CLINICAL STUDY

Embolization of the Periprostatic Venous Plexus for Erectile Dysfunction Resulting from Venous Leakage

Alberto Rebonato, PhD, MD, Alessio Auci, MD, Franco Sanguinetti, MD, Daniele Maiettini, MD, Michele Rossi, MD, Luca Brunese, MD, Gianpaolo Carrafiello, MD, and Tito Torri, MD

ABSTRACT

Purpose: To evaluate retrospectively the safety and efficacy of anterograde embolization of the periprostatic venous plexus (AEPV) via percutaneous access of the deep dorsal vein of the penis for erectile dysfunction (ED) resulting from veno-occlusive dysfunction (VOD).

Materials and Methods: From September 2009 through December 2012, 18 patients with moderate to severe ED secondary to insufficiency of physiologic venous occlusion as diagnosed by color Doppler evaluation of the penis after direct pharmacologic stimulation were treated. Preliminary diagnoses were also confirmed with dynamic cavernosography. Selective AEPV was achieved using a combination of *N*-butyl cyanoacrylate and endovascular coils. Follow-up consisted of collecting International Index of Erectile Function questionnaire (IIEF-6) scores and repeated color Doppler evaluation.

Results: Immediate technical success was achieved in 16 of 18 patients (88.8%). Follow-up data were obtained at a mean of 13.3 months ± 7.5. In 12 of the patients with technical success, the mean IIEF-6 score improved from 10.5 ± 5.2 to 20.6 ± 8.4 after the procedure ($P = .0069$). At 3-month short-term follow-up, clinical success defined by an end-diastolic velocity of < 5 cm/s on color Doppler was noted in 81% (13 of 16 patients). Of these 13 patients, 7 patients had continued erectile function at the end of follow-up, and the other 6 patients reported progressive diminishment in the benefit over time. No major complications and two minor complications were encountered.

Conclusions: AEPV for ED secondary to VOD is a safe alternative to surgical treatment that demonstrates promising short-term and midterm efficacy.

ABBREVIATIONS

AEPV = anterograde embolization of the periprostatic venous plexus, DDV = deep dorsal vein, ED = erectile dysfunction, EDV = end-diastolic velocity, IIEF-6 = International Index of Erectile Function questionnaire, NBCA = *N*-butyl cyanoacrylate, PDE5 = phosphodiesterase 5, VOD = veno-occlusive dysfunction

Erectile dysfunction (ED) is defined as the persistent inability to achieve and maintain an erection sufficient for sexual intercourse. ED is estimated to affect 20 million men in the United States (1). Organic ED most commonly has a vascular etiology (2). Vasculogenic ED may be due to either arterial insufficiency or veno-occlusive dysfunction (VOD). VOD pertains to the inability of the erectile chambers to retain a physiologically requisite volume of blood. VOD is most common in elderly patients and has been associated with diabetes, radical prostatectomy, pelvic radiation, and androgen deprivation therapy (3,4). Insufficiency of venous outlet occlusion has been proposed as the primary cause of VOD (5). However, the precise pathophysiology leading to VOD is unclear. Patients with VOD fare poorly with oral pharmacotherapy (phosphodiesterase 5 [PDE5] inhibitors) and often require long-term intracavernosal injection therapy or other adjunctive, noninvasive measures, such as the application of elastic penile rings or vacuum devices. The aim of this retrospective

From the Department of Surgery, Radiology and Odontostomatology Sciences (A.R., D.M.), Santa Maria della Misericordia University Hospital, University of Perugia, Sant'Andrea delle Fratte, Perugia, 06132; Radiology Department (A.A., F.S., T.T.), ASL 1 Massa Carrara, Carrara; Radiology Department (M.R.), St. Andrea Hospital, Faculty of Medicine and Psychology, University of Rome "La Sapienza," Rome; Department of Medicine and Health Sciences (L.B.), Università del Molise, Contrada Tappino, Campobasso; and Department of Human Morphology (G.C.), Insubria University Hospital, Varese, Italy. Received August 15, 2013; final revision received January 12, 2014; accepted January 14, 2014. **Address correspondence to** A.R.; E-mail: rebonatoalberto@gmail.com

None of the authors have identified a conflict of interest.

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J Vasc Interv Radiol 2014; XX:▮▮▮–▮▮▮

http://dx.doi.org/10.1016/j.jvir.2014.01.015

investigation was to evaluate the safety and short-term and midterm efficacy of anterograde embolization of the periprostatic venous plexus (AEPV), using ultrasound guidance for percutaneous access and subselective catheter delivery of liquid embolic agents.

MATERIALS AND METHODS

Between September 2009 and December 2012, 18 patients with moderate to severe ED secondary to VOD underwent AEVP. After obtaining institutional review board approval for this study, we retrospectively collected the data from the clinical records of our institution. The ages of the patients ranged from 29–64, the mean age was 51, and the mean duration of ED was 4.4 years. Erectile function was assessed before and after the procedure, using the International Index of Erectile Function questionnaire (IIEF-6) score (1). All patients with ED first underwent a clinical evaluation by an endocrinologist to rule out psychogenic causes. Two patients had a history of hypertension, one patient had insulin-dependent diabetes, and one patient had ischemic coronary disease. All treated patients were no longer responsive to pharmacologic treatment with PDE5 inhibitors.

