

High-flow priapism: an overview of diagnostic and therapeutic concepts

Yves Caumartin, MD,¹ Luc Lacoursière, MD,² Alain Naud, MD¹

¹Service d'urologie, Centre Hospitalier Universitaire de Québec, pavillon Hôtel-Dieu de Québec, Québec, Canada

²Département de radiologie, Centre Hospitalier Universitaire de Québec, pavillon Hôtel-Dieu de Québec, Québec, Canada

CAUMARTIN Y, LACOURSIERE L, NAUD A. High-flow priapism: an overview of diagnostic and therapeutic concepts. *The Canadian Journal of Urology*. 2006;13(5):3283-3290.

We describe the case of a 23 year-old man with high-flow priapism following blunt perineal trauma. He was treated successfully with super-selective

embolization with a resorbable material (gel foam). We will review the pathophysiology, etiology, and diagnosis and treatment approaches of high-flow priapism based on a review of the medical literature.

Key Words: priapism, embolization, therapeutic, ultrasonography, erectile dysfunction, therapy

Introduction

Priapism is defined as a prolonged pathologic erection not temporally related to sexual stimulation. Studies of the physiopathology of priapism have described two distinct types.¹⁻³

Most cases of priapism occur as a result of engorgement of the corpora cavernosa from intrinsic or extrinsic venous outflow obstruction, called veno-occlusive priapism (VOP). This type of priapism is

characterized by little or no cavernous blood flow due to obstruction of the subtunical venules with associated venous stasis, hypoxia, acidosis and tissue ischemia. Cavernous blood gases are abnormal and patients typically report a fully erect and rigid penis that is quite painful. This represents an emergency situation because failure to achieve quick detumescence can be associated with cavernous fibrosis and a high rate of erectile dysfunction (ED).

Less frequently, priapism may be due to uncontrolled arterial inflow to the corporal sinusoids, usually as the direct result of trauma to a cavernous artery, and this is termed arterial priapism or high-flow priapism (HFP). In this case, venous outflow is not completely occluded and tissue hypoxia does not occur. Typically, the penis is neither fully rigid nor

Accepted for publication May 2006

Address correspondence to Dr. Alain Naud, CHUQ, Hôtel-Dieu de Québec, 11, Côte du Palais, Québec, Québec G1R 2J6 Canada

painful. In absence of severe pain, there may be a prolonged time period (months to years reported) before the patient seeks medical attention.^{4,5} This situation does not require immediate treatment and is associated with a good prognosis. Differentiating these two types of priapism is important because treatments and outcomes are different. We report our limited experience with HFP in a 23 year-old man and also review the medical literature on the different diagnostic and therapeutic strategies.

Case report

A 23 year-old healthy Caucasian male presented to the emergency room complaining of painless penile tumescence for 2 weeks. He had not experienced priapism in the past. He reported a history of straddle-like injury after a skateboard fall. He noticed penile rigidity 7 days following the trauma. There was no history of hematuria or dysuria. He reported that he didn't take any medication but did smoke marijuana occasionally and drank alcohol on weekends. On physical examination, the corporal bodies were turgid and non-tender, while the glans and spongiosum were soft and compressible. There were no hematomas. Complete blood count and urinalysis were both normal.

The priapism was initially investigated and treated with corporal aspiration. The blood was a bright-red color. Unfortunately, the blood gases were lost and so these results were never received. No improvement was seen after this intervention. We then performed a gray-scale and color Doppler ultrasound (CDUS) of the penis showing an irregular hypoechoic and mainly

cystic cavity at the base of the right corporal body representing a ruptured right cavernous artery with high flow arterio-lacunar fistula, Figures 1a and 1b. The patient was reassured and discharged with direction to apply ice locally for a few days, with no subsequent improvement.

Angiography was performed 7 days later with the intention to localize and embolize the fistula. A left femoral artery approach was used to perform the aortogram, which confirmed the persistence of an arterio-lacunar fistula. A 5 French catheter was used selectively for pudental artery injections and a microcatheter system was inserted coaxially and later advanced in the common penile artery which showed a ruptured cavernous artery and an arterio-lacunar fistula with early venous drainage, Figure 2. This right cavernous artery was supra-selectively embolized with gelfoam until the fistula was completely occluded while the dorsal artery of the penis was preserved, Figure 3. Non-selective injections later showed a persistent fistula at the bulbo-urethral artery, which was also selectively embolized, Figure 4.

