Ketamine Saves the Day

Priapism in a Pediatric Psychiatric Patient

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Abstract: Priapism is an adverse effect of medications used to treat psychiatric disorders. Often, this condition is self-limiting but may require urologic intervention involving aspiration and injection to induce detumescence. A case of a 15-year-old patient with priapism secondary to a long-acting stimulant is presented to describe the effectiveness of ketamine treatment for priapism.

Key Words: priapism, ketamine, procedural sedation
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CASE

A 15-year-old boy with a medical history significant for autism, attention deficit/hyperactivity disorder, and mild mental retardation was transferred from a children's psychiatric hospital to the pediatric emergency department (PED) with a chief complaint of priapism. The patient had a long-standing history of extreme hyperactivity despite trials of multiple medications. Duloxetine (Focalin XR, 30 mg daily) was recently discontinued because of facial tics and several self-resolving episodes of priapism. The patient had experienced 2 previous episodes of priapism related to trazodone (Oleptro) that was subsequently stopped as a result. He remained only on the following 2 other medications for a history of poor sleep: doxepin (Silenor) 20 mg daily (tricyclic antidepressant) and melatonin 10 mg daily at night. However, since discontinuing the stimulant medication, the patient had become overwhelmingly hyperactive to the point of self-injury and he was voluntarily admitted to the children's psychiatric hospital for evaluation and treatment. On the first day of hospitalization, the doxepin dose was decreased and Focalin XR was reinitiated at midday with subsequent development of priapism later that night. The following morning, a second dose of Focalin XR 20 mg was given. By the early afternoon, the erection had persisted. Application of an ice pack to the penis and oral pseudoephedrine (Sudafed) 60 mg was administered without detumescence. The patient was then transferred to the PED after approximately 14 hours of priapism.

On arrival to the PED, the patient was in visible pain and was, at times, combative with staff. His vital signs were the following: temperature was 37°C; pulse rate, 97 beats per minute; respiratory rate, 17 breaths per minute; blood pressure, 155/75 mm Hg; and oxygen saturation, 100% in room air. Physical examination revealed a normal rate and heart sounds, strong femoral pulses bilaterally, and brisk capillary refill. The abdomen was neither distended nor tender. Genitourinary examination revealed a circumcised phallus with an orthotopic urethral meatus with a small blind appearing pit at the ventral aspect of the glans, bilateral descended testicles, and an erect penis extremely tender to palpation. No lower extremity edema was revealed. Neurological examination was nonfocal.

The pediatric urology service was consulted and recommended aspiration of the corpora and injection with phenylephrine to induce detumescence. Sedation was deemed necessary to perform a safe procedure given the patient's progressively worsening hyperactivity and inability to cooperate with medical staff.

Because the patient was uncooperative for placement of an intravenous catheter, 4-mg/kg intramuscular ketamine sedation was provided and used. Routine monitoring including end-tidal CO2 was performed according to standard departmental protocol. The patient's phallus softened within 2 to 3 minutes of administration of ketamine, and an aspiration of the corpora was not performed. A simple penile block was performed and a pressure dressing was applied. The dressing was removed after 1 hour and the phallus remained detumesced. The patient was admitted to the pediatric urology service for observation overnight. Priapism did not recur. Dextroamphetamine (Adderall) 10 mg 3 times daily resulted in acceptable hyperactivity control. There was no report of recurrence of priapism at the time of 6-week follow-up with the pediatric urology service.

DISCUSSION

Priapism is defined as a persistent erection, unrelated to sexual stimulation, lasting longer than 4 hours.1 It is considered a urologic emergency that can lead to permanent and irreversible erectile dysfunction if not managed rapidly. Urgency in management is required because patients with ischemic priapism with a duration of less than 24 hours have a 50% chance of maintaining erectile function, whereas beyond 24 hours, the risk of permanent impotence is greater than 90%.2 Priapism may be seen in the following 2 forms: ischemic and nonischemic. Ischemic priapism (veno-occlusive or low flow) makes up 95% of cases and may lead to compartment syndrome of the penis and subsequent tissue fibrosis if ischemia is prolonged.3 In contrast, nonischemic priapism (arterial, high flow) is a persistent erection caused by unregulated inflow of blood. Recurring or “stuttering” priapism is considered a variant of ischemic priapism in which erections occur repeatedly with intervening periods of detumescence.1 Often, stuttering priapism is reported in patients with sickle cell disease. In the general population, the most common etiology of ischemic priapism is idiopathic. The remaining cases are accounted for by prescription and recreational drugs. Drugs known to cause priapism include but are not exclusive to the following: anticoagulants, antihypertensives, antidepressants, antipsychotics, intracavernous medications, and illicit substances including cocaine and marijuana. Malignancy and neurological disorders make up an even smaller percentage of cases. In the pediatric population, the most common cause of
ischemic priapism is sickle cell disease. The pathophysiology is likely related to prevention of venous outflow due to functional and structural abnormalities of the erythrocytes.

