

## CASE REPORT

# Microcoil embolization of an arteriovenous fistula from the arteria bulbi penis to the corpus spongiosum penis in the treatment of erectile dysfunction: normal function regained immediately after intervention

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**ABSTRACT.** A 39-year-old man presented with erectile dysfunction that had persisted since its sudden onset 5 years ago. He exhibited none of the classic risk factors, and all attempts at medication had been unsuccessful. An ultrasound examination revealed the presence of an arteriovenous shunt in the corpus spongiosum penis. Selective digital subtraction angiography of the left internal pudendal artery showed an arteriovenous fistula from the arteria bulbi penis to the corpus spongiosum penis. The outflow of venous blood took place via the penile veins into the periprostatic vein plexus. Superselective catheterization of the arteria bulbi penis was performed with a 3 French coaxial catheter (Topaz Micro Coils; Micro Therapeutics, Inc, Irvine, CA) and it was occluded by inserting several platinum coils. 1 week after the procedure, the patient reported normal erectile function, which was subsequently maintained.

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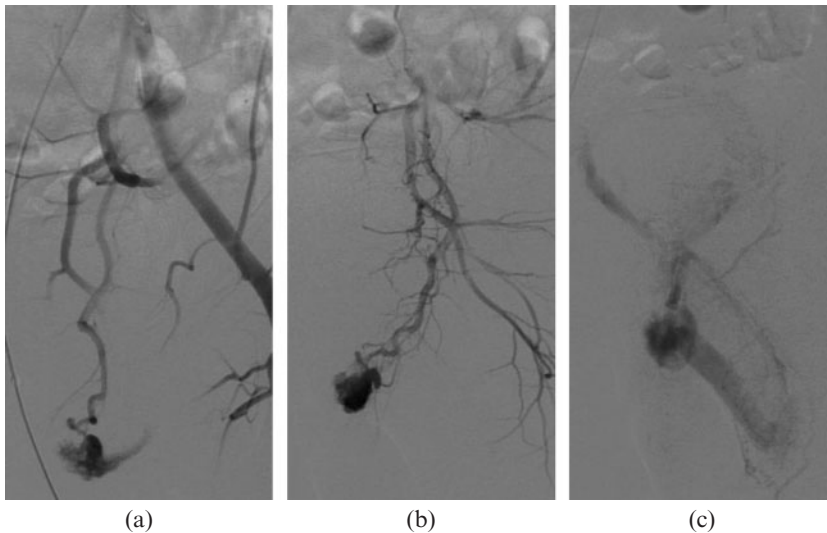
Erectile dysfunction is a common disorder; the reported prevalence is variable [1], occurring in various populations between 20% and 52% [1–3]. In the United States alone, between 10 million and 20 million men are affected [4]. Risk factors include the classic cardiovascular factors, such as hyperlipidemia, arterial hypertension, diabetes, smoking and obesity [2, 5, 6]. The resulting vascular defects can lead to obstructive arterial disease or to veno-occlusive insufficiency which, for approximately 25 years, have been treated using various techniques of arterial revascularization or therapeutic venous obstruction [7, 8]. Pre-procedure preparation includes a clinical examination, laboratory tests, tumescence tests and, finally, ultrasound. Several angiographic measures such as pharmacarteriography, pharmacocavernosometry or pharmacocavernosography may be necessary. In some selected cases of arteriogenic impotence, transluminal angioplasty can produce good results [9]; the distal internal pudendal artery and the proximal cavernous artery have a predilection for arteriosclerotic changes [10, 11]. Veno-occlusive insufficiency can also be treated by transluminal angioplasty [10, 11]; however, endoluminal therapy has not been used previously to treat arteriovenogenic impotence.

## Case report

A 39-year-old man presented with erectile dysfunction that had persisted for 5 years. Classic risk factors for erectile dysfunction were ruled out, and the patient could not recall any traumatic event. He had undergone numerous unsuccessful attempts to discover the cause of the disorder, including ultrasound and laboratory examinations. In addition, several attempts at medication brought no results. In 2002, lymph nodes were removed from the groin because of slight pain on the right side, but the histology was unremarkable. Finally, a diagnosis of “chronic prostatitis” was made, but its treatment did not give rise to any improvement.

The penis was congested and semi-rigid, suggesting increased penile blood flow, with arterial inflow equaling venous outflow. Sexual arousal did not further increase rigidity, thus leading to impotence. Conversely, the tumescence was not sufficient to meet the criteria for painful priapism. The initial Doppler examination 2 years previously had already indicated increased flow rates, but a fistula could not be confirmed at that time. Urine culture was sterile and urine status unremarkable. The laboratory parameters were also unremarkable, except for slightly elevated glutamate pyruvate transaminase (GPT) and  $\gamma$ -glutamyl transpeptidase ( $\gamma$ -GT). In particular, C-reactive protein was within the normal range. Levels of prolactin, follicle-stimulating hormone, luteinizing hormone, free testosterone and total testosterone were normal, thus eliminating an endocrine

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**Figure 1.** (a) Crossover angiography to the left common iliac artery showing an arteriovenous fistula fed by the arteria bulbi penis. (b) Arteriovenous fistula in the median third of the corpus spongiosum penis, fed by the arteria bulbi penis, after selective catheterization of the left internal pudendal artery. (c) Opacification of the corpus spongiosum penis, and venous outflow into the periprostatic vein plexus.

