The Occurrence of Priapism as a Result of the Use of a Single Dose of Quetiapine

Tek Doz Ketiapin Kullanımı Sonrası Gelişen Priapizm Olgusu

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Abstract

Priapism is a prolonged pathologic erection situation that occurs after sexual stimulation or without sexual stimulation. This condition is divided into two types, ischemic (low-flow, veno-occlusive) and non-ischemic (high-flow, arterial). A 68-year-old male patient applied to our clinic with the complaints of hardness and pain in the penis. Three days before he applied to our clinic, he was prescribed a single dose of 200 mg quetiapine by a psychiatry polyclinic for the complication of insomnia. Nearly 6 hours after the first dose of quetiapine, an involuntary erection occurred with accompanying pain in the penis. The patient waited for spontaneous detumescence without consulting a psychiatrist. After the patient had waited for 48 hours without any change, he applied to our clinic. Other etiologic factors of priapism were excluded (such as malignancy, blood dyscrasia, leukemia, and trauma). In blood gas samples obtained from the corpus cavernosum, hypoxia, hypercarbia, and acidosis were diagnosed. Ischemic priapism was supposed. We conclude that priapism can be viewed as a new possible side effect of quetiapine and that patients should be warned about this side effect.

Key Words: Antipsychotic, Priapism, Quetiapine

Introduction

Priapism is a state of extended, pathological erection after or without sexual arousal. It is generally divided into ischemic (low-flow, veno-occlusive) and non-ischemic (high-flow, arterial) forms [1].

This report describes a case of priapism that developed in a 68-year-old male patient following a single dose of 200 mg quetiapine for insomnia.

Case Report

A 68-year-old male patient presented to our clinic with hardness and pain in the penis. Three days before applying to our urology polyclinic, the patient had been started on a single daily dose of 200 mg quetiapine for insomnia by the psychiatry department. Approximately 6 hours after the first dose of quetiapine, an involuntary erection and pain began in the penis. The patient stopped the drug without consulting a psychiatrist and waited for these effects to pass spontaneously. He presented to our clinic when no detumescence occurred after 48 hours. His general condition was average upon physical examination. Systemic examinations were normal. The penis was in an erect state. There was nothing remarkable in his personal or family medical history. The case was re-evaluated by the psychiatry department. The patient had no psychiatric disease, such as substance dependence, depression or schizoaffective disorder, merely sleep impairment.
A complete urine sample was normal upon laboratory analysis. In a full blood count, Hb was 13.2 g/dL, the white cell count 14,500/mm³ and the thrombocyte number 249,000/mm³. The serum biochemistry and electrolytes were at normal values. There was no growth in a urine culture. The PO₂ was 22 mmHg, PCO₂ 70 mmHg and pH 7.21 in a blood gas specimen taken from the corpus cavernosum. All of the other etiological factors that might account for priapism (such as malignancy, blood dyscrasia, leukemia, and trauma) were excluded. Ischemic priapism was suspected, and hypoxia, hypercarbia and acidosis were determined in a blood gas specimen from the corpus cavernosum. Aspiration was performed by entering the corpus cavernosum with a 21G needle, and diluted intracavernosal adrenalin was injected three times at 5 min intervals. There was still no detumescence at subsequent follow-up, and distal spongio-cavernosal shunt (Winter Shunt) surgery was performed. A single 25 mg dose of sildenafil was administered, and antibiotic and anti-inflammatory therapies were initiated. The penis contracted to a semi-rigid state in the follow-up. Penile prosthesis implantation was recommended to the patient, who developed permanent erectile dysfunction, but he has not to date returned to our clinic.

**Discussion**

Low-flow priapism is the most frequently observed type, being an involuntary and prolonged state of erection associated with stasis in the corpus cavernosum. This condition stems from a veno-occlusion-associated lack of detumescence. Hypoxia (PO₂<30 mmHg), hypercarbia (PCO₂>60 mmHg) and acidosis (pH<7.30) are present in blood gas specimens from the corpus cavernosum in these cases [2]. The priapism in our case was ischemic. The PO₂ was 22 mmHg, PCO₂ 70 mmHg and pH 7.21 in a blood gas specimen taken from the corpus cavernosum.

Various factors, such as trauma, hematological diseases (such as blood dyscrasias), malignancies and drugs have been reported to play a role in the etiology of priapism. Causes such as malignancy, blood dyscrasias, leukemia and trauma were excluded in our case.

Thirty percent of priapism cases are drug-related, with atypical antipsychotics causing 50% of these [1]. Antihypertensives, such as hydralazine and prazosin, and antidepressants, such as chlorpromazine and trazodone, can also cause priapism. Quetiapine was used by the patient in our case.

Cases of priapism developing after quetiapine use are very rare. In the first case, reported by Pais and Ayvazian, priapism developed after a single 675-mg (25 mgx27 tb) dose of quetiapine taken in an attempted suicide [2]. The youngest reported age in the literature is 21 years old, and the oldest is 77 years old [3, 4]. In Turkey, a case of priapism that developed after the use of a single 300-mg dose of quetiapine was reported by Torun et al. In our case, priapism developed 6 h after the use of a single 200-mg dose of quetiapine taken due to the inability to sleep [5].

In conclusion, we wish to emphasize that priapism can be a possible side-effect of quetiapine use.

**Conflict of interest statement:** The authors declare that they have no conflict of interest to the publication of this article.

**References**