Treatment Strategies for Varying Patterns and Presentations of Pelvic Venous Disorder

There is not a one-size-fits-all solution!

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Pelvic venous disorder (PeVD) is described in patients with noncyclic chronic pelvic pain (CPP) associated with dilated incompetent intra- and extrapelvic veins, with or without associated venous compression. It remains a diagnostic and therapeutic challenge and has a significant impact on a woman's quality of life and delay in proper diagnosis.[1] The extent of the disease is best evaluated with venography, which remains the gold standard method, but noninvasive ultrasonography and MRI are acceptable in the early diagnostic workup (Figure 1).[2]

The various treatment options range from partial suppression of ovarian function with medroxyprogesterone acetate,[3] which helps counteract the hormonal influence in venous valves, to more invasive options such as hysterectomy with oophorectomy.[4,5] Embolization techniques have been described for more than 20 years—with moderate or complete early pelvic pain relief and improved, sustained symptom relief over time in approximately 75% of women[6] and long-term overall clinical improvement after embolization in approximately 87% of women.[7] At the same time, the heterogeneity of data combined with the different anatomic patterns of PeVD hinders the robust scientific rigor of the evaluation of this entity. In addition, there is a substantial variation in interventional techniques described in the literature, ranging from treatment of venous incompetence with gonadal (ovarian) vein and internal iliac vein (IIV) embolization to treatment of venous compression with stent placement. This article provides an understanding of different patterns and presentations of PeVD, including classic gonadal vein incompetence, venous compression, pelvic escape points, and anatomic variants. We also provide our optimized approach based on a patient's clinical presentation.

PATTERNS OF PeVD: ANATOMY, CLINICAL FEATURES, AND TREATMENT

To understand PeVD, it is imperative to evaluate patients in the clinic and rule out nonvascular conditions

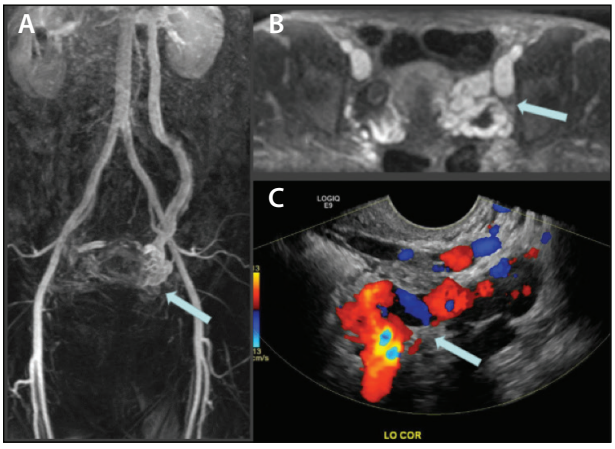

Figure 1. MRA (A, B) demonstrates dilated left OV and enlarged tortuous venous varicosities around the adnexa and in the pelvic floor (arrows). A transvaginal Doppler ultrasound (C) shows multiple dilated veins ("venous lakes") around the ovaries and uterus (arrow).

that lead to CPP (eg, endometriosis, chronic infection, pelvic floor disease). Although much of the literature describes ovarian vein (OV) incompetence as the main mechanism for PeVD, venous compression of the left renal vein and left common iliac veins (LCIVs) is being increasingly recognized as a factor leading to ovarian and internal iliac venous reflux, respectively.[8] As a result, patients need to be evaluated for reflux and obstruction with either ultrasonography and/or cross-sectional imaging. Moreover, when evaluating a patient's clinical history, it is necessary to differentiate between the reflux and the obstructive patterns of PeVD to understand which symptoms are affecting the patient the most. Typically, in our experience, a classic presentation of OV incompetence is seen in younger, multiparous women, whereas older women with associated lower extremity varicosities present more frequently with obstructive patterns of PeVD.[9] Clinical evaluation should include a detailed gynecologic history, history of deep venous thrombosis, renal symptoms (hematuria/flank pain), pelvic symptoms (postural/postcoital pain), urinary urgency/frequency, and presence of vulvar, gluteal, or lower extremity varicosities. Similarly, the physical examination should include evaluation of escape points (gluteal/labial), nonsaphenous causes of varicose veins, and the presence of ovarian point tenderness on abdominal palpation.