CDUS performed at the end of the embolization and repeated a few hours later confirmed the occlusion of the largest cavity (fistula) with a few millimetric residual fistula around the cavernous artery. The ipsilateral cavernous artery was patent on CDUS, most likely from contralateral collateral vessels not seen on ipsilateral angiogram.

The patient was discharged at the end of the day with only a slight partial tumescence. Flaccid state was normally achieved spontaneously 24 hours later. At follow up, however, the patient complained of mild

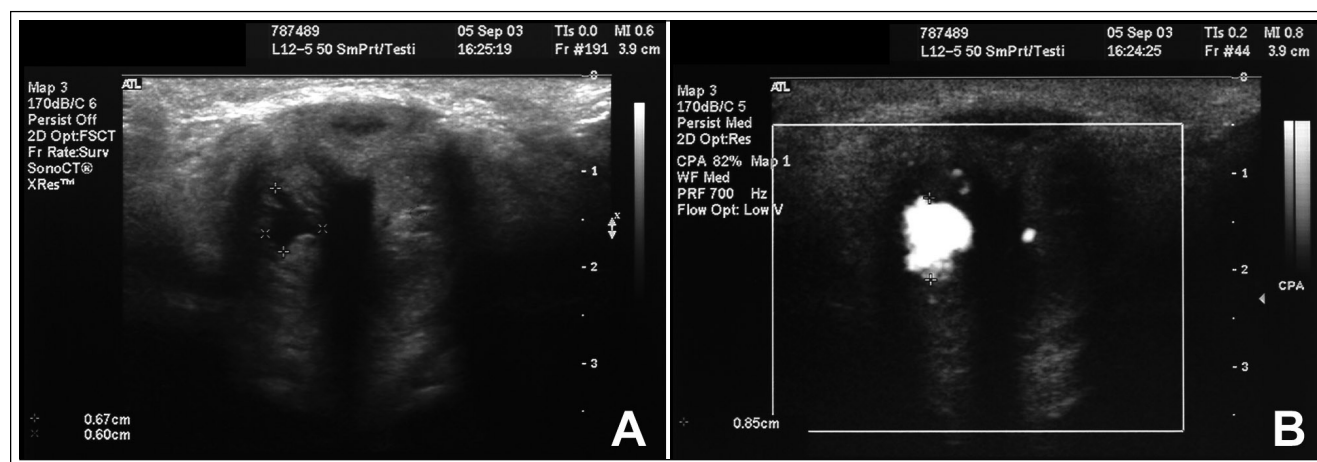


Figure 1. Ultrasound imaging of the proximal penis. a) Gray-scale ultrasound shows an irregular cystic cavity (+) corresponding to the ruptured cavernous artery. b) Color Doppler ultrasound of an adjacent region confirms the high flow arterio-lacunar fistula (+) from the right cavernous artery.

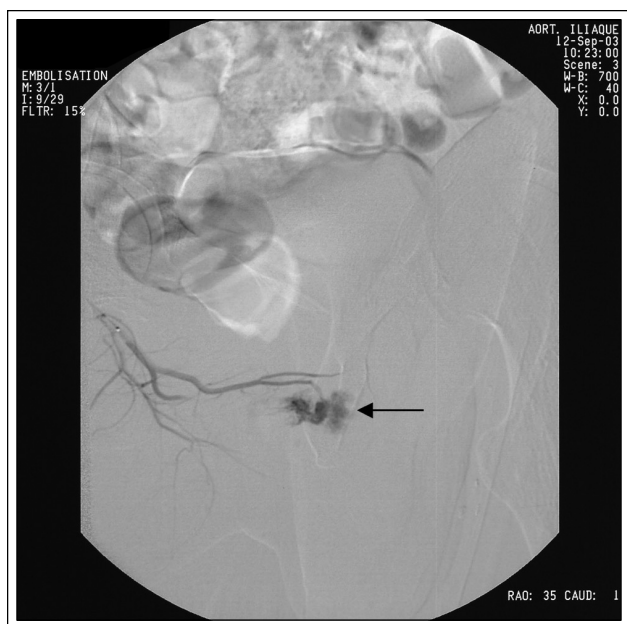


Figure 2. Internal pudendal angiogram shows the extravasation arising from the right cavernous artery (black arrow).

ED compared to baseline. Complete normalization of this patient's sexual function finally resolved approximately 3 months post-embolization.

