Mal de Debarquement Syndrome Complicated by Psychiatric Comorbidities

Özge Ceren Amuk Williams, MD[a]; Emmanuelle J. Caraballo-Rivera, MSc[b]; Sathya Narasimhan, MBBS[c]; Ashrith Challa[d]; and Anil K. Bachu, MD[e,*]

Mal de debarquement syndrome (MdDS), which implies concretely "landing sickness" in French, is a rare neurologic condition of prolonged subjective perception of self-motion from 3 days up to several years that occurs after exposure to passive motion, in most cases accompanying sea travel, or rarely spontaneously.[1,2] The common presentation of patients with MdDS includes symptoms with a continued sensation of motion and imbalance in the forms of rocking, bobbing, and swaying. MdDS is a diagnosis of exclusion and currently has no conclusive treatment. Confirmation of the diagnosis requires a neuropsychiatric history, clinical examination of all systems, and vestibular function tests and brain imaging to exclude other structural causes. The therapeutic options for prolonged symptoms include benzodiazepines, selective serotonin reuptake inhibitors, stress reduction, and physical and vestibular therapy.[3] We present the case of a man who presented with these symptoms and whose follow-up was complicated by psychiatric comorbidities.

Case Report

A 30-year-old man presented to the neurology clinic with episodes of dizziness and poor balance. The patient stated his chief complaint as, "I felt like I was in a boat and had twitching of both arms, legs, and toes." The neurologic examination showed no findings indicating central vertigo, vestibular neuritis, or benign paroxysmal positional vertigo. The electrodiagnostic tests showed mild carpal tunnel syndrome. The neurophysiologic examination showed that the patient manifested 0.2 Hz of rocking and 0.5 Hz of sway, with an amplitude of ± 20 mm in static posturography frequency and 0.67 Hz of rocking in mimicking movement frequency. His cranial and cervical spine magnetic resonance imaging (MRI) scans excluded intracranial tumors, vestibular schwannoma, neurovascular conflicts, and demyelinating and inflammatory lesions such as in multiple sclerosis or cerebellitis. This clinical picture led to a high-grade clinical suspicion of MdDS due to stress-induced vertiginous symptoms.

One month after symptom onset at the neurologic outpatient visit, he was prescribed amitriptyline 25 mg daily. After 6 months, the patient underwent optokinetic stimulation over 4 days in a neurology clinic and responded well, as no post treatment static posturography frequency was detectable.

However, the alleviation of symptoms with amitriptyline diminished after 2 years of treatment, for which a follow-up visit at that time was arranged. At the follow-up visit, the patient reported multiple complaints including constant vertigo, ear pain, mild tremors in both upper extremities, tightness in the neck, numbness, weakness of the left arm from shoulder to elbow, "constant buzzing" of both feet, and chest pain. In addition, he complained of weight gain of 22 lb, which was determined to be a side effect of amitriptyline, and dysphoric mood, for which a change of medication was suggested. These diffuse neurologic symptoms were not related to a central sensory or motor deficit. The episodes of vegetative and thoracic symptoms and unspecific paresthesia were evaluated clinically as anxiety attacks. The neurologic examination was normal at the time of the visit. Referral to the psychiatry clinic and vestibular rehabilitation were recommended through the evaluating neurologist.

Five months later, the patient presented to the psychiatry clinic with depression and psychomotor agitation and a constant sense of motion and intermittent anxiety. The patient took his medications regularly and received outpatient physical and vestibular therapy. Amitriptyline was discontinued due to weight gain. Venlafaxine 37.5 mg daily was prescribed for anxiety and the depressive component of his symptoms, as he had previously taken a selective serotonin reuptake inhibitor with minimal benefit. Clonazepam 0.5 mg nightly as needed was prescribed for severe anxiety and panic attacks. He was provided psychoeducation and recommended to start cognitive-behavioral therapy (CBT). The patient responded well to the combination of venlafaxine and CBT. Anxiety and the depressive component of his symptoms remitted. He tolerated the episodes of panic attack well and was prescribed clonazepam 0.5 mg to take during those episodes at the suggestion of his prior neurologist. The

[a]Department of Psychiatry, Ozark Center, Joplin, Missouri
[b]Ponce Health Sciences University, Ponce, Providence, Rhode Island
[c]Kakatiya Medical College, Telangana, India
[d]University of Pennsylvania, Philadelphia, Pennsylvania
[e]Department of Behavioral Health, Baptist Health, North Little Rock, Arkansas
*Corresponding author: Anil K. Bachu, MD, Department of Behavioral Health, Baptist Health, 3500 Springhill Dr, North Little Rock, AR 72117 (anilkbachu@gmail.com).

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patient denied side effects from venlafaxine or clonazepam and was recommended to stay in neurologic follow-up for the episodes of constant sense of motion. No further psychiatric follow-up visits were required as of this writing.

Discussion

MdDS is a diagnosis of exclusion for episodic dizziness and disequilibrium. Since the physical examination and diagnostic findings are negative, the unexplained dizziness can be misinterpreted as a functional neurologic syndrome or as a manifestation of generalized anxiety.[4,5] Therefore, a careful assessment of the psychiatric symptoms with their onset, as well as situational correlates, and evaluation of psychological traits are significant to detect a potential underlying neurologic cause.

The patient presented here had the tendency to have anxious thoughts due to job-related stress long before the presenting neurologic symptoms. The vertigo precipitated his trait of anxiety, which was treated with benzodiazepines during the panic attacks.

Many clinicians are unaware of MdDs, and patients often consult an average of 19 clinicians before receiving a diagnosis of MdDS, which significantly decreases the patient's quality of life.[6] The diagnostic distinction between non–motion-triggered MdDS and persistent postural perceptual dizziness is unclear and requires in-depth history taking.[7] The history should include questions regarding the chief complaint, the timing of symptoms, and exacerbating and alleviating factors such as movement or position, which could contribute positively to the diagnostic evaluation. In this case, the initial evaluating neurologist pinpointed the diagnosis of MdDS due to the patient's young age, normal clinical examination, absence of acute neurologic disease, and prolonged sense of imbalance over weeks.

There are several different hypotheses regarding the pathogenesis of MdDS. According to one theory, MdDS is a neuroplastic and vestibular adaptation condition.[8] Additional explanations include genetic susceptibility and visual-vestibular conflict.[9] Recent insights into the biological basis of MdDS using functional MRI and 18-F fluorodeoxyglucose–positron emission tomography showed that patients with persistent MdDS have increased glucose metabolism in the amygdala and the left entorhinal cortex and decreased metabolism in the left prefrontal and temporal cortices.[10]

Due to the rarity of MdDS, the clinical guidelines for the treatment steps are lacking. A case series showed that amitriptyline from 25 mg/d to 150 mg/d resulted in consistent remission of MdDS symptoms, although it was unclear if the effect was psychogenic or due to a neurotransmitter regulation in the vestibular system.[11] Similarly, after the clinical diagnosis, our patient was started on the reported minimum effective dose of 25 mg/d, and his symptoms were alleviated for approximately 2 years at the time of this writing.

Conclusion

Our case report emphasizes the necessity of early diagnosis and management of MdDS to prevent the chronification or exacerbation of the vertiginous complaints, leading to increased clinical consultations and worsening of depressive symptoms. We aim to increase awareness among clinicians to be able to identify this underrecognized condition in the early stage and manage MdDS efficiently to reduce the burden on the health care system.

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