QUETIAPINE-INDUCED TRANSIENT LINGUAL PAPILLITIS AND BILATERAL ANKLE EDEMA

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ABSTRACT

Antipsychotic drug quetiapine is frequently prescribed for the treatment of psychiatric disorders. It is an atypical antipsychotic drug that, because of its sedative qualities, is also used outside of its approved uses to treat anxiety and insomnia. Peripheral edema may occur more frequently than indicated in clinical trials, despite having a relatively low incidence of side effects. Peripheral edema caused by additional second-generation antipsychotics has the potential to affect patient adherence to therapy. Another side effect of quetiapine is oral ulceration, while transient lingual papillitis is a less common side effect. Small bumps that form on the tongue as a result of this condition can be uncomfortable and make eating and speaking challenging. However, it quickly subsided after the medication was stopped. It is crucial for medical professionals to be aware of quetiapine's potential side effects and to inform their patients about what to anticipate while taking the drug. Patient compliance and treatment outcomes as a whole may benefit from this.

KEYWORDS: Peripheral edema, Transient lingual papillitis, Quetiapine, Atypical antipsychotic drug

INTRODUCTION

Being a second-generation antipsychotic, quetiapine is frequently used off-label for a wide variety of psychiatric disorders [(1,2)]. Not everything about quetiapine's mechanism of action has been figured out, but we know that it involves antagonism at serotonin type 1 (5-hydroxytryptamine [5-HT1A]) and type 2 (5-HT2A, 5-HT2C) receptors and only modest antagonism at dopamine (D1 and D2 receptors)[(3)]. Although the medicine has a high safety profile, there are a few potential drawbacks that have been reported thus far. Many atypical antipsychotics, such as risperidone[(4)] and ziprasidone[(5)], have been associated with rare occurrences of bilateral leg edoema.

Patients may also experience pain and discomfort from another side effect, oral ulceration[(6)]. Transient lingual papillitis is a painful inflammatory disorder that affects one or more of the fungiform papillae on the tongue, and it is another less common adverse effect of quetiapine. This medical issue, often known as "lie bumps," has been linked to eruptive (familial) lingual papillitis and fungiform papillary glossitis, both of which are rather prevalent[(7)].

We provide a case of a patient who took quetiapine and afterwards suffered bilateral ankle edema along with transitory lingual papillitis, demonstrating the significance of taking into account all possible adverse effects while administering this medication.

CASE PRESENTATION

A woman who was 23 years old and had a history of gradually worsening depression, anxiety, loss of confidence and self-esteem, loss of interest, diminished energy, loss of appetite, early and middle insomnia, and other symptoms that were consistent with a moderate depressive episode with somatic symptoms but no active medical problems presented herself in the emergency room. On admission, the patient was started on a combination of Quetiapine (25 mg) and Escitalopram (10 mg) to help with their anxiety and depression. She got bilateral ankle edema eight days later, along with a rash and a burning tongue sensation. The rash eventually went away, but the pitting edema exacerbated despite torsemide treatment. Due to inflamed papules on the tongue, the patient experienced considerable pain when eating, which persisted after therapy with vitamin B complex and probiotics. Over the course of four weeks, the daily dose of quetiapine was gradually increased to 50 mg for the treatment of anxiety. The cardiac and pulmonary exams, as well as blood pressure measures, were all normal. All laboratory tests, including a complete blood count and differential, thyroid stimulating hormone, albumin, electrolytes, blood urea nitrogen, creatinine, lithium, erythrocyte sedimentation rate, and N-terminal fragment brain-type natriuretic peptide, were within normal ranges. She was evaluated in the outpatient clinic one month after starting quetiapine for ongoing bilateral ankle edema. The patient was worried that the edema was being brought on by the quetiapine. Her physician suspected escitalopram but agreed to reduce her quetiapine dose from 50 mg to 25 mg per day. Three days later, the patient called to report that his edema had improved. Torsemide 10 mg and Spironolactone 50mg/day were recommended symptomatically for edema, which cleared in about a week with no recurrence of edema recorded. She expressed improved quality of life because the papules on her tongue had disappeared with little to no inflammation. The NADRPS score was calculated to be 7 (probable).

