

# Pelvic Congestion Syndrome: Etiology of Pain, Diagnosis, and Clinical Management

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## ABSTRACT

Pelvic congestion syndrome is associated with pelvic varicosities that result in chronic pelvic pain, especially in the setting of prolonged standing, coitus, menstruation, and pregnancy. Although the underlying pathophysiology of pelvic congestion syndrome is unclear, it probably results from a combination of dysfunctional venous valves, retrograde blood flow, venous hypertension, and dilatation. Asymptomatic women may also have pelvic varicosities, making pelvic congestion syndrome difficult to diagnose. This article explores the etiologies of pain, use of imaging techniques, and clinical management of pelvic congestion syndrome. Possible explanations for the spectrum of pain among women with pelvic varicosities are also discussed.

## ABBREVIATIONS

CGRP = calcitonin gene-related peptide, CPP = chronic pelvic pain, IVC = inferior vena cava, PCS = pelvic congestion syndrome

Chronic pelvic pain (CPP) affects approximately one third of all women and accounts for 20% of outpatient gynecology appointments (1). The causes of CPP are varied and can involve endometriosis, pelvic inflammatory disease, pelvic varicosities, and many other conditions (Table 1). Even with extensive diagnostic testing and exploratory laparoscopic studies, the etiology of CPP generally remains elusive. Pelvic congestion syndrome (PCS) occurs when varicose veins develop around the ovaries in a setting of CPP. Similar to varicose veins in the legs, pelvic varicosities are thought to result from a combination of dysfunctional venous valves, retrograde blood flow, and venous engorgement. Congested pelvic veins can be very painful and account for approximately one third of cases of CPP (1). Although enlarged pelvic veins and pain are the hallmark features of PCS, asymptomatic

women also have been found to have pelvic varicosities (2–4), making PCS a challenging disorder to diagnose. Within this context, the clinical efficacy of ovarian vein embolization for treating PCS has yielded mixed results suggesting that there is a clear imperative to identify patient subpopulations for which endovascular intervention would be most beneficial. In this article, we explore the mechanisms by which pelvic varicosities can lead to pelvic pain, the imaging criteria used to confirm venous dilatation, and the clinical management of PCS.

## CLINICAL MANIFESTATIONS OF PCS

PCS typically affects multiparous women of reproductive age. Most women present with noncyclic lower abdominal or pelvic pain, usually described as a dull ache or fullness that persists for > 6 months. This pain is often exacerbated by prolonged standing, coitus, menstruation, and pregnancy. Associated symptoms are nonspecific and include headache, bloating, nausea, vaginal discharge, vulvar swelling, feeling of leg fullness, lower backache, rectal discomfort, urinary urgency, generalized lethargy, and depression. Some women with PCS present with minimal to no pelvic pain but instead have progressive hip pain (5), lower extremity varicose veins (6), or persistent genital arousal (7) as the sole manifestation. Most commonly, varicose veins of the vulva, perineum, buttocks, and lower extremities are found on physical examination. One study found that the combination of

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None of the authors have identified a conflict of interest.

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*J Vasc Interv Radiol* 2014; 25:725–733

