Possible Propofol-Induced Priapism Following Cardiac Catheter Ablation in a Teenager

Ming-Lon Young

Suying Lam

Corresponding Author: Ming-Lon Young, e-mail: Mingyoung@MHS.net

Conflict of interest: None declared

Patient: Male, 16-year-old
Final Diagnosis: Priapism
Symptoms: Erectile
Medication: Propofol
Clinical Procedure: Catheter ablation
Specialty: Cardiology

Objective: Diagnostic/therapeutic accidents
Background: Priapism is rarely reported as a potential complication after the cardiac ablation procedure. We report the case of a teenager admitted for atrial flutter ablation who developed priapism following the procedure.

Case Report: A 16-year-old male with episodic atrial flutter came to our hospital for an electrophysiological study and catheter ablation. During the procedure, he was given IV propofol for anesthesia and IV heparin for anticoagulation. After the procedure, nursing noted that he had an erection, which persisted for 5 h, with complaints of discomfort. There was no known history of sickle cell disease or trauma to the perineum, nor did he endorse any prior prolonged erections. On physical examination, he had a circumcised phallus with rigid and non-tender corpora cavernosa. He was given 5 mg terbutaline PO, without improvement. Three hours later, a second dose of terbutaline was given. In addition, a penis corporal venous blood gas was taken, and the result was consistent with an ischemic priapism. He had detumescence 1–2 min later. The total duration of his priapism was 8 h. There was no swelling, pain, or any sequelae after detumescence.

Conclusions: Although priapism rarely occurs as a complication following catheter ablation procedures due to propofol use, prolonged priapism can result in corporal fibrosis and cause future erectile dysfunction. Recognition and treatment of priapism in the postoperative period may be delayed due to a patient’s hesitance to express concerns. To prevent future erectile dysfunction, signs of priapism should be included in routine postoperative evaluation in male patients.

MeSH Keywords: Catheter Ablation • Heparin • Priapism • Propofol
Background

Cardiac catheter ablation is an established procedure for treatment of cardiac arrhythmias in both adults and children. The procedure complications reported in the literature usually include vascular and/or conduction system injuries. Priapism, although reported as a possible complication of catheter ablation due to propofol use [1], is usually not mentioned as a potential complication of the procedure to the male patients or to their parents. We report such a case in a teenager admitted for atrial flutter catheter ablation, in whom priapism was treated successfully.

Case Report

A 16-year-old male had several episodes of palpitations at age 15. He had an episode of palpitations while he was running in school at age 16. He went to an emergency department, with an ECG showing a narrow QRS tachycardia of 163 bpm (Figure 1A). The heart rate slowed from 163 to 148 bpm before adenosine was given, which revealed underlying atrial flutter waves at 300 bpm (Figure 1B). He was admitted to the hospital and had several episodes of nonsustained atrial flutter shown on the telemetry recordings.

He was discharged home without medication and was scheduled for an electrophysiological study and catheter ablation.

In the baseline electrophysiological study, there were no dual atrioventricular nodal pathways, no accessory pathway, no ventricular-atrial conduction, and no inducible supraventricular tachycardia or atrial flutter. With isoproterenol challenge, there was still no tachycardia induced. By using an irrigated tip radiofrequency ablation catheter, cavotricuspid isthmus linear ablation resulted in bi-directional conduction block through the ablation line. The procedure lasted for 2 h, during which he was given IV propofol for anesthesia (total dose of bolus plus infusion: 3215 mg) and IV heparin for anticoagulation (total dose: 90 units/kg).

Shortly after arrival to the intermediate care unit after the procedure, a nurse noted that the patient had an erection. Five hours later, his mother reported that his erection persisted and he started having some discomfort. There was no known history of sickle cell disease or trauma to the perineum, nor did he endorse any prior prolonged erections. He denied any previous history of using alcohol or illicit drugs.

On physical examination, he had a circumcised phallus with rigid and non-tender corpora cavernosa, glans soft, testes descended and without masses bilaterally. He was given a dose of 5 mg terbutaline PO as a sympathomimetic, and 3 h later he still had an erection. A second dose was given and a penis corporal venous blood gas was withdrawn, which showed: pH 7.05 (low; normal 7.26–7.43), pCO2 100 mmHg (high; normal 41–51 mmHg), %O2 saturation: 26% (low; normal 70–80%), pO2 26 mmHg, HCO3 28.4, and BE –2. This study confirmed low-flow (ischemic) priapism. He had detumescence 1–2 min later. The total duration of his priapism was 8 h. There was no swelling, pain, or any sequelae after detumescence.

At 8-month follow-up, there was no reported arrhythmia or sexual dysfunction.

Discussion

Priapism is defined as a penile erection that persists beyond 4 h and is unrelated to sexual interest or stimulation [2]. It can be classified into ischemic (low-flow), arterial (high-flow), or stuttering (recurrent or intermittent) [2]. Ischemic priapism is the most common form, accounting for >95% of all priapism episodes [3]. This condition can occur in males of all ages, including neonates. While cavernous blood gas values in men with nonischemic priapism are similar to the blood gas values of arterial blood, it typically has a pO2 of <30 mmHg, pCO2 of > 60 mmHg, and pH <7.25 in patients with ischemic priapism [4]. This is similar to the cavernous blood gas analysis of our case, and confirmed the diagnosis of ischemic priapism.

