

Risperidone-induced priapism: a case report and literature review

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Abstract: Priapism is a rare pathological condition defined as painful and persistent penile erection that is unrelated to sexual stimulation. It can be classified as ischaemic or non-ischaemic. Many causes have been attributed to ischaemic priapism, including the use of some medications such as antipsychotics. The mechanism of priapism associated with antipsychotics is thought to be related to alpha-adrenergic blockage that is mediated by the alpha receptors in the corpora cavernosa of the penis. In this paper, we describe a case of a patient who suffered from Risperidone-induced priapism, and how this adverse effect was resolved by switching to olanzapine followed by olanzapine pamoate. A literature search on PubMed/Medline up to 2011 was conducted by some doctors from London and found 30 cases of priapism associated with risperidone. Based on this work, we searched PubMed through 2021, using the keywords 'priapism' and 'risperidone' and found a total of 43 cases of priapism involving risperidone. Priapism is not correlated with the dosage of this psychotropic drug, and has also occasionally occurred when risperidone has been used in conjunction with another drug. The question of choosing a replacement antipsychotic after the first one has induced priapism, remains problematic. It would be preferable to switch to a drug with less marked alpha1-blocking properties, but no consensus has been reached as to the best choice of medication. Finally, any prescription of an antipsychotic treatment must be preceded by a careful interrogation in search of risk factors for priapism, and the patient should be made aware of the possible occurrence of this side effect and the need to then seek urgent medical advice.

Keywords: olanzapine pamoate monohydrate, priapism, risperidone, side effects

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Introduction

Priapism is a rare pathological condition of painful, prolonged and sustained penile erection that occurs without any sexual stimulation.¹ It is a urological emergency, because, in 30% to 90% of patients and particularly in cases of delayed treatment, it can lead to persistent erectile dysfunction due to ischemia and fibrosis of the corpus cavernosa.^{2,3}

Several causes have been attributed to ischaemic priapism, including hemoglobinopathies, neoplastic syndromes, compressive pelviabdominal masses, and use of some recreational drugs and

medications.⁴ Antipsychotics are responsible for about half of priapism cases that are triggered by medication.⁵ This is due to antipsychotics creating an imbalance in alpha-1-adrenoreceptors (A1As), which in turn induces intracavernosal blood stasis.^{6,7}

The occurrence of antipsychotic-induced priapism is a critical short-term event in patients with alarming behavioral disorders secondary to their psychoses. In the longer term, even if we manage to treat priapism, its memory could negatively impact compliance with treatment, which is a difficult issue in patients with schizophrenia.

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In this paper, we report a case of a patient who suffered from risperidone-induced priapism, and how this adverse effect was resolved. Written informed consent of the patient was taken before the case was submitted for publishing.

Case report

Mr. X was 22-year-old, single, unemployed, and living with his mother. The onset of his illness is believed to be in adolescence, but his first psychiatric hospitalization was in 2018, when he was diagnosed with schizophrenia. The patient was put on 150 mg of haloperidol decanoate every 28 days. Later that year, he was rehospitalized following a psychotic relapse caused by the discontinuation of his treatment, and he received the same antipsychotic.

In October 2019, he again stopped taking his medication and had a relapse that required hospitalization in a private hospital. During this relapse, the patient presented alarming symptoms, including heteroaggressive threats against his mother that were underpinned by a delusion of persecution. He also presented behavioral oddities (he tore his pajamas and tried to swallow pieces of them, and he presented an excoriation disorder brought on by delusions). He was put on risperidone (4 mg), diazepam (10 mg) and trihexyphenidyl (5 mg). Seven days after the start of this treatment, and upon developing priapism, the patient was referred to the emergency room of a hospital, where treatment could not be carried out due to the lack of an available urologist. After being transferred to the emergency room at another hospital, an intracavernous injection of Etilefrine (10 mg) was administered without success, and a cavernous puncture in the operating room was proposed but refused by the patient. After the spontaneous resolution of the priapism, an magnetic resonance imaging (MRI) of the corpora cavernosa was requested in order to evaluate the possible sequelae.

This MRI was performed 1 month after the priapism episode, and it did not detect any significant abnormality. During a follow-up urological consultation, a urologist explained to the patient the possibility of further erectile dysfunction despite the normal imaging. Immediately after this episode of priapism, the patient's antipsychotics were stopped, and he was prescribed benzodiazepines only.

