Pelvic Venous Insufficiency: Imaging Diagnosis, Treatment Approaches, and Therapeutic Issues

OBJECTIVE. The purposes of this article are to review the causes of pelvic congestion syndrome and the imaging used to make the diagnosis and to summarize the treatment options.

CONCLUSION. Pelvic congestion syndrome is one of many causes of chronic pelvic pain. It is thought to arise from ovarian and pelvic venous incompetence. Findings from various noninvasive imaging studies, such as Doppler ultrasound and MRI, in association with the clinical symptoms are critical in establishing the diagnosis.

Chronic pelvic pain is a common health problem that affects millions of women worldwide, but it can be a difficult condition to address. As many as 40% of all women seen in gynecology outpatient clinics report chronic pelvic pain. One fourth of all hysterectomies and one third of exploratory laparoscopic procedures are performed to investigate chronic pelvic pain [1]. Pelvic congestion syndrome (PCS) is a diagnosis that should be considered in many women after other pelvic conditions have been ruled out. Imaging may aid in diagnosis, and the imaging findings may facilitate precise preprocedure planning. We describe our current imaging workup for the diagnosis of PCS and the therapeutic approaches.

Description and Terms
Pelvic venous congestion is a commonly overlooked condition that can be severely painful and debilitating for many women. The term “pelvic congestion syndrome” specifically refers to the condition first described by Louis Alfred Richet in 1857 and characterized by chronic, dull pelvic pain, pressure, and heaviness that persist for more than 6 months with no other cause [2]. These symptoms are thought to be attributable to dilated, tortuous, and congested veins within the pelvis [3–5]. PCS is analogous to varicocele in men, but because the pelvic varicosities are not externally visible, the diagnosis can be overlooked. Symptoms are exacerbated with menses, prolonged standing, and activities that increase abdominal pressure [3–5]. The symptoms are worse at the end of the day. The presence of these symptoms in conjunction with a clinical examination finding of ovarian point tenderness is 94% sensitive and 77% specific for the diagnosis of PCS [4].

Pelvic venous insufficiency (PVI) is the standardized definition that specifically refers to the pathophysiologic mechanism of retrograde flow through incompetent ovarian and pelvic veins. The pathogenesis of PVI is multifactorial. Predisposing factors for the development of PVI include a family history of varicose veins, hormonal influences, previous pelvic surgery, a retroverted uterus, and multiple pregnancies [1, 3]. Specifically, the increased demand on venous return as a result of pregnancy and hormonal influences leads to chronic venous distention that can render the valves incompetent, leading to reflux of blood down the ovarian veins and into the internal iliac veins of the pelvis. The accumulation of blood within the pelvic veins can cause further engorgement, thrombosis, and mass effect on nearby nerves, which can lead to worsening pelvic pain. Dilated pelvic veins are more commonly seen on the left, where the left ovarian vein drains into the left renal vein before reaching the inferior vena cava, whereas the right ovarian vein drains directly into the inferior vena cava (Fig. 1). In addition, venous outflow obstruction from anatomic variants, such as compression of the left renal vein by the superior mesenteric artery (nutcracker syndrome) may result in engorgement of the left ovarian vein. Portal hypertension and acquired inferior vena cava syndrome may be related to the development of secondary pelvic venous congestion [2, 5, 6].
Disturbances in hormonal regulation also play a role in the development of PVI. Stones [7] found that in mice from which the ovaries had been removed, the uterine and ovarian veins selectively enlarged in response to estradiol administration with no change in the caliber of the femoral or iliac veins or the vena cava [7]. This finding suggests that the ovarian and uterine veins are selectively sensitized to ovary-produced hormones. During menstruation, the ovarian veins are exposed to a markedly larger concentration of estrone and estradiol than is present in the peripheral plasma. The symptoms of PVI are known to be exacerbated during menses and pregnancy but generally diminish with menopause. This theory is also supported by the accepted medical treatment of PVI by pharmacologic ovarian suppression [4, 8].