Patients with a diagnosis of true VOD were initially screened using color Doppler flow analysis after direct pharmacologic stimulation with an intracavernosal injection of 20 μg alprostadil (prostaglandin E_1) (Fig 1). The criteria for the diagnosis of VOD were a high systolic flow rate of $>$ 25 cm/s (peak systolic velocity) and a persistent end-diastolic velocity (EDV) $>$ 5 cm/s 15 minutes after the injection (rigid phase) with a resistive index $<$ 0.75 (6).

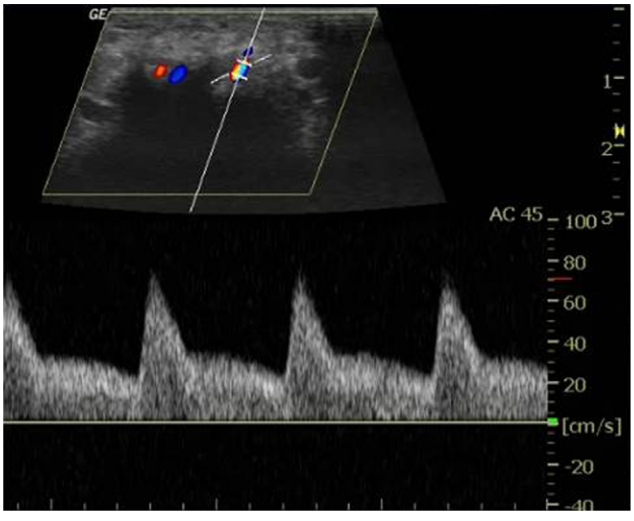

Figure 1. Penile color Doppler waveform analysis (in the rigid phase) performed 15 minutes after intracavernosal pharmacologic stimulation with prostaglandin E_1. The peak systolic velocity is $>$ 25 cm/s, which reflects the integrity of the arterial dilation, and the persistent EDV is $>$ 5 cm/s, which reflects low resistance. Both of these measurements are diagnostic criteria for VOD.

The diagnoses of VOD were secondarily confirmed by dynamic pharmacologic cavernosography that was performed by the same investigators. This procedure involved the insertion of a 21-gauge butterfly needle into the corpora and intracavernosal infusion of 20 μg alprostadil to induce complete smooth muscle relaxation. After 30 minutes, 120 mL of a 50% solution of nonionic contrast agent was power injected into the cavernosa at a flow rate of 2 mL/s. The resulting diagnostic cavernosogram demonstrated the sites of venous leakage, as defined by opacification of any of the following: deep dorsal vein (DDV), cavernosal veins, internal pudendal veins, periprostatic plexus, external pudendal veins, or hypogastric veins (Figs 2, 3). Our formal criteria for VOD by dynamic cavernosography consisted of the demonstration of venous leakage with an associated absence of penile rigidity.

The endpoint of the study was the complete occlusion of the periprostatic venous plexus and related efferents (ie, internal and external pudendal veins). AEPV was performed in the angiographic suite as a same-day procedure and had a mean procedural time of 48 minutes ± 10. After the patient was prepared and draped in the supine position, local anesthesia (lidocaine 2%) was administered, and ultrasound-guided puncture of the DDV was performed using a 20-gauge needle. After gentle advancement of a 0.018-inch Torq-Flex (Cook, Inc, Bloomington, Indiana) guide wire under

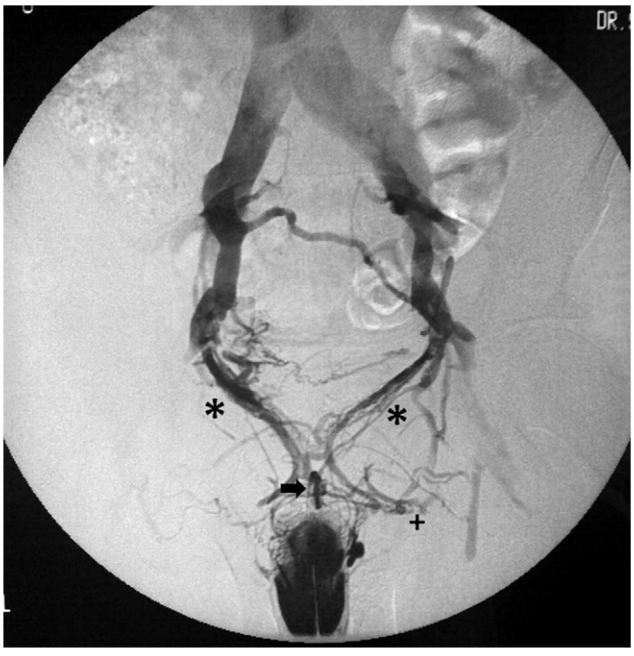

Figure 2. Pharmacologic cavernosography. After a vasodilator-induced erection, the corpora cavernosa is infused with diluted contrast medium using a single-syringe power injector. The leakage sites of the DDV and the cavernous and crural veins determine the opacification of the periprostatic venous plexus (arrow), bilateral internal pudendal veins (asterisks), and left external pudendal vein (cross). The absence of final rigidity of the penis was associated with this pattern of opacification.

