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Facial Edema that Develops with Olanzapine

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Olanzapine is an atypical antipsychotic drug that has been approved for use in the treatment of primarily schizophrenia and bipolar disorder and various other psychiatric disorders. Olanzapine use could cause peripheral edema in 3% of the patients. There are cases in the literature where a peripheral edema occurred after olanzapine mono-therapy. In these cases, edema occurred mostly in peripheral mode, in the lower extremities such as wrists, dorsum of the feet and in the limbs up to knees. However, there are only rare cases that reported edema in the facial region. This article will scrutinize a female case where a dense edema localized in the facial region restricting the movement of facial muscles after olanzapine treatment.

Keywords: Olanzapine; peripheral edema; facial edema.

1. INTRODUCTION

Olanzapine is an atypical antipsychotic drug that has been approved for use in the treatment of primarily schizophrenia and bipolar disorder and various other psychiatric disorders. It is effective as a serotonergic, dopaminergic, adrenergic, muscarinic, and histaminergic receptor blocker [1]. Olanzapine use could cause peripheral edema in 3% of the patients. There are cases in

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the literature that developed peripheral edema in their bodies pursuant to olanzapine mono-therapy or olanzapine use with mirtazapine [2–5]. In these cases, edema occurred mostly in peripheral mode, in the lower extremities such as wrists, dorsum of the feet and in the limbs up to knees [6]. In rare cases reported in the literature, non-peripheral edema developed as a result of olanzapine use, and the symptoms receded after the cessation of the drug use within weeks. In both cases, it was reported that edema was mild and limited to the eye region [4,5]. This article will scrutinize a female case where a dense edema localized in the facial region restricting the movement of facial muscles after olanzapine treatment.

2. THE CASE

18 years old, unmarried, high school senior male patient was brought to the polyclinic by his relatives with absurd speech and behavior, distress, nervousness and petulance complaints. Based on the anamnesis taken from his relatives, the patient was diagnosed with "psychotic disorder" due to adductive behavior, distress, horrific hallucinations and paranoiac thoughts approximately 2 months prior in a private clinic, and was administered olanzapine and diazepam irregularly. In his psychiatric examination audio and visual hallucinations, paranoid and reference thoughts, distractibility, anxiety, irritability and agitation symptoms were identified and the patient was accepted in the clinic as an inpatient. Laboratory test results conducted to exclude any possible organic factor were considered as normal. After the psychiatric examination, psychiatric assessment scale and various psychometric tests were applied to the patient. Furthermore, other psychotic disorders that should be considered during diagnosis were excluded using the clinical scales and BTA psychotic disorder was diagnosed based on DSM-IV-TR [7]. The patient, who refused oral drug intake and had agitations, was started on 10 mg/day intramuscular Haloperidol amp, 10 mg/day intramuscular Biperiden amp, 2.5 mg/day tablet diazepam treatment. On the 5th day of the therapy, with the partial decrease of the complaints, Haloperidol and Biperiden doses were lowered to 5 mg/day and stopped on the 7th day and 5 mg/day olanzapine was added to the therapy. On the 9th day, edema commenced to develop in the face of the patient, and ulceration, rash and change in color on the skin were identified in his physical examination. It was observed that the edema was dense and widespread in the face and the patient had difficulties in moving his facial muscles. Consultation was requested from dermatology and internal medicine departments to determine the reasons for edema in the patient. Hemogram, prothrombin time (PTZ), international normalized ratio (INR), time of hemorrhage tests, electrolytes, blood urea nitrogen (BUN), creatinine, albumin, total protein, hepatitis panel, thyroid function test results were all considered normal. No pathological evidence was found in electrocardiography, abdomen ultrasonography tests and in peripheral lymphadenopathy examination. Dermatology and internal medicine departments reported that there were no factors to explain edema other than the drug the patient was using. Based on the effect of olanzapine to cause edema, the drug was dropped on the 12th day of the treatment and replaced by 2.5 mg/day Aripiprazole treatment. After the aripiprazole treatment has started facial edema of the patient gradually decreased and disappeared. It was concluded that the occurrence of edema that disappeared after the exchange of olanzapine treatment with aripiprazole was due to olanzapine. The aripiprazole doze was increased to 10 mg/day, and the after the complaints of the patient decreased, he was discharged with a follow up examination appointment. A written disclosure approval for the case was obtained from the patient.

3. DISCUSSION

Edema could occur as a result of several systemic diseases such as cirrhosis of the liver, kidney diseases, and congestive heart failure and with the use of many drugs such as non-steroid anti-inflammatory, steroid and anti-hypertensive drugs. Due to the facts that, the patient did not use any drugs with a side effect of edema, had experienced simultaneous development of edema with olanzapine treatment, the regression of edema after the discontinuation of the medicine, and the lack of a systemic disease that could explain the edema, it was deducted that the development of edema was induced by olanzapine use. In a study where patients on olanzapine were compared with placebo group, it was found that when olanzapine was used in doses of 2.5 mg/day or over, it caused peripheral edema in 3% of the patients and this rate was 1% in the placebo group [8]. Based on previous research, olanzapine causes peripheral edema by causing decrease in vein resistance and vasodilation by blocking peripheral α1, 5HT2 receptors [9].

Another view proposed that the changes in fluid and electrolyte balance in the kidneys during post-dopaminergic blockage were effective in development of edema. There are several case studies in the literature where olanzapine use resulted in development of peripheral edema. In these cases, it was mentioned that olanzapine when used in the dose interval of 2.5–20 mg/g could cause pretibial edema and the edema receded after a little while when the treatment was halted [9]. However, the edema observed in our case occurred only in the facial region, different from the edema observed in peripheral regions such as hands, feet and knees, and regressed rapidly when the drug was discontinued. The general approach against olanzapine-induced edema is to discontinue the medicine and continue the treatment with another antipsychotic drug. In certain cases in the literature, olanzapine was replaced with risperidone and it was observed that edema did not recur [2,9]. In some cases, furosemide was added without discontinuing olanzapine and it was observed that the edema cleared up, however it started again when furosemide was discontinued. Scholars reported that furosemide was effective in the treatment of olanzapine-induced edema, but there was no sufficient evidence on its efficiency and reliability for prolonged use [6]. In our case, olanzapine was discontinued and aripiprazol treatment was selected and it was observed that the edema rapidly cleared up and did not redevelop as determined during the follow-up examinations.

4. CONCLUSION

Pathophysiology of peripheral edema due to olanzapine is still being discussed. There are different views on the frequency and the related factors of this side effect clinically observed very rarely. Thus, only future studies that would be conducted with several patient groups would clarify this conundrum. Although facial edema could be perceived as self-limiting petty side effect, it usually disturbs the functionality of the patient and reduces the patient's motivation in treatment. It is the opinion of the authors that reporting such side affects that are rarely observed clinically would contribute to the literature.

CONSENT

All authors declare that 'written informed consent was obtained from the patient (or other approved parties) for publication of this case report and accompanying images.

ETHICAL APPROVAL

All authors hereby declare that all experiments have been examined and approved by the appropriate ethics committee and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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