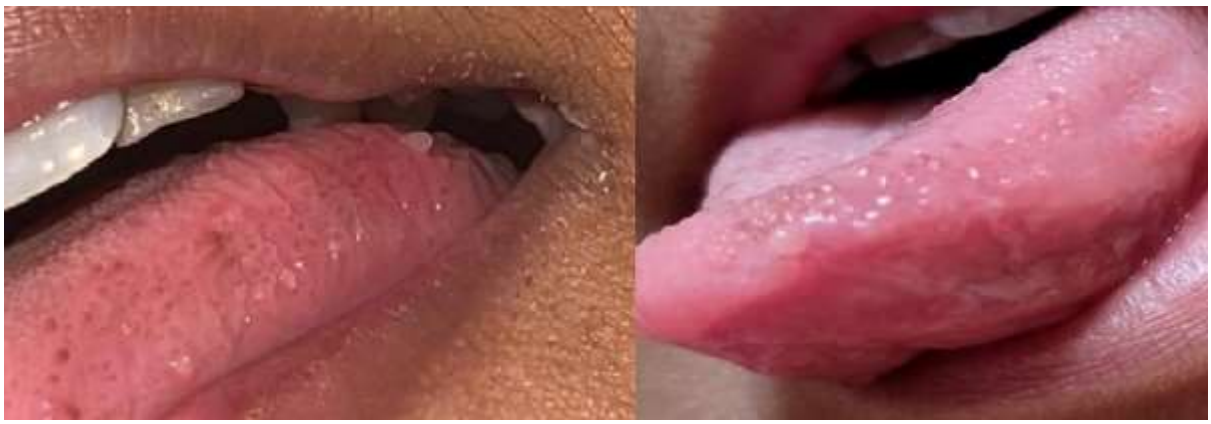

Figure 1: Transient lingual papillitis due to quetiapine

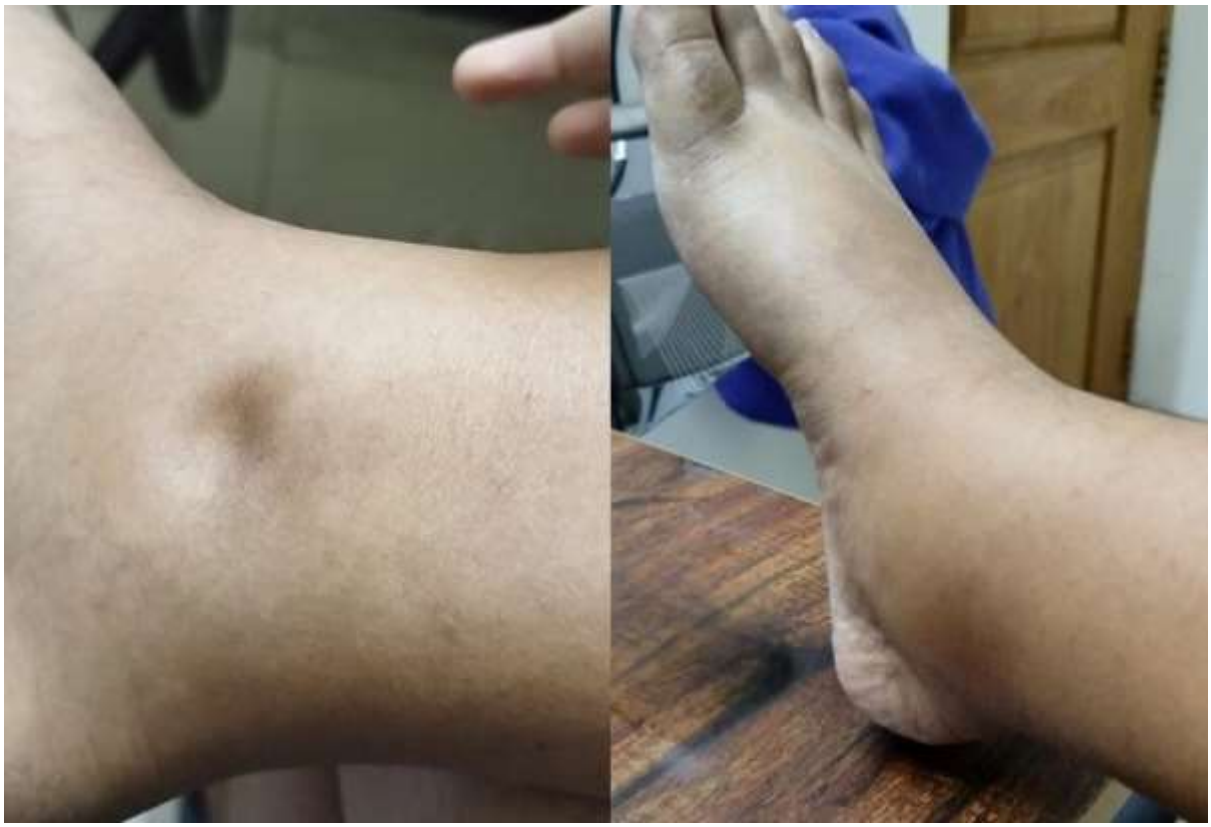

Figure 2: Bilateral pitting edema due to quetiapine

DISCUSSION

The case reported here involves a 23-year-old female who developed significant edema shortly after the initiation of quetiapine. The patients described the leg edoema as uncomfortable and distressing. This case reports bilateral ankle edema as explained by Koleva et al. and also a rare occurrence like transient lingual papillitis [(8)].