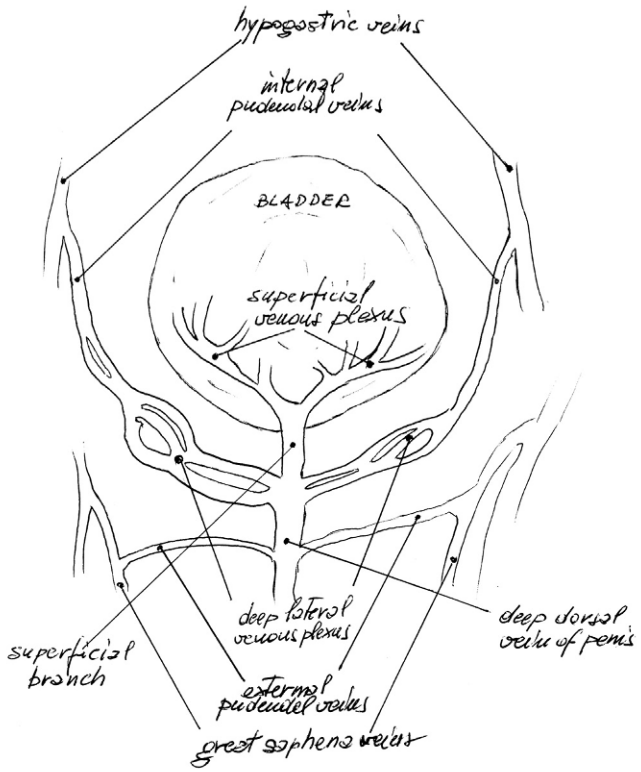

Figure 3. Illustration of the direct communication between the DDV, the external pudendal veins, the periprostatic plexus (which is composed of the superficial branch and the right and left deep lateral venous plexuses), the internal pudendal veins, and the hypogastric veins.

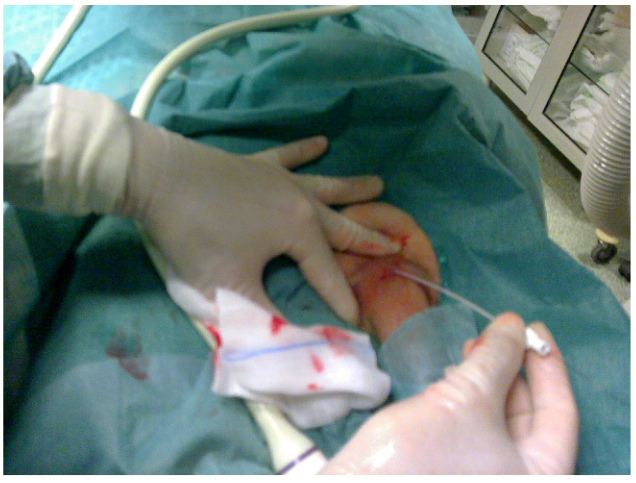

Figure 4. Ultrasound-guided puncture of DDV with a linear probe. (Available in color online at www.jvir.org.)

fluoroscopic guidance, a 9-cm-long 4.5-F peel-away introducer sheath (peripherally inserted central venous catheter Micropuncture introduction system, Cook, Inc) was inserted into the DDV (Figs 4, 5). Using a 0.035-inch Glidewire (Terumo Corporation, Leuven, Belgium), a 4-F Bern catheter (Imager II; Boston Scientific, Natick, Massachusetts) was advanced into the DDV, and venography was performed to confirm the intravenous location and to obtain complete visualization of the periprostatic

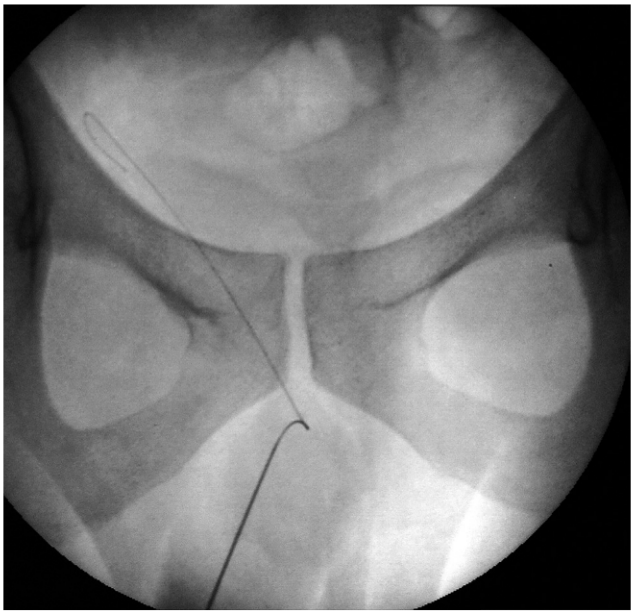

Figure 5. After the vein was punctured with a 20-gauge needle, a 0.018-inch guide wire was gently advanced under fluoroscopic guidance.

