Case series - Selective arterial embolization for non-ischemic priapism

JennaKay Colquitt[1], Sahith Mukku[1], Connor Mayes[1], Sailendra Naidu[2], Nahid Punjani[3]
[1]Mayo Clinic Alix School of Medicine, Scottsdale, AZ, United States; [2]Mayo Clinic Department of Interventional Radiology, Scottsdale, AZ, United States; [3]Mayo Clinic Department of Urology, Phoenix, AZ, United States

Cite as: Colquitt J, Mukku S, Mayes C, et al. Case series - Selective arterial embolization for non-ischemic priapism. *Can Urol Assoc J* 2026 July 20; Epub ahead of print. http://dx.doi.org/10.5489/cuaj.9688

Published online July 20, 2026

Corresponding author: Dr. Nahid Punjani, Mayo Clinic Department of Urology, Phoenix, AZ, United States; Punjani.Nahid@mayo.edu

***

INTRODUCTION

Non-ischemic (high flow) priapism, most commonly results from perineal or penile trauma leading to arteriocavernosal fistula formation, producing a painless, partially rigid erection.[1,2] Initial management is conservative given the potential for spontaneous resolution occurring in around 62% of patients.[3] However, supraphysiological oxygen tension within the corpora can promote free radical formation and subsequent fibrosis, encouraging prompt treatment.[4] Selective arterial embolization (SAE) with angiography has become the preferred treatment for refractory non-ischemic priapism. Embolization may be performed using permanent agents (microcoils, polyvinyl alcohol particles, spherical embolics, and acrylic glue) or temporary agents (autologous blood clots or gelfoam).[5] Temporary agents achieve resolution in approximately 74% of cases but are associated with higher recurrence, whereas permanent agents achieve resolution in approximately 78% of cases but have historically been associated with higher rates of ED.[6]

Due to the rarity of non-ischemic priapism, there is limited data on optimal embolic material across differing lesion phenotypes and clinical scenarios. We present a case-series of five adult patients with non-ischemic priapism refractory to conservative management who underwent angiography with intent to treat.

METHODS

We conducted a retrospective case series of five males with non-ischemic priapism treated from 2007 to 2024 at Mayo Clinic Arizona. Patients were evaluated by Urology and Interventional Radiology. This study was IRB-approved.

Eligibility criteria

Inclusion criteria were: males ≥18 years with (1) clinical suspicion of non-ischemic priapism; (2) confirmatory testing by duplex Doppler and/or angiography; and (3) diagnostic angiography with intent to embolize after failed conservative management.

Data collection

Extracted data included demographics, precipitating events, symptom duration, imaging findings, lesion phenotype, vascular supply, embolic approach, complications, and outcomes. Technical success was defined as angiographic cessation of fistulous filling with preservation of non-target penile arterial flow. Primary outcomes included change in resting partial tumescence, patient-reported erectile function, and need for re-intervention. Analyses were descriptive.

RESULTS

Cohort and presentation

Five adults (22 to 51 years) presented with non-ischemic priapism (Table 1). Mechanisms of injury included bicycle trauma, rodeo trauma, intercourse, syncope, and one non-traumatic presentation following exertional heat stress. Mean time from inciting event to presentation was 5.2 months. Four patients presented with painless symptoms; one reported tender tumescence (Case 4). Erectile function varied, with four patients reporting partial tumescence.

Lesion phenotype

Four patients demonstrated identifiable vascular lesions (Table 2). Two patients had pseudoaneurysms (Case 1, 13 mm; Case 3, 11 mm), while two cases demonstrated diffuse arteriocavernosal blushes (Case 2, Case 4). Duplex ultrasonography in Case 5 confirmed non-ischemic priapism despite normal angiography.

Vascular anatomy

Two patients demonstrated an accessory pudendal artery arising from the obturator that supplied the lesion (Case 2, Case 3). Case 1 had a pseudoaneurysm fed by a cavernosal branch of the bulbourethral artery with reverse filling from the dorsal penile artery. Case 4 showed right-sided inflow from the internal pudendal artery with trauma-related segmental stenoses. Case 5 had ultrasound confirmed non-ischemic priapism with normal angiography; pelvic MRI demonstrated corporal fibrosis consistent with prolonged high-flow physiology.

