CASE REPORT

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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.

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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 from hypertension. Our patient scored 27 points on the

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Hamilton Depression Rating Scale and reached a score of 20 on the Abnormal Involuntary Movement Scale (AIMS). On the basis of her medical history and the findings according to the diagnostic criteria of the DSM-5, she was admitted to our inpatient ward with a preliminary diagnosis of major depression plus tardive dyskinesia. Her treatment was changed to citalopram 40 mg/day, mirtazapine 30 mg/day, and alprazolam 1.5 mg/day. To exclude potential causes of dyskinesia other than the drug, a neurological consultation was carried out. In the neurological examination, EEG and MR results indicated no etiological cause. As a treatment for TD, vitamin E 200 mg/day was added. On day 20 of her hospitalization, the patient's depressive complaints declined (HAMD: 12) and she was discharged with a recommendation for outpatient follow-up. At the time of discharge, her AIMS score was still 20. At follow-up 1 month later, the dyskinesia complaints had not declined; therefore, vitamin E treatment was increased to 400 mg/day. There was no significant regression in TD.

Written informed consent was obtained from the patient.

DISCUSSION

As particular risk factors for TD, advanced age, female sex, long-term use of antipsychotics, a diagnosis of mood disorder, a history of parkinsonism, and comorbid alcohol and substance disorder or diabetes have been reported [8]. In our case, too, the presence of risk factors such as age, sex, and mood disorder may have facilitated the development of TD with low-dose quetiapine.

It has been pointed out that age is the most consistent variable in the development of TD. While TD may occur at any age, its incidence increases in the elderly in a linear way, posing a 3.2 times greater risk compared to young persons [9], with a female-to-male ratio of 1.3/1 [10]. The post-menopausal loss of the protective effect of estrogen on the dopaminergic motor system in women is considered the main determinant for this sex difference [11] and assess its interaction with a Mn superoxide dismutase (MnSOD. Accordingly, elderly women like our patient, being the most sensitive group to develop TD, require particular attention.

While antipsychotic drugs are often used as first-line treatment in patients with psychotic disorders, it is known that they may cause extrapyramidal symptoms especially in the presence of mood disorders. Among mood disorders, major depression 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.

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