Clinical Characteristics

Patients with PVI are typically of childbearing age and multiparous [1, 3–5, 9, 10]. Women often present with unilateral or bilateral lower abdominal and pelvic pain, which is generally chronic and dull, but acute and severe pain may also occur. The pain, described as heaviness and fullness in the lower pelvis, vulvar region, and thighs, is exacerbated during menses and may be associated with dyspareunia. Patients may present with atypical nonsymptomatic pudendal, vulvar, and perianal varicosities. Incompetent pelvic varices also can collateralize along the posterolateral thigh and gluteal regions [11]. Signs of PVI include postcoital ache, dyspareunia, dysmenorrhea, perineal pain, urgency, and vulval and lower extremity varicosities [2, 9, 11–13]. Depression and anxiety are common comorbidities, in part owing to a prolonged time to diagnosis, but may also be physiologic. In response to the shearing and stretching of the congested ovarian veins, endothelial and smooth-muscle cells release vasodilator substances, which may play a key role in regulating emotions and stress [1]. Because of the negative psychosocial associations with the term pelvic congestion syndrome, pelvic venous insufficiency is now the preferred term because it describes the actual pathophysiologic mechanism [11].

Imaging Diagnosis

Various imaging modalities are useful in the evaluation of PVI, revealing multiple imaging features, such as ovarian vein size, flow velocity, flow direction, and the anatomic relations between the ovarian vein and the surrounding structures. Although transcatheter venography remains the standard for diagnosing dilation and congestion of veins, other, less invasive modalities exist to indicate the presence of pelvic vein engorgement and suggest the diagnosis of PVI. Furthermore, it is important to rule out other potential pathologic conditions that could account for chronic pelvic pain. We discuss the imaging findings of ultrasound, MRI, and venography (Table 1). CT should not be a primary modality for imaging women of childbearing age, but its findings are similar to MRI findings and are discussed in the MRI section.

Ultrasound

Ultrasound is widely available and does not involve radiation exposure. Multiple dilated tubular structures around the uterus and ovaries can be identified with transvaginal ultrasound. Color Doppler ultrasound technique is used for real-time analysis of arterial and venous flow. Three distinguishing sonographic criteria are important to adequately suggest the diagnosis of PVI: a dilated tortuous pelvic vein with a diameter greater than 4 mm, slow blood flow (≤ 3 cm/s), and a dilated arcuate vein in the myometrium that communicates with pelvic varicosities [5] (Fig. 2).

Doppler ultrasound is important because the gray-scale sonographic appearance of dilated veins can be similar to that of cystic adnexal masses [14, 15]. During a Valsalva maneuver or upright positioning, Doppler waveforms in the dilated pelvic veins may stop or reverse. Transabdominal sonographic measurement of the left ovarian vein with evaluation for reflux can also be performed [16]. The positive predictive value of a left ovarian vein diameter of 5 mm was 71%, and of 6 mm was 83%. Patients with PVI also tend to have polycystic ovaries, a larger uterus, and a thicker endometrium [17]. The sensitivity, specificity, and accuracy of ultrasound have not been reported, to our knowledge. Ultrasound has its limitations, depending on patient factors such as habitus and the presence of bowel gas, and is operator dependent. Furthermore, normal findings at ultrasound examination do not exclude the diagnosis of pelvic varices.

MRI

Multiplanar pelvic MRI has superb image quality, providing high tissue contrast and spatial resolution in depicting pelvic anatomic detail and vasculature. MRI may reveal evidence of other causes of chronic pelvic pain, such as endometriosis, which may not be visible at ultrasound, or other uterine, adnexal, urologic, gastrointestinal, or musculoskeletal causes of pain. In one study [18], the sensitivity and specificity of MRI for congestion according to the criteria developed by Kauppila et al. [19] with venography as a reference standard were 88% and 67% for ovarian veins, 100% and 38% for internal iliac veins, and 91% and 42% for the pelvic plexus. In another study [20], the sensitivity of time-resolved MR angiography for ovarian reflux ranged from 67% to 75%; the specificity was 100%; and the accuracy was 79–84%. At our institution, we use MRI of the abdomen and pelvis to identify the location and size of the ovarian veins and the presence of pelvic vari-
cosities. The diagnostic criteria for MRI and CT proposed by Coakley et al. [10] consist of at least four ipsilateral paranaute veins of varying calibers, at least one measuring more than 4 mm in diameter, or an ovarian vein diameter greater than 8 mm.