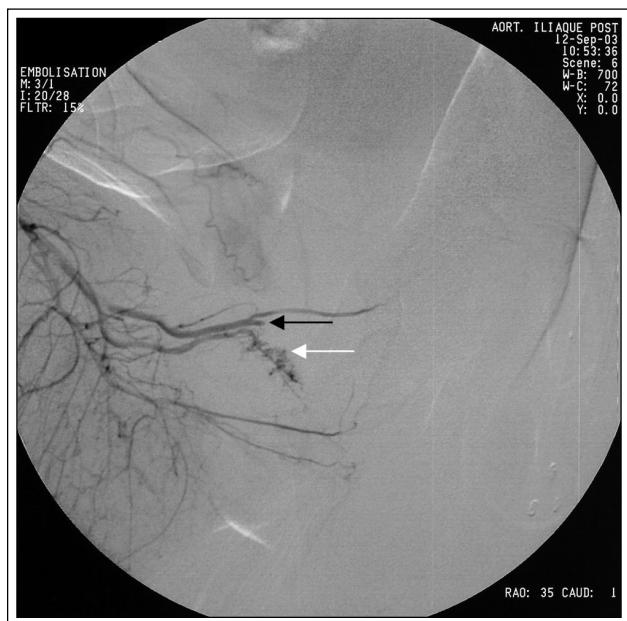


Figure 3. Post-embolization angiogram shows no further filling of the fistula from the occluded cavernous artery (black arrow). However, a new fistula is seen arising from the bulbo-urethral artery (white arrow).

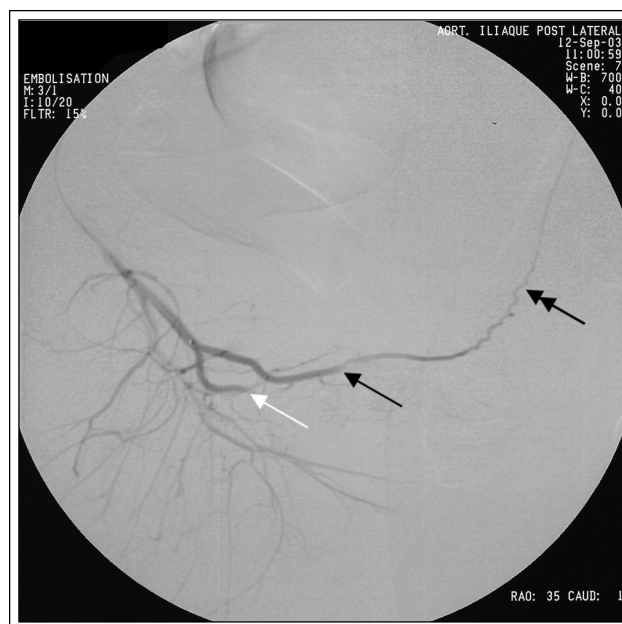


Figure 4. Post-embolization angiogram shows no further filling of the fistula from occluded cavernous (small black arrow) and bulbo-urethral artery (white arrow). Injection also shows the patent dorsal artery (double black arrow).

Discussion

Priapism is a mysterious disorder known from many hundred years but modern diagnosis and therapeutic rational approach has been credited to Franck Hinman.⁶ The first published case of suspected HFP was reported by Burt et al in 1960, who successfully treated the priapism by ligation of the right internal pudendal artery.⁷ Wheeler and Simmons used angiography to demonstrate that traumatic priapism was the result of an arteriovenous fistula.⁸ The pathophysiology of HFP was first suggested by Hauri et al in 1983 and shown to originate at the level of the helicine arteries by a loss of nervous regulation.² Witt et al further elaborated on this condition and, based on their results associated with angiography and embolization, concluded that injury to the artery creates a direct fistula to the lacunar spaces, bypassing the regulatory and highly resistant helicine arteries.³ Because the subtunical venules are not uniformly and completely compressed, adequate venous outflow occurs and this prevents blood stasis, hypoxia and the tissue damage that has been associated with VOP.⁹

Distinguishing between the two subtypes of priapism is crucial for good medical management. In HFP, the patient typically seeks medical attention for a persisting painless erection. Patients typically reveal

a history of penile or perineal trauma as the main etiology. There is often a delay in the onset of the priapism after the traumatic event. Less frequently, HFP may be spontaneous or idiopathic,¹⁰ may be linked to laceration of the cavernous artery secondary to intracavernous injection,^{1,3,11} may occur post-arteriolization of the deep dorsal vein for vasculogenic impotence¹² or can be provoked by surgical interventions (shunts) performed to treat VOP.¹³

Moreover, rare cases of high-flow state occurring after resolution of ischemic priapism have been reported but the pathophysiology is not well understood. Dysregulation of vasorelaxing/ vasoconstrictive factors resulting from ischemic damage have been suggested as a possible mechanism.¹⁴ Sickle cell disease,¹⁵ Fabry disease,¹⁶ cocaine injection¹⁷ and neoplasms of the lung,¹⁸ prostate¹⁹ and bladder²⁰ are examples. Patients with these conditions usually report past episodes of VOP.