<http://dx.doi.org/10.1016/j.jvir.2014.01.030>

**Table 1.** Differential Diagnosis of Chronic Pelvic Pain

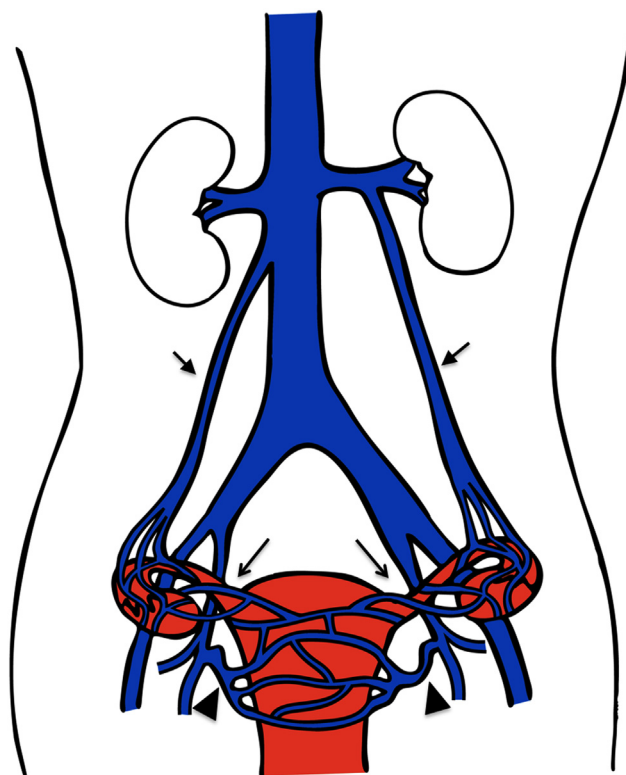
<b>Gynecology</b>	<b>Gastroenterology</b>
Endometriosis	Irritable bowel syndrome
Chronic pelvic inflammatory disease	Inflammatory bowel disease
Pelvic varicosities (pelvic congestion syndrome)	Diverticular disease
Fibroids	Chronic constipation
Ovarian cysts	Hernia
Adhesions	<b>Hematology/oncology</b>
Uterine prolapse	Cancer or metastases
Adenomyosis	Porphyria
<b>Urology</b>	<b>Musculoskeletal</b>
Interstitial cystitis	Pelvic floor myalgia
Recurrent urinary tract infections	Myofascial pain (trigger points)
Urethral diverticulum	Piriformis syndrome
<b>Neurology</b>	Psoas inflammation
Neuralgia of ilioinguinal, genitofemoral, or pudendal nerves	Sacroiliac joint inflammation
Neuropathic pain	Hip joint pathology
Herniated nucleus pulposus	Fractured coccyx
Abdominal epilepsy/migraine	Fibromyalgia
<b>Psychiatry</b>	
Major depression	
Somatization	
Sleep disorders	
Physical, sexual, or substance abuse	

ovarian point tenderness and a history of postcoital pain is 94% sensitive and 77% specific for PCS (8).

## RISK FACTORS FOR PCS

A combination of environmental, anatomic, and genetic risk factors contributes to the pelvic varicosities associated with PCS. Environmental factors include pregnancy, previous pelvic surgery, estrogen therapy, obesity, phlebitis, and engaging in careers that involve prolonged standing or heavy lifting. During pregnancy, pelvic vein capacity increases by 60% owing to the mechanical compression of the gravid uterus and the vasodilator action of progesterone (9). This venous distention persists for months after delivery and can render the venous valves incompetent, leading to venous hypertension and retrograde flow. Additionally, the weight gain and positional changes of the gravid uterus that occur during pregnancy can cause kinking of the ovarian veins and subsequent venous congestion.

Anomalies in pelvic venous anatomy also contribute to the development of PCS (Fig 1). In a normal individual, the ovarian veins originate from the pampiniform venous plexus in the broad ligament and communicate with the uterine plexus. The right ovarian vein drains into the inferior vena cava (IVC),



**Figure 1.** Gonadal veins (short arrows), uterine venous plexus (long arrows), and uterine veins (arrowheads) originating from the internal iliac veins. (Available in color online at [www.jvir.org](http://www.jvir.org).)

whereas the left ovarian vein drains into the left renal vein. These veins are usually 3–4 mm in diameter. A rich anastomotic venous plexus is responsible for draining the pelvic viscera, including connections between ovarian, uterine, vulvar, rectal, vesicle, and upper thigh venous systems. Valves are absent from ovarian veins in 15% of women and incompetent on the left and right in 40% and 35%, respectively (10). Only 10% of internal iliac veins have valves (11), leading to a degree of reflux in normal, healthy individuals.

In PCS, the left ovarian vein is most commonly dilated, presumably because it joins the left renal vein at a right angle facilitating reflux. When the right ovarian vein is affected, its junction with the IVC is usually anomalous (4). PCS has also been associated with mechanical compression that leads to obstruction in draining veins, including nutcracker syndrome (13) and May-Thurner syndrome (1). Additionally, obstruction of flow (ie, IVC thrombosis) or external forces (ie, endometriosis, fibroids, postsurgical or infectious adhesions) can increase ovarian vein pressure and subsequently cause reflux. Hypervascular pelvic tumors, including uterine leiomyomas, gestational trophoblastic neoplasms, ovarian solid tumors, and mesenteric tumors, can also cause compression and increase pelvic venous return via collateral vessels (14).

