Case Report
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Late-onset Quetiapine-related Tardive Dyskinesia Side Effects in a Patient with Psychotic Depression

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The atypical antipsychotics were believed to induce less extrapyramidal syndrome, including tardive dyskinesia (TD). Since the introduction of the quetiapine, it is also reported with less TD side effects. It even can relieve the symptoms of severe TD and reduce the risk of TD. The quetiapine's low affinity and fast dissociation from postsynaptic dopamine D2 receptors should give the least risk of producing the symptoms of TD. The quetiapine even can reduce the TD side effects related to clozapine, which has the lowest risk for TD. However, since the first case report of TD side effects related to quetiapine published on 1999, the safety of quetiapine in TD aspect has been questioned. Therefore, we want to share this case report, which was written to describe the severe late-onset TD side effects after long-term use of quetiapine in a patient with psychotic depression. The patient had no significant findings after concurrent comprehensive neurological examinations, magnetic resonance imaging of brain and electroencephalogram since the onset of TD.

KEY WORDS: Tardive dyskinesia, Quetiapine, Psychotic depression.

INTRODUCTION

Atypical antipsychotics are believed to reduce the risk of extrapyramidal syndrome, including tardive dyskinesia (TD). Quetiapine is reported to be less likely to induce TD side effects and, in fact, has been shown to relieve symptoms of severe TD[1)] and reduce TD risk.[2)] Quetiapine's low affinity and fast dissociation from postsynaptic dopamine D2 receptors should contribute to low TD symptom production.[3)] Additionally, quetiapine reduces TD side effects from clozapine, a drug already associated with very low TD risk.[4)] However, since the first case report of quetiapine-related TD side effects was published in 1999,[5)] quetiapine's safety has been questioned. This case report describes severe, late-onset TD side effects after long-term quetiapine use in a patient with psychotic depression. Since TD onset, the patient has had no significant findings based on comprehensive neurological examinations, brain magnetic resonance imaging, and electroencephalogram.

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CASE

The patient was a female homemaker who has suffered with psychotic depression for 3 years. Her symptoms included depressed mood, lack of interest, lack of energy, suicidal ideation, insomnia, feelings of worthlessness, auditory hallucinations (voices commenting around her ears and commanding her to die), and delusions of poverty. Diagnosis was based on criteria of the Diagnostic and Statistical Manual of Mental Disorders IV text revision (DSM-IV-TR) and the Structured Clinical Interview for DSM-IV-TR. Symptom severity was as follows: Brief Psychiatric Rating Scale-18 items (BPRS-18) score=31, especially for hallucinatory behaviors (5 points), and Hamilton Rating Scales for Depression (HRSD) score=27. Quetiapine and duloxetine were delivered using gradual titration to relieve symptoms, and the final stable medication doses were quetiapine 600 mg/day and duloxetine 90 mg/day across 2.5 years. The patient's depressive and psychotic symptoms responded to psychotropic medication and reached partial remission status (BPRS score=9; HRSD score=12). She did not have extrapyramidal symptoms during the first two years after initiating the above medications. However, she began to experience severe perioral tremors with involuntary, repetitive,

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and irregular movements of tongue starting in the third year of quetiapine treatment. The protruding tongue with irregular movements was a significant detriment to her quality of life, as irregular movements occurred all day and were not relieved by anticholinergic medications. Quetiapine-related TD was suspected, and the quetiapine dose was tapered to 150 mg/day. However, after 3 months, TD severity remained the same and seemed unresponsive to interventions, including switching to other atypical antipsychotics such as aripiprazole or ziprasidone. No significant exacerbations of psychotic or mood symptoms were observed after TD onset and antipsychotic switch. The patient also received concurrent comprehensive neurological examinations, brain magnetic resonance imaging, and electroencephalograms. No significant alterations in brain anatomy or function were observed, and according to the neurological report, no organic etiology could be found to explain the presence of TD.

DISCUSSION

In this case, we observed unique, late-onset, and irreversible TD side effects after long-term quetiapine use. Late-onset TD after 3 years might suggest independent factors. However, comprehensive neurological examinations and imaging studies could not pinpoint organic evidence to suggest that this patient's TD was independent of quetiapine use. Late-onset TD side effects in this patient appear irreversible even after decreasing quetiapine dose and switching antipsychotics. However, case reports of late-onset TD have not been included in previous reports. Rather, these reports mention the long-term safety of quetiapine in adolescent[6] and adult patients.[3] Sacchetti and Valsecchi[7] showed that quetiapine has the lowest risk for TD side effects when compared with olanzapine and clozapine in their long-term trial (154 weeks). Quetiapine-related TD was noted and described in an early case report about 15 years ago.[5] However, some reports also mention contrary findings of quetiapine's ability to relieve TD.[1,2,8-13] Emsley *et al*.[14] found that quetiapine could effectively reduce TD severity in patients with established TD, but the mechanism of this action is unclear. Quetiapine is the atypical antipsychotic most similar to clozapine (without its hematologic side effects) based on receptor and pharmacologic profile, which may explain its treatment effects for TD.[11] Quetiapine is believed to express lower affinity for D2 receptors in striatal and extrastriatal regions of brain,[15] which could explain the mechanisms by which it can treat or relieve TD side effects. The presence of mood disorders may be a possible reason for quetiapine-related TD in this patient. Sharma[16] reported that patients with mood disorders were more prone to treatment-emergent TD with quetiapine. Recently, reports reveal quetiapine's TD side effects and suggest precautionary attention to TD risk characteristics.[17] A recent dopamine receptor imaging study showed that quetiapine occupies dopamine D2 receptors extensively in striatal regions, which likely contributes to TD side effects.[18] Furthermore, low-dose quetiapine also seems to induce early-onset TD in the neuroleptic-naïve patient.[19] Recent reports provide evidence of quetiapine's TD risk as well as possible mechanisms. Previously, it was suggested that quetiapine-related TD could be relieved by tapering the dose or switching to another atypical antipsychotic such as aripiprazole.[15] However, in this case study, TD was not abated with quetiapine dose tapering or with a switch to other atypical antipsychotics. Therefore, use of quetiapine should be approached with caution, with close attention paid to TD risk factors.

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