On physical examination, genitalia bruises or swelling can be noticed. The penis is partially erect with sparing of the corpus spongiosum. The peisis sign is partial or complete penile detumescence with compression of the perineum with the fingers. This was described in two pediatric patients where it was suggested to be a specific physical examination sign of arterial priapism.²¹

Corporal aspiration has been used routinely to distinguish HFP from VOP. In HFP, bright red blood with an oxygen content similar to arterial blood has been found. This manoeuvre should only be conducted when therapeutically necessary because blood aspiration has not demonstrated therapeutic efficacy in HFP.²² Since corporal aspiration is an invasive technique with potential complications (iatrogenic cavernous artery laceration), we recommend using it only if the veno-occlusive subtype is a concern after anamnesis and physical examination (painful erection, non-traumatic etiology, recurrent priapism, and risk factors for ischemic subtype). In classical HFP, aspiration is of no additional diagnostic or prognostic value. Nonetheless, in difficult cases it can provide important diagnostic information or be part of the treatment if veno-occlusive symptoms are present.²²

The CDUS is the imaging modality of choice for confirming the diagnosis.^{23,24} It is a non-invasive procedure that correlates well with the angiogram.^{4,11} It usually allows for precise localization of the fistula if intervention is planned²³ and it is an adjunct to intervention during embolization or surgery.^{25,26} It also helps when performing guided-doppler compressions²⁷ and is an essential tool for the follow up of both watchful waiting and treated patients.^{4,11} CDUS can miss fistula

localized proximally under the pubis because of sonic attenuation of pubic bone.²⁴ Also, the number and origin of the feeding vessels of the fistula may not be accurate since the vasculature of a corpus cavernosum can occasionally arise from the contralateral side.^{4,28}

Several treatment options, such as shunts, intracavernous injection of alpha-adrenergic agonists or methylene blue, guided-doppler compressions, direct percutaneous injection of occlusive material in the fistula, surgical ligation of the cavernous artery and selective embolization of the cavernous artery have been tried. The optimal treatment for HFP is still a matter of debate. An example of management of HFP is presented in Figure 5. Among outcomes of these therapies, therapeutic efficacy and sexual function are the main considerations. Many authors propose a conservative strategy to manage HFP. Unfortunately, the natural history of this condition is unclear. Only a few authors have reported spontaneous occlusion of the fistula. Several published cases are quite remarkable in demonstrating that time from trauma to patient presentation, ranging from days to years, has no significant impact on subsequent outcome, and that many patients remain potent after spontaneous resolution of priapism.

On the other hand, the long-term effect of HFP on erectile tissue still needs to be clarified. Hakim et al followed five patients who underwent a period of watchful waiting during their course of treatment. In none of these cases did spontaneous closure of the fistula occur. The duration of priapism was up to 36 years. One watchful waiting period of almost 31 years without a significant negative outcome was observed.⁴ However, in two of the five cases, the quality of the erection changed over the long-term follow up. Recently, the American Urological Association published guidelines on the management of priapism. They concluded that 33% of patients experienced erectile difficulties among those patients who received conservative treatment (ice, rest, observation).²² Savoca et al reported on sexual function over the long term following super-selective embolization for 11 patients with HFP. Three patients experienced decreased potency, including two patients with long-standing priapism (12 and 30 years) and one patient with fibrosis and veno-occlusive ED.⁵ It is unclear if this high-flow state of prolonged superphysiologic oxygen tension damages the underlying cavernosal tissue in any way when the penis, in its flaccid state, normally receives a venous oxygen level. Most of the time, post-traumatic ED secondary to blunt trauma to the perineum or pelvis has been linked to corporal veno-occlusive dysfunction and cavernous artery insufficiency.²⁹