Although a genetic basis for PCS has not been established, the fact that this syndrome affects young

women with no predisposing risk factors suggests that inborn genetic traits are involved in the underlying venous pathology. There are several reports of familial clustering in patients with varicose veins (15,16), and it is estimated that 50% of patients with varicose veins have some genetic linkage (16). The *FOXC2* gene was the first to be implicated in the etiology of varicose veins, and it plays a key role in the development and function of venous valves (17). Other reports have found associations between development of varicose veins and mutations in *TIE2*, *NOTCH3*, thrombomodulin, and the type 2 transforming growth factor- $\beta$  receptor (18), which suggest a genetic component for the venous disease associated with PCS.

## **PATHOGENIC MECHANISMS OF VENOUS DISEASE ASSOCIATED WITH PCS**

Reflux through incompetent valves in the ovarian and pelvic veins appears to be the major defect in PCS (19). The mechanisms by which the valves become incompetent are poorly defined. On the one hand, there may be primary changes in valve structure, which lead to leaky valves, progressive reflux, and ultimately venous dilatation. On the other hand, there may be underlying structural abnormalities in the vein wall leading to dilated veins that subsequently cause valves to become distorted, dysfunctional, and leaky. Regardless of the inciting events, prolonged venous dilatation causes inflammation that further destroys valve structure, leading to significant reflux.

Disruption of vein wall integrity can also contribute to pelvic varicosities associated with PCS. Venous hypertension increases the expression of matrix metalloproteinases, which degrade the underlying endothelium and smooth muscle (20). These changes impair the ability of veins to constrict and relax leading to increased venous pressure, which further promotes endothelial cell injury by triggering leukocyte infiltration and inflammation, resulting in chronic venous distention and reflux (19).

Estrogen hyperstimulation is frequently implicated in PCS, even though its significance is unclear. Up to 50% of women with PCS have polycystic ovaries (identified on ultrasound) but do not have hirsutism or amenorrhea (1). Worsening of PCS symptoms during menstruation, increased prevalence of PCS in multiparous and premenopausal women, the positive therapeutic effects of hormonal substitution on PCS symptoms (21), and the high concentration of sex hormones in blood refluxing to the groin (22) all imply that hormonal factors play a critical role in the pathophysiology of symptomatic pelvic varicosities. Estrogen is known to weaken veins and induce venous dilatation through nitric oxide release, whereas progesterone weakens venous valves (20), which may collectively promote the development of incompetent ovarian and pelvic veins and subsequent reflux.

## **ETIOLOGY OF PAIN IN PCS**

By definition, PCS involves pelvic varicosities that are associated with CPP. Although the previous discussion highlights the potential determinants of varicose vein development, it does not adequately explain the reasons for pelvic pain. Although venous distention does not universally cause pain, stretch and stasis of the engorged ovarian and pelvic veins may activate selective pain receptors within the venous walls (23) causing a diffuse ache secondary to the low concentration of nociceptive afferents within viscera. Supporting evidence that distention of the ovarian veins leads to pain receptor activation is that gabapentin and amitriptyline, standard treatments for neuropathic pain, are more effective than opioid or nonsteroidal analgesia at relieving pelvic pain (24). Additionally, patients with both pelvic and lower limb varicosities report higher pain levels compared with patients with isolated lower limb varicosities (25). These studies suggest a direct relationship between the severity of pain and the degree of venous dilatation and reflux.

The release of neurotransmitters from the walls of dilated pelvic veins is postulated as another source of pain in PCS (26–28). Increased levels of substance P, a neurotransmitter involved in nociception, have been detected in symptomatic patients with PCS (28). Additional studies show that pharmacologic antagonism of substance P attenuates pelvic pain, providing further evidence that substance P contributes to PCS symptoms (29). Similarly, calcitonin gene-related peptide (CGRP), a neurotransmitter associated with autonomic feedback and sensory nerves of the reproductive tract, has been associated with pain. Studies show that CGRP infusion significantly exacerbates pelvic pain in women with PCS compared with control subjects, suggesting that PCS is associated with supersensitivity to CGRP (27). The following neurotransmitters also have been linked to PCS symptoms: adenosine triphosphate, endothelin, vasopressin, and nitric oxide (26–28). Treatment with medroxyprogesterone acetate reduces pain by inhibiting neurotransmitter release (30), further evidence that neurotransmitter release from dilated veins contributes to pelvic pain.

In addition to the activation of pain receptors and release of nociceptive factors, external mechanical compression causes pelvic pain. Pelvic structures are anatomically compact within the cavity. Dilatation of ovarian and pelvic veins, coupled with the associated local inflammation, can compress nearby nerves against adjacent anatomic structures and lead to ischemia and the visceral pain of PCS.