Descriptions of the most common variants of PeVD are presented here.

Variant 1: Classic Multiparous Women

In this variant, pelvic varices are caused by incompetence of the OVs. These arise from the ovarian venous plexus and communicate with the uterine plexus in the broad ligament (Figure 2).

It is estimated that 15% of women do not have valves in the OVs, and 35% to 40% of existent gonadal venous valves are incompetent.[10] The left OV and the right IIV are most frequently affected by reflux. Combined reflux in more than one pelvic vein and an extension of reflux through incompetent branches of the obturator and circumflex femoral veins are common.[11] Patients usually present with noncyclical postural pelvic pain characterized by heaviness, dyspareunia, and postcoital pain that starts in the second or third trimester of pregnancy and then progresses to debilitating CPP. The pain is worse with exercise and prolonged periods of standing. The treatment approach often requires embolization of unilateral or bilateral OVs depending on the patient's symptoms, with or without simultaneous or staged IIV sclerotherapy.

Variant 2: Compression Syndromes

Secondary PeVD is due to venous compression of the IIVs or left renal veins, as seen in May-Thurner syndrome and Nutcracker syndrome, respectively. Other etiologies include tumor thrombosis of the inferior vena cava (IVC), portal vein thrombosis, renal cell carcinoma with left renal vein thrombosis, and left renal arteriovenous fistula. In Nutcracker syndrome, the left renal vein is compressed between the aorta and the superior mesenteric artery, resulting in increased renal vein pressure and reflux of blood into the left OV, which causes hematuria and left flank pain that may be associated with CPP. Similarly, in May-Thurner syndrome, the LCIV is compressed by the right common iliac artery (RCIA), with consequent reflux of blood and increased pressure in the IIVs and pelvic collaterals (Figure 3).

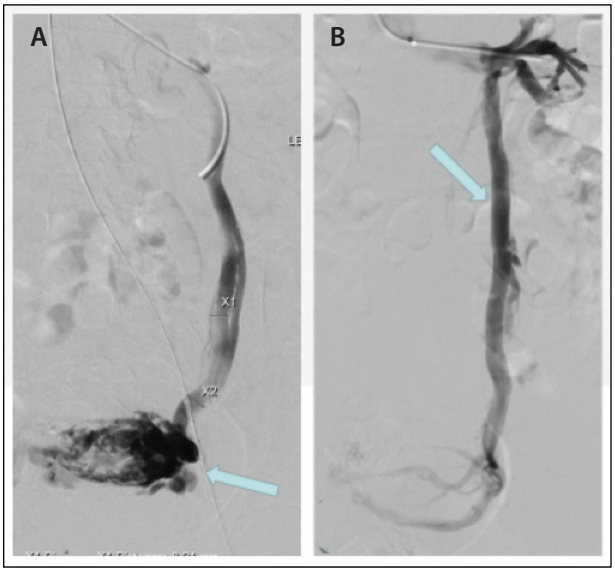

Figure 2. Left ovarian venography shows reflux of contrast into large pelvic veins (arrow) with crosspelvic collaterals (A). Left renal venography demonstrates reflux of contrast into the left OV (arrow) and pelvic veins (B).

