Olanzapine-related Hypersexuality: A Case Report

Sexual dysfunction associated with antipsychotic treatment has significant negative impact on medication adherence [1]. But hypersexuality is less commonly seen in antipsychotic treatment. In this report, we describe a case of a patient with possible hypersexuality related to olanzapine.

Case report

A 43-year-old single Taiwanese male patient was diagnosed with schizophrenia when he was 34 years old. He had been treated with haloperidol, risperidone, and sulpiride in the past. But his psychotic symptoms had never fully remitted. Owing to worsened auditory hallucinations and persecutory delusion for two weeks, he was admitted to our acute psychiatric ward. Initially, he received risperidone (6 mg/day) and trihexyphenidyl (2 mg/day). Because of insufficient response to risperidone, the antipsychotic medication was switched to olanzapine four weeks later, while trihexyphenidyl prescription was kept with the same dosage. Olanzapine was titrated to 15 mg/day in 10 days. One week after olanzapine treatment, the patient reported to have an increased sexual desire and needed for more frequent masturbation. He did not disturb other patients, but spent more time in his room masturbating instead of participating in therapeutic activities on the ward. He did not show any manic-like symptoms or sexually related psychotic symptoms. He denied any painful long-lasting erection. The patient had never had similar experience before. Without improving psychotic symptoms and with persisting hypersexuality, patient's antipsychotic drug was shifted from olanzapine to zotepine five weeks later. The symptoms of hypersexuality were resolved within one week after discontinuation of olanzapine. Under zotepine (150 mg/day) treatment, his psychotic symptoms had been improved without clinically significant side effects.

Comment

Olanzapine is a second-generation antipsychotic drug with less risk of sexual dysfunction compared with other antipsychotic drugs. But previous studies have shown a prevalence of 10% - 35% of olanzapine-related sexual dysfunction, including decreased libido, as well as erectile and ejaculatory dysfunction [2].

Olanzapine is an antagonist of dopamine D2, but it also consists of the property of antagonistic effects on serotonin 5-HT2A, muscarine M1, histamine H1, and alpha adrenergic receptors. Activating of 5-HT2A receptors can impair sexual function. In contrast, olanzapine's blockage on 5-HT2A receptors may play a rôle to cause an increased libido [3]. Like yohimbine, olanzapine is an antagonist of alpha-2 adrenergic receptors, which have genital stimulation effects [4]. In addition, the antagonistic activity of alpha-1 adrenergic receptors of olanzapine may cause priapism. But risperidone has higher affinity for 5-HT2A and alpha adrenergic receptors than olanzapine. Compared with other antipsychotic drugs (amisul-
piride, risperidone, and zotepine), olanzapine has lower rate of hyperprolactinemia due to fewer dopamine blockade in the dopamine receptors of the tuberoinfundibular pathway [5,6]. Hyperprolactinemia inhibits sexual drive. This may explain that our patient had hypersexuality after switching risperidone with olanzapine in our patient.

This case report has three limitations. First, the hypersexuality in our patient was solely determined by clinical observations and patient’s subjective report. Second, endocrine factors including serum prolactin, cortisol, testosterone were not examined in this patient. And third, olanzapine was not re-challenged in our patient to prove its effect on sexual drive for ethical reason.

Some case reports exist on increased sexual desire related to second-generation antipsychotic drugs, including risperidone, quetiapine, and aripiprazole [7]. So far, hypersexuality has not been reported in olanzapine treatment. According to Naranjo nomogram (total score = 4), this case showed a possible relationship between olanzapine and hypersexuality. Further research is required to examine the risk of olanzapine-related hypersexuality, and to elucidate the underlying mechanisms. (The authors declare no conflicts of interest in writing this letter-to-the-editor.)

**References**


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