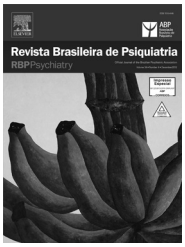
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Letter to the Editors

Depersonalization and derealization syndrome: report on a case study and pharmacological management

Dear Editor,

Depersonalization disorder (DPD), a chronic condition characterized by a profound disruption of self-awareness, appears to be more common than previously thought and may possibly affect 1% of the general population.[1,2] DPD usually presents with severe distress and functional disability. Because some of the symptoms may resemble those of psychotic and anxiety disorders (affect numbing, disembodiment, and social anxiety), DPD is often misdiagnosed.[1,2] There is no established treatment for this disorder.[2] We describe a case that illustrates the particularities of DPD and proposals for treatment.

M., 27, single, with a university degree, sought psychiatric treatment with complaints that she "did not feel her body". She reported that she felt strange and empty, that her body seemed to be somewhere else and hollow, with nothing but the skin, and it seemed to be someone else's body. She had come to the point of wearing numerous bracelets to mark the boundaries of her own limbs. She also suffered from affective detachment, frequently stating “I feel like I was dead” or “I feel nothing”, but complained of intense anxiety in social situations. Reality testing was intact. Slightly depressed mood and mitigated panic-like symptoms were also identified; however, she did not fulfill the criteria for any other DSM axis I disorder, as confirmed by the MINI-plus. There were no comorbid conditions or history of drug abuse. She had a normal neurological examination, and an EEG showed no abnormalities.

As the patient did not respond to risperidone 2 mg/day, it was replaced with a selective serotonin reuptake inhibitor (SSRI), which led to anxiety improvement, but the specific symptoms of DPD grew worse. A subsequent change to venlafaxine 225 mg/day led to a significant mood improvement and a reduction in panic-like episodes; however, depersonalization and derealization remained unchanged.

Lamotrigine was then added at an initial dose of 25 mg/day, with a gradual increase to up to 200 mg/day. The patient had significant improvement in different aspects, such as affect, interpersonal contact and social interaction. The depersonalization symptoms gradually decreased, which made it possible for the patient to go back to work. During that period, a Portuguese version of the Cambridge Depersonalization Scale (CDS) was used.[3] The scale contains 29 items, each one rated from 0-10, and a score of > 70 suggests the disorder. Before the introduction of lamotrigine, the patient scored 230. After 12 weeks of 100 mg/day and 12 weeks of 200 mg/day, her scores were 198 and 175, respectively, which represents an overall reduction of 24% compared to the initial evaluation.

The literature on DPD shows that it is often refractory to pharmacological approaches.[2,5] Lamotrigine, a glutamate release inhibitor, seems to be a promising alternative because it has been shown to reverse depersonalization-related phenomena induced by the N-methyl-D-aspartate (NMDA) receptor antagonist ketamine in healthy individuals.[2,5] A recent placebo-controlled study showed that a 12-week treatment regimen of lamotrigine produced a good response in male patients with less intense symptoms than our patient (CDS baseline mean = 185).[4] This result contrasts with a smaller previous study, in which patients of both genders (CDS baseline = 128.4) showed no improvement.[5] Further studies are required to define whether there is a specific patient profile that predicts a good response to lamotrigine. Our case report provides evidence that lamotrigine may also be beneficial, albeit partially, in female patients and those with more severe symptoms.

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Disclosures

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** Modest*
*** Significant*
**** Significant. Grants were not awarded directly to the author but rather to a co-researcher or to the author's employer.*

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