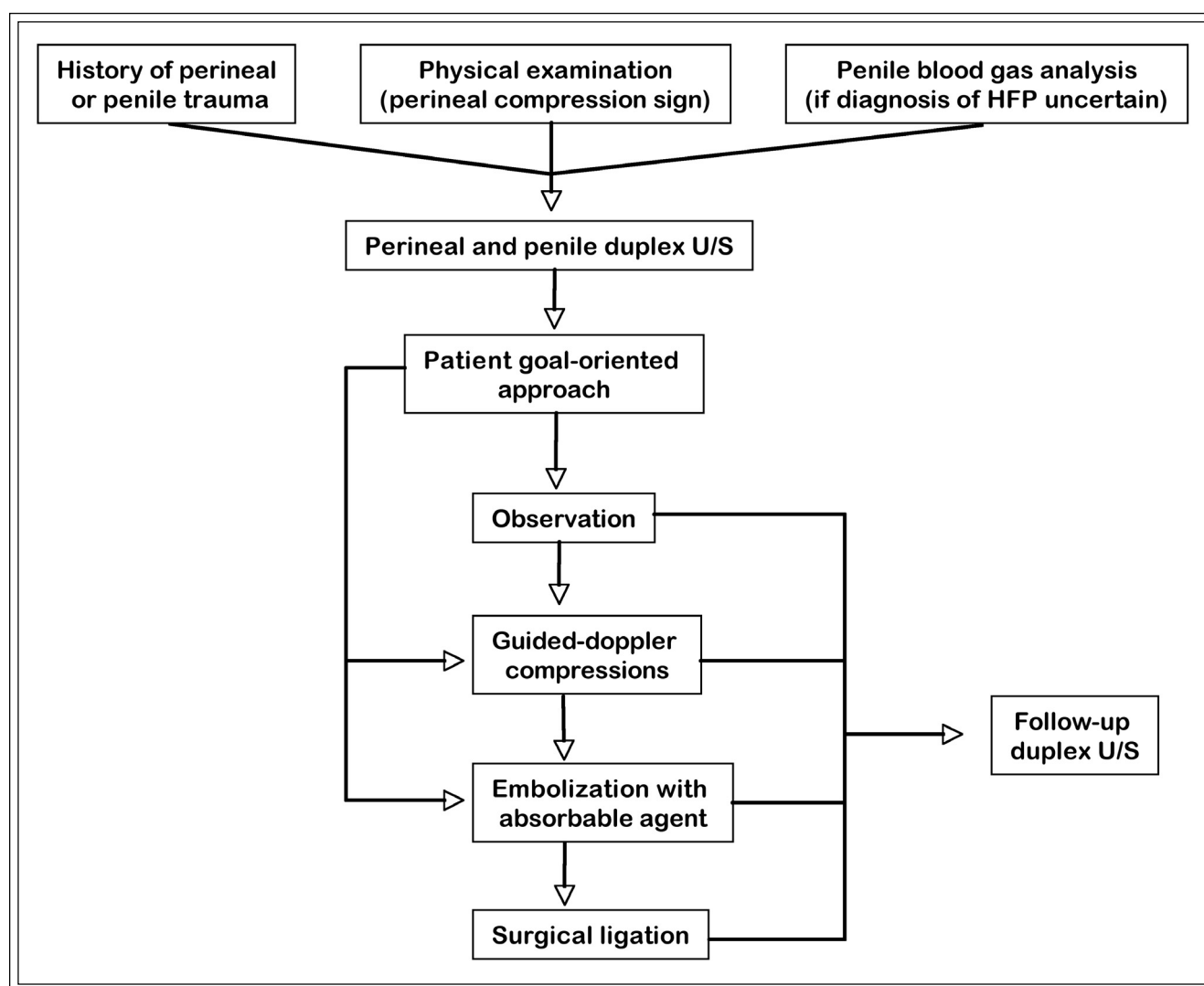


Figure 5. Management of high-flow priapism.

A variety of conservative modalities have been used (compression, ice, rest). However, only a few cases of spontaneous resolution of the fistula have been published. Most of these were in children or else occurred many years later. Recently, conservative management with guided-doppler compressions have been reported to be successful.^{27,30} This strategy has been used in the treatment of pseudoaneurysm resulting from arterial puncture.³¹ We, unfortunately, have limited experience with this modality of treatment. Based on these results, we believe that an observational period, with or without adjunct modalities, can be useful for small fistulas to allow for possible spontaneous closure. If priapism does not resolve after a reasonable period, still to be defined, highly selective embolization should be performed. Conversely, for large fistulas in which spontaneous

occlusion is unlikely, a long observational period is considered hazardous as we really do not understand the long-term effects of excessive arterial inflow on the erectile tissue.