One can infer an evolutionary advantage for the pain caused by the dilatation of pelvic veins. Given the high incidence of PCS in pregnant women, it is conceivable that pelvic pain initially served to protect pregnant women from environmental dangers by making them seek shelter because the pain would limit their mobility.

Maladaptation of this evolutionary signal, possibly from genetic or anatomic variations, may be responsible for continued pelvic pain after childbirth.

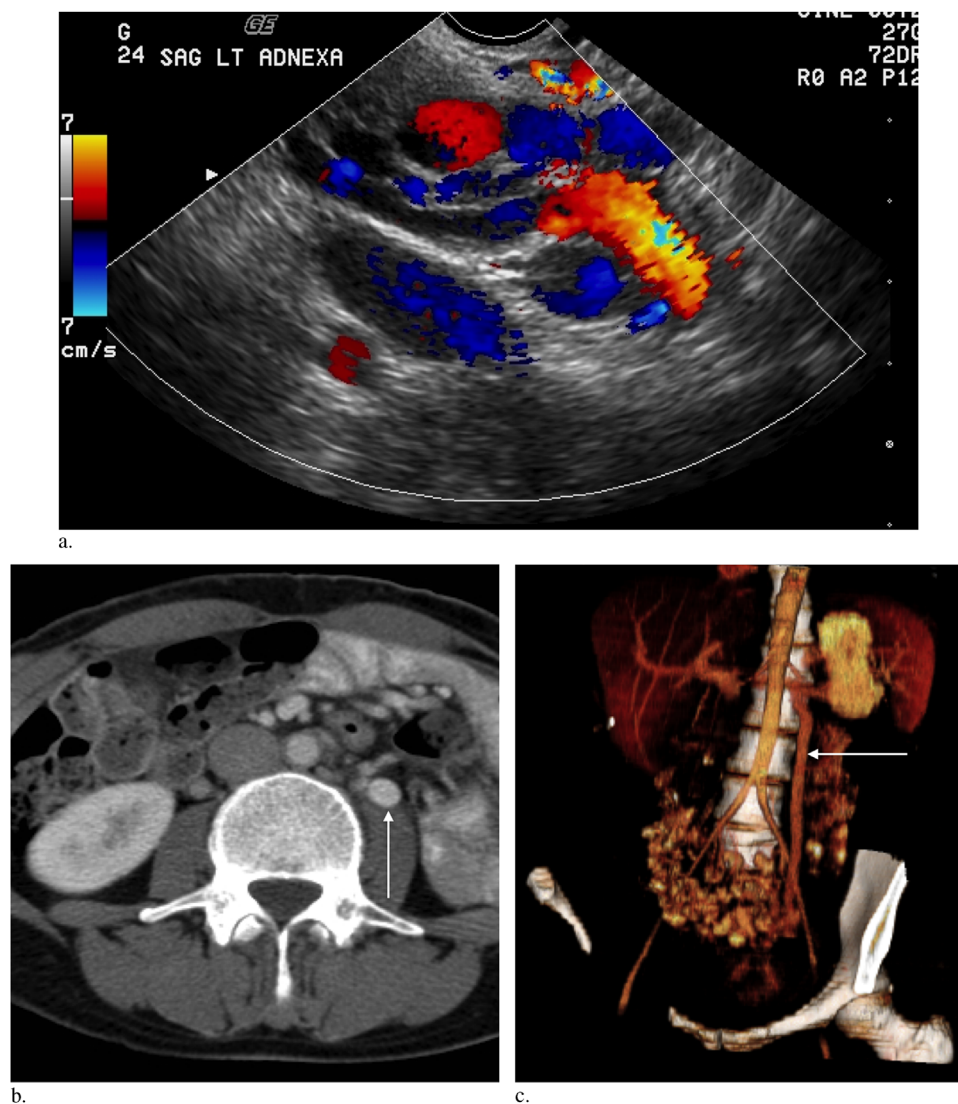
## IMAGING TECHNIQUES: SCREENING FOR PELVIC VENOUS DILATATION

Imaging studies cannot diagnose PCS but can confirm a distinctive clinical pattern of varicosities that when present in the setting of pelvic symptoms can help identify patients who require further diagnostic work-up. Different imaging modalities adhere to different diagnostic criteria for pelvic varicosities.

