

Case Report

A rare case of normal erections in a patient with recurrent priapism treated with multiple shunt surgeries and anti-platelets

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ABSTRACT

This case report discusses a 23-year-old male with recurrent priapism and its management. Priapism, defined as prolonged and painful penile erection, can lead to severe complications including permanent erectile dysfunction (ED) and penile deformities. The patient presented with priapism lasting 34 hours, despite previous spontaneous resolution of similar episodes. Initial treatment involved blood aspiration and irrigation with epinephrine, but the priapism recurred. Various shunting procedures were attempted, yet recurrent priapism persisted. Discussion highlights the challenge of maintaining shunt patency due to thrombosis and the importance of blood flow through the shunt for healing. The report introduces the notion of pre-operative aspirin and post-operative heparin to prevent shunt closure, though guidelines do not currently support this approach. Prior research suggests aspirin's positive impact on erectile function through enhanced nitric oxide production and platelet inhibition. The report concludes by recommending a treatment protocol involving initial aspiration and irrigation, followed by pre-operative aspirin, shunt surgery, and post-operative aspirin for 6 months to prevent shunt closure. This case emphasizes the need for further studies to validate the efficacy of anti-platelet therapy as part of priapism management.

Keywords: Ischemic priapism, Shunt surgery, Refractory, Anti-platelets therapy, Aspirin

INTRODUCTION

The word “priapism” originated in Greek mythology. Priapus was the god of fertility, and his large phallus was the symbol of masculine power. The first case of priapism was published by Callos in 1845.¹⁻³ Priapism is defined as a prolonged penile erection lasting more than 4 hours, remaining despite orgasm and in the absence of sexual stimulation. There are three types of priapism: Ischaemic, non-ischaemic and stuttering. The most common of these is ischaemic priapism (IP). It represents a compartment syndrome of the corpus cavernosa with minimal or no arterial flow into the penis. It causes time-dependent damage to the smooth muscle, which results in significant long-term morbidities-permanent erectile dysfunction (ED), penile shortening, penile curvature and loss of girth. The first step in the management of priapism is to aspirate the blood of the corpus cavernosa with a large bore 16-18G butterfly needle. A sample of the aspirate should be sent

for arterial blood gas analysis. A hypoxic, hypercapnic and acidotic blood gas sample confirms the diagnosis of IP. Following aspiration, the corpus cavernosa should be irrigated with 0.9% normal saline. This may lead to successful de-tumescence in approximately 30% cases. If this fails, injection a sympathomimetic agent into the corpus cavernosa with simultaneous cardiac monitoring should be done. Shunt procedures are undertaken in patients with IP that are refractory to these initial management steps.⁴⁻⁶

CASE REPORT

Our index case is a 23-year-old male, who presented to casualty with complains of sustained painful erection for last 34 hours. He had history of similar complaints twice in past (1 month back), which resolved spontaneously after 6-8 hours. There was no history of trauma to the pelvis/perineum or penis. Patient was immediately taken up for

aspiration of pooled blood and irrigation with epinephrine (Figure 1). But patient developed recurrent priapism. Winter shunt, T shunts and Al Ghorab shunt (Figure 1C) was also attempted but, patient continued to develop recurrent priapism. Sub cutaneous heparin 5000 IU stat dose was given and the patient was started on anti-platelets, the patient achieved penile flaccidity. Patient was discharged on anti-platelets for 6 months. On 6 month follow up, patient was doing well and had no episode of recurrent priapism and had normal erections.

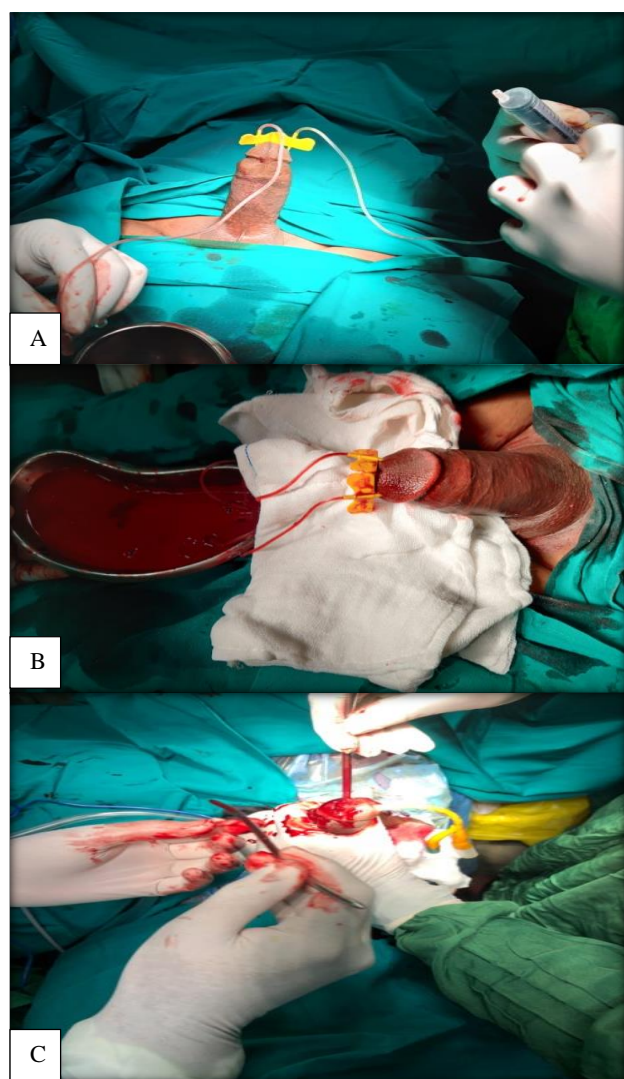


Figure 2: Irrigation of corpora cavernosa; aspiration of clots and old blood and Al Ghorab shunt.