Ovarian vein and pelvic varices are hypointense on T1-weighted images, appearing as flow voids. On T2-weighted images, dilated ovarian and pelvic veins are hyperintense and of decreased flow velocity [10]. On MR images, pelvic varices appear as multiple dilated tubular structures around the uterus, ovaries, and pelvic sidewall. They are hypointense on T1-weighted images and hyperintense on T2-weighted images [10]. On contrast-enhanced T1-weighted gradient-echo MR images, dilated ovarian and pelvic varices are best visualized in the venous phase [15]. Subtracted maximum-intensity-projection reconstruction images are used for 3D visualization of the course of the dilated veins (Fig. 3).

Late arterial phase MRI can show retrograde filling of the ovarian veins from the renal veins and early draining of the pelvic veins into the iliac veins. Pelvic varices can vary in size and number and may extend into the broad ligament, reach the pelvic sidewall, or extend inferiorly to communicate with the paravaginal and thigh venous plexus (Figs. 4 and 5).

**Venography**

Transcatheter venography is performed when findings at noninvasive imaging are inconclusive for diagnosis in a patient with suspected PVI or when interventional treatment is pursued. Confirmatory venographic findings include dilated ovarian veins with diameters of at least 5 mm; retrograde ovarian vein reflux; uterine venous engorgement; and cross filling of pelvic veins across the midline with filling of vulvovaginal or thigh varicosities [11]. Although these findings are helpful, not all of them need be present in all patients being treated. Some authors [11] believe that strict diameter measurements should not preclude treatment. Conversely, correlation with clinical symptoms is essential because dilatation of the ovarian veins and ovarian venous reflux were seen on CT images of as many as 47% of women without symptoms [21]. PVI therefore should not be diagnosed solely with imaging.

The venographic scoring system of Beard et al. [5] consists of three components: maximum diameter of the ovarian vein, time to disappearance of contrast material, and degree of congestion. Each component is scored from 1 to 3 on the basis of degree. A score of 5 or more has been found to be an objective measure of pelvic congestion, having 91% sensitivity and 89% specificity. Ovarian vein diameter was considered normal if it measured 1–4 mm; moderate, 5–8 mm; and severe, greater than 8 mm. Time to disappearance after transuterine injection of contrast medium was scored on the basis of a time of 0, 20, or 40 seconds. Degree of congestion was determined as follows: Normal veins were small, straight, and easily seen. In moderate congestion, veins were tortuous with variable caliber. In severe congestion, veins were wide with great variation in caliper and were highly tortuous [19].

**Principles and Selection of Treatment**

Multiple methods of therapy for PVI have been used, including medical management with hormone analogues and surgical options, such as ligation of the ovarian veins and hysterectomy with or without bilateral salpingo-oophorectomy. Less invasive and modern approaches to treatment include transcatheter embolization of the ovarian or internal iliac veins.

**Medical Options**

The medical treatment of PVI consists in treating the entity as one of ovarian dysfunction. Proponents of this theory argue that estrogen is a venous dilator and that counteracting this effect can resolve symptoms. A review of the literature spans the use of hormone therapies, such as medroxyprogesterone acetate (MPA) and goserelin, a gonadotropin-releasing hormone analogue, in an effort to suppress ovarian function or increase venous contraction [9, 22]. MPA acts as an agonist of progesterone, causing an inhibitory effect on estrogen levels through suppression of the hypothalamic-pituitary axis. The net effect is to suppress ovarian function. The long-term benefits of MPA with doses of 30 mg/d for 4–6 months have been found to produce an optimal hormonal milieu of hypoestrogenism [17, 23] with success in pelvic pain reduction. Soysal et al. [22] performed a randomized controlled trial of MPA and goserelin in a group of 47 women with symptoms suggestive of PVI. They looked at the longer-term effects after 6 months of medical treatment in either the MPA (30-mg/d regimen) or the goserelin acetate (3.6 mg/mo) arm. Comparative objectives (peruterine venography) and subjective measures (symptom improvement) were measured at 6 months and 1 year. Goserelin was found to be more effective: Subjective improvements in pelvic symptoms persisted for 1 year after treatment [22]. The authors did not report on adverse effects. It remains unclear whether a temporary artificial menopausal state of 6 months can effectively yield a permanent cure of PVI. The limitations of gonadotropin-releasing hormone analogues are the side effects, cost, and lack of feasibility of long-term use because of the risk of menopausal symptoms and osteoporosis.