Historically, corporospongiosal shunts have been used extensively in HFP with very poor results. Now that we better understand the pathophysiology of this condition, it is clear that this treatment is inappropriate for HFP because adequate venous outflow already exists. Moreover, intracavernous injection of alpha-adrenergic agonists have been attempted, and two reports show documented resolution of post-traumatic priapism using metaraminol bitartrate injection. In one of these reports, however, six injections over a 10 day period were required,³² and in the other, the injection was associated with severe systolic hypertension up to 200 mmHg.³³ This

technique, therefore, is associated with undesirable effects on the cardiovascular system due to the pathophysiology of unregulated arterial inflow and large venous outflow that are characteristic of this condition. In contrast, many investigators have reported a transient detumescence associated with injection of alpha stimulants with eventual recurrence of the high-flow state.^{1,34} Because of these restrictions and weak efficacy of this treatment, intracavernous injection of alpha-adrenergic agonists is discouraged.²² Intracavernous methylene blue, an inhibitor of guanylate cyclase, may have some efficacy but experience is very limited.³⁵

In the last years, super-selective embolization of the internal pudental or cavernous artery has evolved as the strategy of choice in the treatment of HFP. This was first described by Wear et al who used autologous clot to embolize the internal pudental artery.³⁶ Many authors have reported good results with resolution of priapism and preservation of potency. So far, different occlusive materials have been used which can be divided in two groups, absorbable and permanent. Absorbable materials (autologous clot, gelatin sponge, gel foam) have been used extensively.^{4,11,37,38} The advantage of using absorbable material is that this permits early recanalization of the artery after healing of the fistula. Theoretical disadvantages include increased recurrence rate and a greater number of sessions needed to occlude the fistula. After an embolization session without complete detumescence, combination therapy with guided-doppler compressions has been used successfully to achieve fistula occlusion, avoiding the need for further embolization treatment.^{39,40} Permanent embolization techniques involve the use of coil, polyvinyl alcohol particules and n-cyano-butyl-acrylate.^{41,42} They have been used with success and have the theoretical advantage of less recurrence. This technique is thought to be associated with more ED than non-permanent embolization. When recanalization did not occur, vascularization of the cavernous body is usually warranted by the contralateral cavernous artery through septal vascular communications. According to the American Urological Association guidelines on the management of priapism, there is more ED associated with permanent embolization materials than with temporary techniques (39% versus 5%, respectively) with a comparable resolution rate (78% versus 74%, respectively).²² Savoca et al reported their experience with 15 patients with HFP treated with polyvinyl alcohol embolization. Seventy-three percent of these patients showed no fistula recurrence and 20% reported a slight change in the quality of erections.⁵

Ciampalini et al showed, on a series of nine patients treated with embolization using absorbable material, a 44% recurrence rate and 11% ED rate.¹⁰ Since this is a rare condition, no randomized studies have been done so far to clarify the question that remains which is what material should be used to achieve the best occlusion rate of the fistula with optimal preservation of potency. Embolization is usually well tolerated even among patients requiring many embolizations but complications associated with the angiography technique must not be underestimated. Well-known complications such as leg emboli, pseudoaneurysm, femoral artery access bleeding, allergic reaction to contrast medium, femoral neuropathy are all possible. Perineal abscess formation and migration of infected emboli causing cavernositis have both been reported following pudental super-selective embolization.^{4,43}

Surgical ligation, as an alternative to angiographic embolization, could be performed by ligating the cavernous artery at the outlet from Alcock's canal or by performing exploratory corporotomy followed by microsurgical closure of the arterial fistula and feeding vessels.²⁵ In both procedures, CDUS would be useful to localize and confirm resolution of the fistula. Because there is a high ED rate of up to 50% associated with the surgical treatment, it should be reserved for recurrent HFP in patients who failed other forms of treatment.²²

The first procedure resolves the priapism but the ipsilateral cavernous artery is irreversibly occluded. Retrograde revascularization through the contralateral cavernous artery may be possible. However, in this case, it would be necessary to perform an ultrasound study to confirm the presence of a contralateral cavernous artery before definitive ligation.

The second approach is a more selective but a more complex surgery. Dissection of the corpora places the patient at higher risk of ED due to scarring of the tunica albuginea and secondary venous leakage. This second surgical method is preferred for long duration priapism, because it allows formation of a vascular pseudocapsule and development of significant collateral circulation originating from the contralateral corpora to occur. This pseudocapsule can be documented by CDUS. In these cases, ligation of all the feeding vessels within the fistula itself decreased the risk of failure.^{1,2,25}