Given the persistence of the patient's behavioral disorders, which were difficult to manage in a private hospital, the patient was involuntarily admitted to our psychiatric inpatient unit for further care. Due to the severity of the auto- and hetero-aggressive behaviors underpinned by delusions and bizarre behavior, the patient was given antipsychotics. Olanzapine was chosen, as it is theoretically one of the least likely drugs to cause priapism.^{3,5}

The patient began his treatment on extremely low doses (2.5 mg/day), with a very careful increase (2.5 mg per day every 3 days) to reach an effective dose (20 mg/day). Diazepam was concurrently administered, for its sedative effect. This treatment program was well tolerated from a general and urological standpoint. After 1 month, the continued tolerance enabled a switch from oral treatment to an extended-release injection (300 mg). The patient tolerated these therapeutic changes without any sign of priapism.

In all, 20 mg of olanzapine, followed by 300 mg of olanzapine pamoate monohydrate every 28 days, then 405 mg of olanzapine pamoate monohydrate every 28 days, led to a significant improvement in symptoms, with the disappearance of hostile behavior, a reduction in the frequency and intensity of delusions, and the disappearance of bizarre behaviors, particularly excoriation disorder.

This patient had had no history of priapism before the episode previously described. He had no risk factors for priapism other than taking antipsychotic medication. He had never had a hematological disorder, had never taken medication for erectile dysfunction, and had had no history of consuming illegal substances such as cocaine or cannabis. At the time of the priapism, he was not taking any medication other than diazepam, risperidone, and trihexyphenidyl. He was a nonsmoker, and had never had a pelvic or genital trauma. He reported no specific family history of priapism, and denied having any allergies to medication.

Regarding the imputability of priapism to risperidone in our patient, a score of 7 was found on Naranjo *et al.*'s⁸ Adverse-Reaction Probability Scale. The reaction was therefore considered 'Probable'. During his outpatient follow-up, clinical improvement and good tolerance were confirmed. Currently, the patient regularly takes his long-acting olanzapine injections and reports satisfaction with this treatment.

Discussion

Priapism can be classified as ischaemic (low flow) or non-ischaemic (high flow).⁴ The non-ischaemic form is rare and usually follows trauma to the penis or perineum. The most common causes of low-flow priapism are sickle cell disease, spinal cord injury, substance use (including alcohol and cocaine), and medication. Psychotropic medications (including atypical and typical antipsychotics, anticonvulsants, and antidepressants) are the most common causes of drug-induced priapism.⁹

The exact origin of antipsychotic-induced priapism remains unknown. Nevertheless, some etiopathogenic theories have been suggested. Among these is a neuromuscular hypothesis involving the antagonism of α -adrenergic receptors, which is the most widely accepted theory.^{10,11} Indeed, the contraction of smooth muscle cells of the resistance arteries and the trabecular system, mediated by noradrenaline, causes detumescence and penile flaccidity, and their relaxation leads to an increase in blood flow and erection. Norepinephrine acts via the antagonism of $\alpha 1$ and, to a smaller degree, $\alpha 2$ adrenergic receptors.¹² Blocking these receptors could therefore result in a prolonged erection and intracavernous blood stasis. This would cause hypoxia and acidosis that could lead to irreversible fibrosis.¹³ Usually, this vicious cycle can only be interrupted by surgical interventions, such as the aspiration of blood from the corpora cavernosa, an injection of phenylephrine, and the creation of a shunt.¹⁴

Several drugs have been linked to priapism, including urology and cardiology medication such as prazosin, tamsulosin, doxazosin, nifedipine and labetalol. Priapism is also a documented side effect of trazadone, an antidepressant which can act as an α -adrenergic receptor antagonist. In addition, anticoagulant drugs, including warfarin and intravenous heparin, corticosteroids, and oral hypoglycemic drugs (e.g. tolbutamide), may increase the risk of priapism.¹⁵ Despite early reports, however, there has been little research into the frequency with which priapism occurs secondary to psychotropic medications. The available literature indicates that priapism has been attributed to the use of antipsychotics in around 15% to 26% of cases.¹⁶

The affinity of antipsychotics to α -adrenergic receptors varies from one antipsychotic to another.