Interventions

Four patients underwent SAE. Initially, two patients underwent temporary gelfoam embolization (Case 2, Case 4) and two underwent permanent coil embolization (Case 1, Case 3). Two cases required reintervention (Cases 1, Case 4). Case 1 underwent coil embolization, percutaneous thrombin injection, repeat coil embolization and thrombin injection, and eventual FloSeal injection. (Figure 1). Case 4 required permanent coil embolization after failure of temporary gelfoam.

Outcomes

Among embolized patients (n=4), resting tumescence decreased in all patients and resolved in 3 patients; one patient (Case 1) had persistent low-grade fullness. Erectile function improved in 3 patients: Case 2 achieved near-baseline erections, Case 3 reported 75–85% rigidity, and Case 1 attained 75% rigidity with medication assistance; Case 4 had persistent erectile dysfunction despite PDE-5 inhibitor therapy. Recurrence was observed in one embolized patient requiring reintervention (Case 1; Figure 1). No ischemic tissue loss, cutaneous changes, infection, or complications were observed.

DISCUSSION

Non-ischemic priapism is often managed conservatively, but up to one-third of patients develop ED, underscoring the value of timely, definitive therapy.[5] SAE success rates approach 89%, supporting its role as an effective first line intervention after observation.[7] In our cohort SAE achieved dependable hemodynamic control with acceptable functional outcomes.
Discrete pseudoaneurysms responded best to superselective coil occlusion, while diffuse arteriocavernosal blush was effectively treated with temporary gelfoam. Failure of temporary embolization warranted escalation to coils without ischemic sequelae, favoring an absorbable-first approach to minimize ED risk. Conversely, when permanent coil embolization resulted in recurrence, sac-directed adjuncts (thrombin matrix) yielded transient benefits, but their use has met success in described reports.[8] We hypothesize that collateral reperfusion and endothelization of the pseudoaneurysm may cause failure of percutaneous interventions.

Persistent symptoms of priapism may signal variant inflow that is unlikely to involute spontaneously.[9] Two patients demonstrated accessory pudendal supply from the obturator artery, and one case showed reverse filling via the dorsal penile artery. Accessory pudendal arteries are reported in 28.5-44.7% of men, and in one study nearly half originated from the obturator artery.[10] Targeted-vessel embolization can be challenged by aberrant pathways**,** supporting a systematic angiographic survey and a low threshold for stepwise escalation.

Our study illustrates the value of separating hemodynamic from functional endpoints: reduction of partial tumescence reflects control of abnormal arterial inflow, whereas erectile function captures the patient-centered outcome. These domains can move independently as Case 4 achieved hemodynamic resolution yet had persistent ED, whereas Case 1 achieved medication-assisted intercourse despite low-grade fullness. For patients with persistent ED, PDE-5 inhibitor therapy is commonly employed. In our cohort, 3 patients trialed PDE-5 inhibitors with only one patient achieving improvement. By contrast, Zacharakis et al. reported higher medication responsiveness with ED resolving in 5 of 6 patients.[11] This supports counseling patients that erectile recovery is often gradual and warrants structured follow-up and early pharmacologic support.

In our series, time to intervention averaged 5.2 months, indicating longer untreated intervals than reported elsewhere (1.25 months, Zacharis et al; 20 days, Kim et al.).[11,12] Although historically considered non-urgent, as oxygenated blood continues to perfuse the corpus

cavernosum, prolonged exposure has been linked to corporal fibrosis in 30-40% of patients.[4,13] Prolonged supraphysiologic oxygen tension may paradoxically induce oxidative stress. Experimental models of sustained hyperoxia at oxygen concentrations of 40 percent or higher have demonstrated increased reactive oxygen species production, resulting in smooth muscle injury, apoptosis, and necrosis, mechanisms that promote corporal fibrosis in non-ischemic priapism.[11,14] These observations support structured observation with defined reassessment and patient education on warning signs to minimize avoidable delays.