In a 2014 Cochrane review [24], 13 randomized controlled trials were evaluated to assess the effectiveness and safety of nonsurgical interventions for women with chronic pelvic pain, which included women with PVI or adhesions. The authors concluded that there is evidence of moderate quality to support progesterone as an option for the treatment of chronic pelvic pain. However, the authors stated that the limitations of this therapy are the adverse side effects, such as weight gain and bloating. The quality of evidence was considered very low for most comparisons of medical treatments because they involved single small studies. Furthermore, it is difficult to draw meaningful conclusions about quality-of-life and functional outcomes because of the large variation in outcome measures used in these studies.

The efficacy of a synthetic steroid consisting of a single-rod nonbiodegradable implant containing and releasing the desogestrel metabolite etonogestrel (Implanon, Merck) has been evaluated for the treatment of pelvic pain in women with PVI [25]. This implant has been widely used for long-term contraception. It inhibits follicle-stimulating hormone activity and thus ovulation and produces a hypoestrogenic state. In this small study, which included only 23 women, nearly 80% of the women with PVI who received the implant were satisfied with symptom relief after only 3 months of treatment. It is difficult to compare this device with other medical treatment options, because the other options are used for a shorter duration than the implant. The etonogestrel implant is a viable option for long-term medical treatment of PVI, but a long-term study with more subjects is necessary to evaluate the effectiveness and recurrence of PVI after removal.

**Surgical Options**

Surgical treatment options are based on counteracting the mechanical factors that af-
Pelvic Venous Insufficiency: Imaging and Treatment

Effects of pelvic varicosities. A marked increase in the capacity and laxity of pelvic veins later in gestation causes these vessels to become more vulnerable to enlargement and venous incompetence. This increased dilatation with venous stasis likely results in the chronic pelvic pain that women may feel. In the 1980s Rundquist et al. [26] found that resection of the left ovarian vein for PVI had a positive effect in relieving PVI symptoms. More recently, laparoscopic ligation of the ovarian veins also has had positive effects, but only a few case studies are available [7, 27]. Damage to nearby pelvic nerves and the development of collateral channels are risk factors. Hysterectomy was initially considered 100% curative, but several studies have shown residual pain in 30% of women and a 20% recurrence rate [28]. Overall, surgery is rarely used because more modern endovascular treatments are much more effective and less invasive.

Interventional Options

Transcatheter pelvic vein embolization was pioneered in 1993 by Edwards et al. [12] and has developed into a well-recognized procedure that is safe and efficacious in the treatment of women with PVI [11]. Numerous studies have shown clinical success rates ranging from 60% to 80% in reducing chronic pelvic pain. In one long-term study, Kim et al. [29] found that 83% of women had significant improvement in overall pain perception in a mean of 45 months. The goal of transcatheter embolization is to occlude the diseased, refluxing veins, which eliminates the increased hydrostatic pressure in the pelvis and resolves the varicosities. After occlusion of the refluxing vein, follow-up pelvic venography is performed to document resolution of the reflux into the varicosities. Clinical success is measured by long-term symptomatic relief.

Embolization Materials

Edwards et al. [12] described the first case of transcatheter embolization of ovarian varices in 1993. They used coils as the embolic agent. Today, embolizing agents include coils, sclerosants, and glue alone or in various combinations. Although the literature reports data on the use of different embolic agents, to our knowledge, no controlled studies have directly compared embolic agents for the treatment of PVI. Therefore, the choice of embolic agent is largely determined by the operator and institution. A review of the literature [30] shows that a combination of coils and sclerosing agents seems to be the most frequently reported method.

Historically, mechanical occlusion with metallic coils is one of the most common techniques of permanent blood vessel occlusion in endovascular therapy. Typical coils comprise either a stainless steel or platinum core and synthetic thrombogenic fibers funneling out from the core. Coils come in a variety of shapes and sizes and are typically used to treat vessels wider than 2 mm in diameter. Once deployed from the catheter, the coil assumes its shape in the vessel and causes mechanical occlusion and thrombosis, which lead to sclerosis and eventual permanent occlusion of the vessel [31, 32]. The optimal size and number of coils used for embolization are determined by the size of the ovarian vein being treated. Coil size can be 1–3 mm greater than the diameter of the vein to be embolized. Kwon et al. [33] in 2006 reported results of a study of 67 patients treated for PVI by coil embolization alone with an average of 5.8 coils per ovarian vein (Figs. 6 and 7).