Richelson¹⁷ studied a series of antipsychotics and established a specific affinity for each drug by determining the equilibrium dissociation constants (KD's) of the $\alpha 1$ -receptors. He took prazosin, the molecule with the highest $\alpha 1$ -adrenergic affinity, estimated at 250 ($10^{-7} \times 1/\text{KD}$) as a standard. This affinity was 38.5 for chlorpromazine, 37 for risperidone, but only 2.3 for olanzapine.

An American study conducted by Andersohn *et al.*,³ which included 144 cases of antipsychotic-induced priapism, studied the relationship between the degree of affinity to the α -adrenergic receptors of antipsychotics and the occurrence of priapism by calculating the reporting odds ratios (RORs) for each drug, being 52.6 for chlorpromazine, 16.4 for risperidone, 10 for aripiprazole, and 1.5 for olanzapine. The question of choosing a replacement antipsychotic for one that induced a priapism, remains problematic, and few authors have focused on alternatives for these patients.

Another challenge for clinicians is how to manage priapism when a patient is in the midst of a psychotic relapse. First, non-recognition of priapism and its potentially serious consequences may cause a diagnostic and therapeutic delay. Second, the patient may not cooperate with the urology team, which would hinder the implementation of the proposed therapeutic measures.¹¹ Indeed, our patient refused the cavernous puncture that the urologists recommended. However, the longer the duration of the priapism, the higher the risk of ischemia, acidosis and long-term fibrosis of penile tissues. Penile ischemia following priapism could eventually lead to penile amputation.¹⁵ Early management is essential to improve the outcome.

Risperidone, which caused the priapism in our patient, has been frequently associated with this adverse effect. Paklet *et al.*¹⁵ conducted a literature search on PubMed/Medline up to 2011, and found 30 cases of priapism associated with risperidone. Based on this work, we searched PubMed through 2021, without time or language restrictions, using the keywords 'priapism' and 'risperidone' and found 13 additional case reports of priapism involving risperidone (see Table 1).

Numerous case reports on risperidone-induced priapism have shown that this phenomenon can occur days or even years after starting this drug, even at low

Table 1. Reports of antipsychotics induced priapism.

Case report	Age	Risperidone dose	Time to onset	Association with other treatment(s)	History of priapism with other molecules	Type and dose of antipsychotic after priapism
Makesar and Thome ¹⁸	31 years	1 single dose of 16 mg	24 hours	No	No	Unknown
Koirala <i>et al.</i> ¹⁹	Middle aged	Switch from oral to Risperdal Consta	1 week	No	No	Unknown
Koirala <i>et al.</i> ¹⁹	14 years	1 mg/day	Unknown	No	No	Unknown
Ankem <i>et al.</i> ²⁰	47 years	4 mg/day	Unknown	No	No	Unknown
Maizel <i>et al.</i> ²¹	44 years	Unknown	Unknown	No	No	Unknown
Nicolson and McCurley ²²	46 years	8 mg/day	Unknown	Lorazepam	No	Unknown
du Toit <i>et al.</i> ²³	44 years	8 mg/day	Unknown	Trazodone	Quetiapine, Olanzapine	Unknown
Stauson and LoVecchio ²⁴	28 years	Unknown	4 days	Venlafaxine	No	Unknown
Haberfeller ²⁵	22 years	4 mg/day	4 weeks	Sertraline	No	Unknown
Sharma and Fleisher ¹⁶	31 years	5 mg/day	8 years	No	No	Aripiprazole
Madhusoodanan <i>et al.</i> ²⁶	65 years	1 mg/day	6 weeks	No	No	Unknown
Kirshner and Davis ²⁷	50 years	Risperdal Consta 25 mg/15days + Oral 6 mg/day	24 hours	No	No	Risperdal Consta 25 mg/15 days
Emes and Millson ²⁸	50 years	10 mg/day	12 weeks	Lithium + Lorazepam	No	Clozapine
Sirota and Bogdanov ²⁹	19 years	2 mg/day	5 days	No	No	Olanzapine 10 mg/day
Bourgeois and Mundh ¹³	26 years	3 mg/day	1 year	Divalproex sodium	No	Olanzapine 10 mg/day
Relan <i>et al.</i> ³⁰	32 years	5 mg/day	2 weeks	No	No	Flupenthixol 1 mg/day
Reeves and Mack ³¹	22 years	4 mg/day	5 years	Clonazepam + Vitamin E + Multivitamins	Ziprasidone	Olanzapine 10 mg then 25 mg/day
Penaskovic <i>et al.</i> ³²	21 years	Unknown	Unknown	No	Olanzapine, Quetiapine	Olanzapine 15 mg/day
Yang and Tsai ³³	13 years	2 mg/day	2 months	Paroxetine	No	Unknown
Dodds <i>et al.</i> ³⁴	49 years	Risperdal Consta dose Unknown	1 month	No	No	Oral fluphenazine
Lin <i>et al.</i> ³⁵	26 years	3 mg/day	3 years	Ginkgo Biloba	No	Risperidone 3 mg/day