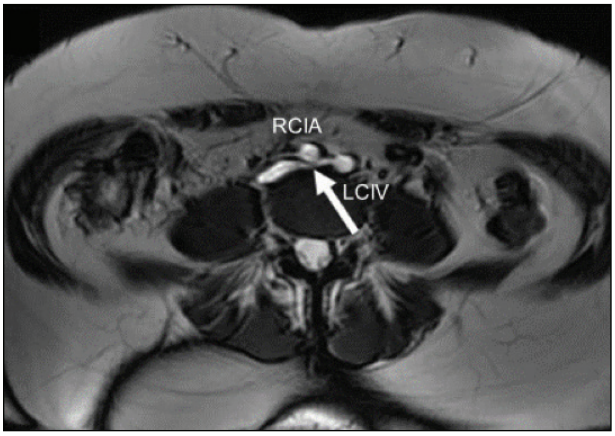

Figure 3. T2-weighted MRI demonstrates LCIV compression by the RCIA (arrow), characteristic of May-Thurner syndrome.

Aside from the classic symptoms of PeVD previously described, patients may also present with signs and symptoms of venous hypertension, with persistent isolated edema that is refractory to superficial ablation treatment, or sometimes with advanced venous disease. There should be high clinical suspicion of compression patterns in patients who are nulliparous and present with signs and symptoms of PeVD, prompting evaluation with cross-sectional imaging.

Variant 3: Escape Points

Pelvic-derived vulvar and lower extremity varicose veins result from pelvic venous hypertension that causes downstream reflux to the legs and groin through one of four common escape points: (1) perineal (the connection between internal and external pudendal veins), causing inner thigh and posterior labial varicose veins; (2) inguinal (recanalized round ligament vein), causing groin and labial varicose veins; (3) gluteal; and (4) sciatic nerve varicose veins. These patients may present with classic pelvic symptoms, and they may have more lower pelvic, buttock, thigh, and leg pain without ovarian point tenderness. Venous-directed treatment usually involves direct puncture and sclerotherapy under ultrasound or venographic guidance.

Variant 4: Anatomic Variation

Although anatomic venous variations are rare, IVC or iliac venous system variants are present in up to 3% of patients.[12] The gonadal veins have extensive collateral communication with the ascending lumbar and peritoneal veins; large retroperitoneal aberrant veins may drain into the OV and increase intravascular pressure. Moreover, anatomic variants in some patients with PeVD may have different compression points, resulting in pelvic varices (Figure 4).

VENOGRAPHIC EVALUATION OF PATIENTS WITH PEVD

Venographic evaluation is reserved for intention to treat and, as such, should include evaluation of venous reflux in the OVs and IIVs, as well as venous compression at the left common iliac and left renal veins. In our practice, we routinely perform intravascular ultrasound (IVUS) of the common iliac and left renal veins, as well as a venographic evaluation of venous reflux during the Valsalva maneuver.

PERCUTANEOUS EMBOLIZATION TECHNIQUES

When the angiographic diagnosis is performed and incompetence of the OVs and/or IIVs are established, the goal of the endovascular procedure is to achieve endothelial damage by mechanical, detergent, or osmotic action. It is important to embolize the entire length of the veins, including their tributaries, to prevent new collateralization.[13] The choice of embolic agents includes coils, foam, glue, and liquid sclerosants. Although coils and sclerosants are currently endorsed as level 2B evidence for treatment of OV incompetence,[14] the choice of embolic depends mainly on an operator's preference and experience. Coils demonstrate a controlled level of proximal occlusion, while liquid sclerosant agents produce more extensive embolus and are less expensive. That said, there are situations when a single dilated OV without venous collaterals or venous compression can be treated with coils alone, particularly if the coils are tightly packed as seen in Figure 5, which shows a patient with a classic presentation of PeVD and a single left OV.