Conclusions

In conclusion, diagnosis of HFP is usually made clinically. Cavernous blood gases should be reserved for atypical presentation in patients with a past history or with risk factors for VOP. CDUS is the best radiological modality to confirm and localize the fistula

and allows for precise follow up. It is very sensitive, non-invasive and widely available. Guided-doppler compression is a conservative treatment option that can be explored initially. If conservative treatment fails or if a more aggressive treatment is required, embolization should be the first line strategy because of the good resolution rate and low rate of associated ED. Resorbable materials have potential benefits on preserving erectile function possibly at the price of a higher recurrence rate. Surgical procedures must be reserved for cases that fail less invasive therapy. □

References

1. Brock G, Breza J, Lue TF, Tanagho EA. High flow priapism: a spectrum of disease. *J Urol* 1993;150:968-971.
2. Hauri D, Spycher M, Bruhlmann W. Erection and priapism: a new physiopathological concept. *Urol Int* 1983;38:138-145.
3. Witt MA, Goldstein I, Saenz de Tejada I, Greenfield A, Krane RJ. Traumatic laceration of intracavernosal arteries: the pathophysiology of nonischemic, high flow, arterial priapism. *J Urol* 1990;143:129-132.
4. Hakim LS, Kulaksizoglu H, Mulligan R, Greenfield A, Goldstein I. Evolving concepts in the diagnosis and treatment of arterial high flow priapism. *J Urol* 1996;155:541-548.
5. Savoca G, Pietropaolo F, Scieri F, Bertolotto M, Mucelli FP, Belgrano E. Sexual function after highly selective embolization of cavernous artery in patients with high flow priapism: long-term followup. *J Urol* 2004;172:644-647.
6. Hinman F. Priapism: report of cases in a clinical study of the literature with reference to its pathogenesis and surgical treatments. *Ann Surg* 1914;60:689.
7. Burt FB, Schirmer HK, Scott WW. A new concept in the management of priapism. *J Urol* 1960;83:60-61.
8. Wheeler GW, Simmons CR. Angiography in post-traumatic priapism. A case report. *Am J Roentgenol Radium Ther Nucl Med* 1973;119:619-620.
9. Spycher MA, Hauri D. The ultrastructure of the erectile tissue in priapism. *J Urol* 1986;135:142-147.
10. Ciampalini S, Savoca G, Buttazzi L, Gattuccio I, Mucelli FP, Bertolotto M, De Stefani S, Belgrano E. High-flow priapism: treatment and long-term follow-up. *Urology* 2002;59:110-113.
11. Bastuba MD, Saenz de Tejada I, Dinlenc CZ, Sarazen A, Krane RJ, Goldstein I. Arterial priapism: diagnosis, treatment and long-term followup. *J Urol* 1994;151:1231-1237.
12. Wolf JS Jr, Lue TF. High-flow priapism and glans hypervascularization following deep dorsal vein arterialization for vasculogenic impotence. *Urol Int* 1992;49:227-229.
13. Rodriguez J, Cuadrado JM, Frances A, Franco E. High-flow priapism as a complication of a veno-occlusive priapism: two case reports. *Int J Impot Res* 2005.
14. Burnett AL. Pathophysiology of priapism: dysregulatory erection physiology thesis. *J Urol* 2003;170:26-34.
15. Hoffman S, Kaynan AM, Melman A. Priapism of ambiguous classification in a sickle cell patient. *Int J Impot Res* 2000;12:59-63.
16. Foda MM, Mahmood K, Rasuli P, Dunlap H, Kiruluta G, Schillinger JF. High-flow priapism associated with Fabry's disease in a child: a case report and review of the literature. *Urology* 1996;48:949-952.
17. Altman AL, Seftel AD, Brown SL, Hampel N. Cocaine associated priapism. *J Urol* 1999;161:1817-1818.
18. Greschner M, Krautschick A, Alken P. High-flow priapism leading to the diagnosis of lung cancer. *Urol Int* 1998;60:126-127.
19. Schroeder-Printzen I, Vosschenrich R, Weidner W, Ringert RH. Malignant priapism in a patient with metastatic prostate adenocarcinoma. *Urol Int* 1994;52:52-54.
20. Dubocq FM, Tefilli MV, Grignon DJ, Pontes JE, Dhabuwala CB. High flow malignant priapism with isolated metastasis to the corpora cavernosa. *Urology* 1998;51:324-326.
21. Hatzichristou D, Salpiggidis G, Hatzimouratidis K, Apostolidis A, Tzortzis V, Bekos A, Saripoulos D. Management strategy for arterial priapism: therapeutic dilemmas. *J Urol* 2002;168:2074-2077.