Psychotropic agents such as antipsychotics, antiepileptics, and antidepressants have emerged as indispensable agents in the management of self-injury, irritability, and aggression in children with autism. Unfortunately, these medications are known to cause priapism. Trazodone, an atypical antidepressant and hypnotic, is the psychiatric medication most commonly associated with ischemic priapism with an incidence of 1:6000. The most likely mechanism by which this occurs is alpha-adrenergic blockage in the corpora cavernosa leading to a parasympathetic-mediated arteriolar dilatation as well as inhibition of the sympathetic system responsible for detumescence. Hofmann et al describe a case of penile amputation after a late presentation of trazodone-induced priapism. Risperidone (Risperdal), an atypical antipsychotic, has a particularly high affinity for the alpha-adrenergic receptor and has been associated with priapism. Other antipsychotic medications such as phenothiazines (Perphenazine) may cause ischemic priapism by a similar mechanism in addition to its antihistamine, anticholinergic, and antiserotonergic activity. Serotonin-specific reuptake inhibitors and tricyclic antidepressants have also been implicated in inducing priapism. Awareness of the risk of priapism with various psychotropic medications is important for the provider who may encounter children with psychiatric disorders in the emergency department. Furthermore, priapism can occur irrespective of when the medication was initiated, dose titrated, or combined with another medication.

The teenager in this case had been treated with multiple medications mentioned previously and had experienced several episodes of priapism while on both trazodone and dexamethasone. During this particular episode of priapism, he had taken his routine midday dose of dexamethasone at a long-acting central nervous system stimulant, with an onset of action of 30 minutes and duration of 12 hours. The absorption pattern of dexamethasone is bi-modal with the first peak of activity at 1.5 hours and a second peak at 6.5 hours. The therapeutic effects of central nervous system stimulants such as Focalin and Concerta are attributed to the inhibition of dopamine and norepinephrine reuptake. These same neurotransmitters are thought to play integral roles in sexual stimulation and may potentiate penile tumescence. A similar mechanism is described in cases of immediate release methylphenidate (Concerta)-related priapism.

Treatment of priapism involves prompt surgical aspiration of the penis with or without intracavernous administration of sympathomimetic agents (ie, phenylephrine, epinephrine, ephedrine). Surgical intervention may be necessary for an ineffective pharmacological approach. A simultaneous evaluation for underlying etiologies such as sickle cell anemia and the presence of other medications or illicit substances should be conducted. The prognosis of priapism depends on the speed with which detumescence is achieved, but despite successful treatment up to 40% to 50% of patients, they can become intermittent, secondary to ischemia and fibrosis of the corpus cavernous. In this case, the patient underwent sedation with intramuscular ketamine, which unexpectedly produced detumescence within 2 to 3 minutes of administration. While there are no reports within the pediatric emergency medicine literature of ketamine-induced detumescence, there are rare case reports of successful treatment of priapism using ketamine. Priapism secondary to general or spinal anesthesia in adult patients has been successfully treated with ketamine. In another case report, intravenous ketamine hydrochloride (0.5 mg/kg) was used to treat priapism that developed during a hypospadias repair. Prompt detumescence of idiopathic priapism in a 3.6-kg newborn was similarly described using intravenous ketamine.

The decision to use ketamine in the patient’s sedation was not made lightly. Ketamine is contraindicated in patients with known schizophrenia or psychotic disorders. Studies have suggested that ketamine exacerbates schizophrenia, although its effect on other forms of psychosis is unknown. It is important to note that our patient was uncooperative but not actively psychotic. His inpatient hospitalization was secondary to his autism and extreme hyperactivity. While caution was advised because of his preprocedural hyperactivity and agitation, he did not present a contraindication to a ketamine sedation.

One may argue that the act of sedating the patient, regardless of pharmacologic agent, may have induced detumescence in this case. While theoretically possible, there is an absence of adult and pediatric literature regarding detumescence with nonketamine agents used for procedural sedation. The paucity of effective options in the unique clinical scenario of a child with priapism requiring sedation may strengthen the argument for the use of ketamine when not contraindicated.

While ketamine effectively treated the patient’s priapism in this case, it is still not considered to be a first-line therapy. However, if a pediatric procedural sedation was planned to surgically manage ongoing priapism, ketamine may be a preferred pharmacologic agent for the procedure and may even obviate the need to aspirate.

REFERENCES