venous plexus and internal and external pudendal veins. Consecutive series of selective catheterization of these veins followed by embolization were performed to obtain complete occlusion. All of the embolization procedures were performed by injecting 1–2 mL of *N*-butyl cyanoacrylate (NBCA; Glubran 2; GEM Srl, Viareggio, Italy) and ethiodized oil (Lipiodol; Guebert, Aulnay-sous-Bois, France) prepared in 1:1 ratio through the 4-F catheter under continuous fluoroscopic control and continuous Valsalva maneuver to reduce the incidence of inadvertent distal progression of the glue (Figs 6, 7). In two cases, the pudendal internal veins had particularly large diameters, and the plexus exhibited fast outflows; before glue injection, 0.035-inch fibered coils were distally released into the pudendal internal veins. A total volume of approximately 5–7 mL of NBCA was administered in each procedure. The main working projection was the neutral one; however, the oblique and laterolateral projections were also used to control the optimal distribution of embolic agents.

Follow-up after the procedure consisted of pharmacologic color Doppler evaluation and administration of the IIEF-6 questionnaire. The mean follow-up time was 13.3 months ± 7.5. Variations between the preoperative and postoperative IIEF-6 domain scores were analyzed using a Student *t* test ($P < .05$), and the SPSS statistical package, release 14.0 (SPSS, Inc, Chicago, Illinois), was used for all statistical analyses.

RESULTS

The follow-up periods ranged from 3–26 months, and the mean follow-up time was 13.3 months ± 7.5. Technical

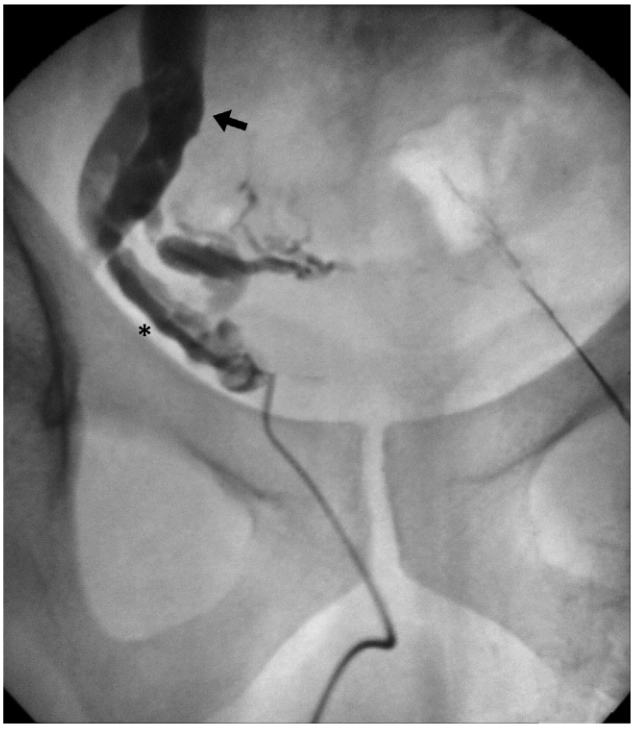

Figure 6. Selective catheterization of the right internal pudendal vein (asterisk) and opacification of the draining right hypogastric vein (arrow).

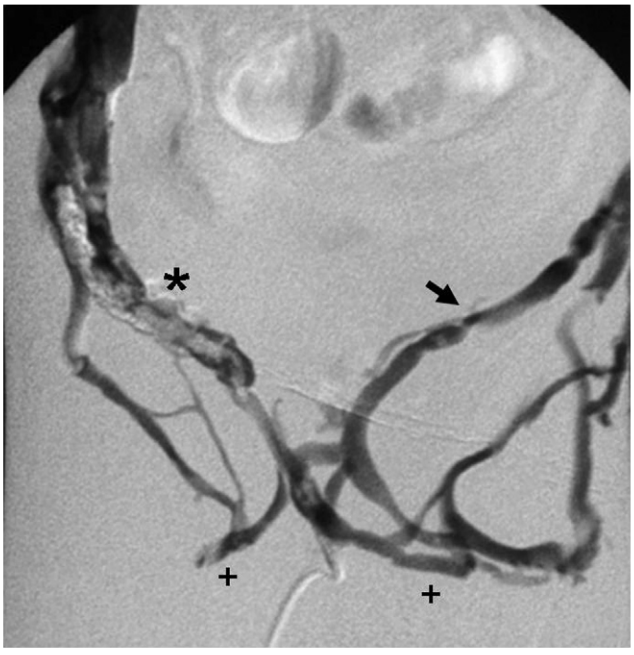

Figure 7. Contrast venography after partial embolization (Glubran 2 and Lipiodol in a 1:1 ratio) of the right internal pudendal vein (asterisk) showing the maintained patency of the right internal pudendal vein, bilateral external pudendal vein (crosses), and left internal pudendal vein (arrow).