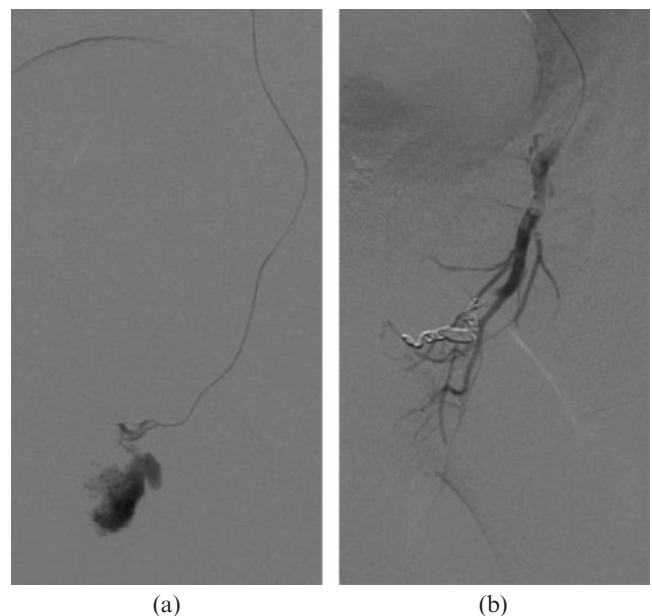
cause. Neurography showed normal bulbocavernosus reflex latency times, and the needle electromyography gave no indication of a lesion of the pudendal nerve. Proctological examination was unremarkable. Ultrasound of the genitals showed a prostate volume of 17 ml. There was pronounced prostatic calcification. There was no sign of infection and the scrotal contents were unremarkable.

Duplex sonography revealed an arteriovenous shunt in the corpus spongiosum penis with high diastolic arterial flow, as well as pulsatile flow patterns in the corpus spongiosum penis. This prompted the decision to perform an angiography. The bilateral common iliac artery and the bilateral internal and external iliac arteries were visualized in anteroposterior, and in left and right oblique, projections by means of right transfemoral access. The left internal pudendal artery had a larger calibre than the right; it fed an arteriovenous fistula via the arteria bulbi penis to the corpus spongiosum penis, which is visible in an overview image (Figure 1a). We therefore proceeded with the superselective insertion into the internal pudendal artery. After contrasting the internal pudendal artery and the arteria bulbi penis, the fistula was immediately visualized in the median third of the corpus spongiosum penis (Figure 1b). The venous outflow was into Santorini's plexus (Figure 1c). The venous vessels exhibited pronounced congestion with delayed venous outflow. An additional supply to the fistula from the right arteria bulbi penis was ruled out by non-selective depiction of the right internal pudendal artery, as were other possible parasitic inflows. The left arteria bulbi penis was then catheterized by superselective techniques using a 3F coaxial catheter (Prowler Plus; Cordis Neurovascular, Inc, Miami Lakes, FL) (Figure 2a). The arteria bulbi penis was occluded at the fistula (Figure 2b) with Topaz Micro Coils (Micro Therapeutics, Inc, Irvine, CA), taking care not to damage the proximal third. Follow-up angiography revealed that the fistula had been completely excluded from circulation. 1 week after the procedure, the patient reported normal erectile function, which was subsequently maintained for a follow-up of 6 months.

## Discussion

A wide variety of surgical options for revascularization are available in cases of arteriogenic impotence. All of these methods have a poor outcome, except in young patients with traumatic arterial lesions due to pelvic injuries [12]. For these patients, good results can be obtained by anastomosis of the dorsal penis artery with an inferior epigastric artery [12]. In general, venous leakage surgery has only minor significance in the treatment of venogenic impotence [12, 13]. In contrast to arteriogenic impotence, which is common, and to venogenic impotence, there have only been a few cases of arteriovenogenic impotence discussed in the literature thus far [14].

In the case of an arteriospongious fistula presented here, the procedure led to complete restoration of



**Figure 2.** (a) Selective catheterization of the left arteria bulbi penis. (b) Complete elimination of the fistula by insertion of platinum coils.

function. It is thus the first case of arteriovenogenic impotence of non-traumatic, or not verifiably traumatic, origin, and also the first case of an arteriospongioid fistula treated by endovascular methods. However, as most cases of arteriovenogenic impotence are of traumatic origin, this was also assumed in this case. Whereas most arteriocavernous fistulae seem to be supplied bilaterally, the arteriospongioid fistula presented here was supplied from the left side only. The source of the patient's impotence had been the high-flow arteriospongioid fistula, with the outflow from the corpus spongiosum equalling the inflow. This did not result in high-flow priapism as in most cases of arteriocavernous fistulae. As our patient was already suffering from impotence before the procedure, microcoils were chosen for occlusion of the relatively large high-flow arteriospongioid fistula, with a good outcome. However, there could also be valid arguments made for the use of autologous clot or gel foam.