22. Montague DK, Jarow J, Broderick GA, Dmochowski RR, Heaton JP, Lue TF, Nehra A, Sharlip ID. American Urological Association guideline on the management of priapism. *J Urol* 2003;170:1318-1324.
23. Feldstein VA. Posttraumatic "high-flow" priapism evaluation with color flow Doppler sonography. *J Ultrasound Med* 1993;12:589-593.
24. Kang BC, Lee DY, Byun JY, Baek SY, Lee SW, Kim KW. Post-traumatic arterial priapism: colour Doppler examination and superselective arterial embolization. *Clin Radiol* 1998;53:830-834.
25. Shapiro RH, Berger RE. Post-traumatic priapism treated with selective cavernosal artery ligation. *Urology* 1997;49:638-643.
26. Volkmer BG, Nessler T, Kuefer R, Kraemer S, Goerich J, Gottfried HW. High-flow priapism: a combined interventional approach with angiography and colour Doppler. *Ultrasound Med Biol* 2002;28:165-169.
27. Mabjeesh NJ, Shemesh D, Abramowitz HB. Posttraumatic high flow priapism: successful management using duplex guided compression. *J Urol* 1999;161:215-216.
28. Bertolotto M, Quaia E, Mucelli FP, Ciampalini S, Forgacs B, Gattuccio I. Color Doppler imaging of posttraumatic priapism before and after selective embolization. *Radiographics* 2003;23:495-503.
29. Munarriz RM, Yan QR, A ZN, Udelson D, Goldstein I. Blunt trauma: the pathophysiology of hemodynamic injury leading to erectile dysfunction. *J Urol* 1995;153:1831-1840.
30. Sancak T, Conkbayir I. Post-traumatic high-flow priapism: management by superselective transcatheter autologous clot embolization and duplex sonography-guided compression. *J Clin Ultrasound* 2001;29:349-353.
31. Hood DB, Mattos MA, Douglas MG, Barkmeier LD, Hodgson KJ, Ramsey DE, Sumner DS. Determinants of success of color-flow duplex-guided compression repair of femoral pseudoaneurysms. *Surgery* 1996;120:585-588;discussion 588-590.
32. Koga S, Shiraishi K, Saito Y. Post-traumatic priapism treated with metaraminol bitartrate: case report. *J Trauma* 1990;30:1591-1593.
33. Mizutani M, Nakano H, Sagami K, Nihira H. Treatment of post-traumatic priapism by intracavernous injection of alpha-stimulant. *Urol Int* 1986;41:312-314.
34. Ricciardi R Jr, Bhatt GM, Cynamon J, Bakal CW, Melman A. Delayed high flow priapism: pathophysiology and management. *J Urol* 1993;149:119-121.
35. Martinez Portillo F, Hoang-Boehm J, Weiss J, Alken P, Junemann K. Methylene blue as a successful treatment alternative for pharmacologically induced priapism. *Eur Urol* 2001;39:20-23.
36. Wear JB Jr, Crummy AB, Munson BO. A new approach to the treatment of priapism. *J Urol* 1977;117:252-254.
37. Gorich J, Ermis C, Kramer SC, Fleiter T, Wisianowsky C, Basche S, Gottfried HW, Volkmer BG. Interventional treatment of traumatic priapism. *J Endovasc Ther* 2002;9:614-617.
38. Roussel G, Robert Y, Mahe P, Soret R, Legeais D, Meria P, Biserte J, Mazeman E. High-flow priapism in children: immediate treatment by selective embolization. *Eur J Pediatr Surg* 2001;11:350-353.

39. Cakan M, Altu G, Ucaron U, Aldemir M. Is the combination of superselective transcatheter autologous clot embolization and duplex sonography-guided compression therapy useful treatment option for the patients with high-flow priapism? *Int J Impot Res* 2005.
40. Liguori G, Garaffa G, Trombetta C, Capone M, Bertolotto M, Pozzi-Mucelli F, Belgrano E. High-flow priapism (HFP) secondary to Nesbit operation: management by percutaneous embolization and colour Doppler-guided compression. *Int J Impot Res* 2005;17:304-306.
41. Bennett JD. Evidence-based radiology problems. High-flow post-traumatic priapism: transcatheter embolotherapy: October 2003-September 2004. *Can Assoc Radiol J* 2003;54:207-210.
42. Gujral S, MacDonagh RP, Cavanagh PM. Bilateral superselective arterial microcoil embolisation in delayed post-traumatic high flow priapism. *Postgrad Med J* 2001;77:193-194.
43. Sandock DS, Seftel AD, Herbener TE, Goldstein I, Greenfield AJ. Perineal abscess after embolization for high-flow priapism. *Urology* 1996;48:308-311.