In situations when extensive venous collaterals and/or duplicated OVs are present, we recommend a combination of sclerosant agent and coil embolization for the OVs (ie, the sandwich technique) and sclerosants for the IIVs. These two procedures can be performed in one setting or in a staged fashion. In our practice, we opt for staging

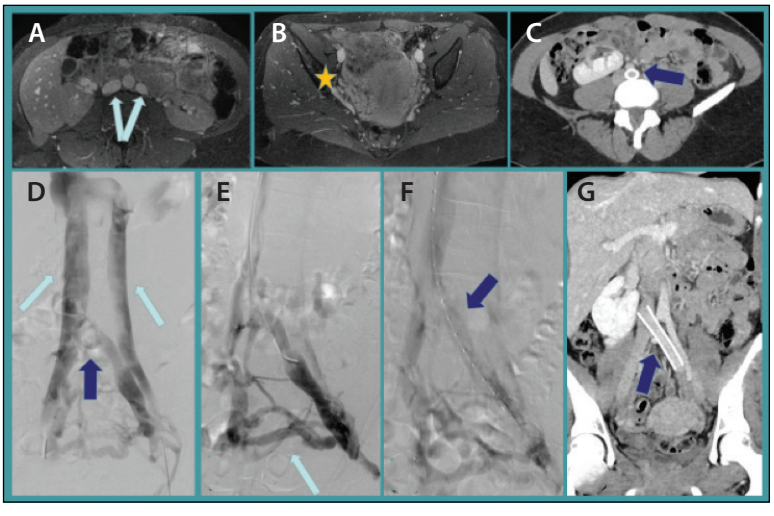

Figure 4. MR venography demonstrates a duplicated IVC (blue arrows, A) and significant pelvic varices (star, B) with crosspelvic collaterals. Duplicated IVC venography (D) shows compression of the limb (dark-blue arrow) connecting the left IIV and right IVC (light-blue arrows), resulting in significant retrograde contrast flow into the pelvic varices (light-blue arrow, E). Successful stenting (dark-blue arrow, F). Complete resolution of pelvic pain and patent stent (dark-blue arrows, C and G), as seen on CT venography at 16-month follow-up in axial and coronal views, respectively.

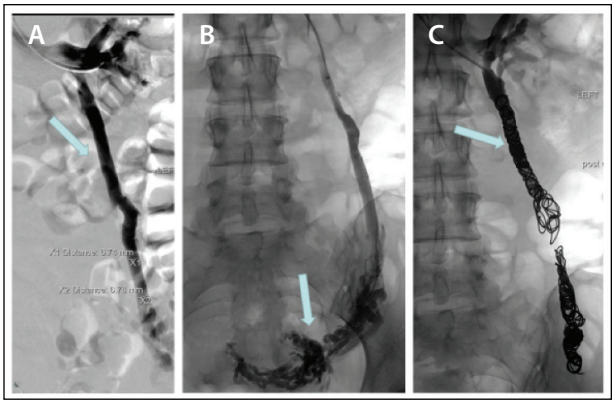

Figure 5. Left renal venography demonstrates reflux of contrast (arrow) into the enlarged left OV (A). Left ovarian venography shows reflux of contrast into large pelvic veins (arrow), but no other proximal collateral flow to the pelvis is visualized (B). Tight coil packing of the proximal left OV (arrow) with complete cessation of blood flow into the OVs and pelvic veins after embolization (C).

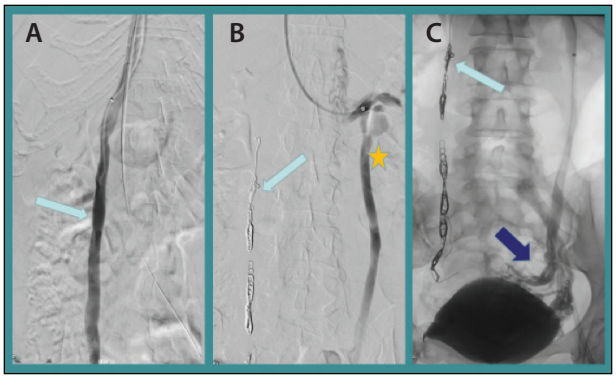

Figure 6. Dilated right OV (light-blue arrow) venography (A). Embolization of the right OV performed with coils and 3% STS foam, with the coils extending throughout the vessel length up to 2 to 3 cm proximal to the origin of the IVC (light-blue arrow, B). Note the contralateral left renal artery venography with retrograde flow of contrast into the left OV (star). Left gonadal venography (C) shows retrograde flow of contrast into the dilated tortuous parauterine veins (dark-blue arrow).

procedures, starting with embolization of the OV, with approximately 20% to 30% of patients presenting with persistent or recurrent CPP after the initial embolization, requiring IIV balloon-occlusion sclerotherapy for optimal clinical relief of pelvic pain.