Sodium tetradecyl sulfate (STS) and sodium morrhuate are commonly used sclerosing agents. Detergent-based chemicals act on the lipid molecules in the cells of the vein wall, causing intimal inflammation and thrombus formation, which lead to sclerosis and complete vessel occlusion. Sclerosing agents can be delivered in a variety of forms, including liquid, foam, or a slurry of sclerosant and gelatin foam sponge. When the agent is used in liquid form, small aliquots of the sclerosant (2 mL) are injected slowly with balloon occlusion of the proximal vessel until coagulation of the vessel is achieved. Often, metallic coils are placed behind the sclerosant to ensure the sclerosant stays in place and to contribute to complete vessel occlusion [34]. The preparation and use of STS foam for the treatment of varicose veins was introduced by Tessari in France in 1999 [35]. Using STS in the form of foam takes advantage of the fact that foam spreads on the endothelium so that there is larger surface contact, which induces more effective sclerosis. In addition, owing to its high viscosity, STS foam is associated with lower risk of extravasation and allows use of smaller quantities of sclerosing agent, thereby reducing the rate of systemic dispersion and adverse reactions. Gandini et al. [36] reported that the malleable nature of the foam allows complete filling of the varices via the ovarian vein, eliminating the need for selective catheterization of the internal iliac veins for embolization of eventual collaterals. Gandini et al. also stated that use of STS foam eliminated the need for concomitant use of metallic coils, so that the procedure is less invasive with overall less fluoroscopic time and therefore a lower radiation dose.

The use of glue as a venous embolic agent in the treatment of PVI has also been described in the literature. In 2000, Maleux et al. [37] reported the use of a mixture of 0.5 mL enbucrilate and 0.4 mL iodized oil, injecting approximately 2 mL of the mixture into each ovarian vein, occluding from distal to proximal. This technique was used in conjunction with coil embolization. Flushing the catheter with a 10% dextrose solution both before and after injection of the glue helps to prevent immediate coagulation of glue in the catheter. In very large ovarian veins (>2 cm), a sandwich technique with both glue and coils has been described [37]. The advantages of using glue include its ability to reflux into various branches of any eventual collaterals owing to its liquid state and its local inflammatory effect on the veins themselves, which contribute to effective thrombosis. The disadvantage of using glue for venous occlusion is the risk of migration of glue fragments. One of the more dangerous outcomes is pulmonary embolus.

Although numerous studies have shown the efficacy of percutaneous transcatheter embolization for the treatment of PVI, to our knowledge no study has directly compared the choices of embolic agents. Therefore, this choice is based more on the experience and preference of the operator than on objective data directly related to embolic treatment of PVI. A randomized clinical trial is warranted to further investigate the efficacy of various embolic agents in the treatment of PVI.

Technique for Pelvic Vein Embolization

Preoperative image analysis (ultrasound and MRI) is crucial for delineating the ovarian venous anatomy and for documenting the pelvic varicosities. Localized pelvic varices from dilated ovarian veins are the simplest to treat. Contribution to pelvic varices via the internal iliac veins may require a staged approach. Before embolization, adequate visualization of pelvic varicosities and their contributors is critical. We perform our transcatheter embolization technique with moderate sedation using fentanyl and midazolam with adequate monitoring by nursing staff. The ovarian veins are approached from either a femoral or a jugular route; we prefer the jugular approach. A 7-French sheath is placed into the inferior vena cava, and a
pelvis. If the patient experiences persistent problem of excessive blood flow within the vein. The primary refluxing vein, the gonadal vein. In such cases, we are conservation to localize the feeding vein. Variocisties can originate in the inguinal region, arising from the external iliac vein. It is frequently noted that internal iliac vein reflux documented before gonadal vein embolization resolves after embolization of the gonadal vein. In such cases, we are conservative about localizing and emoblizing the varicosties arising from the internal iliac vein. The primary refluxing vein, the gonadal vein, has been obliterated, eliminating the problem of excessive blood flow within the pelvis. If the patient experiences persistent pelvic discomfort, she can return for further emoblization of the hypogastric varicosties.

**Follow-Up**

After the procedure, patients may experience mild to moderate pain that is significantly less than that of arterial emoblization. Access to oral or IV pain medication may be needed. Emoblization procedures are typically performed in an outpatient setting. In a small cohort study (24 patients), Creton et al. [38] reported a high rate of technical success and clinical improvement in 80% of patients 3 years after embolization. Complications are rare, occurring at a rate of 0–4%, and include migration of coils to the pulmonary bed and thrombophlebitis and pain [39, 40].