Pelvic ultrasound is usually the first-line imaging modality in patients with suspected PCS. Although both transabdominal and transvaginal ultrasound can be used, the transvaginal approach with Doppler evaluation

is generally preferred because it provides better visualization of the pelvic venous plexus and allows dynamic examination of blood flow through tortuous pelvic veins. Ultrasound allows patients to be imaged in a standing position or while performing a Valsalva maneuver, conditions that accentuate venous filling and enable better visualization of pelvic varicosities. The normal venous plexus appears as straight tubular structures with a normal diameter of < 4 mm. In patients with pelvic varicosities and suspected PCS, ultrasound typically shows dilated veins  $\geq 6$  mm in diameter, slowed and reversed blood flow in the ovarian veins, dilated arcuate veins communicating with bilateral pelvic varicose veins across the myometrium (**Fig 2a**), or associated polycystic ovaries (31).

On cross-sectional computed tomography (CT) and magnetic resonance (MR) imaging, pelvic varicosities appear as dilated, tortuous, tubular structures in



**Figure 2.** A 37-year-old woman with chronic pelvic pain. (a) Transvaginal pelvic ultrasound image demonstrates marked engorgement of veins surrounding the left ovary. Corresponding contrast-enhanced CT scan of the abdomen shows an enlarged left gonadal vein (arrow) in cross section (b) and in a reformatted three-dimensional image (c). (Available in color online at [www.jvir.org](http://www.jvir.org).)

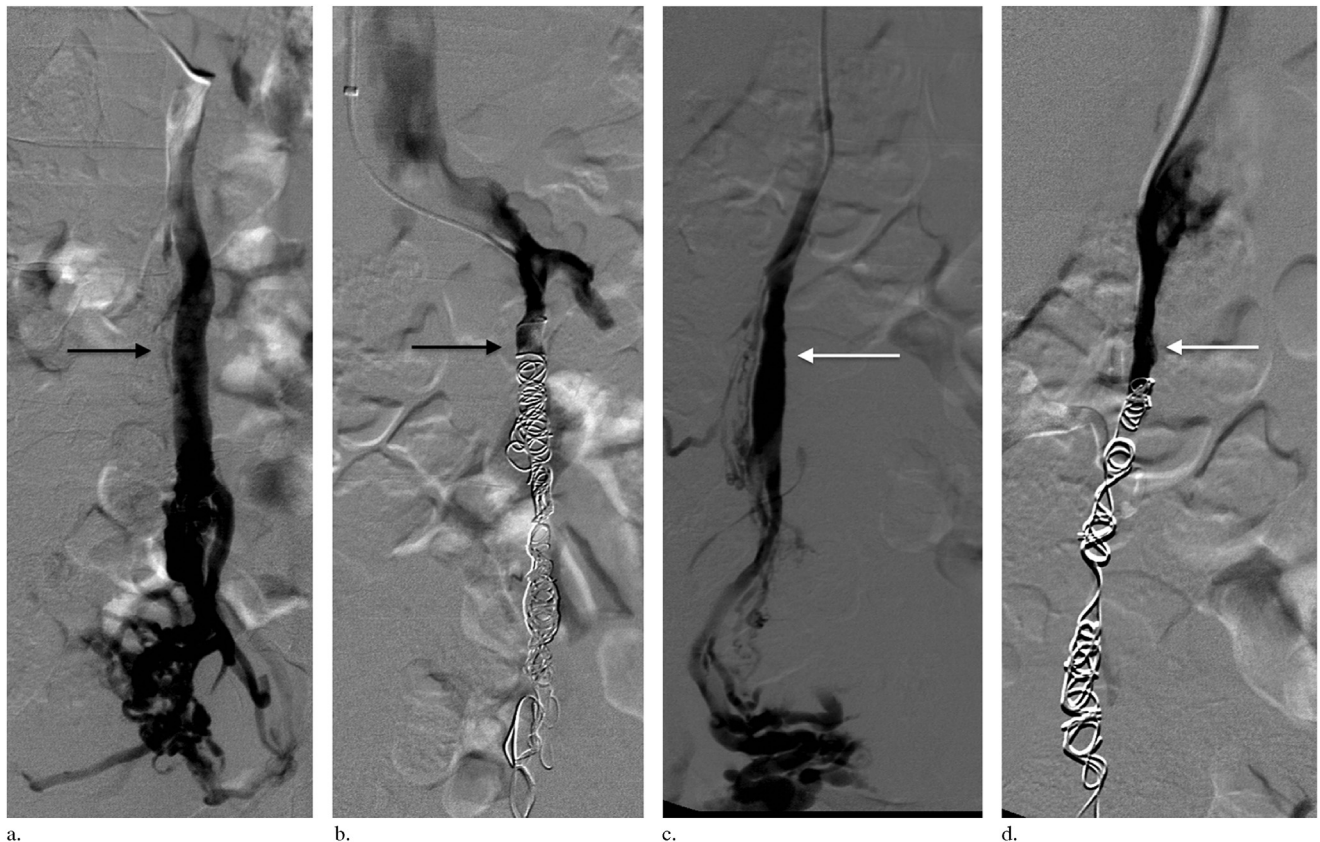
the uterine adnexa. Additionally, CT and MR imaging provide complete examination of pelvic anatomy and can identify coexisting pathology, such as compressive tumors. On CT, varicosities are isodense relative to other abdominal veins, whereas on MR imaging, varicosities are typically hyperintense on T2-weighted sequences (32). Various MR imaging sequences are used to highlight the varicosities associated with PCS, including gradient echo sequences showing high signal intensity within the ovarian and pelvic varices. The criteria for diagnosing pelvic varices with cross-sectional CT and MR imaging include the presence of at least four ipsilateral pelvic veins varying in caliber, with at least one measuring  $> 4$  mm in maximum diameter or an ovarian vein diameter of  $> 8$  mm (Fig 2b, c) (32). Despite these criteria, the significance of pelvic varices on CT or MR imaging is most often subjectively assessed and reported.

