

A Case of Psychogenic Movement Disorder Mimicking Acute Cerebellar Syndrome

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ABSTRACT

A case of psychogenic movement disorder mimicking acute cerebellar syndrome. Psychogenic movement disorders (PMDs) are involuntary movements of various kinds without any underlying organic etiology. They can occur as tremor, spasm, dystonia, parkinsonism or myoclonus. A detailed history and neurological examination is essential to differentiate these disorders from organic neurological etiologies. Since PMDs are challenging entities in clinical practice, we presented this case of psychogenic tremor and gait disorder mimicking acute cerebellar syndrome in order to emphasize the importance of diagnostic clinical clues of PMDs in the differentiation of organic diseases, and to give accurate treatment.

Keywords: Differential diagnosis, gait disorder, psychogenic movement disorder, tremor

ÖZET

Akut serebellar sendromu taklit eden bir psikojenik hareket bozukluğu olgusu

Psikojenik hareket bozuklukları (PHB) altta yatan herhangi bir organik etiyoloji olmaksızın ortaya çıkan farklı tiplerdeki istemsiz hareketlerdir. Tremor, spazm, distoni, parkinsonizm veya miyoklonus şeklinde görülebilirler. Bu hastalıkları organik nörolojik etiyolojilerden ayırmak için detaylı bir öykü ve nörolojik muayene gereklidir. PHB'nin klinik pratikte zorlayıcı bir antite olmaları nedeniyle, akut serebellar sendromu taklit eden psikojenik tremor ve yürüyüş bozukluğu olan bu olguyu, PHB'nin tanısız klinik ipuçlarının farkında olmanın, organik hastalıklardan ayırmada ve doğru tedavi uygulamadaki önemini vurgulamak amacıyla sunduk.

Anahtar kelimeler: Ayırıcı tanı, yürüyüş bozukluğu, psikojenik hareket bozukluğu, tremor

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INTRODUCTION

Psychogenic movement disorders (PMD) are a part of the spectrum of neurological movement disorders, in which the movements are held as the result of psychological or psychiatric etiologies rather than neurologic disturbances (1). They account for the 1.5% proportion of all patients seen in neurology clinics, and 2% to 20% of the patients referred to movement disorder outpatient clinics, with a gender dominance of women (2).

The pathophysiology of PMDs is thought to be multi-factorial, and the most considered theory is the biopsychosocial model. However, the exact pathophysiology is still unclear (3).

Since they are one of the most challenging neurological diseases and difficult to diagnose, some clinical clues are needed to differentiate the spectrum

of PMD than neurological movement disorders with organic origin including sudden onset, spontaneous remissions, and variability over time or during clinical examination (4). Additionally, the broad phenomenological spectrum of PMDs which can mimic tremor, parkinsonism, spasms, tics, chorea, athetosis, myoclonus, and dystonias, make the differential diagnosis more challenging.

On this aspect, we presented a young female with PMDs suffering from tremor-like involuntary movements and bizarre speech disturbance mimicking acute cerebellar syndrome.

CASE

A-27 year old woman presented to the emergency room of our faculty with sudden onset speech

disturbance and involuntary movements in her right lower limb within a few hours. She was married and house wife. Her medical history was unremarkable, except a mild depression which was declared by her husband. There was no drug and/or substance abuse, systemic and/or neurological disease. Her family history was also not remarkable. Since there was a mild deterioration in the rapid alternating movements in her right upper limb with dysmetria, and an involuntary movement resembling tremor in her right lower limb, she was hospitalized with the pre-diagnosis of acute cerebellar syndrome. Cranial magnetic resonance and magnetic resonance angiography of the patient was normal, and there was no contrast enhancement. When the neurological examination of the patient was detailed with prolonged observations, her speech pattern was found to be different from dysarthria with burst of verbal gibberish. She was changing her dialect in some parts of her spontaneous speech. When she was asked to perform voluntary activity, her speech was turning to normal. The tremor on her right limb was found to vary in direction, frequency, and amplitude. Moreover, tremor was found to improve when she performed motor tasks with the contralateral limb. She had a bizarre gait with knee-buckling, and was unable to perform tandem-walk. She was suffering from excessive fatigue and anxiety during the neurological examination. Her deep tendon reflexes were normal, and plantar responses were flexor, bilaterally. Laboratory investigations including complete blood count, kidney and liver functions, electrolytes, blood sugar, thyroid function tests, serum vitamin B12 and folic acid levels were normal. Markers of vasculitis, serum ceruloplasmin and copper levels were also normal. There was no acanthocytes or any other abnormal finding in her peripheral blood smear. Since she had normal neuro-imaging, and laboratory investigation, and her symptoms were compatible with psychogenic movement disorders including bizarre speech and involuntary, arrhythmic, irregular movements with high amplitude and varying patterns differing from any kind of organic tremor with a pause when she was distracted, she was diagnosed as psychogenic movement disorder, rather than a