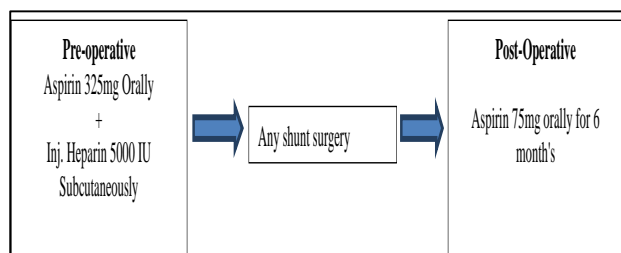


Figure 1: Priapism treatment regime.

DISCUSSION

In acute ischemic priapism that fail to respond to alpha-adrenergic agonists, various shunting procedures have been developed to re-establish circulation of the corpora cavernosa and prevent necrosis of erectile tissues. However, early closure of the newly created shunt, resulting in recurrent priapism, is a common complication which leads to repeated shunting procedures. In subsequent re-shunting, blood clots at the site of shunting and the un-coagulated “crank-case oil”-like old blood are removed. The old blood inside the corpora cavernosa does not clot due to the abundance of endothelium-derived anti coagulating and fibrinolytic factors within the corpora cavernosa. But, the newly created shunt is not lined and protected by endothelium. In fact, the shunts created, cuts a new wound through the collagen-rich tunica albuginea. Hence, the collagen-activated platelets and fibrin begin to form a clot within minutes to seal off the shunt. So, keeping the newly created shunt patent requires a continuous high blood flow through a large caliber shunt. This helps the shunt to remain open for hours to days until the smooth muscles of the corpora cavernosa and helicine arteries regain normal contractile capacity. Premature closure of the shunt is due to inadequate size and stagnant blood flow, which enhances the adherence of platelets and fibrin to collagen, and eventually forms a thrombus at the site of shunting and causes early recurrence of priapism. Hence, early postoperative shunt closure is a postoperative thrombotic complication.⁷ Hence, pre-operatively oral aspirin and injectable Heparin 5000 IU subcutaneously should be started 1 day prior to shunt surgery and post-operatively Aspirin should be continued for 6 months in order to prevent shunt closure (Figure 2).

According to AUA guidelines, they have not mentioned about starting anti-platelets pre-operatively as a treatment protocol for shunt surgery in order to prevent premature shunt closure.¹ But, in our practical experience, starting aspirin and heparin proved to be helpful.

A study conducted by Dilip et al reported a 20% preservation of normal erectile function after distal shunt procedures.⁸ A meta-analysis of randomized controlled trials (RCTs) revealed a significant improvement in erectile function following aspirin intake.

The process of achieving and maintaining a normal erection is regulated by various molecules released during sexual stimulation. One of these crucial molecules is nitric oxide (NO), which is released from nerves in the corpora cavernosa. NO diffuses into the smooth muscle cells of the cavernosa, increasing the concentration of cyclic guanosine monophosphate (cGMP) within these cells. Elevated levels of cGMP then lead to muscle relaxation by reducing calcium ion concentrations in the smooth muscle cells. This entire mechanism results in increased blood flow and better blood entrapment in the penile tissues, thereby strengthening the hydrostatic skeleton of the penis for an erection.⁹

Study done by Arthur et al commented that in clinical situations where recovery of natural erection is considered possible (i.e., ischemic priapism lasting less than 36 hours in a previously potent patient), intervention should ideally aim for this possibility. Treatment then consists of applying minimally invasive procedures initially followed by successively invasive procedures as necessary. On the opposite end of the spectrum, for clinical situations in which natural erection recovery is considered to be all but impossible (i.e., ischemic priapism lasting continuously for more than 72 hours), immediate penile prosthesis surgery may be indicated as an ideal therapeutic option. The management of ischemic priapism that occurs between these time lines may be acceptably managed by either surgical approach.¹⁰ In our patient, the patient had refused for penile prosthesis. But, pre-operative treatment with aspirin and administering aspirin for 6 months post-operatively helped him in achieving normal erections.

However, the cGMP pathway is affected by cGMP-specific phosphodiesterase (PDE) type 5 enzyme, which is abundantly expressed in the cavernosa. This PDE-5 enzyme deactivates cGMP, impacting the duration of the erection. Interestingly, the NO pathway has other vital functions in the body, including the prevention of platelet aggregation. Aspirin contributes to increased blood flow in vessels not only by inhibiting platelet aggregation but also by directly stimulating the activity of endothelial NO synthase, leading to enhance NO production for smooth muscle relaxation.⁹ Thus, administering aspirin to our patient helped him achieve normal erections.

CONCLUSION

In cases of ischemic priapism, initiate treatment with aspiration and irrigation using alpha-adrenergic blockers. If ineffective, begin preoperative oral aspirin and subcutaneous heparin 5000 IU. Follow with shunt surgery and continue postoperative aspirin for 6 months to prevent shunt closure. Given the single patient nature of this report, further studies are warranted to ascertain the efficacy of preoperative anti-platelet therapy in priapism management.

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