The neurotransmitter receptors quetiapine blocks include those for serotonin 5-HT1A and 5-HT2, dopamine D1 and D2, histamine H1, and 1- and 2-adrenergic receptors [(9)]. While the exact processes by which antipsychotics cause edoema are still unknown, earlier research has revealed a connection between idiopathic edoema and dopaminergic antagonism [(10,11,12)]. Dopamine may influence natriuresis, epithelial fluid resorption, vascular smooth muscle relaxation, and the renin-angiotensin system through a number of receptor subtypes [(13,14)]. An alternative mechanism could appear more likely given that quetiapine is regarded as a rather weak dopaminergic antagonist [(15)]. Quetiapine notably has 5-HT2 antagonistic effects. According to some writers, olanzapine-induced leg edoema may be caused by 5-HT2 receptor blockage because it raises levels of cyclic adenosine monophosphate, which can eventually relax vascular smooth muscle [(16,17)]. Alternately, it has been proposed that the atypical antipsychotics' 1-adrenergic blocking activity explains the cardiovascular adverse effects of orthostatic hypertension, vertigo, and reflex tachycardia [(18)]. Olanzapine-induced edoema may potentially be caused via peripheral vasodilation mediated by alpha-adrenergic receptors [(16,17)]. An analogous mechanism for quetiapine-induced edoema might be hypothesized given that the two drugs have comparable affinities for the 1-adrenergic

receptor and similar propensities for orthostatic hypertension. Risperidone and olanzapine were shown to have the highest risk of peripheral edoema in a review by Chen et al. [(19)], followed by quetiapine with a risk of 27.8%. Elderly patients may have developed peripheral edoema due to the existence of additional medical conditions or deteriorating thyroid, heart, liver, and kidney function [(17)]. Given that CYP3A4 is the main enzyme responsible for metabolizing quetiapine, senility is a significant predictor of a higher quetiapine concentration [(20)]. The patients in this case series ranged in age from 22 to 54 years old, had no further disorders, and had completely normal thyroid, liver, and kidney functions.

The atypical antipsychotic medication quetiapine is prescribed to patients suffering from a variety of mental health conditions, including schizophrenia, major depressive disorder, and bipolar disorder. Transient lingual papillitis is one of the infrequent side effects connected to quetiapine use (TLP). The benign disorder known as TLP is characterized by the swelling of one or more of the tongue's fungiform papillae, which are tiny bumps that house taste receptors. TLP often results in discomfort or agony and can briefly affect how you perceive flavours. Usually self-limited, the ailment becomes better on its own in a few days to a few weeks. There are case reports of BMS that are thought to be brought on by drug usage in the literature. The most often documented side effects are angiotensin-converting enzyme inhibitors, albeit the pathogenic mechanism is still not completely understood [(21,22,23)]. Some authors blame the inflammatory response brought on by an elevated amount of bradykinin. Antipsychotics, antiretrovirals, and benzodiazepines are additional medications that have been documented to promote BMS-like symptoms [(21)]. In order to identify cytological and cytomorphometric methods, oral smears taken from the mucosa with a normal clinical appearance were examined in a study involving 20 BMS patients and 20 healthy control subjects. The authors came to the conclusion that these changes are probably related to oral symptoms such as epithelial atrophy and an uncontrolled maturation process of the oral mucosa [(24)]. The anterior dorsal tongue's fungiform papillae can become inflamed ('lingual papillitis'), resulting in a reddish appearance of the tongue's surface and frequently accompanied by burning sensations that resemble BMS symptoms but pass quickly [(25)].

It is currently unclear what role the inactive ingredients of antipsychotic medications have in edema formation. However, we anticipate that our report will make doctors aware of this potential vascular complication, encouraging early detection and treatment.

CONCLUSION

More clinical inquiry and research is necessary to explain the features, risk factors, dosage dependency, and probable causes of the edema associated with AAP as well as the optimal therapy for transient lingual papillitis. This is due to the fact that a feasible mechanism of AAP-induced edema is still not well understood. This case report aims to educate medical professionals about this potential vascular complication, encouraging early detection and prompt treatment.

CONFLICT OF INTEREST

None

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