Contrast-enhanced MR imaging may become the initial imaging study for diagnosing pelvic venous incompetence because of its superior functional imaging and the fact that, in contrast to CT, it does not involve radiation exposure. Velocity-encoded phase contrast imaging and time-resolved MR angiography have significantly improved the detection of venous reflux (33–36).

A study comparing time-resolved MR angiography and conventional venography for grading ovarian venous reflux found no significant difference between techniques (34), establishing time-resolved MR angiography as an accurate, noninvasive method for assessing venous dilatation and reflux.

Venography has long been considered the diagnostic gold standard for assessing pelvic venous dilatation and reflux as well as planning for embolization treatment. Catheter-directed venography is performed by guiding a catheter from the jugular, brachial, or femoral veins to the ovarian or internal iliac veins and injecting contrast material. Venographic diagnostic criteria for pelvic venous incompetence include ovarian vein diameter  $> 10$  mm; congestion of the ovarian, pelvic, vulvovaginal, or thigh veins; and retrograde filling (Fig 3a–d) (37). In addition, venography from a catheter in the distal left renal vein may reveal reflux of contrast material into the left ovarian vein, further supporting the diagnosis of PCS. Besides excellent visualization of incompetent pelvic veins, a key advantage of catheter-directed venography is the option of performing interventional treatment if needed.

Laparoscopy is often used to search for an etiology in patients with CPP. Although it is useful for detecting conditions such as endometriosis or adhesions,



**Figure 3.** A 37-year-old woman with chronic pelvic pain and pelvic congestion syndrome (the same patient as in Fig 2). (a) Following right internal jugular vein access, a 5-French Davis catheter was brought through the left renal vein and into the left gonadal vein near its origin. A slow injection of contrast material demonstrated immediate filling of the markedly dilated left gonadal vein (arrow). (b) Coil embolization of the left gonadal vein (arrow). (c) Venogram in the same patient demonstrating immediate filling of the right gonadal vein (arrow) after slow injection of contrast material. (d) Coil embolization of the right gonadal vein (arrow).

laparoscopy is negative for detecting pelvic varicosities in 80%–90% of patients with PCS (1,38). Because laparoscopy is performed with the patient in the supine position and requires insufflation of carbon dioxide gas, intraperitoneal pressure is increased, which compresses (and often conceals) pelvic varicosities.

## SPECTRUM OF DILATATION AND REFLUX OF PELVIC VEINS: SELECTING PATIENTS FOR EMBOLIZATION THERAPY

Three studies on healthy female kidney donors identified “asymptomatic” women with pelvic varicosities on imaging (2–4), which further confuses the diagnosis and etiology of pain in PCS. Belenky et al (2) evaluated 273 healthy women for kidney donation using preoperative abdominal aortography. Left ovarian vein varices were incidentally found in 27 of these women (9.9%). However, in retrospect, 59% of these women acknowledged CPP and dysmenorrhea before surgery, although none of these women sought treatment for CPP, which is usually the case in PCS. All donors underwent left nephrectomy and left ovarian vein ligation. In patients with reflux in the left ovarian vein and pelvic pain, symptoms improved in 77% of patients (54% experienced complete relief and 23% experienced partial relief). Although this study included a patient population presumed to be normal from the standpoint of kidney donation, a significant number of the patients who demonstrated pelvic congestion had symptoms of CPP, which improved after harvesting of their normal left kidney for transplantation.