How We Do It: OV Embolization

For OV embolization, we routinely use right internal jugular vein access due to the shorter and straighter pathway to the pelvic veins, with consequent simpler venous catheterization and catheter exchanges. In cases of retroaortic left renal vein or compromised catheter access from the superior vena cava, the femoral vein can be used as an access point. Initial IVC, left renal, and LCIV venography is performed, and a road map is obtained for further selective catheterization of the OVs and IIVs. This is performed under fluoroscopic guidance with a multipurpose catheter (80–125 cm, 4–5 F) to measure the pressures between the renal and left iliac veins and the IVC. Significant left renal and LCIV lesions are defined by either significant vein compression, opacification of the collateral veins, or retrograde flow through the gonadal veins. Next, selective cannulation of the left OV is performed with a Cobra-shaped or multipurpose catheter. A 10-mL contrast medium is hand injected into the vessel while the patient is asked to perform a Valsalva maneuver. Alternatively, the table can be tilted into the reverse Trendelenburg position. Reflux into the pelvic veins, dilated gonadal veins (> 5 mm), and contrast pooling in the pelvis should be documented as criteria for PeVD. Typically, each encountered diseased vein is treated before evaluating the next vein.

Embolization is performed using a combination of 0.035- or 0.018-inch detachable coils and 3% sodium tetradecyl sulfate (STS) (Sotradecol, Mylan Pharma Group Limited) mixed with air via the Tessari method. The injection should be done during the Valsalva maneuver. Coil deployment is extended throughout the vessel length, 2 to 3 cm proximal from the origin of the OVs off the left renal vein or IVC. If coils are used alone, tight packing of the vein is preferred (Figure 6). Diameters of coils range from 8 to 20 mm in size, depending on the size of the target vessel, as measured by venography.

Next, the right OV followed by the bilateral IIVs are examined and, if needed, treated. It is extremely important to also evaluate the pelvic venous vasculature for any escape points to the lower limbs or groin because reflux would warrant simultaneous treatment to ensure complete clinical improvement. Similarly, IVUS is extremely important to evaluate for left iliac or left renal vein compression (see **How We Do It: Stenting**). Postembolization venography should confirm vein occlusion. Complications are rare, with venous perforation due to a combination of venous spasm and hydrophilic guidewire manipulation being the most common and often self-limiting. Another rare but important complication is coil migration; to prevent this, we recommend using detachable coils, at least in the upper segment of OV embolization.

How We Do It: IIV Balloon-Assisted Sclerotherapy

In high-flow internal iliac varicoceles, an occlusion balloon can prevent systemic dispersion of the sclerosing agent, help completely fill pelvic varices, exclude existent collaterals, and

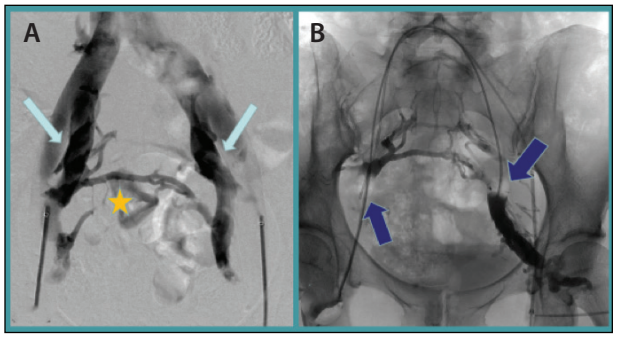

Figure 7. Bilateral femoral vein venography is performed with significant contrast reflux in the dilated bilateral IIVs (light-blue arrows) and crosspelvic collaterals (star) (A). Bilateral balloons are inflated (dark-blue arrows), and the selective left IIV is injected with contrast until normal veins are visualized to estimate the necessary volume of the sclerosant for embolization (B).

