CASE REPORT

Peripheral and Facial Edema Associated with Olanzapine: A Case Report

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Abstract

Olanzapine is a potent second-generation antipsychotic commonly used in the treatment of many psychiatric illnesses. It is considered more potent and has less incidence of extrapyramidal symptoms compared with first-generation antipsychotics. With sedation and weight gain being considered as the most common adverse effects, it has also been reported that peripheral edema is associated with olanzapine therapy, but only a few cases of facial edema have been reported so far. However, a definitive cause and consequence of edema are not established, as it is not commonly encountered in medical practice. As olanzapine is a commonly used atypical antipsychotic, we report a case of bilateral pitting pedal edema and facial edema in a woman with no medical comorbidities after initiating olanzapine therapy. All systemic causes of edema were ruled out, and it was completely resolved after discontinuation of olanzapine.

Keywords: Atypical antipsychotics, Edema, Olanzapine, Side effects.

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Introduction

Olanzapine was first approved by FDA in 1996 for the treatment of schizophrenia[1]. It is also used for the treatment of acute mania, bipolar depression, and other psychotic disorders. It has an antagonistic action on dopaminergic, muscarinic, serotonergic, and histaminergic receptors. Constipation, dyspepsia, weight gain, somnolence, asthenia, dry mouth, and dizziness are the most common side effects associated with olanzapine.[2]

In premarketing trials, peripheral edema was reported as an infrequent side effect, which affected 3% of the 532 olanzapine-treated patients, when compared with 1% of the 294 subjects on placebo.[3] Edema can occur because of congestive heart failure, anemia, renal disease, protein deficiency, or as a drug reaction.

Several atypical antipsychotics can occasionally cause peripheral edema. Among atypical antipsychotics, edema has been reported in individuals using risperidone, quetiapine, ziprasidone, amisulpride, clozapine, and paliperidone.[4]

We present here the case of a middle-aged woman who developed bilateral lower-limb edema along with facial edema during the course of olanzapine to emphasize its relatively frequent association with edema.

Case Description

A 31-year-old widowed lady with two children, premorbidly well-adjusted was brought to the psychiatry outpatient department (OPD) by her cousin brother, and she complained that she felt sad most of the time in a day, had no interest in activities, and has wished to end her life for the past 7 months. The symptoms followed soon after the death of her husband, who committed suicide by hanging. She said that he was suffering from a psychiatric condition with psychotic symptoms and was on medications for the same. For the past 2 months, she reported that there is worsening of low mood which is persistent and pervasive and she had taken no medical consultations for her symptoms. She also had trouble sleeping at night as it took her several hours to fall asleep, some nights she could not fall asleep at all. She also reported that she would cry excessively without any reason, had no appetite, and was not interested in doing anything, including taking care of her children and household activities as she used to feel tired most of the time. She said she would sit alone and would think about killing herself by overdosing with medications, but could control herself from doing so by thinking about her children. She complained that she could not concentrate on activities and used to ruminate about all the past events. She denies any history of substance use, hearing voices not heard by others, suspiciousness thought phenomenon. Denies any history of similar or manic episodes in the past.

She reported she has never been diagnosed with any medical or mental disorder and denied any family history of medical illness including diabetes, hypertension, stroke, hypothyroidism, or any depression or suicide in the family.

Physical examination revealed a blood pressure of 110/80 mm Hg, and a body mass index of 19.9. Her vitals were normal and no abnormal results were found from his cardiopulmonary auscultation, results of the neurological examination were normal. The mental status examination was in clear consciousness with psychomotor retardation and poor eye-to-eye contact. She had a reduced volume of speech and reaction time was increased and she was able to stay on topic during conversations. The mood was low, affect was restricted to sadness. There was no formal thought or perceptual disorder or evidence of cognitive impairment. There were no signs of hallucinations and delusions. And her insight about the illness was from grade six.

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Her Hamilton depression scale (HAM D) was 24, indicating moderate to severe depression, and a psychiatric diagnosis of the severe depressive episode without psychotic symptoms (F32.2) was made based on the International classification of diseases (ICD 10). The patient was treated on an outpatient basis as she refused admission, attenders were explained about the risks, and the medications were given under supervision. The initial treatment was a combination of tablet escitalopram 5 mg, clonazepam 0.5 mg, and supportive therapy and reported a 30% improvement in low mood within 10 days. Treatment with escitalopram was 10 mg continued for another month but she reported that she still feels low in mood and has started hearing voices whenever she is alone usually incoherent, the second person, fragmented a male voice and mood-congruent but there was no history of any delusions. Brief psychiatric rating scale (BPRS) score was 30, HAM D score was 28 during the current interview and a diagnosis of the severe depressive episode with psychotic symptoms (F 32.3) was made and olanzapine 5 mg orally at night along with escitalopram 10 mg in the morning was started. She came back to the OPD immediately after 3 days of treatment with complaints of bilateral swelling in her ankles, legs, and face but her auditory hallucinations had reduced and her mood symptoms improved. A physician opinion was taken and a detailed examination was done. Edema was localized to the ankle, foot, and face which was pitting type and non-dependent. There was also no diurnal variation and was nontender but it caused significant distress to patients and attenders. Echocardiogram, thyroid function test, lipid function test, fasting blood sugar, postprandial blood sugar, serum electrolytes, lipid profile came to be normal. Since no other cause for edema could be found and as she was improving symptomatically it was advised to reduce the olanzapine dose to 2.5 mg and review within a week.

The patient came back after 5 days with worsening of auditory hallucinations and sleep disturbances and reported that pedal and facial edema still persisted due to which olanzapine was discontinued and typical antipsychotic haloperidol 2.5 mg was started in the evening along with escitalopram 10 mg and trihexyphenidyl 1 mg at morning. The patient reported that peripheral and facial edema subsided after 2 days of discontinuation of olanzapine.

After 15 days of treatment, HAM D score was 17 and BPRS was 22 and she reported improvement of 70% in mood symptoms and denied any psychotic symptoms.

Discussion

In this case, the cause of edema was attributed to olanzapine, because there was a significant temporal association between the onset of edema and the initiation of olanzapine therapy, as well as the resolution of edema after the discontinuation of olanzapine, and the lack of a systemic disease, did not use any other medications with a side effect of edema, it was concluded that the development of edema was secondary to olanzapine use.

The time to onset of edema after ingestion of atypical antipsychotic differs, from a day to several months.[5] In most of the reported cases, edema develops in the lower limbs. But can also affect other parts of the body either alone or in combination with pedal edema. These include the eyelids, face, hands, periorbital region, forearms, lower trunk, forearm, and a combination of face, eyelid, and lower extremity.[6] Even though various theories have been postulated like supersensitivity and vasodilation theory, dose-response relationship, rate of dose escalation, immune reaction, and old age has also been suggested as a risk factors.[4,7,8]

Conclusion

The main purpose of this case report is to create awareness about the rare side effects, and also to avoid poor compliance, unnecessary investigations, or relapses.

We recommend that routine inquiries on this side effect should be part of a follow-up action in treating these patients. Patient education is also vital in preventing and treating these side effects. Further studies regarding olanzapine and peripheral edema are also warranted.

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