterized by short episodes of involuntary movement attacks, which are precipitated by sudden voluntary movement. The most common etiology of reported PKD cases is idiopathic or familial. But, several neurologic conditions such as multiple sclerosis, head trauma or basal ganglia calcification have been reported to be associated with symptomatic PKD. Hypoparathyroidism counts one of the causes of symptomatic PKD.

Aims: Our aim is to report an exacerbation of idiopathic PKD in remission state caused by secondary hypoparathyroidism after a thyroidectomy.

Methods: Single case report.

Results: A 34-year-old man presented with relapse and aggravation of paroxysmal involuntary movements after thyroidectomy. He experienced intermittently mild dystonia and brief choreoathetosis induced by sudden movements in his late teens. Duration was very short, ceased within 5 seconds and only right limbs were involved. Symptom gradually diminished since early twenty. However, symptoms of PKD has aggravated after a thyroidectomy for treatment of thyroid papillary cancer. Episodes occurred more frequently and severity and duration of symptoms increased, lasting up to a few minutes. EEG and brain imaging study were unremarkable. Laboratory studies revealed hypoparathyroidism with hypocalcemia. Treatment with additional oral calcium replacement resulted in the normalization of serum calcium level and amelioration of PKD episodes.

Conclusions: This report can be a support for the hypothesis that PKD has an association with neuronal ion regulation and that threshold of dyskinesia in idiopathic PKD patient can be influenced by serum calcium level just as in hypocalcemic secondary PKD.