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Priapism associated with quetiapine in an elderly patient

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It still has not been possible to synthesize a molecule selective enough to act only on the desired receptor in order to produce the necessary therapeutic effect and that lacks side effects. However, the risk-benefit ratio is being evaluated increasingly more when a prescription is written.

Priapism is defined as a prolonged penis erection, usually painful and unrelated to sexual stimulation. Detumescence of the penis would be mediated by the sympathetic system while the α_1-adrenergic antagonism would inhibit detumescence and would consequently prolong erection. Atypical antipsychotics would be involved in 15%-20% of drug-associated priapism[1]. The predisposition of neuroleptics to induce prolonged erection would presumably be due to an α_1-adrenergic block[2]. Appearance of this side effect has been documented in patients who receive olanzapine[3], risperidone[4], ziprasidone[5] and clozapine[6]. Not all the antipsychotics are bond to the α_1-adrenergic receptor with the same intensity, quetiapine presenting an intermediate affinity in relationship to other neuroleptics[7].

The case of a 77 year old male with a history of Alzheimer's disease (DMS IV-TR criteria[8]), who was hospitalized in a psychogeriatrics unit for 3 months, is presented below. Among his personal background, we find hypercholesterolemia, benign prostate hypertrophy, hip fracture and ischemic heart disease. Among his family psychiatric background, his mother had been treated for depression and a brother who had also been under treatment for depression died after a precipitation.

The reason neuroleptic treatment was initiated was to try to control his restlessness and the occasional episodes of aggressiveness towards his family. Constant wandering and insomnia stood out within his clinical picture. Prior to his hospitalization, he had received treatment with olanzapine, 2.5 mg per day. This medication was withdrawn when he was admitted to the hospital (the Spanish Drug Agency recommends not prescribing olanzapine after this time to treat behavior disorders in those over 75 years of age with dementia[9]) and treatment was initiated with quetiapine, gradually increasing the dose until reaching 175 mg per day. Coinciding with this treatment, Mr. L. began to have intermittent prolonged erections of up to 4 hours long, every day for 2 weeks, that were treated with local ice (4 weeks after introducing quetiapine). The neuroleptic dose was also decreased, but the priapism continued. Thus, this medication was discontinued and 5 days later, the erections disappeared. The patient received up to 9 different drugs prior to his arrival to the site. These were not modified except for the neuroleptic treatment (furosemide, diltiazem, simvastatin, finasteride, acetylsalicylic acid, isosorbide mononitrate, omeprazole, bromazepam and donepezil). He had not suffered previous episodes of prolonged erections with any of these medications.

In our clinical case, the therapeutic procedure used when priapism appeared was that explained by Montejo, and the Geopte group[10] when sexual dysfunction secondary to antipsychotics appeared: first we waited for the spontaneous resolution of the side effect, applying physical measures, since tolerance could appear over time. However, the priapism did not disappear and was a significant problem for the patient's wife as she was ashamed that the health care staff would see her husband in this situation. We then reduced the drug dose by 50%, but there was still no improvement. The final decision was to withdraw treatment with quetiapine.

We observe a time relationship between the initiation of treatment with quetiapine and the appearance of the erections. These also ended when treatment was discontinued. Although it is true that this patient received multiple medications, these medications were not changed or withdrawn during this period. However, we do not rule out that any of these drugs may have favored the appearance of the erections. Quetiapine has an affinity for the α_1-adrenergic receptor and blocks it. This would be the mechanism that would explain the inhibition of the penis detumescence and the appearance of this side effect.

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Sexual dysfunction is often not approached by the patients or investigated by the doctors, in spite of the fact that it seriously affects the patient's quality of life, his relationship with his partner and very often his own self-esteem[11]. The psychiatrist should keep these important side effects in mind and know how to face them since they often hinder treatment compliance of the patients and even affect long term therapeutic compliance[10]. Finally, we consider it necessary to maintain an alert attitude to identify the possible adverse effects caused by the medications prescribed to our patients. There are no ideal molecules with a specific affinity for a single receptor, located in a certain area and that, therefore, do not cause side effects.

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