PHOBIC POSTURAL VERTIGO

A cognitive-behavior approach

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Dizziness is one of the most common symptoms in primary care and can be associated with otologic, neurologic, and psychiatric conditions^{1,2}. Phobic postural vertigo (PPV) is a specific anxiety-related, chronic and incapacitating condition with subjective imbalance and short attacks of dizziness, and has been described as a prevalent cause of chronic subjective dizziness that cannot be sufficiently explained by patient's vestibular condition³. Behavioral therapy, vestibular rehabilitation and serotonin selective re-uptake inhibitor antidepressants (SS-RIs) have been pointed as useful treatments⁴. Few cognitive-behavior (CBT) studies for chronic dizziness have been described.

We report a patient with chronic dizziness that only achieved complete symptom relieve after adding CBT to the conventional medication treatment.

CASE

At the age of 17 this male, patient presented vestibular neuritis episodes due to peripheral vestibular dysfunction, diagnosed by otolaryngologic examinations that showed predominance of the left ear. Differential diagnosis was made by neurological examinations, blood biochemical tests, EEG, MRI, audiometry, negative Dix-Hallpike maneuver responses and absence of nystagmus. He responded positively to treatment with flunarizine, achieving complete relief of symptoms in few weeks.

Eight months later, the complaints of dizziness returned with atypical presentation that could not be explained by remaining vestibular lesions. Episodes occurred several times a day, lasting 3–10 seconds and followed by an intense sense of fear, which turned out to be very incapacitating. He completely withdraw from daily activities, including school, avoided moving the head, reading and being alone, and adopted safety behaviors such as only leaving the house with a family member.

The patient was medicated with sertraline 50 mg/day and clonazepam 2 mg/day, and referred to a clinical psychologist

(A.S.) for CBT. He fulfilled the DSM-IV criteria for agoraphobia, but not for panic disorder, since only dizziness, rarely accompanied by tachycardia, was present during the episodes. Patient was clinically diagnosed with PPV⁵.

Treatment sessions were conducted once a week. The initial goal was to reduce the sense of vulnerability and self-devaluation. Cognitive interventions focused on putting this unpleasant life moment in perspective and making plans for the short and the long run. Euthymic mood was yield after 8 weeks of treatment.

Information on the etiology of the symptoms and how discomfort was caused by the experience of anxiety was emphasized. He was instructed on how avoidance and hypervigilance behaviors maintained the problem, as the attention focused in every balance change triggered anxiety responses and dizziness, and realized that focusing on the occurrence of symptoms worsened discomfort. Although it restricted the evaluation of intensity and duration of the crisis to the patient's subjective reports, objective monitoring was thus discouraged by the therapist.

Once the patient perceived the symptoms as uncontrollable, he was encouraged to slowly resume his activities, despite the dizziness. Each week the patient endured one or more activities previously avoided. The rationale for this intervention was that: focusing on other activities would distract him from the symptoms and break the hypervigilance cycle⁶; slowly moving his head again in a daily-activity level could help promote internal ear habituation and desensitization⁴, and exposure therapy is the gold-standard treatment for agoraphobia⁷.

Exposure therapy was proposed to the patient. After two weeks, he resumed activities inside the house, such as reading or using the computer. On the third week, he was encouraged to leave the house with a family member. His sense of self-efficacy was sharply increased and the patient started to feel less vulnerable. After four weeks, he reported decreased anticipatory anxiety of the episodes and a reduction in their intensity, but not on its frequency. At the same time, he stopped experienc-

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ing dizziness episodes every time he left the house. In the fifth week, the patient initiated private lessons at home.

Symptom relief concurred with the ninth week of treatment. Along with a decrease in the frequency and intensity of the episodes, an improvement in his subjective sense of well-being has reported. In the eleventh week, dizziness symptoms completely disappeared. The otolaryngologic examinations showed no more alterations. On the sixtieth week of treatment, he started going out alone. Passed two more weeks with no symptoms, therapy was interrupted (18 weeks).

DISCUSSION

This case is of high interest in two senses: it describes the CBT management of PPV, as well as secondary agoraphobia, and also contributes to the discussion on the interaction between otologic and psychiatric symptoms, as the patient presented an actual vestibular dysfunction that we consider to be the trigger to agoraphobia and PPV.

Although the first presentation of the vestibular dysfunction responded to regular pharmacologic treatment, it draws attention due to the association of the initial dizziness with anxiety symptoms. The co-occurrence of anxiety may be thought as a clinical indicator for maintenance of dizziness after treatment, as patients who associate vestibular symptoms to anxiety at first may be more prone

to present chronic dizziness and PPV. It also sheds light over the possibility of reducing the dizziness symptom.

Despite the methodological limitations of a case report, that does not allow the conclusion that CBT intervention caused the observed improvement in symptoms, and the lack of objective outcome data due to the therapeutic focus of the intervention, the positive results yield provide additional supporting evidence for the current preliminary literature recommendations for associating CBT to the treatment of chronic dizziness.

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