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LETTER TO THE EDITOR

Priapism: a rare and acute-onset side effect of paliperidone

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Dear Editor,

Priapism is an uncontrolled and prolonged penile erection state, emerging regardless of the presence of any sexual stimulation. Several various etiological factors of priapism have been reported including sickle cell anemia, thalassemia, fat embolism, vasculitis, spinal cord stenosis, malignancies, and medications (1). It is generally known to occur as a side effect of trazodone, but it can also be seen with the use of various antipsychotics (2). Reportedly, more than 50% of drug-induced priapism cases are caused by antipsychotics (3). Medications may induce an ischemic and painful type of priapism. Failure of providing immediate treatment may lead to fibrosis, resulting in a subsequent persistent erectile dysfunction. To the best of our knowledge, acute-onset priapism after a single dose of oral paliperidone has not been reported in the literature to date. In this letter, we aimed to draw clinicians' attention to this rare and acute-onset side effect of paliperidone.

Mr. K. was an unemployed 36-year-old single male. The patient had a history of persecutory delusions and auditory hallucinations, for which over the past 11 years he had received several antipsychotic medications, including aripiprazole, olanzapine, haloperidol, and quetiapine. There was no history of any medical illness or any substance use. The results of the patient's hemogram and biochemistry tests at the time of admission were normal. The patient was started on oral paliperidone 6 mg/day. On the second day of hospitalization, he presented with a complaint of painful penile erection that lasted 12 to 13 hours. The patient was seen by the urology department and no underlying factors were identified except for paliperidone usage. The patient did not benefit from conservative treatments including aspiration, application of cold compresses, and intracavernous ephedrine injections. The penile erection resolved with a spongiosum-cavernosum shunt.

Priapism associated with paliperidone has been reported less frequently. Only two case reports are available in the literature (4,5). In one of these patients, priapism developed three days after a 150 mg paliperidone palmitate injection, and priapism episodes recurred three times during the effect of this long-acting drug (4). The other patient developed priapism after an almost 2-year use of a 9 mg dose of oral paliperidone (5). In the first case, priapism resolved with conservative treatments and medication switch (4). Conservative management failed in the second case (5). Our case is different from the above two cases as acute-onset priapism developed after the use of a single dose of oral paliperidone. The necessity of surgical intervention was also similar to the second case. To the best of our knowledge, this is the first case of acute-onset priapism after a single dose of oral paliperidone described in the literature.

The mechanism of drug-induced priapism is an occurrence of hypoxia and ischemia due to the inappropriate tonus of the smooth muscles in the penile vascular structures (6). The major pharmacological

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mechanism causing the inappropriate tonus in these vessels is reportedly an alpha-1 adrenergic receptor blockage. This is also emphasized as the main factor in priapism caused by antipsychotics (7). As this type of priapism resolves by treatment with α-1 adrenergic receptor-stimulating medications like pseudoephedrine and phenylephrine, the role of the α-1 adrenergic blockade in priapism is strongly supported (2). The affinity of atypical antipsychotics to adrenergic receptors varies. Ziprasidone and risperidone have the highest affinity for these receptors, whereas the affinity of clozapine and quetiapine is at moderate levels. Paliperidone, aripiprazole, and olanzapine have the lowest affinity compared to other atypical antipsychotics (8). The relatively low alpha-1 receptor blockade effect of the paliperidone molecule may explain the low frequency of priapism with paliperidone.

There is no relationship between atypical antipsychotic-associated priapism and the treatment dose or duration; therefore, this adverse effect may emerge at any time during the treatment with any dose. In our patient, priapism developed with the use of a single dose of 6 mg oral paliperidone; however, as reported in the literature, in one case priapism developed after two years of treatment with a 9 mg/day oral dose of paliperidone (4). Medication switch is the usual intervention strategy in patients with priapism association with the use of atypical antipsychotics. On the other hand, Kartalci et al. (2) presented the case of a patient developing priapism in association with the use of 300 mg quetiapine. In that patient, the dose of quetiapine was reduced to 100 mg/day and no recurrences of priapism were observed during the follow-up period. Although the side effect of drug-induced priapism is considered to be idiosyncratic, the patient presented by Kartalci et al. (2) suggests that there might be a dose-response relationship in the development of priapism. It is suggested that some patients might be relatively more sensitive to the α-1 adrenergic receptor blocking effects of antipsychotics (2). Therefore, priapism may recur despite switching to a different antipsychotic treatment. It is important to inform and follow patients that might be susceptible to the development of priapism.

In conclusion, priapism can lead to permanent damage to the penis such as ischemia and fibrosis. The most common assumption is that priapism occurs due to the alpha-1 adrenergic blockade effects of drugs. Paliperidone is low in alpha-1 adrenergic receptor blockade but even a single oral dose use may rarely lead to acute-onset priapism as in our case. Therefore, clinicians should be aware that priapism may develop after a single dose of oral paliperidone.

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