angiographic success, defined as complete occlusion of the periprostatic venous plexus (Figs 8, 9), was obtained in 16 of 18 patients (88.8%). Access cannulation of the DDV was unsuccessful in two patients. One of these

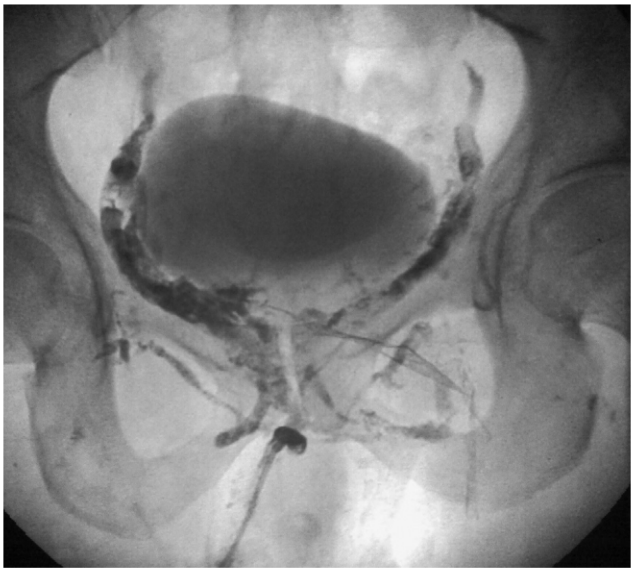

Figure 8. Final result of complete embolization of the periprostatic venous plexus (note the radiopaque embolic mixture without subtraction).

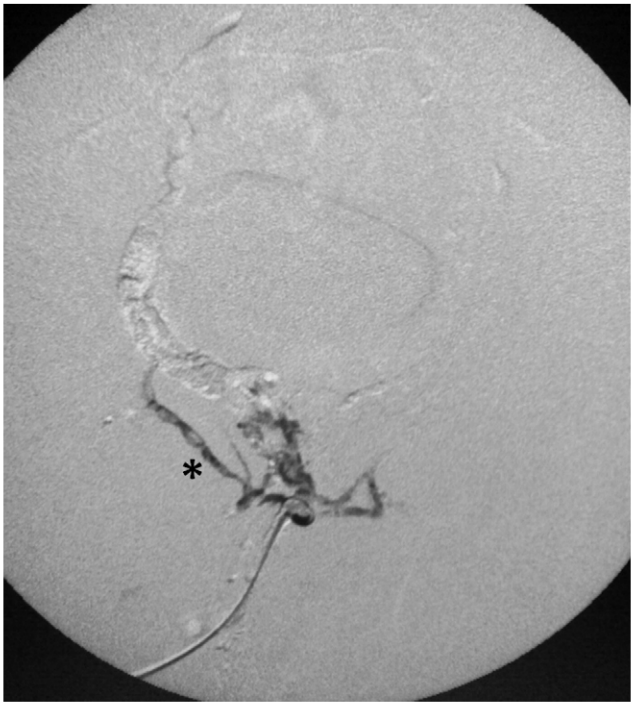

Figure 9. Contrast injections confirmed the complete occlusion of the target vessels (ie, periprostatic venous plexus, internal pudendal veins, and right external pudendal vein) and revealed a faint opacification of the partially occluded right external pudendal vein (asterisk).

patients had previously undergone an unsuccessful DDV ligation that compromised the ultrasound appearance of the DDV. The second patient exhibited hypoplasia of the DDV.

In 13 of the 16 patients (81%) who were evaluated at 3-month follow-up, initial clinical improvements in Doppler EDV (improvements were defined as flow rates

Table 1. Patient Characteristics and Treatment Success

Patient age, mean (range)	51 y (29–64 y)
Patients enrolled	18
ED duration, mean	4.4 y
Follow-up period, mean (range)	13.3 mo (3–26 mo)
Technical angiographic success	16
EDV at 3-mo follow-up:	
EDV < 5 cm/s; EDV > 5 cm/s	
All patients	13/16 (81%); 3/16 (19%)
Patients with preoperative and postoperative IIEF-6	12/16 (69%); 1/16 (6%)
Patient with only postoperative IIEF-6	1/16 (6%); 2/16 (13%)
ED improvement at end of follow-up	7 (44%)
Patients with progressive reduction in clinical benefit at end of follow-up	6 (38%)

ED = erectile dysfunction; EDV = end-diastolic velocity; IIEF-6 = International Index of Erectile Function questionnaire.

