NÖRO PSİKİYATRİ ARŞİVİ

Letter to the Editor

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Reversible Anorgasmia with Acetazolamide Treatment for Idiopathic Intracranial Hypertension

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Dear Editor,

Idiopathic intracranial hypertension (IHH) is a clinical disorder characterized by symptoms and signs of increased intracranial pressure without abnormal cerebrospinal fluid composition and structural parenchymal abnormalities (1,2). There is no related systemic disorder and its etiology is unknown yet. Patients usually present with headache, vomiting, visual loss, and diplopia. Bilateral papilledema is found in most cases as a neurological sign and acetazolamide is an effective drug for the treatment of IIH.

Acetazolamide is a carbonic anhydrase inhibitor and has been used for many relatively common disorders such as congestive heart failure, some forms of epilepsy, glaucoma, IHH, and also for some rare diseases such as acute mountain sickness. Paresthesia, fatigue, taste alterations, vomiting, and polyuria are common side effects of the treatment with acetazolamide. Usually acetazolamide is well tolerated, and sometimes this treatment may be associated with anorgasmia and this effect is thought to be dose related. Organic impotence has been reported in glaucoma patient therapy with acetazolamide. In this paper, we report a case admitted to our hospital that developed anorgasmia during treatment with acetazolamide.

The patient, a 25-year-old female was admitted to the neurology department of our university hospital due to presence of anorgasmia. Previously, the patient had applied to a center for headache, nausea and vomiting, and visual loss, which had persisted for the past three months. The patient was evaluated with brain magnetic resonance imaging, in addition to examination of the cerebrospinal fluid and the other possible reasons of headache. Other possible causes of bilateral papilledema were ruled out; therefore, she was diagnosed with IIH and was started with appropriate treatment at our center. The patient reported that she was taking acetazolamide for IIH treatment orally during the last 15 days with an onset dosage of 750 mg/day, increased to 1500 mg/day after the 7th day of usage.

She was not using antidepressants, antipsychotics, or any other medications. The patient reported that the anorgasmia appeared during the last 5 days of acetazolamide treatment (three days after the onset of 1500 mg/days dosage). Except for the bilateral papilledema and light visual loss, the physical, psychiatric, gynecological, and neurological examinations as well as the brain magnetic resonance imaging were normal. Routine blood and hormonal tests were also normal. When the acetazolamide was decreased to 1000 mg/day, the anorgasmia resolved within 7 days.

Female orgasmic disorder is characterized by the persistent or recurrent delay in, or absence of orgasm following a normal sexual excitement phase. Anorgasmia is defined as failure to achieve orgasm (climax) during sexual intercourse. Anorgasmia has many causes, it is believed that approximately 90% of anorgasmia problems are related to psychological issues, and some cases may result from the use of certain drugs such as serotoninergic drugs including antidepressants (particularly selective serotonin reuptake inhibitors and serotonin-norepinephrine reuptake inhibitors), antiepileptic, and antipsychotic drugs (3,4).

The new onset of the patient's complaints, the lack of any additional drug use, and the absence of previous similar complaints suggested that the present anorgasmia was due to acetazolamide. We believe that acetazolamide treatment produced a dose-related anorgasmia in our patient. To the best of our knowledge, no other case of acetazolamide induced reversible anorgasmia has been reported previously for a female patient. It is difficult to present the precise mechanism between acetazolamide and anorgasmia.

Acetazolamide is an enzyme inhibitor that acts on carbonic anhydrase specifically and catalyzes the reversible reaction of hydration of carbon dioxide and dehydration of carbonic acid. Although the underlying neurochemical changes of anorgasmia are not fully understood, orgasm, in both sexes, is particularly controlled by noradrenergic activity. Serotonin and dopamine are also essential

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neurotransmitters for the propagation of sexual response (5). Perhaps inhibition of carbonic anhydrase interferes with production of vasoactive intestinal peptide and nitric oxide and subsequently reduces genital blood flow (6). Sexual dysfunction and anorgasmia are rarely observed due to carbonic anhydrase inhibitors (5,7,8). We suggest that the anorgasmia of our patient was related to the carbonic anhydrase inhibition caused by the use of acetazolamide.

A previous study shows that only one third of women with sexual dysfunction report their situation to a family physician or gynecologist. Female patients' sexual problems were understood by physicians due to questions posed by physicians in 80% of interviews, rather than the patients reporting themselves (9). Neurologists, cardiologists, and ophthalmologists require education, regarding potential dose-dependent acetazolamide-induced anorgasmia that may occur at high doses than previously reported.

In conclusion, this case demonstrates that the use of acetazolamide can lead to sexual side effects in some cases. However, prospective studies are needed to determine the frequency of this side effect. In addition, the benefits and risks of side effects in patients should be considered before the recommendation of acetazolamide.

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ERRATUM

In the article by Nurhan Fıstıkcı et al., entitled "Attentional Bias and Training in Social Anxiety Disorder" (Arch Neuropsychiatr 2015; 52: 4-7) that was published in the March 2015 issue of Archives of Neuropsychiatry, one of the contributing authors was erroneously omitted from the author list during the productiction process. Upon receipt of the written request of the contributing authors, the Editorial Board reviewed the case and approved the author list to be corrected as follows.

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