Diagnosis and Endovascular Management of PVI

Pelvic venous insufficiency remains an underdiagnosed but treatable cause of chronic pelvic pain.

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Chronic pelvic pain is a common challenge for obstetricians, gynecologists, family physicians, and other providers of women's health care. Up to 39% of women will experience chronic pelvic pain at some time in their lives.[1] Unfortunately, pelvic venous insufficiency (PVI) often goes unconsidered among the many differential etiologies of chronic pelvic pain. It is estimated that 30% of women for whom there is no determined cause of pelvic pain in fact suffer from symptomatic PVI.[2] Pelvic congestion with pelvic varices was first described in 1857, and the first association of PVI with chronic pelvic pain was described in 1949.[3,4]

The hemodynamic mechanism of PVI is anatomically analogous to the male varicocele, but because the associated pelvic varicosities may not be externally visible or palpable, the diagnosis may be elusive.[4,5] Although psychosocial factors have been associated with PVI,[6,7] it is likely that these are the result of rather than the cause of symptoms related to PVI.[8] Because of the negative psychosocial implications historically associated with the traditional term of *pelvic congestion syndrome* (PCS), PVI is a preferable term because it defines the pathophysiology of the condition.

PATIENT POPULATION

Affected patients (usually aged 20–30 years) generally present with complaints of chronic pelvic pain of > 6-month duration. The classic presentation typically includes positional pelvic and lower back pain that is exacerbated by prolonged standing and strenuous activity. The pain is often described as heaviness and fullness in the lower pelvis, vulvar region, and thighs. The pain is typically exacerbated with menses and may be associated with dyspareunia and prolonged postcoital discomfort. Symptoms are usually most severe at the end of the day, and patients frequently describe relief with supine positioning.[9] Patients often present with atypical nonsaphenous pudendal, vulvar, and perilabial varicosities. Pelvic varices often collateralize into posterolateral thigh and gluteal regions.[8,10] An increased incidence of symptomatic PVI has been reported in patients suffering from complex nonsaphenous patterns of lower extremity superficial venous insufficiency.[11]

ANATOMIC CONSIDERATIONS AND PATHOPHYSIOLOGY

Pelvic venous outflow occurs via branches of the internal iliac and ovarian veins. The left ovarian vein typically empties into the left renal vein. The right ovarian vein usually drains directly into the inferior vena cava but may drain into the right renal vein in approximately 9% of cases. The ovarian plexus drains through the ovarian veins. The internal iliac venous drainage includes the obturator, internal pudendal, labial and gluteal veins, the uterine plexus, and the vaginal plexus. There is potential collateral communication between the ovarian plexus, uterine plexus, and the internal iliac veins.[12]

The etiology of PVI is multifactorial, including primary valvular insufficiency, venous outflow obstruction, and hormonally mediated vasomotor dysfunction. Anatomical studies show that up to 15% of women lack valves in the cephalad segment of the left ovarian vein, and 6% lack cephalad valves on the right. Even when they are present, valves are incompetent on the left in up to 43% of patients and on the right in up to 41%.[13] The frequency of incompetent valves and the diameter of the gonadal veins are significantly greater in multiparous women compared to the nulliparous population. This may be due to the increased venous capacity that occurs during pregnancy.[14] Venous outflow obstruction, which is seen in retroaortic

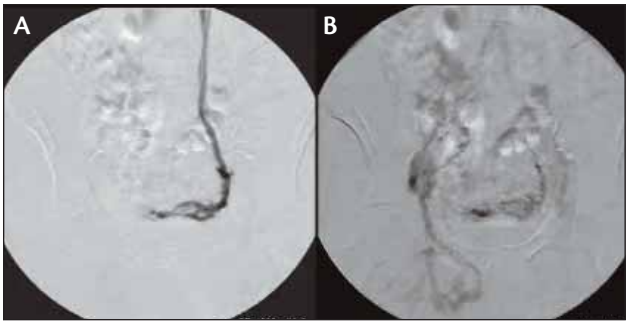

Figure 1. Reflux into the left ovarian vein and ovarian plexus with Valsalva (A). Delayed imaging from the same injection shows reflux across midline into the right upper thigh (B).

left renal vein and mesoaortic compression of the left renal vein, may also play a role in the development of PVI.