< 5 cm/s) after direct pharmacologic stimulation were observed. In the remaining three patients, no improvements were reported in erectile function (EDV > 5 cm/s) despite technical success. Of the 16 successfully treated patients, 12 (69%) were evaluated with IIEF-6 before the procedure; 11 of these 12 patients exhibited improvement in EDV. The initial mean score was 10.5 ± 5.2, and after a mean follow-up 13.3 months ± 7.5, this score was significantly improved to 20.6 ± 8.4 (Student *t* test, $P = .0069$). In regard to the duration of the benefits, 7 of 16 patients (44%) who experienced clinical benefits reported no diminishment in the improvements of erectile function over time. The remaining six patients (38%) who exhibited improvement in EDV described progressive reductions in the initial benefit over time; five of these six patients later resorted to adjunctive oral pharmacotherapy (PDE5 inhibitors). The results are summarized in **Table 1**.

Regarding the Society of Interventional Radiology (SIR) classification (7), no major complications were noted either in the period immediately after the procedure or at follow-up. An asymptomatic pulmonary glue embolism was noted in one patient. In another patient, a partial reflux of the glue was noted in the subcutaneous tissue after removal of the sheath; this patient was referred for pain 3 weeks after the procedure. Two patients developed mild curvature of the penis with erection by 5 months and 8 months of follow-up, respectively, that did not affect sexual intercourse.

DISCUSSION

Penile erection is achieved by the following series of closely related synergistic mechanisms: smooth muscle cell relaxation, increased arterial flow, and increased venous outflow restriction (8). However, the precise interplay of these physiologic mechanisms is not completely understood. Patients who do not respond adequately to intracavernosal prostaglandin administration typically have damage to the corporeal smooth musculature or the tunica albuginea, or both, which results in impairments of vascular dilation. This smooth muscle cell damage can be secondary to toxins (eg, nicotine or hyperlipidemia) or to denervation resulting from surgery, trauma, or diabetes mellitus (4). Additionally, atrophy and degeneration of the collagen matrix of the tunica albuginea may lead to decreases in structural rigidity (4,8,9). Whatever the precise mechanisms are, venous leakage is the end manifestation of a complex and multifactorial process.

The American Urological Association recommends surgical therapy for ED for erections of short duration or tumescence only with sexual stimulation, failure to obtain or maintain an erection with intracavernosal injection agents, normal cavernous arterial inflow in response to an intracavernosal injection agent (as measured by penile color Doppler or systolic occlusion pressure evaluation), and venous leakage from the corpora cavernosa on pharmacocavernosography (3). In 1902, Wooten performed the first surgical ligation of the deep dorsal vein for atonic impotence (10). Since then, other open surgical approaches have been described that include stripping or excision of the deep dorsal vein, cavernous vein, and crural vein (11,12) or penile revascularization (13). More recently, embedding of the DDV has been proposed as a promising therapy for ED with good outcome; 82% of the treated patients experienced spontaneous erections during the follow-up period (14). However, the long-term efficacy of this procedure remains disappointing; recurrence rates of 25% have been reported (14). These delayed-onset recurrences likely stem from the difficulty of ligating numerous tiny collateral veins that cannot be observed intraoperatively and from the complexity of surgically exposing the deep veins of the pelvis (ie, the periprostatic and pudendal veins). Additionally, these surgical procedures are sometimes associated with significant complications, including wound infection, hematoma, penile shortening, decreased penile sensation, and penile deviation and tethering owing to wound scar contraction (15).

Alternatively, different endovascular treatment approaches have been described (**Table 2**). These approaches include embolization with subsequent surgical ligation, bilateral coil occlusion of the crural veins, and venous ablation with sclerosing agents such as sodium morrhuate and hot contrast medium (16,17).

DDV and prostatic venous plexus embolization with NBCA has been described previously as a safe and effective procedure that results in the recovery of sexual function in 68% of patients at a median follow-up time of 25 months (3,18). However, in both reports detailing this procedure, vascular access was obtained via surgical exposure of the DDV, and the injection of embolic material was made through a flexible cannula (18-gauge to 20-gauge) directly into the DDV without any catheterization

Table 2. Previous Reports on Embolization for ED from Veno-Occlusive Dysfunction

Reference	No. Patients	DDV Approach	Selective Catheterization	Embolic Agent
Peşkircioğlu et al, 2000 (3)	32	Surgical	No	NBCA
Kutlu and Soylu, 2009 (18)	32	Surgical	No	NBCA + Lipiodol
Zhang et al, 2009 (14)	17	Surgical	No catheterization	—
Chocholatv et al, 2010 (23)	15	Surgical	No	Ethanol
Aschenbach et al, 2013 (20)	29	Transfemoral approach	Yes	NBCA

DDV = deep dorsal vein; ED = erectile dysfunction; NBCA = *N*-butyl cyanoacrylate.

of the periprostatic plexus. In our series, all DDV accesses were obtained percutaneously by direct puncture with ultrasound guidance, which allowed this procedure to be performed on an outpatient basis and avoided any surgical isolation of veins and the associated postoperative complications (eg, wound scar contraction).

