

Spontaneous resolution of delayed onset, posttraumatic high-flow priapism

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ABSTRACT

Priapism is an uncommon but serious condition with major long-term sequelae. The commoner, ischemic variety is a medical emergency requiring immediate intervention in order to avoid erectile dysfunction. However, the high flow variety is non-ischemic and the patient may not seek immediate therapy. The options of management for this rare, painless form of priapism vary from conservative therapy to embolization of the internal pudendal artery or, in some cases, surgery that may result in subsequent erectile dysfunction. We present a case of a 24-year-old man who presented with priapism of six-day duration, 10 days after perineal injury. Doppler ultrasonogram of the penis revealed a cavernosal artery pseudoaneurysm. Pudendal artery angiogram done four days later revealed no leak and the priapism subsided spontaneously with subsequent return of normal erections.

KEY WORDS: Embolization, erectile dysfunction, high-flow, priapism, spontaneous, trauma

High-flow priapism usually results from trauma to the perineum with injury of the cavernous tissue or the cavernosal artery. These patients are often asymptomatic at the time of trauma but develop delayed onset priapism following a nocturnal erection.^[1] This priapism is painless and not a medical emergency since the penile blood is well-oxygenated.^[2] The options for management include conservative measures, selective embolization of pudendal artery or its branches and surgery. The major complications of embolization and surgery are penile gangrene and erectile dysfunction.^[3] Prudent selection of the management strategy is important to avoid these drastic complications. We describe a patient who presented with post-traumatic delayed onset priapism that resolved spontaneously with return of normal erections. This report strengthens the philosophy of initial conservative management for these patients.^[4]

Case History

A 24-year-old male presented to us with a 10-day history of blunt trauma to the perineum with a bicycle handlebar. He was asymptomatic for the initial four days but then developed persistent painless erection that did not subside. Examination revealed a tumescent penis that was compressible. Blood gas analysis of corporal blood showed well-oxygenated arterial blood with pH 7.3. Doppler ultrasonogram of the penis showed a pseudoaneurysm arising from the left cavernosal artery

[Figure 1]. Since the erection was painless with well-oxygenated corporal blood, conservative management was chosen. Ice compresses were applied for the first two days with no effect. When the erection failed to subside completely at four days, selective pudendal artery angiogram was performed [Figure 2]. The angiogram did not reveal any aneurysm or leak and embolization was not done. Three days after the angiogram, the priapism spontaneously subsided. Two weeks following

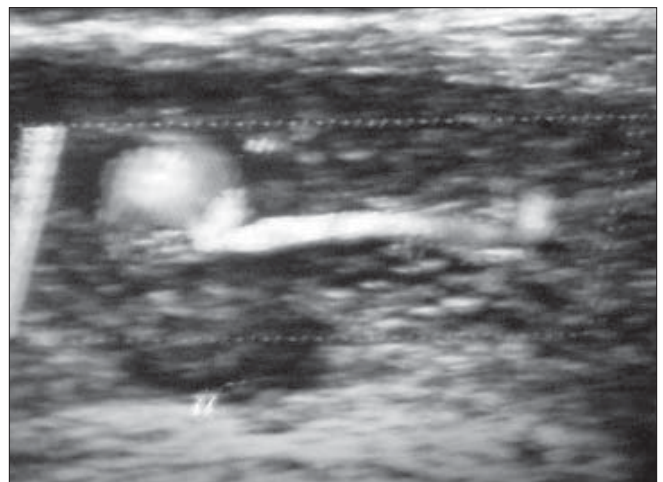


Figure 1: Duplex ultrasonogram of the cavernosal artery showing a pseudoaneurysm with flow



Figure 2: Selective left pudendal artery angiogram showing no leak

subside of priapism the patient reported normal erections.

Discussion

Priapism following blunt perineal trauma usually has a delayed onset. It results from rupture of the damaged cavernosal vessel following a natural erection, usually a nocturnal erection.^[1] Since the venous channels are normal, they are able to compensate partially for the increased blood flow and the erection is incomplete and compressible. The presence of this outflow mechanism also ensures that the blood within the penis is arterial and well-oxygenated and, unlike the ischemic variety, is not a medical emergency.^[2]

This condition is fairly uncommon and its management has changed over time as more experience is accumulated. It has been documented that early onset priapism following trauma may subside spontaneously.^[5] The management of delayed onset high-flow priapism has included pudendal angiography and embolization because it was believed that these cases are less likely to have a spontaneous resolution.^[6] However, recent work on the etiology and outcomes of therapy for this condition have led to a shift towards conservative measures.^[4] Spycher *et al*^[7] have shown that unlike in the ischemic variety, these cases do not undergo fibroblast-like cellular

transformation of the smooth muscle cells which could predispose to subsequent erectile dysfunction. The corporal tissue is well-oxygenated and the erection is incomplete, compressible and painless. Unlike traditional arteriovenous fistulae, this condition possibly has an arterio-lacunar fistula with blood flowing directly from the injured cavernosal artery into the lacunar spaces of the cavernosa.^[3] This may also explain the spontaneous resolution since these fistulae with less rigid walls of the lacunar spaces may be more prone to spontaneous thrombosis. A recent review and guidelines by the American Urological Association suggests that up to 62% cases may have a spontaneous resolution.^[4] Moreover, interventions such as embolization or surgery may be associated with erectile dysfunction in up to 50% cases.^[4]

Our patient had a delayed onset high-flow priapism with a documented injury to the cavernosal vessel on the ultrasonogram. Our initial decision to manage the patient conservatively was based on the above recommendations. When the erection did not subside after six days, we suggested an angiogram with possible embolization. However, no abnormality was seen on the angiogram and the patient had a spontaneous resolution subsequently. This case, thus reinforces the decision to manage these patients conservatively and avoid angiographic embolization.

References

1. Ilkay AK, Levine LA. Conservative management of high flow priapism. *Urology* 1995;46:419-24.
2. Sadeghi-Nejad H, Dogra V, Seftel AD, Mohamed MA. Priapism. *Radiol Clin North Am* 2004;42:427-43.
3. Bastuba MD, Saenz de Tejada I, Dinlenc CZ, Sarazen A, Krane RJ, Goldstein I. Arettrial priapism: Diagnosis, treatment and long-term follow-up. *J Urol* 1994;151:1231-7.
4. Montague DK, Jarow J, Broderick GA, Dmochowski RR, Heaton JP, Lue TF, *et al.* American Urological Association guideline on the management of priapism. *J Urol* 2003;170:1318-24.
5. Ricciardi R Jr, Bhatt GM, Cynamon J, Bakal, CW, Melman A. Delayed high flow priapism: Pathophysiology and management. *J Urol* 1993;149:119-21.
6. Walker TG, Grant PW, Goldstein I. 'High-flow' priapism: Treatment with superselective transcatheter embolization. *Radiology* 1990;174:1053-4.
7. Spycher MA, Hauri D. The ultrastructure of the erectile tissue in priapism. *J Urol* 1986;135:142-7.

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