Disturbances of hormonal vasomotor regulation also likely contribute to the development of PVI. Animal studies have shown that uterine and ovarian veins have disproportionate sensitivity to ovarian-produced hormones. During a normal menstrual cycle, the ovarian veins are exposed to a near 100-fold higher concentration of estrone and estradiol compared with peripheral plasma.[13] The resulting ovarian venous distension is thought to exacerbate the symptoms of PVI. This theory is supported by the observation that the signs and symptoms of PVI are exacerbated with menstruation and pregnancy and generally diminish with menopause. This concept is further supported by reports of decreased pain associated with PVI after pharmacologic ovarian suppression.[9,15]

DIAGNOSTIC EVALUATION

Venography remains the gold standard in confirming the suspected clinical diagnosis of PVI. Venographic evaluation should include assessment of the inferior vena cava, left renal vein, gonadal veins, and the common and internal iliac veins. When possible, the patient should be asked to Valsalva to maximize physiologic venous pressure and distension while on the procedure table. Described diagnostic criteria for PVI by venography include venous ectasia with diameters of > 5 mm in the ovarian, uterine, and utero-ovarian arcade veins; free reflux in the ovarian vein with valvular incompetence; contralateral reflux of contrast medium across the midline; opacification of vulvar or thigh varices; and stagnation of contrast medium in the pelvic veins (Figure 1).[2,12,16,17] Although absolute venous dimensions may help in confirming the diagnosis of PVI, it is our opinion that strict diameter measurements should not preclude treatment if the overall clinical picture otherwise suggests PVI.

The primary goal and utility of ultrasound (US), magnetic resonance imaging (MRI), computed tomography (CT), and laparoscopy in the workup of PVI are to exclude concurrent pelvic pathology. Although cross-sectional imaging may demonstrate pelvic venous ectasia, there are limited published data comparing laparoscopy, US, CT, and MRI to the generally accepted venographic gold standard.[16,18,19] Multiplanar pelvic MRI may allow exclusion of uterine and ovarian pathology, endometriosis, adenomyosis, and lumbosacral disease. The anatomic information obtained from the MRI has also been described as useful in assisting preprocedure planning before the embolization.[20] Up to 80% of pelvic varices go undetected by laparoscopy due to technical limitations, including compression of the varices from peritoneal CO_2 insufflation and the resultant decompression of varices while the patient is in the Trendelenburg position.[17] In a longer-term 2-year study of 131 patients with a confirmed diagnosis of PCS, the sensitivities of MRI, laparoscopy, US, and CT were found to be 58.6%, 40%, 20%, and 12.5%, respectively. The investigators found no difference in clinical outcomes from embolization for PVI between those patients who had positive pre-embolization cross-sectional imaging or directly visualized pelvic varices by laparoscopy compared to those with negative laparoscopy and prevenographic imaging. They attributed this observation to relatively low sensitivities for MRI, laparoscopy, US, and CT.[2]

EMBOLIZATION

Although various approaches to treatment have been employed, such as hormonal ovarian suppression, hysterectomy with bilateral oophorectomy, and psychotherapy, the treatment of choice is selective transcatheter ovarian and pelvic venous embolization. A single, small, prospective, randomized trial compared embolization to combined hysterectomy and oophorectomy or hormonal ovarian suppression. Embolization was found to be significantly more effective at reducing pelvic pain in PCS.[21] Since the first description of embolization for treating PCS in 1993, several case reports and larger patient series have reported that embolization offers an effective, minimally invasive, safe therapeutic option with a > 95% technical success rate and significant relief of symptoms in 68% to 100% of patients with mean clinical follow-up ranging from 1 to 48 months.[2,14,22-29]

Embolization is generally performed on an outpatient basis. Once gonadal venous insufficiency is confirmed by venography, transcatheter embolization is performed. Access to the gonadal veins is traditionally gained via a transjugular or transfemoral route, depending on operator experience and preference. The ovarian veins often have multiple tributaries (Figure 2) that generally collateralize into the utero-ovarian venous arcade with contralateral reflux of contrast medium across the midline. Incomplete embolization of the gonadal vein tributaries and associated pelvic collaterals may result in clinical failure.