In general, arterial revascularization, regardless of whether it is produced endoluminally [9] or surgically [15], produces poor results with frequent arterial occlusion. As a result, in 1996, the Clinical Guidelines Panel of the American Urological Association decided that revascularization should not be carried out on a routine basis, especially for patients with arteriosclerosis [16]. The reasoning could have been that, although arteriosclerosis is a generalized disease, the penis is only a "barometer of the body's endothelial function" [5], and satisfactory results cannot be anticipated from localized treatment alone. This is all the more true as the underlying causes of arteriosclerosis, such as diabetes, not only impair vascular function but also can lead to neuropathic erectile dysfunction [17].

New medications are now available [18] for treating the sharply rising incidence and prevalence of arteriosclerotic erectile dysfunction [19]. Complex aetiologies call for a multidisciplinary approach, even including psychotherapy [20, 21]. The generally poor results attained by revascularization, coupled with the usually good prospects from medication, should not, however, lead us to refrain from performing an angiography because of the lack of clinical consequences. This is particularly true, as in the case presented here, when the lack of risk factors coincides with the failure to respond to medication.

Firstly, a complete evaluation of every case should be made to eliminate causes that are treatable by other means, so that a curable endocrine factor or (as in the case presented here) a treatable cause of any kind is not overlooked. Only after that should a staged scheme be implemented, initially consisting of medication and psychotherapy, followed by more invasive methods such as penile self-injection or penile prosthetics, depending upon which treatment appears most appropriate for the patient.

## References

1. Papatsoris AG, Triantafyllidis A, Gekas A. Prevalence of erectile dysfunction in the European Union. *Asian J Androl* 2003;5:255.
2. Bai Q, Xu QQ, Jiang H, Zhang WL, Wang XH, Zhu JC. Prevalence and risk factors of erectile dysfunction in three cities of China: a community-based study. *Asian J Androl* 2004;6:343-8.
3. Wespes E, Wildschutz T, Roumeguere T, Schulman CC. The place of surgery for vascular impotence in the third millennium. *J Urol* 2003;170:1284-6.
4. Russell ST, Khandheria BK, Nehra A. Erectile dysfunction and cardiovascular disease. *Mayo Clin Proc* 2004;79:782-94.
5. Kendirci M, Nowfar S, Hellstrom WJ. The impact of vascular risk factors on erectile function. *Drugs Today (Barc)* 2005;41:65-74.
6. Montorsi P, Ravagnani PM, Galli S, Rotatori F, Briganti A, Salonia A, et al. Common grounds for erectile dysfunction and coronary artery disease. *Curr Opin Urol* 2004;14:361-5.
7. Wespes E, Schulman CC. Venous leakage: surgical treatment of a curable cause of impotence. *J Urol* 1985;133:796-8.
8. Rao DS, Donatucci CF. Vasculogenic impotence. Arterial and venous surgery. *Urol Clin North Am* 2001;28:309-19.
9. Dehmer GJ. Another piece of the fish oil puzzle. *Circulation* 1990;82:639-42.
10. Bookstein JJ. Penile vascular catheterization in the diagnosis and treatment of impotence. *Cardiovasc Intervent Radiol* 1988;11:183-4.
11. Rosen MP, Greenfield AJ, Walker TG, Grant P, Guben JK, Dubrow J, et al. Arteriogenic impotence: findings in 195 impotent men examined with selective internal pudendal angiography. *Radiology* 1990;174:1043-8.
12. Wespes E, Wildschutz T, Roumeguere T, Schulman CC. The place of surgery for vascular impotence in the third millennium. *J Urol* 2003;170:1284-6.
13. Siroky MB, Azadzi KM. Vasculogenic erectile dysfunction: newer therapeutic strategies. *J Urol* 2003;170:S24-9.
14. Lurie AL, Bookstein JJ, Kessler WO. Posttraumatic impotence: angiographic evaluation. *Radiology* 1988;166:115-9.
15. Goldstein I. Overview of types and results of vascular surgical procedures for impotence. *Cardiovasc Intervent Radiol* 1988;11:240-4.
16. Rao DS, Donatucci CF. Vasculogenic impotence. Arterial and venous surgery. *Urol Clin North Am* 2001;28:309-19.
17. Klein R, Klein BE, Moss SE. Ten-year incidence of self-reported erectile dysfunction in people with long-term type 1 diabetes. *J Diabetes Complications* 2005;19:35-41.
18. Anderson PC, Gommersall L, Hayne D, Arya M, Patel HR. New phosphodiesterase inhibitors in the treatment of erectile dysfunction. *Expert Opin Pharmacother* 2004;5:2241-9.
19. Milbank AJ, Goldfarb DA. Urological manifestations of vascular disease. *Urol Clin North Am* 2003;30:13-26.
20. Stief CG. Is there a common pathophysiology of erectile dysfunction and how does this relate to new pharmacotherapies? *Int J Impot Res*. 2002;14(Suppl 1):S11-6.
21. Levine SB. Erectile dysfunction: why drug therapy isn't always enough. *Cleve Clin J Med* 2003;70:241-6.