Patients are usually seen in an outpatient facility within 1 week for short-term clinical follow-up and again within 3–6 months to confirm and document symptom relief. Clinical improvement markers to be reported before and after treatment should include overall pelvic pain level, pelvic and leg pain on standing and lying down, dyspareunia, menstrual pain, and the use of pain medications. The primary clinical endpoint should be to improve a woman's quality of life through relief of chronic pelvic pain. Emoblization of the ovarian veins should cause the pelvic varicosties to shrink within a few weeks. Only recently have a few prospective observational studies, conducted with a visual analog scale, shown relief of pelvic pain symptoms [32, 41]. Further studies conducted with objective scores are needed to determine the long-term beneficial effect of endovascular embolotherpy on symptom improvement.

**Conclusion**

PVI is difficult to diagnose in many women. Its identification is based on the presence of typical symptoms and imaging findings with various diagnostic modalities and parameters. The diagnostic and therapeutic approach to PVI must be individually tailored and must take into account the severity of symptoms. Multiple treatment approaches, varying from medical therapies to aggressive surgical options, have been used in the past. More recently, there have been encouraging technical and clinical success rates for selective endovascular emoblization of incompetent veins. However, additional clinical studies are needed to address the long-term outcomes and to clarify which patients may benefit from each treatment approach.

**Knuttinen et al.**

**References**

Pelvic Venous Insufficiency: Imaging and Treatment


(Figures start on next page)
Fig. 1—Drawing depicts ovarian venous anatomy. Right ovarian vein (v.) drains directly into inferior vena cava and courses inferiorly in relation to pelvic venous plexus. Left ovarian vein drains into left renal vein and then courses inferiorly. Internal iliac veins also communicate with pelvic venous plexus. (Courtesy of Natalie Doolittle, Medical Illustrator and Animator)

Fig. 2—45-year-old woman with pelvic venous insufficiency. A and B, Gray-scale (A) and color Doppler (B) ultrasound images show dilated left adnexal veins (arrow, B).

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Pelvic Venous Insufficiency: Imaging and Treatment

Fig. 2 (continued)—45-year-old woman with pelvic venous insufficiency. C–E, CT images show dilated left pelvic veins (white arrow) and left ovarian vein (black arrow).

Fig. 3—48-year-old woman with pelvic venous insufficiency. A–C, Venous phase (A and B) and maximum-intensity-projection MR angiographic (C) images show dilated left gonadal vein (arrow, A and C) and dilated pelvic collaterals (arrows, B) consistent with pelvic venous insufficiency.

Fig. 4—48-year-old woman with pelvic venous insufficiency. A and B, Late arterial phase MR images show retrograde filling of ovarian veins (white arrow) from renal vein (black arrow).
Fig. 5—49-year-old woman with pelvic venous insufficiency.
A–C, Thigh varicose veins (arrows, A), vulvar varicose veins (arrow, B), and dilated internal iliac veins and pelvic veins (arrows, C).

Fig. 6—48-year-old woman with pelvic venous insufficiency.
A, Maximum-intensity-projection MR angiogram shows enlarged left ovarian vein coursing inferiorly in pelvis.
B, Transcatheter venogram confirms dilatation of left ovarian vein.
C, Transcatheter venogram shows dilated ovarian vein with several collateral pelvic veins and cross filling into right internal iliac venous system.
Pelvic Venous Insufficiency: Imaging and Treatment

Fig. 7—48-year-old woman with pelvic venous insufficiency.
A, Venogram shows enlargement of right ovarian vein with engorgement of uterine veins.
B, Venogram obtained with catheter in left renal vein shows coils within right ovarian vein extending to origin from inferior vena cava and retrograde reflux down left ovarian vein.

Fig. 8—48-year-old woman with pelvic venous insufficiency.
A, Venogram obtained with catheter advanced distally into left ovarian vein shows engorged uterine venous plexus.
B, Venogram shows aftermath of coil embolization of left ovarian vein up to its origin with left renal vein.
Fig. 9—39-year-old woman with pelvic venous insufficiency.

A, Venogram shows large collateral pelvic vein arising from left internal iliac vein.

B, Venogram shows aftermath of coil embolization performed for venous varicosity.