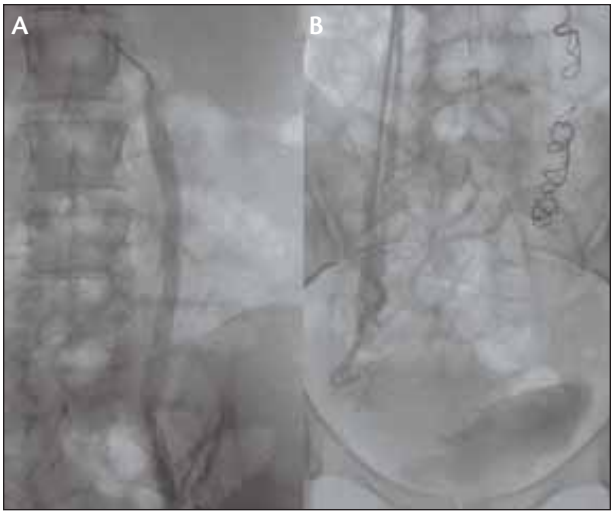

Figure 2. A selective left ovarian venogram with Valsalva shows ovarian venous ectasia with reflux (A). A selective right ovarian venogram shows tributaries with reflux into the right adnexa. The left ovarian vein has been embolized (B).

The goal of embolization is to eliminate the hydrostatic pressure that is generated from gonadal and pelvic venous collateral venous insufficiency. Foamed sclerosant combined with Gelfoam (Pfizer Pharmaceutical, New York, NY) is often used as an adjunct to coil embolization and has been reported to reduce recanalization and treatment failure.[2] Commonly used sclerosant agents include 3% sodium tetradecyl sulfate and 5% sodium morrhuate.

A safe injection volume of sclerosant can be estimated based on a test injection of contrast. To avoid nontarget embolization, the volume of embolic agent should be less than or equal to the volume of the test injection. Coil embolization is then performed in the main gonadal vein and its principal tributaries to a level within 3 cm from the left renal vein or inferior vena cava on the right. Coils should be oversized to optimize stability and prevent migration. After gonadal vein embolization, a repeat left renal venography should be performed to confirm gonadal vein occlusion. Venography of the inferior vena cava is also performed after embolization of the right ovarian vein.

Selective bilateral internal iliac venography should then be performed to assess for residual collateral flow into the ovarian plexus, vaginal, labial, and thigh-region varices (Figure 3). Selective embolization can then be carried out in a similar fashion. Coaxial temporary balloon occlusion has been described as useful in managing control flow during embolization. If coils are used in the internal iliac distribution, they should be oversized and used with caution, because there is an increased potential for coil migration.[2,27] Postembolization bilateral internal iliac venography is then performed with Valsalva to confirm successful embolization.