(Continued)

Table 1. (Continued)

Case report	Age	Risperidone dose	Time to onset	Association with other treatment(s)	History of priapism with other molecules	Type and dose of antipsychotic after priapism
Freudenreich ³⁶	29 years	3 mg/day	4 weeks	Citalopram	No	Haloperidol
Brichart <i>et al.</i> ⁵	55 years	2 mg/day	Few years	Unknown	No	Risperidone 2 mg/day
Brichart <i>et al.</i> ⁵	26 years	4 mg/day	Few years	Unknown	No	Unknown
Rosenberg <i>et al.</i> ³⁷	49 years	8 mg/day	2 days	Lithium	Quetiapine, Trazodone	Aripiprazole 5 mg/day
Salawu <i>et al.</i> ³⁸	30 years	4 mg/day	Unknown	Sertraline	Sertraline	Risperidone
Owley <i>et al.</i> ³⁹	17 years	1.5 mg/day	12 weeks	Lithium	No	Risperidone
Tekell <i>et al.</i> ⁴⁰	41 years	6 mg/day	6 days	No	No	Unknown
Wang <i>et al.</i> ⁴¹	37 years	2 mg/day	9 months	No	No	Clozapine 100 mg/day
Wang <i>et al.</i> ⁴¹	27 years	Risperdal Consta 37.5 mg	22 days	No	No	Clozapine 250 mg/day
Ginory and Nguyen ⁴²	50 years	6 mg/day	1 month	No	Trazodone	none
Unver <i>et al.</i> ⁴³	12 years	1 mg/day	22 days	Methylphenidate	No	none
Aabbassi <i>et al.</i> ⁴⁴	12 years	2 mg/day	Few hours	No	No	Sulpiride 150 mg/day
Burk and Nelson ⁹	34 years	Unknown	2 years	Trazodone	Chlorpromazine, Trazodone, Quetiapine	Unknown
Baytunca <i>et al.</i> ⁴⁵	13 years	2.5 mg/day	7 years	Methylphenidate	Quetiapine + Methylphenidate, Chlorpromazine	Quetiapine 25 mg/day
Şenormanci <i>et al.</i> ⁴⁶	25 years	4 mg/day	2 years	No	No	Olanzapine 10 mg/day
Paklet <i>et al.</i> ¹⁵	45 years	4 mg/day	3 days	Sodium valproate	No	Aripiprazole
Pradhan and Hardan ⁴⁷	21 years	Between 0.5 and 3 mg/day	9 years	No	No	none
Cruzado and Vallejos ⁴⁸	32 years	3 mg/day	3 years	No	No	Risperidone 2 mg/day
Prabhuswamy <i>et al.</i> ⁴⁹	12 years	6 mg/day	4 months	No	No	Olanzapine
Refai and Nakama ⁵⁰	21 years	6 mg/day	4 days	No	No	Asenapine 10 mg/day
Eslami <i>et al.</i> ⁵¹	35 years	3 mg/day	13 days	Clonazepam, Biperiden, Chlorpromazine	No	Unknown
Segar and Lambert ⁵²	37 years	6 mg/day	3 months	Olanzapine + Fluvoxamine + Gabapentin + Oxazepam	No	Unknown