transient ischemic attack. A placebo treatment of 500cc/day 0.9 sodium chloride was administered intravenously, and her speech began to normalize abruptly within approximately 10 minutes, and a full recovery was seen in the patient within 24 hours.

DISCUSSION

PMDs which can be defined as “functional”, “hysterical” and/or “nonorganic” movement disorders represent a diagnostic and management challenge in neurology practice (5). According to the organic clinical phenomenology they mimic, a wide-spectrum of differential diagnosis is needed for accurate diagnosis and proper approach. They should be diagnosed where possible on the basis of positive aspects of the history and examination whereas a detailed observation can give us the basic clues from the beginning of the examination including phenomenology with mixed movement disorders, paroxysmal attacks, in-clinic variability and distractibility (3,5). Moreover, an abrupt onset with variability over time and accompanying stressor factors or concomitant psychopathology in clinical history are the supporters of PMDs (5). Since PMDs are based on clinical diagnosis, some instrumental findings including electromyography, neuroimaging techniques and laboratory investigations can be helpful to differentiate these disorders from organic etiologies, and neurological movement disorders (5,6).

As in our patient, tremor is the most common form of PMDs (7). Differing from the organic forms of tremor, tremor with psychogenic origin has a tendency to be of equal magnitude at rest, during maintenance of posture, and while performing an action. It can abolish with distracting maneuvers, and can suppress during contralateral movements (8).

Since our patient had an abrupt onset of tremor like involuntary movements in her right limb with variable amplitude, direction and frequency in time, which was abolishing during voluntary tasks, and when she was distracted, this involuntary movement was considered as psychogenic tremor. Her depressive mood was supporting our diagnosis of PMD, as well as her bizarre gait with knee-buckling and speech disturbance. Her

speech was full of bursts of verbal gibberish, and varying dialects in some parts which was turning to normal during voluntary activity and distracted. Although, she was suspected as an acute cerebellar syndrome and hospitalized with the pre-diagnosis of stroke and transient ischemic attack, her clinical manifestation was not compatible with an organic etiology. She had normal neuroimaging which gave us a chance to exclude stroke. We excluded Wilson disease while there was no Kayser-Fleischer ring in our patient, and his serum and 24-hours urinary copper levels were also found to be normal, as well as serum ceruloplasmin levels. Since there was no acanthocytes in the peripheral blood smear of the patient, neuroacanthocytosis was also excluded. As the abrupt onset and rapid, spontaneous remission of these bizarre symptoms were the hallmarks of PMDs, and the laboratory findings with neuroimaging were normal, the patient was diagnosed as a case of PMD.

Since the suggested treatment options for PMDs include non-pharmacologic interventions including cognitive-behavioral therapy, psychodynamic psychotherapy, physical and occupational therapy, transcutaneous electrical stimulation, and acupuncture, as well as pharmacologic interventions including antidepressants (9), we referred our patient to a psychiatric evaluation.

As PMDs are common and disabling issues in

clinical practice, it is important to be aware of diagnostic clinical clues of these disorders in order to differentiate them from organic etiologies, and give accurate treatment. In patients with depressive and/or anxious mood, involuntary movements which are not compatible with neurological and/or anatomic features or distribution, PMDs must be kept in mind in the differential diagnosis. Although prognosis can be poor, associated with delayed diagnosis, treatment with pharmacologic or non-pharmacologic approaches including physical and/or cognitive-behavioral methods can be helpful. Since a developing understanding of the pathophysiology of these disorders has moved away from pure psychological explanations in favor of broader bio-psycho-social models, a collaborative approach between neurologists and psychiatrists is essential.

Contribution Categories	Name of Author
Follow up of the case	A.O., Y.D.
Literature review	Y.D.
Manuscript writing	Y.D., A.O.
Manuscript review and revision	Y.D., A.O.

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