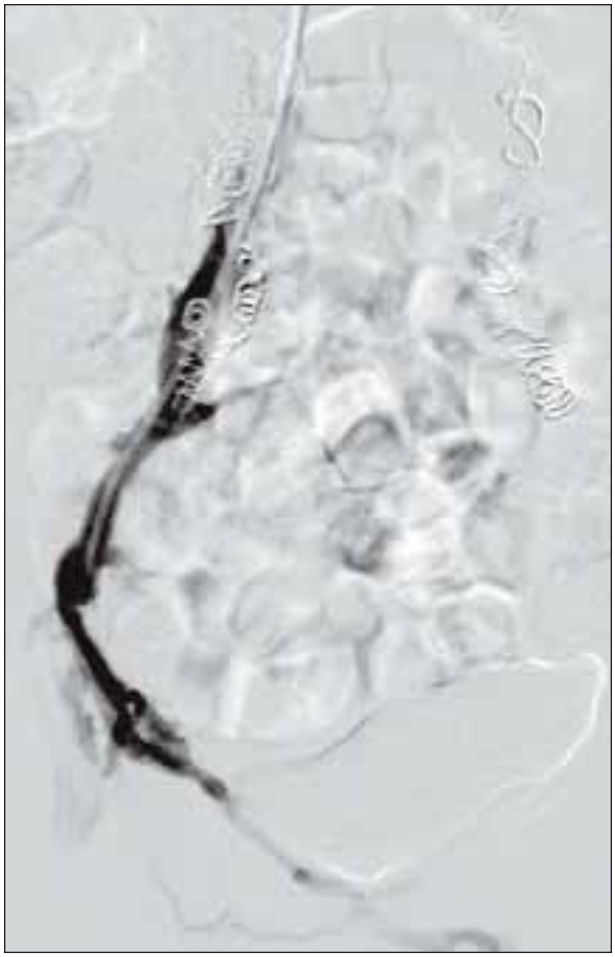

Figure 3. A selective right internal iliac venogram with Valsalva shows reflux into vulvar varices. Both ovarian veins have been embolized.

POSTPROCEDURE ASSESSMENT

The primary clinical endpoint of embolization is improvement of a patient's quality of life through relief of chronic pelvic pain. If available, relief of symptoms would be best documented through the use of an independently validated visual analog scale. However, because no published and validated scale specific to PVI is available, we recommend using a pre- and postprocedure survey, such as the one found in Table 1. It is our recommendation that postprocedure clinical reassessment should occur periodically out to 36 months. Technical success is a secondary endpoint for PVI treatment, and it is defined as the successful occlusion of diseased ovarian and internal iliac branch pelvic varices. Occasionally, there may be a recurrence of symptoms, which could require follow-up cross-sectional imaging, venography, and further embolization. However, technical and clinical failures will be minimized through appro-

TABLE 1. PRE- AND POSTPROCEDURE PATIENT QUESTIONNAIRE[a]

1. How intense is your overall pelvic pain?

0 1 2 3 4 5 6 7 8 9 10

2. How intense is your pelvic pain while lying down?

0 1 2 3 4 5 6 7 8 9 10

3. How intense is your pelvic pain while standing?

0 1 2 3 4 5 6 7 8 9 10

4. How intense is the pain in your leg(s) while lying down?

0 1 2 3 4 5 6 7 8 9 10

5. How intense is the pain in your leg(s) while standing?

0 1 2 3 4 5 6 7 8 9 10

6. How intense is the pain in your leg(s) during menstruation?

0 1 2 3 4 5 6 7 8 9 10

7. How intense is your pain during or after intercourse?

0 1 2 3 4 5 6 7 8 9 10

8. Do you have the urge to urinate more frequently than usual?

0 1 2 3 4 5 6 7 8 9 10

9. Do you take pain medication on a regular basis? (0, no; 10, daily)

0 1 2 3 4 5 6 7 8 9 10

[a]Zero (0) indicates no pain; ten (10) indicates extreme pain.

priate preprocedure patient selection and meticulous venographic and embolotherapy technique.

CONCLUSION

Symptomatic PVI is a significant yet underdiagnosed disorder that when left untreated may result in significant disability. PVI is often present in patients who suffer from complex patterns of lower extremity, nonsaphenous, superficial venous insufficiency. In the setting of a high clinical index of suspicion for PVI, venography remains the gold standard exam to confirm the presence of ovarian and pelvic venous reflux. The role of cross-sectional imaging is primarily to exclude concurrent pelvic pathology. Transcatheter embolization is a safe, established therapy with high reported rates of technical and clinical success. Careful patient selection, thorough venographic evaluation, meticulous embolization technique, and longer-term patient follow-up will help optimize clinical and technical results. ■

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