Venous embolization was obtained after selective catheterization of the target vessels with a 4-F catheter and Terumo Glidewire, which allowed for better visualization of the vascular structures and a more controlled and safe release of NBCA. The anterograde approach improves catheter placement and maneuverability owing to the short distance between the vascular access and the point of glue delivery; the catheterization of target vessel with a Terumo Glidewire and a 4-F catheter was sufficiently easy that we were never required to use a microcatheter.

Other studies have reported on venous plexus embolization via a transfemoral approach (19,20). With transfemoral access, the target vessels are farther from the puncture site; catheter maneuverability is reduced, and the selective catheterization of the periprostatic venous plexus is more difficult. Aschenbach et al (20) reported that this procedure failed in two cases because of the impossibility of reaching the target vessels using any catheter system.

As a liquid embolic agent, NBCA glue has the advantage of deep penetration into very small vessels and the induction of a sclerotic, inflammatory response. The rate of initial clinical improvement observed at 3 months was high (81%, 13 of 16 patients), and the increase in IIEF-6 scores at a mean follow-up of 13.3 months ± 7.5 was significant. The IIEF-6 consists of six questions, and the maximum and minimum final scores are 30 and 6, respectively. The domain scores were classified as normal (≥ 26), mild (22–25), mild to moderate (17–21), moderate (11–16), and severe (6–10). However, 6 of the 13 patients who experienced improved erectile function reported subsequent progressive recurrence of ED, and 5 of these patients eventually resorted to oral PDE5 inhibitors to achieve erections. AEPV can be considered to be a useful tool for restoring the pharmacotherapeutic responsiveness in patients with ED secondary to VOD. The angiographic venous embolization procedures of the three patients who reported no improvement in erectile function all were successful and complete. This finding underscores the multifactorial nature of ED. Regarding the potential complications of embolization, previous reports in the literature have described symptomatic pulmonary migrations of the coils and glue (21,22). In our series, one of our first cases was complicated by asymptomatic pulmonary glue embolism. This complication led to an alteration of our technique; the patients were instructed to perform the Valsalva maneuver during the glue injection to decrease central venous return. We also used a 1:1 NBCA-to-Lipiodol ratio mixture, which may have a more technically advantageous profile than the 1:7 mixture ratio used by Kutlu et al (22). A second minor complication that we encountered was an inadvertent proximal reflux of glue that was suspected based on the patient's symptoms, but it was not confirmed (nontarget glue foci were evident on fluoroscopy after sheath removal). We suspect that the symptoms were due to nerve irritation and partial reflux into the subcutaneous tissue. The patient did well with standard nonsteroidal antiinflammatory medications. A viable embolic alternative to NBCA is Onyx (ev3, Covidien Vascular Therapies, Mansfield, Massachusetts). Owing to its characteristics of deep penetration, final solidification in 5 minutes, and excellent fluoroscopic visualization, this liquid nonadhesive embolic agent could produce a more precise and safe embolization of the periprostatic venous plexus. Onyx could be considered for use in embolization procedures via the transfemoral approach rather than NBCA to avoid the risk of catheter entrapment secondary to the retrograde diffusion of glue with the venous blood flow.

This study is limited by the small number of patients enrolled. Also, the retrospective protocol did not allow us to collect a complete set of data (eg, IIEF-6 scores) for all patients or to program a long-term follow-up on the order of several years. Nevertheless, the experience gained from this small initial series has allowed our group to develop a technique that aims to obtain angiographic success. This protocol includes several key measures. First, the penis must be lightly stretched and well secured with surgical tape on its distal aspect because the extreme mobility of the flaccid penis can make puncture and access difficult. Second, optimal ultrasound focus and depth is essential to visualize the DDV accurately and allow for safe and easy percutaneus

access. Third, we routinely administered a small intracavernosal injection of prostaglandin E_1 to distend the DDV; otherwise the DDV often collapsed and became difficult to visualize. Fourth, we have found that fluoroscopy techniques, such as mapping with multiple lateral and oblique projections, immensely aided the often difficult advancement of the micro–guide wire. Fifth, we found that a 1:1 NBCA-to-Lipiodol ratio embolic mixture delivered by the classic "glue bullet" technique to be ideal in combination with selective catheterization. Sixth, because of the bending of the DDV beneath the symphysis pubis, we preferred to use the 4.5-F peel-away introducer sheath rather than a conventional 4-F vascular sheath to obtain stable vascular access. The peel-away introducer provided a smoother transition during the introduction and stable support for the embolization procedure.

Whether surgical or endovascular, all current modalities of intervention are ultimately limited by their focus on occluding penile venous outflow. VOD likely represents a final common pathophysiology. More permanent cures for ED must ultimately focus on a causative etiology that is further upstream. Given the multifactorial nature of VOD, we should evaluate the results obtained in light of the possible placebo effect of AEVP.

In conclusion, AEVP via ultrasound-guided percutaneous access with selective catheterization is a safe, minimally invasive alternative for the treatment of ED resulting from venous leakage. Although this case series is limited in size, the intermediate-term results are encouraging.

