CASE REPORT

Quetiapine-induced priapism

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Abstract

Priapism is a urologic emergency characterized by a prolonged, persistent, and painful penile erection in the absence of sexual stimulation. The reports indicate that several conditions can cause priapism such as psychotropic medication. One of the mechanisms by which antipsychotics are believed to induce priapism is through alpha 1-antagonism. This case is of a 28-year-old male with a history of bipolar disorder with priapism related to quetiapine. In this case, the treatment of priapism includes discontinuation of the offending agent and drainage of the corpus cavernosum twice a day along with intracavernosal phenylephrine injections. Previous episodes of medication-induced priapism may increase the risk of priapism in patients afterwards. It is important to inform patients about the priapism that may occurs as a side effect of medications.

Keywords: Quetiapine, antipsychotic, drug-induced, priapism

Introduction

Greater than 4 hours of penile erection not initiated by sexual stimulation is defined as priapism [1]. Medications, hematologic disorders, substances, malignancies, trauma, and metabolic conditions are several causes of this clinically urologic emergent condition [1,2]. Whether priapism left untreated, could lead to impotence and tissue necrosis even gangrene as a result of intracavernosal stasis and hypoxia [3]. Conservative management (observation, ice, and rest), corporal aspiration, injection of sympathomimetic agents, and, if the former treatments fail, surgical intervention are the treatment choices [4]. Alpha-1 adrenergic antagonism is thought to be the underlying mechanism of priapism [3]. With this case, we report a case of priapism caused by quetiapine in a patient diagnosed bipolar disorder, in euthymic state.

Case Report

Mr. E is a 28-year-old male with a prior psychiatric history of bipolar disorder and no known past medical history. Patient was admitted to emergency unit of our hospital for 11 hours of penile erection with a severe pain. There was no a history of substance use, hematological illnesses, penile trauma, or past family history related to priapism but only medication with quetiapine 300 mg twice a day, which was prescribed before 7 months and ongoing treatment. He never had before these symptoms. He delayed in admitting to the emergency department due to a lack of information on side effects of medication and assumed it would resolve on its own. Vital signs and results of physical examinations were within normal limits. Mental status examination was euthymic.

Complete blood count, electrolytes, liver function test, and urine analysis revealed no abnormalities, and urine drug screen findings were negative. There was no explanation to this clinically emergent issue, but quetiapine use. Mr. E was diagnosed with nonischemic priapism secondary to psychotropic medication, quetiapine. After admission and evaluation of the patient, underwent drainage (aspiration and irrigation) of the corpus cavernosum twice along with intracavernosal adrenaline injections. Swelling penile erection resolved approximately 2 hours.

The decision was made to discontinue quetiapine due to side effect of priapism. Since the patient has no acute symptoms and a prior episode of priapism with a different medication, he was managed by lithium therapy and referred to outpatient follow-up. His symptoms of priapism completely resolved 24 hours after admission. On the follow-up, sexual problems except for rare arousal problems were not reported.

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Discussion

Priapism is observed in two different patterns: high-flow priapism, in which penile or pelvic trauma leads to a change in arterial flow, low-flow priapism, a more commonly encountered situation, in which drugs play a major role. Approximately 40% to 50% of patients who develop priapism become impotent, even after surgical interventions. Priapism caused by drugs accounts for 25%–40% of all cases, with the most commonly associated categories of drugs being antipsychotics and antihypertensives [5].

The ultimate common pathway in the pathophysiology of priapism is decreased venous outflow from the corpora cavernosa of the penis. Antipsychotics’ blocking of the sympathetic alpha 1-receptor appears related to penile detumescence. It has been proposed that psychotropic-induced priapism is caused by the alpha 1-adrenergic antagonism of these medications. For the typical antipsychotics, more cases have been reported with low potency agents, such as chlorpromazine, compared to the high potency agents, such as haloperidol, due to the level of alpha 1-adrenergic antagonism [2]. For the atypical antipsychotics, risperidone and ziprasidone have the highest antagonism at alpha 1 and olanzapine has the lowest [3]. However, there have been several published cases of priapism with other atypical antipsychotics with lower alpha 1-antagonism such as olanzapine, quetiapine, and clozapine [6]. Although alpha 1-adrenergic affinity hypothesis, lower (olanzapine, quetiapine, clozapine) or higher (risperidone, ziprasidone) affinity of the antipsychotics found to be same offenders of priapism.

Priapism is thought to be an idiosyncratic reaction which is not related to drug dose and may be seen during any stage of treatment. Although priapism is encountered in all age groups, it is more often seen in the third and fourth decade of life as in this case [7].

Recent dose and medication changes or reinitiation of medication after noncompliance, concomitant substance use and/or other medications during medication may also be the probable risk factors for the development of priapism [2]. Another issue is that priapism can occur at any time of the medication, at the beginning or sometime later, thus, duration or treatment dose of medication is not found to be associated [8].

There is some evidence to suggest that patients with prior episodes of priapism are at increased risk of subsequent episodes of priapism [9]. A case report by Penaskovic et al. [9] illustrated a case of priapism with several atypical antipsychotics (risperidone, quetiapine, and olanzapine) which was stabilized on loxapine with no further instances of priapism. A medication with less alpha 1-antagonism should be considered and used at the lowest effective dose [10]. A thorough discussion of risk versus benefit of treatment should be made with the patient as well as family, if indicated and consented to by the patient.

Proper patient education and obtaining complete historical information is very important on side effects of medications in order to prevent recurrence.

Conclusion

In conclusion, priapism may occur at any stage of single antipsychotic treatment, at routine doses, without interaction with pharmacological agents. The atypical antipsychotic quetiapine appears to have the potential to induce priapism, not only in the situation of an overdose or concomitant use of other medications. This report may be an example to “idiosyncratic” priapism case.

Conflict of Interest

No conflict of interest was declared by the authors.

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