This series provides case level detail linkage between angiographic anatomy, lesion phenotype, embolic treatment modality, and outcomes in refractory non-ischemic priapism. Limitations include the single center, retrospective design; heterogenous population and follow-up; and non-standardized reported outcomes. Future work should include prospective multicenter comparisons of selective embolization methods with longer follow-up to better quantify outcomes.

CONCLUSIONS

In this case series, successful SAE for refractory non-ischemic priapism appeared to be influenced by arterial inflow anatomy and embolic material selection. Lesion phenotype and vascular variants were associated with differing technical approaches, with coils appearing effective for discrete pseudoaneurysms and temporary gelatin for diffuse arteriocavernosal inflow. These findings suggest that an anatomy-informed, staged approach may help optimize functional outcomes, although larger studies are needed for confirmation. Accordingly, SAE should be reserved for carefully selected patients after failure of conservative management.

REFERENCES

1. Dang H, Perazzini C, Caudron S, et al. High-flow priapism: Highly selective embolization of a traumatic arterio-cavernosal fistula. *J Med Vasc* 2022;47:27-32. https://doi.org/10.1016/j.jdmv.2021.11.001
2. Hakim LS, Kulaksizoglu H, Mulligan R, et al. Evolving concepts in the diagnosis and treatment of arterial high flow priapism. *J Urol* 1996;155:541-8. https://doi.org/10.1016/S0022-5347(01)66444-9
3. Ridgley J, Raison N, Sheikh MI, et al. Ischaemic priapism: A clinical review. *Turk J Urol* 2017;43:1-8. https://doi.org/10.5152/tud.2017.59458
4. Dursun M, Kalkanli A, Tantekin SA, et al. The role of the urologist in managing high flow priapism. *Int J Impot Res* 2025. https://doi.org/10.1038/s41443-025-01017-6
5. Ingram AR, Stillings SA, Jenkins LC. An update on non-ischemic priapism. *Sex Med Rev* 2020;8:140-9. https://doi.org/10.1016/j.sxmr.2019.03.004
6. Montague DK, Jarow J, Broderick GA, et al. American Urological Association guideline on the management of priapism. *J Urol* 2003;170:1318-24. https://doi.org/10.1097/01.ju.0000087608.07371.ca
7. Broderick GA, Kadioglu A, Bivalacqua TJ, et al. Priapism: Pathogenesis, epidemiology, and management. *J Sex Med* 2010;7:476-500. https://doi.org/10.1111/j.1743-6109.2009.01625.x
8. Abu-Suboh Abadia A, Mosquera Seoane L, Hernández Mancera J, et al. Ultrasound-guided percutaneous thrombin/gelatin treatment of high-flow posttraumatic priapism associated with urethrorrhagia in an adolescent patient. *J Vasc Interv Radiol* 2020;31:1902-4. https://doi.org/10.1016/j.jvir.2020.06.003
9. Bassett J, Rajfer J. Diagnostic and therapeutic options for the management of ischemic and nonischemic priapism. *Rev Urol* 2010;12:56-63.
10. Henry BM, Pekala PA, Vikse J, et al. Variations in the arterial blood supply to the penis and the accessory pudendal artery: A meta-analysis and review of implications in radical prostatectomy. *J Urol* 2017;198:345-53. https://doi.org/10.1016/j.juro.2017.01.080
11. Zacharakis E, Ralph DJ, Walkden M, Muneer A. Distal corpus cavernosum fibrosis and erectile dysfunction secondary to non-ischaemic priapism. *Arch Ital Urol Androl* 2015;87:258-9. https://doi.org/10.4081/aiua.2015.3.258
12. Kim KR, Shin JH, Song HY, et al. Treatment of high-flow priapism with superselective transcatheter embolization in 27 patients: A multicenter study. *J Vasc Interv Radiol* 2007;18:1222-6. https://doi.org/10.1016/j.jvir.2007.06.030
13. Kalejaiye O, Fontaine C, Langston J, et al. High flow priapism is associated with high risk of erectile dysfunction and corporal fibrosis. *J Sex Med* 2017;14:S109. https://doi.org/10.1016/j.jsxm.2016.11.253
14. Kazzaz JA, Xu J, Palaia TA, et al. Cellular oxygen toxicity: Oxidant injury without apoptosis. *J Biol Chem* 1996;271:15182-6. https://doi.org/10.1074/jbc.271.25.15182