Nascimento et al (3) and Rozenblit et al (4) retrospectively reviewed cross-sectional images and found reflux in 38% of MR venograms and 47% of CT scans, respectively. Although no evidence of abdominal or gynecologic pain was detected by retrospectively reviewing the medical records of these women in the two studies, a focused pelvic pain history or examination was not performed. It is possible that the women with incompetent and dilated left ovarian veins had symptoms that were not elicited.

These three studies had varying definitions for pelvic varicosities—retrograde flow (3), dilatation of  $\geq 7$  mm (4), or dilatation of  $\geq 8$  mm (2)—and used different retrospective standards for assessing pelvic pain. Despite variations in methodology, these studies highlight that there is generally a broad spectrum of pelvic vein dilatation and associated pain with 4 mm being “normal,” 4–8 mm associated with asymptomatic reflux, and  $> 8$  mm associated with reflux with pelvic pain. Additional studies have shown that the mean diameter of competent ovarian veins ranges from 3.2–3.6 mm compared with 8.7–10.7 mm in patients with PCS (12,39). Such stratification coupled with clinical symptoms may be useful for determining which patients

are most likely to respond to embolization therapy. We generally recommend embolization treatment in patients with pelvic pain and evidence of pelvic vein incompetence as confirmed by venography ( $\geq 10$  mm ovarian vein diameter and reflux).

## CLINICAL MANAGEMENT OF PCS

Since the association between chronic pelvic pain and ovarian varicosities were first noted in the 1950s (40), many treatment modalities have been proposed, with medical and endovascular therapies currently being most commonly used. Medical management with medroxyprogesterone acetate or the gonadotropin-releasing hormone analogue goserelin typically provides symptomatic relief for several weeks, but benefits are rarely sustained (1). These medications are thought to relieve PCS symptoms by suppressing ovarian function, increasing venous contraction, and inhibiting neurotransmitter release (1). Presumably, resistance develops over time, explaining why symptomatic relief is not sustained.

Transcatheter embolization of the ovarian veins typically is performed after failed medical management and is increasingly being used as the primary treatment for PCS. Parameters for undergoing embolization include confirmation of pelvic varicosities with venography; the presence of associated CPP or dyspareunia (in the absence of other gynecologic causes); or the presence of severe labial, perineal, or lower extremity varicosities (10). To perform transcatheter embolization, a 5-F Cobra catheter (Cook, Inc, Bloomington, Indiana) is used to select the left renal vein from either a femoral or a jugular vein approach. Over a guide wire, the catheter is advanced to the ovarian vein plexus where embolic agents are delivered while the catheter is pulled back to occlude the left main ovarian vein. Sclerosants, glue, absorbable gelatin sponge (Gelfoam; Pfizer, New York), coils, and AMPLATZER vascular plugs (St. Jude Medical, St. Paul, Minnesota) have been used in the embolization of ovarian veins. Similarly, a 5-F Cobra catheter or a Simmons catheter (Cook, Inc) is used to select the right ovarian vein from the IVC. Over a guide wire, a Davis catheter (Cook, Inc) is advanced to the ovarian plexus, and the embolization procedure is repeated. Because communication exists between the ovarian and internal iliac veins, bilateral balloon occlusion venography and embolization of pelvic varices may be performed to prevent recurrence.

Embolization therapy is technically successful in 99% of cases (1,41,42), with a recurrence rate of  $< 8\%$  (1,43). Complications are rare and most commonly include coil migration, vessel perforation, and local thrombophlebitis (10). Treatment failure is generally explained by the complex anatomy of the pelvic veins, which show a wide variation in terms of trunks, venous valves, duplications, and crossover connections (44). Coil migration is more

likely to occur with embolization of the internal iliac veins than ovarian veins. Coils typically migrate to the pulmonary circulation and are removed without incident (42). Vessel perforation more commonly occurs in patients with intact valves at the confluence of the inferior vena cava or renal vein; risk of perforation is diminished by using hydrophilic wires and microcatheters, which increase the ease of passage (45). There have been no reports of changes in menstrual cycle or fertility after ovarian vein embolization.

