**INTRODUCTION**

Pregabalin is a gamma-amino butyric acid (GABA) analogue approved for the treatment of partial-onset seizures and neuropathic pain in Turkey. It is also approved for the treatment of generalized anxiety disorder in some European countries (1). It has been suggested that pregabalin is likely to be associated with an abuse potential (2); however, case reports about pregabalin abuse or dependence are rather scarce (3). In Turkey, prescribing pregabalin is not under the control of legislation. Patients can buy the medication from a pharmacy without a prescription. Available formulations of pregabalin in Turkey contain 75, 150, and 300 mg of pregabalin. To our knowledge, this is the first case report of pregabalin abuse in Turkey.

**CASE**

Mr. S. was a 37 year old man seeking treatment for clonazepam abuse. He had a history of grand mal epilepsy for ten years, bipolar disorder for five years and clonazepam abuse for two years. His EEGs had revealed epileptic discharges originating from the left temporal lobe. He was on oxcarbazepine 1800 mg/day which he had been taking for almost ten years. He was still having epileptic seizures 4-5 times per month. He reported having two hypomanic episodes, five and two years ago. These episodes were not triggered by antidepressants, and he was treated with risperidone as an outpatient. He also reported having numerous depressive episodes since his adolescence. Ten years ago he abused ketamine for one year. He reported...
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using cannabis several times but did not like it. He did not have any problems with alcohol, drinking one or two beers occasionally. His father, uncle, and aunt had a history of alcohol dependence. He had been taking lithium irregularly for five years. His plasma lithium level was detected as 0.28mEq/L during the first visit.

He reported that a psychiatrist prescribed clonazepam two years ago to relieve his anxiety and help with epileptic seizures. Initially clonazepam provided relief for both conditions, however later he started taking it irregularly 1-2 times a week to get high. He increased the dose of clonazepam to 60 mg/day, using the entire bottle on the day he got the prescription for clonazepam. His longest period of abstinence from clonazepam was three weeks. He experienced blackouts while on clonazepam.

We switched from oxcarbazepine to valproate gradually. We obtained a valproate blood level of 81.2 ng/ml. As he continued complaining about anxiety, we added pregabalin to his regimen and titrated the dose up to 300 mg/day. He started individual addiction counseling 1-2 times per week. Two months after starting pregabalin, his wife realized that he was abusing pregabalin. The patient admitted that he had consumed approximately 20 capsules (equivalent to 3000 mg) of pregabalin on 6-7 occasions. He said he felt euphoria while on high doses of pregabalin and did not have any blackouts or disturbing behaviors. He took this medication after his wife went to bed and experienced euphoria until he fell asleep several hours later on a couch in the living room, where he was discovered by his wife in the morning. He reported that two days after taking the high dose of pregabalin he had more severe anxiety symptoms that decreased to previous levels in a couple of days. He agreed to be given the pregabalin by his wife and he did that for a month. As pregabalin can be obtained from pharmacies without prescription in Turkey, such a measure could not control his access to the drug.

Six months after the episodes of pregabalin abuse, he was taking valproate and pregabalin regularly at recommended doses. He did not have any signs of alcohol or drug abuse. He continued to see the addiction counselor once a month. He learned alternative behavioral techniques to manage his anxiety. His mood had been euthymic since he was on valproate. He still had occasional epileptic seizures, but significantly less frequently.

DISCUSSION

Gamma amino butyric acid (GABA) is the major inhibitory neurotransmitter of the central nervous system. Ethanol, benzodiazepines, and some anticonvulsant drugs directly affect GABA receptors and induce similar anxiolytic, sedative/hypnotic, and anticonvulsant effects. Pregabalin is a novel medication that modulates GABA-ergic neurotransmission. Pregabalin has been found to be effective in treating withdrawal symptoms at doses of 150-450 mg/day for alcohol dependence and 225-900 mg/day for benzodiazepine dependence (4). Due to its lower abuse potential than that of the benzodiazepines, pregabalin augmentation could be beneficial in the treatment of anxiety disorders unresponsive to other medications, especially in patients with comorbid substance use disorders (5).

Our case, however, cautions us to be careful when using pregabalin in treating patients with a history of drug or alcohol dependence. The abuse potential or addictive nature of pregabalin is still controversial in the literature. Pregabalin is likely to be abused for its euphoric effect. Its GABA-ergic effects may cause positive reinforcement in some patients; however, these effects are weak and not sustained during long-term use (6). The fact that our patient was able to control his pregabalin use, despite his long history of benzodiazepine abuse, supports the notion that its abuse potential might be lower than that of benzodiazepines (7).

References:

