

Dissociative Symptoms Secondary to Piracetam: A Case Report

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ÖZET:

Pirasetam kullanımı sonrası gelişen dissosiyatif belirtiler: Bir olgu sunumu

Pirasetam nöroloji pratiğinde sık kullanılan gama-aminobütirik asit türevi bir ilaçtır. Pirasetamın antitrombotik, nöroprotektif ve bilişsel işlevleri düzeltici bir özellikleri vardır. Vertigo pirasetamın kullanım endikasyonlarından biridir. Disosiyasyonun etyolojisi tam olarak aydınlatılamamıştır. Yapılan ilaç çalışmalarında ortaya çıkan temel disosiyatif semptom depersonalizasyondur.

Bu yazıda periferik vertigo tanısı alan ve eklem tedavisi olarak pirasetam eklenmiş ancak kullanımından hemen sonra depersonalizasyon ve derealizasyon gibi dissosiyatif semptomlar gelişen ve pirasetamın kesilmesinden sonra bu şikâyetlerin kaybolduğu bir olgu sunumu yapılmıştır.

Anahtar sözcükler: Pirasetam, vertigo, disosiyatif belirtiler

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ABSTRACT:

Dissociative symptoms secondary to piracetam: a case report

Piracetam is a cyclic derivative of gamma-aminobutyric acid that is often used in neurology. Piracetam is an antithrombotic, neuroprotective agent which improves cognitive performance. Piracetam is indicated for vertigo in clinical practice. The etiology of dissociation is not precisely defined. In pharmacotherapy studies, depersonalization is the basic dissociative symptom.

In this article, we have reported a case with the diagnosis of peripheral vertigo where piracetam was used as part of combination therapy. However dissociative symptoms like depersonalization and derealization occurred after piracetam use and these symptoms disappeared after discontinuation of piracetam.

Key words: Piracetam, vertigo, dissociative symptoms

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INTRODUCTION

Piracetam is a cyclic derivative of gamma-aminobutyric acid that is often used in neurology (1). Piracetam is an antithrombotic, neuroprotective agent which improves cognitive performance such as learning, memory, attention and consciousness (2,3). Furthermore the clinical benefit of piracetam has been shown in vertigo of both central and peripheral origin (4). In this article, we have reported a case with dissociative symptoms like depersonalization and derealization that occurred after piracetam use as part of combination therapy for peripheral vertigo.

CASE REPORT

A 29-year-old female patient was admitted to our psychiatric outpatient clinic with complaints such as intense discomfort, perceiving herself to be odd, erroneous perception of oversized hands and feet, a feeling of being separate from society, having perceptual defects with respect to colors and dimensions of objects and feelings of alienation from subjects and people in her surroundings. As reported by the patient, these complaints had continued for 10 days and she had never had any psychological complaints before. She also stated that she had been extremely bothered by these

involuntary complaints. Except for these complaints, she did not describe any abnormalities in perception or thought content. She did not have prominent depressive complaints or psychosocial stress in her history. One month previously, she had visited a neurologist because of vertigo; peripheral vertigo was diagnosed and she was prescribed betahistine (Betaserc®) 16 mg/day. Upon use of this medication, she described a marked decline in vertigo symptoms, however since she was not clear of her complaints completely, she was prescribed combined therapy with the addition of piracetam 2400 mg/day on 12th day of her treatment.

On psychiatric examination, she was conscious, normally oriented, anxious and irritable; she presented with depersonalization and derealization; psychomotor activation was normal. A comprehensive chemistry profile, blood counts and thyroid function tests results were all normal. Brain Magnetic Resonance Imaging (MRI) and Electroencephalography (EEG) findings were normal also.

In clinical follow-up, since there was a connection between the patient's administration of piracetam and dissociative indications, use of piracetam was stopped. Betahistine administration was continued at the same dosage. Her dissociative symptoms, that were observed to be on the wane during her "next-day" follow up, totally disappeared in 5 days. During later visits, dissociative symptoms were not observed.

DISCUSSION

The dissociative symptoms like derealisation and depersonalization started upon addition of piracetam to the treatment, and diminished quickly following the discontinuation of piracetam. We considered those dissociative symptoms to be related to the piracetam administration. This relationship has not reported in the literature. The listed adverse effects of piracetam are anxiety, insomnia, agitation, irritability and sleep disturbances (5). Drug-drug interactions have been

reported with warfarin (6). In the literature, piracetam is mentioned to be used for the treatment of cognitive dysfunction in organic personality syndrome and has been reported to worsen behavioral symptoms (3). Its mechanism of action is not related to that of GABA, but is associated with modulation of neurotransmitter function and activation of the alpha-amino-3-hydroxy-5-methylisoxazole-4-propionate type of glutamate receptors and membrane-stabilizing properties (3,7).

Trauma is usually reported to be the root cause of dissociative symptoms. This is generally a trauma experienced in childhood and dissociative symptoms occur in later life. In our case, after piracetam was discontinued, the dissociative symptoms disappeared. Thus, we did not consider this explanation. There is still no etiopathogenesis described related to the development of dissociative symptoms. Traumatic stress and neurobiological theories are the principal models suggested for the etiology (8). In medication studies, depersonalization is the basic dissociative symptom in the neurobiological theory of dissociation. In those studies, it was also shown that serotonergic and glutamate receptors had a role in depersonalization (8). The mechanism through which piracetam might cause dissociative symptoms, merits further investigation. Furthermore depersonalization and derealization symptoms were justified in this case due to severe drug-induced anxiety. The side effect of anxiety is related to piracetam's being a derivative of GABA. But our case did not have severe anxiety and this issue is open to debate. Further case series and case reports are required to examine the association between piracetam and dissociative symptoms. Therefore, if neurobiological theories of dissociation are better understood, piracetam induced dissociative symptoms may be further explained.

As a result, this case report shows that the development of dissociative symptoms upon use of piracetam might contribute to the neurobiological theories about the etiology of dissociative disorder.

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