Topiramate Add-On for Treatment of Migraine-Type Headache Cures Alcohol Dependence: A Case Report

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ABStract:
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Topiramate presents a broad-spectrum efficacy in the treatment of different psychiatric and neurological conditions, as suggested by findings from studies on addiction, epilepsy, and migraine headaches. Here, we present a patient with a diagnosis of alcohol dependence that was treated with topiramate for migraine headaches as an add-on treatment to psychiatric medications. Alcohol craving and consumption were significantly reduced. Additionally, the patient’s migraine symptoms improved, without the appearance of side effects.

Keywords: topiramate, alcohol dependence, migraine-type headache, add-on treatment, male

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INTRODUCTION

Topiramate (TPM) is an anticonvulsant known to contribute to the treatment of epilepsy and use in the prevention of headaches. Recently, TPM has been reported to be effective also in bulimia nervosa, binging disorders (1), smoking addiction (2), and alcohol dependence (3). Since 2003, several randomized controlled clinical trials have examined the value of TPM in the treatment of alcoholism (4). The following case involves a 28-year-old male patient diagnosed with alcohol dependence who was treated with TPM for migraine headaches as an add-on treatment to psychiatric medications.

CASE PRESENTATIONS

A 28-year-old man was admitted to a neurology outpatient clinic with a complaint of headache, which was diagnosed as migraine. He had a history of alcohol consumption for the last 6 years and had developed the features of salience, tolerance, craving, and loss of control over drinking over the last 3 years. The patient was referred to psychiatry for alcohol dependence and was treated with venlafaxine (225 mg/day) and quetiapine (300 mg/day). This regimen did not improve his alcohol problems. After initiation of TPM for migraine headaches—and gradually increasing the dosage—the patient experienced reduced craving and alcohol consumption. At a dose of 200 mg/day of TPM, his headache resolved and the need for pain relievers ceased. After a month of this TPM treatment (200 mg/day), and with the continuation of the previously mentioned psychiatric treatment, the patient’s alcohol dependence behaviors changed. His six-month follow-up showed his alcohol dependence to be cured. Neurological and psychiatric consultation led to the decision to maintain the treatment with the TPM add-on to venlafaxine and quetiapine for one full year.
DISCUSSION

When the results of the three randomized and placebo-control clinical trials were combined, TPM was found more effective than a placebo in the objective measurements of consumption, gamma-glutamyltransferase (GGT) levels, and the self-reported measures of alcohol use and the percentage of high consumption days and abstinence days (4).

Alcohol exerts reinforcing effects on cortico-mesolimbic dopamine pathways through the disinhibition of the inhibitory effects of gamma-aminobutyric acid-A (GABA-A) neurons in the ventral tegmental area (VTA). The neuropharmacological actions of TPM include facilitation of the neurotransmitter GABA’s inhibitory action in its non-benzodiazepine receptor and the reduction of the glutamate excitatory action in the alpha-amino-3 hydroxy-5- methylisoxazole-4 propionic (AMPA) receptor and the kainate receptors (5,6), thus reducing neuronal excitability. TPM shows no apparent effect on the NMDA glutamate receptors, but it seems to reduce the mesolimbic cortical activity of dopamine. This appears to be the principal mechanism for decreasing alcohol consumption by tempering its rewarding effects (7,8). In alcohol-dependent patients, doses of up to 300 mg/day have been found effective for reducing cravings. The optimum dose of TPM for the treatment of alcohol dependence has not been established (four of the five trials, including the three placebo-controlled ones, used 300 mg/day). As the side effects of TPM increase with dose, a study on its efficacy at different doses is necessary (4).

The efficacy of TPM has also been compared with two drugs approved for alcohol use disorders: disulfiram and oral naltrexone. Three studies suggested that TPM might be more effective than standard doses of oral naltrexone. On the other hand, a study with disulfiram showed it to be more effective than TPM. However, in that trial, the TPM dose was 150 mg/day, which is half that was used in the other trials and in this case. In any event, the value of disulfiram versus TPM needs additional research at different doses (9).

TPM has been found to reduce the days of elevated intake, increase the days of abstinence, and improve GGT levels in patients with alcohol dependence (4). TPM has also been shown to reduce alcohol craving and heavy drinking and to improve abstinence among alcohol-dependent individuals. TPM reduces mesocorticolimbic dopamine activity by potentiating the inhibitory effects of gamma-aminobutyric acid (GABA) and depressing glutamate receptors, which are crucial pathways by which alcohol exerts its rewarding effects (7,8).

CONCLUSION

This case reveals that patients with psychiatric and/or neurologic problems having concomitant alcohol dependence or alcohol problems might benefit from TPM add-on treatment. Additional research is needed to establish the optimum doses and TPM’s utility in different alcoholism subtypes.

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