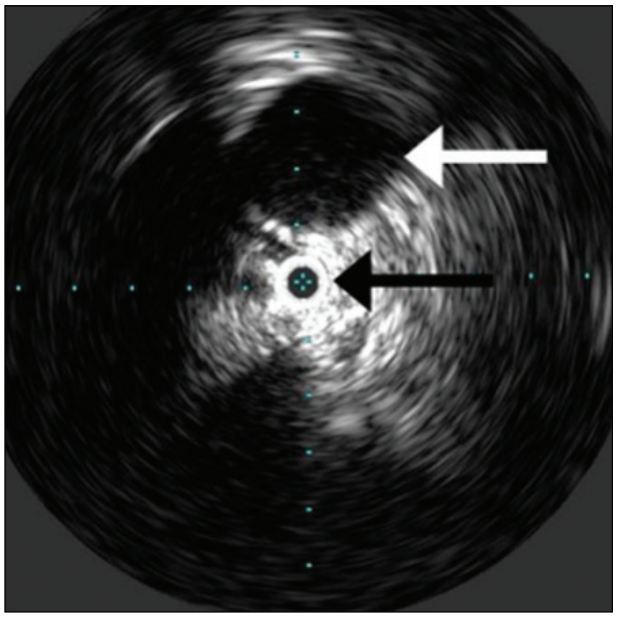

Figure 8. IVUS demonstrates a compressed segment of the LCIV (black arrow) and adjacent crossing RCIA (white arrow).

improve overall procedural efficacy. In our practice, sclerosing agents without coils are used when treating IIVs to minimize nontargeted embolization. Initial right femoral access is achieved with a 9-F short sheath, and the contralateral IIV is catheterized with a Cobra catheter. A 5.5-F Fogarty catheter (Edwards Lifesciences) or a 7-F Standard occlusion balloon catheter (Boston Scientific Corporation) is then exchanged over a 0.035-inch wire and placed just above the true pelvis where the tributaries of the main OV join. The volume of the varicose pelvic venous plexus can be estimated by inflating the balloon and injecting the contrast until normal veins start to be opacified (Figure 7).

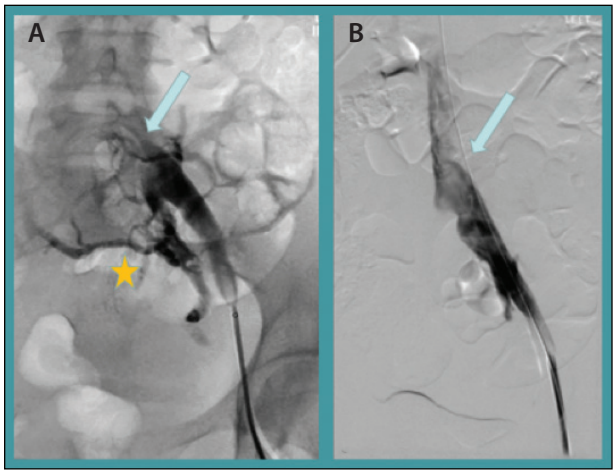

Figure 9. Left femoral venography demonstrates narrowing/compression of the proximal LCIV (arrow), with retrograde flow of contrast into the left IIV and pelvic veins (star) (A). Left femoral venography after Wallstent placement shows restored flow through the LCIV (arrow) and absence of retrograde flow in the left IIV and pelvic collaterals (B).

The volume of the sclerosing agent should be approximately 75% of the measured/injected contrast volume. Next, embolization is performed with a 3% STS foam mixed with air at a 1:4 ratio, and the balloons are kept inflated for 5 minutes. Postembolization venography should be performed to confirm the IIV occlusion. Finally, right (ipsilateral) IIV venography and embolization are performed with the same technique.

