

Recurrent Priapism Related with Low Single Dose Olanzapine

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ABSTRACT

Priapism is defined as persistent prolonged erection of the penis, usually painful, without sexual arousal or desire, generally continuing for hours. Many medical situations and drugs, especially psychotropic agents, may cause priapism. The mechanism of psychotropic drug-induced priapism is not fully known, but α 1- adrenergic receptor blockade in the corpus cavernous is being accused. Priapism is an urgent situation for the health of the patient. Cavernous old blood is drained by a butterfly needle or a diluted solution of an alpha-adrenergic agonist is injected into the corporal space. In the event failure of medical treatment, surgical interference may be needed to avert fibrosis and impotence due to tissue death. For preventing that condition, a fistula between the deep dorsal vein or saphenous vein and the glans penis, the corpus cavernosum, and corpus spongiosum is constituted to open a new way for the venous circulation.

Keywords: Olanzapine, Priapism, Recurrence

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INTRODUCTION

Priapism is a permanent and usually painful penile erection, lasting approximately 4 hours, frequently having no relation to sexual stimuli. Drug onset is the one of the most common reasons, being the cause of priapism in 25-40% of cases. Various groups of medications are included, such as antidepressants, antipsychotics, antihypertensive, anticoagulants, alpha-blockers, and some psychoactive substances (1). In this report, we present a case with olanzapine-induced priapism in order to draw the attention of clinicians to this rare side effect.

CASE REPORT

A 24-year-old male with schizoaffective disorder checked into our outpatient clinic with his parents. He had been using quetiapine 600 mg/day for over two years. He declared that he had stopped using quetiapine for the last three months because it slowed down his his movements, and thought that his present clinical condition was good. However his parents complained that his usage of quetiapine was irregular and that he suffered from insomnia and nervousness, talked to himself and thought that people talked about him. Moreover, he experienced increasing on amount of speech, laughing and movements. During his psychiatric examination, the patient was conscious and his orientation relating to people, place, and time was completely normal. His immediate, recent, and remote memory and mental capacity were also normal. Affect and mood were irritable, while increased self-esteem was observed. The content of his thoughts involved reference and persecutory delusions. The need for sleep decreased in the last month, and hallucination was not reported by him or his parents. Laboratory examinations were normal. The patient also insisted on not using quetiapine, and then olanzapine 10 mg/day was initiated. Priapism occurred in the 6th

hour of first dosage. He consulted with the urology department, and the corpus cavernosum was then drained in accordance with the consultation. Olanzapine 2.5 mg was initiated the following day, and also the dose was set to increase slowly. However the priapism occurred again. The drainage of the corpus cavernosum was repeated and then the erection was ended; shunt was not required. Finally, olanzapine was stopped and risperidone 2 mg/day was initiated – this is the 5th week of patient follow-up with risperidone. The priapism has not occurred again, and affective and psychotic symptoms have decreased.

DISCUSSION

Priapism is an urgent situation that requires intervention via drainage of the corpus cavernosum and using an alpha adrenergic agonist. Additionally, some patients who do not respond to drainage of the corpus cavernosum may require shunt surgery (2). The reason for drug-induced priapism is still completely unknown, but alpha-1 adrenergic receptor blockade in the corpus cavernosum of the penis is suspected (3). In the literature, olanzapine-induced priapism was reported as occurring olanzapine with low and high dosage, single and multiple medications, as well as short-term and long-term uses (4-19). Contrary to others, priapism was observed with single-dose olanzapine in our case; furthermore, priapism occurred with repeated low doses of olanzapine. Quetiapine might cause priapism by itself (19, 20). Another interesting aspect of our case is that the priapism did not occur due to usage of quetiapine during the two-year period, although the 600 mg/day dose of quetiapine is considerably higher than the equivalent dose of olanzapine 2.5 mg/day. Furthermore, olanzapine and quetiapine are second-generation antipsychotics. They also affect similar receptors but their complex pharmacokinetic and pharmacodynamics profiles and binding potentials on alpha-1 adrenergic receptors might be different. These differences might explain

why in our case priapism occurred with olanzapine and without quetiapine. Priapism is a rare idiosyncratic side effect of antipsychotics. Finally, we concluded that medications with weak alpha-1 adrenergic blockage must be used in the treatment of priapism.

* The authors declare that there are no conflicts of interest.

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