A Case of Tardive Dyskinesia Occurring in the Early Stage of Low-Dose Quetiapine in a Major Depression Patient

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Abstract
Quetiapine is an atypical antipsychotic used not only in cases of schizophrenia and bipolar disorder but also in the treatment of major depression. Given its pharmacological profile, extrapyramidal syndromes are rarely observed; therefore, it is often the drug of choice for patients developing extrapyramidal syndromes. It is also known to be frequently prescribed in clinical practice for treating primary and secondary insomnia. This report presents a case of tardive dyskinesia occurring in the presence of predisposing factors in an elderly patient with major depression whose insomnia was treated with low-dose quetiapine.

INTRODUCTION
Tardive dyskinesia (TD) is defined as an iatrogenic movement disorder characterized by repetitive and involuntary choreoathetoid movements of the mouth, tongue, cheek area, or the extremities [1]. In its etiology, because of hypersensitivity in the dopamine receptors, TD occurs with any agent that is blocking dopamine receptor DA2 or causing increased dopaminergic transmission [2]. According to the DSM, minimum exposure should be for 3 months (1 month if >60 years old) and dyskinesia should start during or within 4 weeks of drug exposure, persisting for at least 4 weeks [3].

Quetiapine is recommended in TD therapy not only because it reduces extrapyramidal symptoms [4]. Its low DA2 affinity and rapid detachment from the receptor are thought to be associated with low risk of TD [5]. However, in the literature quetiapine is found in cases with TD, including psychotic disorder patients, while one regarded late onset after quetiapine use in a patient with major depression [6,7]. We present the case of an elderly female patient followed with major depression who developed TD in the early stage after low-dose quetiapine.

CASE
A 60-year-old female homemaker presented to outpatient clinic with complaints including anhedonia, insomnia, and involuntary lip smacking. When she first came to our clinic in September 2018, she was medicated for depressive complaints with duloxetine 60 mg/day and mirtazapine 15 mg/day. At first follow-up one month later, difficulty falling asleep persisted and therefore quetiapine 100 mg was added to her treatment. According to medical documents, in the second month after adding quetiapine movements around the mouth were first noticed, but as no connection was made with the drug, the treatment was continued. At the consultation one month later, the depressive complaints had receded but involuntary movements of the mouth and the tongue increased. Therefore, quetiapine was discontinued with a provisional diagnosis of TD. She also stated that the involuntary movements in the oral region had continued during the 6-month-period in which she had not attended our clinic for follow-up. In the mental state examination, the patient’s affect was dysphoric and her mood depressive. While there were no delusions in her thought content, intense pessimism and feelings of worthlessness were found. In the physical examination, involuntary movements in the orobuccal region were observed, including lip smacking, puckering the lips, sticking her tongue out, and pulling in her cheeks. In the extremities, no movement disorder was found, including signs of extrapyramidal symptoms. In her medical history, there were no abnormalities apart
neuroleptics swings renders the brain more sensitive to the effects of monoamine and catecholamine activity during mood may develop early-stage TD [13]. It is believed that cyclic mood disorder patients have been seen to be more at risk than those with bipolar disorder [12]. In addition, when comparing mood disorder patients to psychotic patients, it has been reported that while the former use fewer antipsychotics in a shorter period of time, they may develop early-stage TD [13]. It is believed that cyclic monoamine and catecholamine activity during mood swings renders the brain more sensitive to the effects of neuroleptics [14]. The early occurrence of TD with low-dose quetiapine (100 mg) in our patient with depression is consistent with these views.

Studies have shown that quetiapine is used in patients with major depression at an average dose of 40.7 mg/ day [15]. Quetiapine is generally selected for insomnia in patients with major depression [16]. Nevertheless, the use of low-dose quetiapine to treat insomnia is not recommended [17]. The occurrence of TD in our case confirms these recommendations. Furthermore, guidelines do not recommend the use of antipsychotics in insomnia therapy [18].

In conclusion, in major depression patients with predisposing factors the choice of antipsychotics needs to be made with particular care. In addition, in this patient group antipsychotics should possibly not be selected for insomnia therapy; if inevitable, careful clinical follow-up for TD needs to be assured.

REFERENCES


Cansiz A. & Taymur F.