How We Do It: Stenting

In patients with PeVD, iliac stenting can improve symptoms in a large subset of patients with chronic pelvic venous insufficiency. A recent study recommends LCIV stent placement to augment the clinical outcome of gonadal vein embolization.[9] In our experience, patients with May-Thurner syndrome can present with isolated symptoms of CPP that, after exclusion of other pathologies, warrant venous stent placement.

After left transfemoral access, initial vessel evaluation with a 9-F IVUS catheter is performed to confirm venous compression (Figure 8) and estimate the size of the stent based on intraluminal diameters.

The stent is generally sized to the level of the proximal landing zone based on the reference lumen area seen on IVUS.[15] Stent size varies from 14 to 22 mm in diameter and can be oversized by 10% to 20%. The stainless steel self-expanding Wallstent (Boston Scientific Corporation) is preferred because of its higher radial force, longer length, and flexibility (Figure 9). Newer dedicated venous stents, such as the Vici (Boston Scientific Corporation)[16]

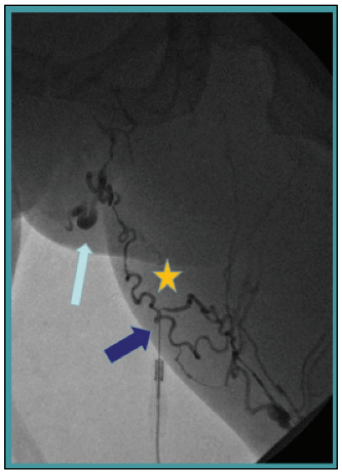

Figure 10. Direct puncture (dark-blue arrow) venography of the left vulvar (light-blue arrow) and left thigh (star) varicosities.

and Venovo (BD Interventional),[17] have high patency rates and excellent precision at the time of deployment. Given the different options available in the market, stent choice will depend on the diameter of the LCIV, length of venous compression, and operator's experience, but more studies are needed to evaluate outcomes in this patient population.

How We Do It: Vulvar and Gluteal Vein Sclerotherapy

Patients usually develop vulvar varices after two or more full-term pregnancies.[18] In addition to the symptoms seen with the classic variant of PeVD, these patients often experience vulvodynia, chronic pain localized to the perineum, and hypogastrium that is worse with heavy exercise. In patients with both CPP and clinically significant pelvic-derived vulvar or gluteal varicose veins, direct puncture embolization should be considered. Injecting foam or liquid sclerosant could be used for the treatment of atypical varicose veins of perineal, vulval, gluteal, or posterior thigh localization.[19] In our practice, fluoroscopic and/or ultrasonographic guidance are used to directly access these veins and perform venography followed by foam sclerotherapy.

Fluoroscopic-guided sclerotherapy has the advantage of titrating the drug dose and controlling the injection up to the visualized normal pelvic veins, thereby enhancing safety. The injection can be manually directed by compressing the connections to a normal vein or guiding the sclerosant to the desired targets. Our preferred sclerosant is 1% to 3% STS mixed with air at a 1:5 ratio. A maximum volume of 10 mL can be safely injected per procedure (Figure 10).

SUMMARY

As minimally invasive endovascular treatment of patients with PeVD becomes more prevalent, it is important for interventional radiologists to recognize the different patterns of disease presentation and be familiar with various embolization techniques. Classic OV incompetence can be sufficiently treated with a combination of sclerotherapy and coil embolization, whereas IIV insufficiency is more often approached with sclerotherapy alone due to the risk of nontargeted embolization. Careful pretreatment venography should always be performed to evaluate and plan for venous anatomic variants. Multiple forms of venous compression variants can be managed with stenting, and escape points to vulvar or lower extremity varicosities may require direct puncture sclerotherapy. In summary, optimizing interventional treatments for PeVD according to patients' symptoms and imaging findings are key for successful clinical outcomes. ■

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