discarded it given no evidence of emesis, dehydration, new interacting medications, or nonadherence. In addition, the temporary symptom resolution after the blood draw could indicate some degree of suggestibility, often seen with somatization. However, this was an isolated neurological symptom for the patient, who was not somatically preoccupied in general—arguing against somatization as its primary cause.

The mechanisms by which SRIs cause electric shock-like sensations are inadequately elucidated. One hypothesis is that abrupt shifts in serotonin concentration at the central nervous system synapses lead to the neuronal hyperexcitability similar to that caused by myelin disruption. This is corroborated by evidence that in most cases the adverse effect fully remits upon resumption of the offending SRI.[5] One would then expect that substituting an alternative SRI would result in symptom alleviation as well; however, this has not consistently been shown to be the case.[5] This suggests a more complex pathophysiology, one possibly involving a number of neurotransmitters and receptor binding profiles (which vary somewhat by specific SRI). Perhaps most importantly, the mechanism may involve volume neurotransmission rather than synaptic neurotransmission alone neuronal hyperexcitability similar to that caused by myelin description.

This is the first case reported in the literature of electric shock-like sensations occurring during active treatment with sertraline. Notably, such adverse effects may be somewhat underreported in the literature; experienced clinicians have likely treated patients reporting electric-shock sensations while on active treatment with selective serotonin reuptake inhibitors and serotonin-norepinephrine reuptake inhibitors. However, this often occurs in the setting of irregularly timed dosing, partial adherence, or shorter half-life agents such as paroxetine and venlafaxine. Our report suggests that these distressing sensations may additionally occur in other contexts.

Sertraline is an SRI approved by the Food and Drug Administration for treatment of major depressive disorder, OCD, panic disorder, social anxiety disorder, premenstrual dysphoric disorder, and posttraumatic stress disorder. It has a half-life of 22 to 36 hours and is metabolized by a number of cytochrome P450 isoforms, including 2B6, 2C9, 2C19, 2D6, and 3A4.[10] Recognized adverse reactions affecting the nervous system include headache, somnolence, fatigue, insomnia, dizziness, agitation, tremor, akathisia, dystonia, oculogyric crisis, psychosis, and, as part of the withdrawal syndrome, dysphoria, irritability, agitation, tremor, seizures, paresthesia, tinnitus, and electric shock sensations.[11] Long-term effects, if any, of these electric shock sensations are unknown.

When encountering a patient with electric shock-like sensations during active treatment with an SRI, it is important to counsel the patient about the likely benign nature of these sensations and eventual symptom remission, both with continued treatment and upon treatment discontinuation. The subjective discomfort and disability brought on by the sensations may inform whether it would be more beneficial to continue the treatment or switch to a different SRI. There have been no reports of patients who have experienced electric shock-like sensations associated with one SRI experiencing such sensations on other SRIs. Both with the continuation and discontinuation of SRI, symptoms may persist for months, although typically over time become less severe in nature.

AUTHOR DISCLOSURE INFORMATION

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Oral Ulcers as an Unpredictable Adverse Reaction to Quetiapine Use in a Patient With Schizophrenia

To the Editor:

Quetiapine is a dibenzothiazepine derivative that is classified as a second-generation antipsychotic, and it is available in both immediate-release (IR) and extended-release (ER) forms. The elimination half-life of quetiapine IR and norquetiapine (active metabolite) are 6 and 12 hours, respectively.[1] The therapeutic dose of quetiapine in schizophrenia ranges from 300 to 800 mg/d. Quetiapine acts on various receptors, such as antagonists at dopamine receptor D_2 and serotonin $5HT_{2c}$ receptor.[2] The predictable adverse reactions of antipsychotics include involuntary oral movement and dry mouth,[3] both of which can contribute to traumatic oral ulceration. Oral ulcers can occur in 49.8 to 217.7 per 1000 person-year among antipsychotic users.[4] However, oral ulcers that result from a nontraumatic cause are rare. There has been only 1 case report of oral lesions with white tongue and burning mouth syndrome, and the patient was taking risperidone.[5] Here, we report a case of oral ulcers caused by quetiapine in a Thai patient with schizophrenia.

CASE REPORT

A 64-year-old Thai woman with schizophrenia was previously treated at another hospital with amisulpride 150 mg/d, and she achieved full remission for more than 1 year. She then switched hospital and came to our hospital because of a change in her