FIGURES AND TABLES

Figure 1. Case 1. (a) Initial penile angiogram demonstrates a pseudoaneurysm arising from a cavernosal artery (black arrow). (b) This was treated with coil embolization (white arrow) with truncation of the cavernous artery; however, persistent filling of the pseudoaneurysm was noted via collaterals from the dorsal penile artery*. (c) To preserve flow to the dorsal penile artery, direct percutaneous thrombin injection into the pseudoaneurysm was undertaken. Ultrasound image of the penis demonstrates percutaneous thrombin injection directly into the pseudoaneurysm (grey arrow). Additional coil embolization was performed in addition to the thrombin injection. Angiography and ultrasound imaging showed resolution of the pseudoaneurysm at time of procedure; however, one-month followup Doppler ultrasound showed recurrence of the pseudoaneurysm (not shown).

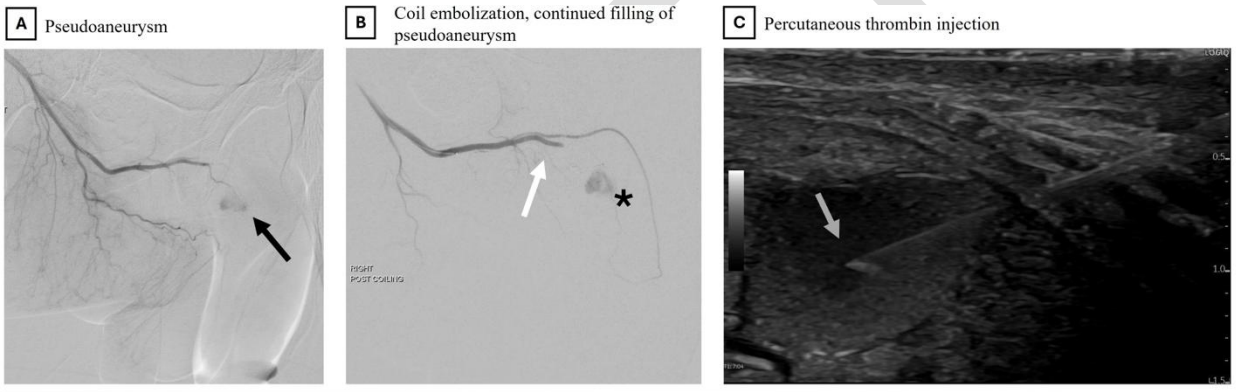

Figure 2. Case 2. (a) Penile angiogram demonstrates a diffuse blush within the cavernosa with contribution from the bulbourethral artery and cavernosal arteries*. Incidentally, this patient has variant anatomy with an accessory pudendal artery arising from the obturator artery. (b) This was treated with gelfoam pledget embolization with truncation of the accessory pudendal artery (white arrow).