## OUTCOMES OF EMBOLIZATION THERAPY

In 1993, Edwards et al (46) published the first case report for treating PCS with ovarian vein embolization. Long-term symptom relief was achieved for this patient, and widespread adoption of ovarian vein embolization followed. From published series in the 1990s, success rates for reduction of chronic pelvic pain ranged from 50%–80% (46–48). With advancements in technique, significant relief is now reported in 60%–100% of patients (Table 2) (41,42,49–52). This variation probably arises from the several definitions used for pelvic vein incompetence as well as the use of different outcome measures (eg, follow-up time, size of patient cohorts, symptom improvement scale).

Venbrux et al (42) studied 56 patients, all of whom underwent technically successful ovarian vein embolization. At mean follow-up of 22 months, 96% of patients reported symptom relief. Two patients experienced complications of coils migrating to the pulmonary circulation, but these coils were subsequently retrieved. Kim et al (41) conducted the first long-term study on ovarian vein embolization. In their study, 127

patients underwent treatment, with 108 receiving combined ovarian and internal iliac vein embolization. At follow-up evaluation at 45 months, 83% of patients showed clinical improvement, with 13% unchanged and 4% worse. Kwon et al (50) followed 67 patients up to 72 months after ovarian vein embolization. Of their patients, 82% reported improved symptoms. The remaining 18% of patients reported no symptom relief and required additional management with surgery or drug therapy. No serious adverse events were reported in these studies. Two more recent, larger cohort publications reported partial or total symptom relief in 93% of patients undergoing embolization (51,52). Monedero et al (52) followed 100 patients 14 months after embolization, and Laborda et al (51) followed 202 patients up to 60 months after embolization.

Although few clinical trials have compared ovarian vein embolization with other therapies, a randomized trial by Chung and Huh (49) showed that embolization was superior to hysterectomy and oophorectomy in providing symptom relief for PCS using a pelvic pain score measured on a visual analogue scale. The mean pain score decreased from 7.8 to 3.2 in the embolization group compared with 4.6 in patients receiving bilateral oophorectomy and 5.6 in patients receiving unilateral oophorectomy.

In conclusion, PCS is a common condition with significant physical and psychosexual consequences. Although the pathophysiology is multifactorial and is still poorly defined, interventional radiologists play a critical role in diagnosing pelvic varicosities and treating PCS-related symptoms with embolization. The spectrum of pelvic venous dilatation and reflux as well as associated pain makes the diagnostic and treatment criteria

**Table 2.** Outcome of Ovarian and Pelvic Vein Embolization to Treat PCS in Studies with  $\geq 50$  Patients

Publication	Patients (n)	Embolization	Material	Mean Follow-up (mo)	Clinical Outcome
Venbrux et al, 2002 (42)	56	56 bilateral ovarian, 43 bilateral internal iliac	Coils + sclerosant	22.1	Significant/partial relief: 96% No relief: 4%
Chung and Huh, 2003 (49)	52	Bilateral ovarian	Coils	6–12	Significant relief: 100%
Kim et al, 2006 (41)	127	106 bilateral ovarian + 95 internal iliac, 20 unilateral ovarian + 13 internal iliac	Coil + sclerosant	45	Improved: 83% Unchanged: 13% Worsened: 4%
Kwon et al, 2007 (50)	67	64 left ovarian, 1 right ovarian, 2 bilateral ovarian	Coils	44.8	Improved: 82% No relief: 15% Worsened: 3%
Monedero et al, 2012 (52)	100	NA	NA	14	Total relief: 64% Partial relief: 29% No relief: 7%
Laborda et al, 2013 (51)	202	Bilateral ovarian + bilateral hypogastric	Coils	60	Improved: 93% Unchanged: 5% Worsened: 1%

NA = not available; PCS = pelvic congestion syndrome.

difficult to define. In our experience, patients with pelvic pain who meet venographic diagnostic criteria for pelvic vein incompetence (ie, ovarian vein diameter  $\geq$  10 mm and reflux) are most likely to respond to embolization therapy. Results in these patients are encouraging, with 60%–100% reporting significant clinical improvement (41,42,49–52). Future studies are needed to address long-term outcomes, clarify which patients benefit from treatment, and improve understanding of the pathophysiology of PCS.

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