REFERENCES

1. Rhoden EL, Telöken C, Sogari PR, Vargas Souto CA. The use of the simplified International Index of Erectile Function (IIEF-5) as a diagnostic tool to study the prevalence of erectile dysfunction. Int J Impot Res 2002; 14:245–250.
2. Mulhall JP, Damaser MS. Development of a mathematical model for the prediction of the area of venous leak. Int J Impot Res 2001; 13: 236–239.
3. Peşkircioğlu L, Tekin I, Boyvat F, Karabulut A, Ozkardeş H. Embolization of the deep dorsal vein for the treatment of erectile impotence due to veno-occlusive dysfunction. J Urol 2000; 163:472–475.
4. Berardinucci D, Morales A, Heaton JP, Fenemore J, Bloom S. Surgical treatment of penile veno-occlusive dysfunction: is it justified? Urology 1996; 47:88–92.
5. Rajfer J, Rosciszewski A, Mehringer M. Prevalence of corporeal venous leakage in impotent men. J Urol 1988; 140:69–71.
6. Burnett AL. Evaluation and management of erectile dysfunction. In: Wein AJ, Partin AW, Peters CA, Kavoussi LR, Novick AC, eds. Campbell-Walsh Urology, 10th ed. Philadelphia: Saunders; 2012. 746–747.
7. Sacks D, McClenny TE, Cardella JF, Lewis CA. Society of Interventional Radiology clinical practice guidelines. J Vasc Interv Radiol 2003; 14(9 Pt 2):S199–S202.
8. Aboseif SR, Wetterauer U, Breza J, et al. The effect of venous incompetence and arterial insufficiency on erectile function: an animal model. J Urol 1990; 144:790–793.
9. Davila HH, Rajfer J, Gonzalez-Cadavid NF. Corporal veno-occlusive dysfunction in aging rats: evaluation by cavernosometry and cavernosography. Urology 2004; 64:1261–1266.
10. Hsu GL, Chen HS, Hsieh CH, et al. Insufficient response to venous stripping surgery: is the penile vein recurrent or residual? J Androl 2006; 27: 700–706.
11. Puech-Leão P, Reis JM, Glina S, Reichelt AC. Leakage through the crural edge of corpus cavernosum: diagnosis and treatment. Eur Urol 1987; 13:163–165.
12. Lewis RW. Venous surgery for impotence. Urol Clin North Am 1988; 15:115–121.
13. Babaei AR, Safarinejad MR, Kolahi AA. Penile revascularization for erectile dysfunction: a systematic review and meta-analysis of effectiveness and complications. Urol J 2009; 6:1–7.
14. Zhang B, Chen J, Xiao H, et al. Treatment of penile deep dorsal venous leakage of erectile dysfunction by embedding the deep dorsal vein of the penis: a single center experience with 17 patients. J Sex Med 2009; 6: 1467–1473.
15. Lewis RW, Munarriz R. Vascular surgery for erectile dysfunction. In: Wein AJ, Partin AW, Peters CA, Kavoussi LR, Novick AC, eds. Campbell-Walsh Urology, 9th ed. Philadelphia: Saunders; 2007. 891–892.
16. Schild HH, Müller SC, Mildenberger P, et al. Percutaneous penile venoablation for treatment of impotence. Cardiovasc Intervent Radiol 1993; 16:280–286.
17. Schwartz AN, Lowe M, Harley JD, Berger RE. Preliminary report: penile vein occlusion therapy: selection criteria and methods used for the transcatheter treatment of impotence caused by venous-sinusoidal incompetence. J Urol 1992; 148:815–820.
18. Kutlu R, Soylu A. Deep dorsal vein embolization with N-butyl-2-cyanoacrylate and Lipiodol mixture in venogenic erectile dysfunction: early and late results. Radiol Oncol 2009; 43:17–25.
19. Schild HH, Müller SC, Mildenberger P, Strunk H, Thelen M. Retrograde venous occlusion-therapy of impotence of venous origin. Rofo 1993; 158: 238–241.
20. Aschenbach R, Steiner T, Kerl MJ, Zangos S, Basche S, Vogl TJ. Endovascular embolisation therapy in men with erectile impotence due to veno-occlusive dysfunction. Eur J Radiol 2013; 82:504–507.
21. Moriel EZ, Mehringer CM, Schwartz M, Rajfer J. Pulmonary migration of coils inserted for treatment of erectile dysfunction caused by venous leakage. J Urol 1993; 149(5 Pt 2):1316–1318.
22. Kutlu R, Soylu A, Alkan A, Turker G. Pulmonary embolism after penile deep dorsal vein embolization with n-butyl-2- cyanoacrylate and Lipiodol mixture. Eur J Radiol 2004; 49:103–106.
23. Chocholatv M, Hanek P, Jarolim L, Padr R. Embolization of the deep dorsal vein using 96% ethanol for treatment of the venogenic erectile dysfunction. Eur Urol Suppl 2010; 9(6):643.