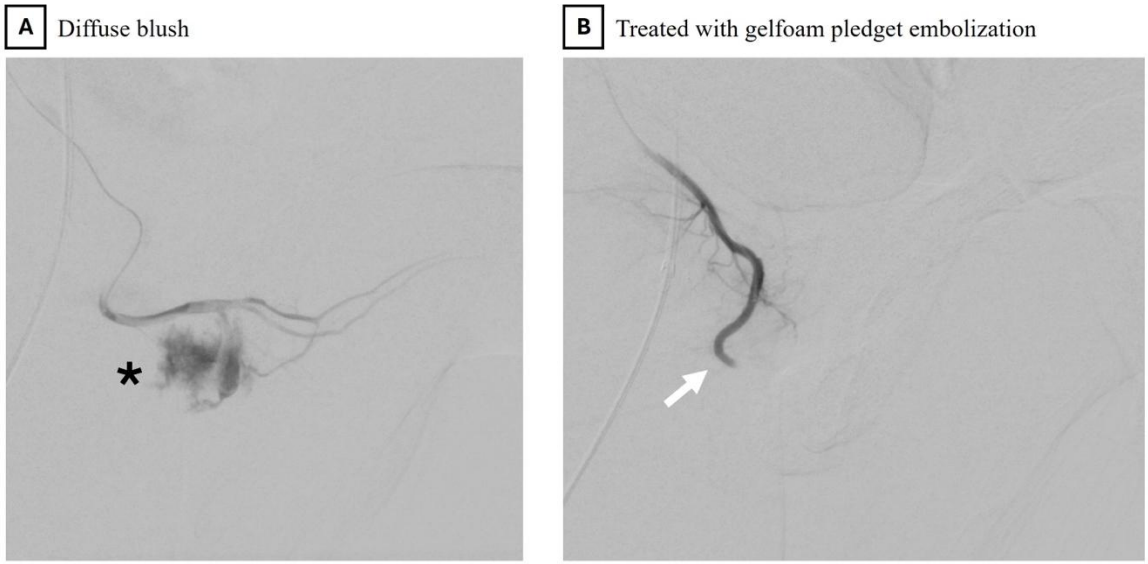

Table 1. Baseline characteristics and presentation

Case	Age (yrs)	Mechanism of injury	Time to presentation	Pain	Presentation	Conservative measures
1	44	Syncopal episode while urinating	3 months	No	Persistent partial tumescence; ejaculation preserved; 15−20% tumescence at rest; 25−30% with stimulation	Observation for 7 months; later PDE-5 trial (tadalafil)
2	25	Bicycle saddle/perineal injury	6 months	No	Constant partial tumescence (30% at rest); preserved sexual function	Ice and finasteride (partial relief)
3	22	Multiple groin impacts (saddle strike; horse kick)	7 months	No	Partial tumescence; preserved sexual function	Tadalafil trial ineffective
4	51	Intercourse related event on	2 months	Yes	Partial tumescence; preserved sexual function	Observation for 2 months

		background of recent groin injury				
5	31	No discrete trauma; exertional heat stress while on supratherapeutic testosterone	8 months	No	Unable to have sexual intercourse; absent nocturnal erections; no baseline tumescence	Oral PDE-5 (partial response)

Table 2. Lesion characteristics and intervention outcomes

Case	Lesion type	Vascular anatomy	Treatment	Recurrence	Tumescence	Erectile function
1	Right arteriocavernosal fistula with 13 mm pseudoaneurysm	Cavernosal branch off bulbourethral artery with collateral/reverse filling from dorsal penile artery	SAE, US-thrombin (200 U), repeat SAE, thrombin (100 U), FloSeal	Yes (pseudoaneurysm persisted)	Baseline tumescence decreased, not resolved	Improved to 75% rigidity with PDE-5 (med-assisted)
2	Diffuse arteriocavernosal blush (no sac)	Accessory pudendal artery arising from right obturator; native internal pudendal did not opacify lesion	Superselective gelfoam pledgets	No	Resolved	Erections near baseline (~90% at 1 mo)
3	Right 11 mm pseudoaneurysm (contralateral blush notes, not treated)	Accessory pudendal from right obturator; feeder via bulbourethral branch with preserved dorsal penile artery	Superselective coils	No	Resolved	Improved; 75–85% rigidity
4	Right penile arteriovenous diffuse blush (no sac)	Supplied via right internal pudendal; trauma-related asymmetry	Gelfoam (failed) → distal penile coil	No	Resolved	Persistent ED (PDE-5 trial)
5	No demonstratable lesion on angiography,	Bilateral selective internal pudendal injections negative for fistula	No embolization; medical mgmt	N/A	Intermittent spontaneous partial	Persistent ED; partial response with meds, no morning

	MRI consistent with corporal fibrosis				erections reported	erections (non-ischemic priapism confirmed; secondary psychogenic contribution considered)