
Letter to the Editor

A CASE OF OXYBUTYNIN ABUSE

Dear Editor,

Oxybutynin is an antimuscarinic medication used to relieve urinary and bladder difficulties, including frequent urination. When administered in high doses, anticholinergic agents cause symptoms that include impairment in cognitive function, confusion, orientation disorders, pressurized speech, delirium, hallucinations (most frequently visual and more rarely auditory and tactile), paranoid thoughts, an increased self-confidence, insomnia and lack of appetite (Hines et al. 2006).

In this article, we state that an antimuscarinic medication containing oxybutynin taken to suppress symptoms related to depression and social anxiety constitute a risk of drug abuse and addiction. Also, we state that its easy accessibility is an important factor paving the way for this risk.

CASE

A male 22 years old, single patient that was a graduate of secondary school and was employed, applied in May 2013 to the Ankara Atatürk Training and Research Hospital psychiatry polyclinic. He stated that he has been consistently taking

a drug containing oxybutynin and wished to discontinue the medication. The patient started to take an oxybutynin-containing drug 2.5 years ago during his military service upon recommendation of a friend in order to get rid of sorrow and to be companionable and talkative in social environments, and had used this drug regularly until 2 days prior to his application to our polyclinic. Whenever he tried to discontinue the medication, he had complaints that included feelings of malaise, aversion, nervousness, inattentiveness, indisposition to speaking, introvertedness, and difficulty in doing his jobs; because of these effects, he restarted the medication. With these complaints, the patient could easily buy this drug over-the-counter from a pharmacy. He had used 5-10 mg/day for the initial three months, and continued to take 100 mg/day (20 tablets). When he used the drug, he felt courageous, companionable, talkative and energetic, which lead to his continuation of the medication. While using the drug at a dose of 100 mg/day, he experienced symptoms that included soliloquizing, insomnia, lack of appetite, and unwillingness to speak to friends. Therefore, two months prior to applying to our polyclinic, he went to another psychiatrist and started olanzapine 5 mg/day, but had not begun the medication. His background does not contain any trauma or medical disease. He smoked (1 pack/day) and used alcohol (2 beers/week). His family history does not contain any psychiatric disorder, other than a brother that smoked marijuana.

During his psychiatric examination he was conscious and oriented to place, time and person. His memory, attention tests and intelligence were clinically normal. His affect was sorrowful and anxious, and speech was of low rhythm and mostly in question-answer mode. Reduction was detected in thought content and self-esteem. Also, a reduction was detected in

psychomotor activity. He described visual and audio hallucinations upon use of drug. He had difficulty falling asleep and maintaining sleep, and experienced a lack of appetite.

No anomaly was detected in routine blood tests. Results of brain magnetic resonance imaging and electroencephalography were declared normal.

Upon arrival, the patient's Hamilton depression score was 20 and Hamilton anxiety score was 13. Total performance score obtained from Weschler Memory Scale (WMS) was within normal limits, and no pathology was observed regarding memory functions. Attention and attention-maintenance functions were within normal limits. Minnesota Multiphasic Personality Inventory scoring indicated depressive, hypersensitive to criticism, and paranoid orientations.

With a provisional diagnosis of depressive disorder, fluoxetine 20 mg/day and olanzapine 2.5 mg/day treatments were started, and polyclinic follow-up was continued. One week after first application, the patient reported no withdrawal syndrome other than a sensation of nausea. The sensation of nausea disappeared after two weeks, and one-month-later the polyclinic examination revealed a Hamilton depression score of 8 and a Hamilton anxiety score of 6. Three months later, an evident improvement was observed in the patient's depressive symptoms and anxiety signs, and he no longer used the drug.

DISCUSSION

Depression and anxiety-linked symptoms that are observed, but not diagnosed constitute an important reason for encouraging the use of self-treatment with drugs and substances due to their anxiety-sedating and euphoria-causing effects (Uzbay 2009). Therefore, such anticholinergic agents as oxybutynin which are prone to addiction or abuse and may easily pass to the central nervous system increase acetylcholine in the synaptic gap and if taken for a long time, increase neurotransmitters such as dopamine, serotonin and GABA (Pietzko et al. 1994). The agent, when taken, causes increase in dopamine in synaptic gap along with mesolimbic dopaminergic pathway elongating from the ventral tegmental area to the nucleus accumbens, which in turn reinforces the repeated desire of taking the drug; this pathway plays an important role in the development of addiction (Pietzko et al. 2006). Oxybutynin particularly affects the postsynaptic M1 receptor found mostly in the central nervous system. These receptors have important functions in regulation of memory functions

between cortex and hippocampus, in dopamine neurotransmission in the striatum, in locomotor activity, in reduction of the stimulant effect of amphetamine, and in epilepsy (Gerber et al. 2001). M2 receptors are mostly located in the brain stem and thalamus, and control acetylcholine release. Stimulation of M4 receptors has been determined to block D1 dopamine receptors in the striatum, and their activation at the midbrain area has been observed to suppress dopamine hyperactivity (Langmead et al. 2008). Due to these reasons, when taken in high doses, anticholinergic agents may lead to impairment in cognitive functions, confusion, lethargy, excitability, coma, impairment in memory, orientation disorder, agitation, pressurized speech, incoherence, inhibition findings in the parasympathetic nervous system (pupil dilatation, mouth dryness, difficulty in swallowing, weak quick pulse, ileus, urine retention), delirium with reduction of cholinergic efficiency and increase of dopaminergic efficiency, and hallucinations (most frequently visual and more rarely auditory and tactile), paranoid thoughts, and EEG anomalies.

In conclusion, oxybutynin-containing drugs that are not regulated, cost little, and are easy to access create an important risk for drug abuse. In cases where drug abuse is unlikely that present with anticholinergic findings, the use of oxybutynin and similar drugs must be taken into consideration in the differential diagnosis.

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