The most common causes of priapism in children are sickle cell disease (65%), leukemia (10%), trauma (10%), idiopathic (10%), and pharmacologically induced (5%) [5,6]. Although it is rare, there are 3 reports of propofol inducing priapism in the literature [1,7,8], 2 of them confirmed by rechallenge [6,8]. Similar to the case reported by Vesta et al. of a 17-year-old with supraventricular tachycardia catheter ablation who developed priapism [1], our patient, without any known hematological disease and who received IV propofol as anesthesia, might have had propofol-induced priapism. The mechanism of propofol-induced priapism remains poorly understood, but could be due to sympathetic vasoconstrictor action or parasympathetic vasodilatory action, or nitric oxide-mediated smooth-muscle relaxation [9–12].

There are rare reports of heparin causing priapism [13–17]. Our patient received IV heparin for anticoagulation during the procedure. The mechanism could be veno-occlusion secondary to platelet aggregation triggered by antibody-platelet antibodies, a clinical sequela of heparin-induced thrombocytopenia [17], but the fact that our patient had no previous exposure to heparin, and because the priapism occurred within 3 h after exposure, argues against this hypothesis. Unfortunately, no platelet count was obtained in our case during his priapism episode.
The mechanism of ischemic priapism is that a persistent corporal smooth-muscle relaxation compresses the subtunical veins and prevents sinusoidal outflow. When the intracorporal pressure is increased above the mean arterial pressure of the cavernosal arteries, no inflow of blood can occur. Because prolonged priapism can result in corporal fibrosis and cause future erectile dysfunction [3,9], it is a true emergency and should be handled with the same urgency as an ischemic heart attack medical emergency.

Initial treatment of priapism includes exercise, urination, a cold bath, ejaculation, and fluids [18]. Second-line treatment includes vasoconstrictive agents such as terbutaline (a β2 agonist) PO or diluted phenylephrine (α-1 agonist for patients ≥11 years old) or epinephrine local injection [3].

Figure 1. Tachycardia ECG. (A) 12-lead ECG showing a narrow QRS tachycardia at 163 bpm (likely a 2:1 atrial flutter with inverted P in inferior leads). (B) Adenosine challenge revealed underlying flutter waves at 300 bpm.
Conclusions

Because patients may be embarrassed to mention this condition, recognition and treatment of ischemic priapism as a postoperative complication may be delayed. Therefore, although this condition is rare, to prevent future erectile dysfunction, signs of priapism should be included in routine postoperative assessment by healthcare professionals in male patients.

References:

1. Vesta KS, Martina SD, Kozlowski EA: Propofol-induced priapism, a case confirmed with rechallenge. Ann Pharmacother, 2006; 40: 980–82
2. Salonia A, Eardley I, Giuliano F et al: European Association of Urology guidelines on priapism. Eur Urol, 2014; 65: 480–89
3. Broderick GA, Kadioglu A, Bivalacqua TJ et al: Priapism: Pathogenesis, epidemiology, and management. J Sex Med, 2010; 7: 476–500
4. Montague DK, Jarow J, Broderick GA et al: American Urological Association guideline on the management of priapism. J Urol, 2003; 170(Suppl. 1): 1318–24
5. Donaldson JF, Rees RW, Slinbrecher HA: Priapism in children: A comprehensive review and clinical guideline. J ped Urol, 2014; 10: 11–25
6. Scherzer ND, Reddy AG, Le TV et al: Unintended consequences: A review of pharmacologically-induced priapism. Sex Med Rev, 2019; 7(2): 283–92
7. Fuentes EJ, Garcia S, Garrido M et al: Successful treatment of propofol-induced priapism with distal glans to corporal cavernosal shunt. Urol, 2009; 74: 113–15
8. Senthilkumaran S, Shah S, Ganapathy-subramanian et al: Propofol and priapism. Indian J Pharmacol, 2010; 42: 238–39
9. Corten B, Aarts F, Harms AS, Vogelaar J: Postoperative drug induced priapism. BMI Case Rep, 2017; 2017: pii: bcr-2016-218060
10. Giuliano F: Neurophysiology of erection and ejaculation. J Sex Med, 2011; 8(Suppl. 4): 310–15
11. Vasileiou I, Xanthos T, Koudouna E et al: Propofol: A review of its non-anesthetic effects. Eur J Pharmacol, 2009; 605: 1–8
12. Andersson KE: Pharmacology of penile erection. Pharmacol Rev, 2001; 53: 417–50
13. Bschleipfer T, Hauck EW, Diemer T et al: Heparin-induced priapism. Int J Impotence Res, 2001; 13: 357–59
14. Lin PH, Bush RL, Lumsden AB: Low molecular weight heparin induced priapism. J Urol, 2004; 172: 263
15. Anastei L, Kwan A: Heparin-induced priapism. Canadian Journal of Hospital Pharmacy, 2018; 52: 378–79
16. De Siati M, Chierigo P, Contin F et al: Priapism as a complication of heparin therapy. Arch Ital Urol Androl, 1999; 71: 201–2
17. Bick RL, Frenkel EP: Clinical aspects of heparin-induced thrombocytopenia and thrombosis and other side effects of heparin therapy. Clin Appl Thromb Hemost, 1999; 5(Suppl. 1): 57–15
18. Maples BL, Hagemann TM: Treatment of priapism in pediatric patients with sickle cell disease. Am J Health Syst Pharm, 2004; 61: 355–63