Source: Adapted and updated from Paklet *et al.*¹⁵

and stable doses.^{5,26} In theory, polypharmacy may increase the risk of priapism, either through the synergy obtained by combining drugs that can independently induce this complication, or by combining drugs such as risperidone with another drug that affects the former's metabolism.¹⁵ Some published cases have reported priapism following concomitant administration of risperidone and other molecules such as sertraline,^{25,38} lithium,^{28,37,39} fluvoxamine,⁵² valproate sodium and divalproex sodium,^{13,15} lorazepam,^{22,28} gabapentin,⁵² trazodone,^{9,23} venlafaxine,²⁴ clonazepam,^{31,51} oxazepam,⁵² chlorpromazine,⁵¹ paroxetine,³³ citalopram,³⁶ methylphenidate,^{43,45} olanzapine,⁵² and so on.

However, it is not clear whether and to what extent the combination of different risk factors or the combination of several drugs increase the risk of priapism.⁵³ Priapism could be considered an idiosyncratic reaction as it is not correlated with either the dosage of a psychotropic drug or the duration of its use.¹⁰ Furthermore, the lack of association between the dose and duration of antipsychotic treatment on the one hand and the onset of priapism on the other makes the phenomenon difficult to predict.³² It is therefore important to be aware of the risk of priapism, and to inform and monitor patients who may be susceptible to this side effect.

Based on the hypothesis that priapism is related to the blocking of $\alpha 1$ -adrenergic receptors, we decided to substitute risperidone for olanzapine, a drug with low $\alpha 1$ -adrenergic affinity, first orally, and then by extended-release injection.^{3,54} This switch was well tolerated by the patient: during the whole follow-up period (of more than 1 year), no further episode of priapism was observed. Donizete da Costa *et al.*⁶ also opted for a switch to olanzapine following an episode of priapism induced by clozapine. To our knowledge, no other medical team had chosen to introduce the extended-release injectable form of olanzapine following an episode of priapism.

In these different cases, the psychiatrists were faced with the challenge of treating the patient's illness in light of the risk of priapism, which had been induced by different types of medication. This emphasizes the importance of considering pharmacodynamic properties of each drug while choosing the most appropriate medication for patients predisposed to this type of urological emergency.¹⁰ According to Aabbassi *et al.*,⁴⁴ if, after an episode of priapism, the patient needs to

continue antipsychotic treatment, the dosage should be decreased, or the medication should be discontinued and replaced by another drug together with a medical follow-up, given the high risk of antipsychotic-induced priapism.

Most authors agree that when priapism is induced by an antipsychotic, it is recommended to switch to another molecule with fewer $\alpha 1$ -blocking properties.^{3,11,44,55}

In the published cases, most of the teams replaced risperidone with olanzapine,^{13,29,31,32,46,49} aripiprazole^{15,16,37} or clozapine.^{28,41} Few teams chose haloperidol,³⁶ sulpiride,⁴⁴ quetiapine,⁴⁵ asenapine,⁵⁰ fluphenazine³⁴ or flupenthixol.³⁰ To date, no consensus has been reached on the best choice of drug.

An alternative to antipsychotics could be the use of electroconvulsive therapy (ECT), which could be viable if psychotic symptoms are severe or disabling.¹¹ Finally, to prevent the occurrence of priapism, some precautions should be taken. Any prescription of an antipsychotic treatment must be preceded by a careful interrogation centered on the patient's sexual and andrological history in search of risk factors for priapism, such as episodes of prolonged erection, or previous episodes of priapism.⁵⁶

Conclusion

Priapism is a urological emergency requiring rapid treatment to avoid erectile sequelae. Priapism can be induced by antipsychotics, particularly those with a strong affinity to α -adrenergic receptors such as risperidone. Although rare, any patient on antipsychotics should be made aware of the possible occurrence of this side effect and the need to then seek urgent medical advice.

Overall, our study shows that the etiopathogenesis of this phenomenon is far from being fully understood. Further studies and research seem necessary to identify patients at higher risk of antipsychotic-induced priapism, and to propose clear alternative treatments should it occur.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Written informed consent of the patient was taken before the case was submitted for publishing.

Author contributions

Sarra Ateb: Conceptualization, Investigation, Methodology, Writing – original draft, Writing – review & editing.

Taoufik Fourati: Methodology, Writing – review & editing.

Hammadi Ben Rejeb: Conceptualization, Writing – review & editing.

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