ELSEVIER

Techniques in Vascular and Interventional Radiology

Pelvic Congestion Syndrome and Pelvic Varicosities

Sonya Koo, MD, PhD, and Chieh-Min Fan, MD, FSIR

Pelvic venous insufficiency (PVI), defined as retrograde flow in the gonadal and internal iliac veins, is the underlying cause of pelvic congestion syndrome (PCS), a common cause of disabling chronic pelvic pain in women of child-bearing age. PCS is a chronic pain syndrome characterized by positional pelvic pain that is worse in the upright position and is associated with pelvic and vulvar varicosities as well as symptoms of dyspareunia and postcoital pain. Through collaterals to the lower extremity venous system, PVI may also contribute to varicose vein formation and recurrence in the lower extremities. Endovascular embolization of the ovarian and internal iliac veins has become the treatment of choice for PVI and PCS. This article reviews the pelvic retroperitoneal venous anatomy, pathophysiology of PCS, treatment options and techniques, and clinical outcomes of embolotherapy for PCS.

KEYWORDS Venous insufficiency, pelvic pain, embolization

Background

Clinical Significance

Pelvic venous congestion has been recognized as a potential cause of chronic pelvic pain (CPP) in women. CPP is defined as noncyclical pain of greater than 6-month duration. It is a common complaint, with an estimated prevalence of 3.8% in the adult female population. It is the impetus for 10% of gynecologic referrals, 12% of hysterectomies, and 40% of laparoscopies.[1] Despite the many potential causes including endometriosis, fibroids, adhesions, urological and gastrointestinal disease, as well as ovarian pathology, the underlying cause of CPP remains unclear in 61% of patients. The socioeconomic cost of CPP is considerable, with estimated direct health care cost of 880 million dollars per year in the United States, and direct and indirect costs of 2 billion dollars per year. Of these women, 15% report missing work, and 45% experiencing reduced productivity.[2] Pelvic venous insufficiency (PVI), defined as incompetence of the ovarian vein or internal iliac vein or both, has been implicated as the cause of pelvic congestion syndrome (PCS), a chronic pain syndrome characterized by chronic positional pelvic pain associated with pelvic and vulvar varicosities. Ovarian vein incompetence is present in 10% of women, up to 60% of whom may develop PCS.[3] As such, PCS may be an underdiagnosed treatable cause of CPP.

In recent years, pelvic venous reflux has captured the attention of venous specialists for another reason, namely for its potential causal relationship to lower extremity venous insufficiency. The internal iliac veins connect to the saphenofemoral junction and lower extremity veins via pudendal and perineal collateral veins, thus transmitting pelvic venous hypertension to the lower extremity venous system. Pelvic venous reflux has been detected in 17% of 170 patients with recurrent varicose veins after surgery.[4] In a series of 100 women with sonographic and clinical signs of PVI, 71% demonstrated reflux on venography, 58% showed communication of the pelvic reflux to the lower extremity superficial venous system, and two-thirds manifested recurrent varicose veins after prior vein stripping.[5] Failure to recognize and treat this potential "highest point of reflux" in the pelvis may compromise the efficacy and outcome of venous treatments lower down.

Anatomy

PVI manifests in the 2 distinct but overlapping venous territories of the gonadal veins and the internal iliac veins. The gonadal veins include the ovarian veins in women and testicular veins in men. These are paired structures originating in the pelvis and terminating in the vicinity

Angiography and Interventional Radiology, Brigham and Women's Hospital, Boston, MA.

Address reprint requests to Chieh-Min Fan, MD, FSIR, Brigham and Women's Hospital, 75 Francis St, Midcampus 3, Boston, MA 02115. E-mail: cfan@partners.org

1089-2516/13/$ - see front matter © 2014 Elsevier Inc. All rights reserved.
http://dx.doi.org/10.1053/j.tvir.2014.02.005

of the renal veins. Valves are present in 80%-90% of gonadal veins, usually at or near the junction with the renal veins or inferior vena cava or both. The left gonadal vein terminates in the midsegment of the left renal vein in >99% of individuals. The right gonadal vein is more variable, terminating in the infrarenal inferior vena cava in 60%, at the right renal vein/inferior vena cava junction in 30%, and with intrarenal branches in the remainder.[6] The gonadal veins are commonly multichanneled networks of veins coursing together toward the pelvis rather than solitary veins. The gonadal veins collateralize extensively with the ascending lumbar and retroperitoneal veins. These features of gonadal veins affect treatment as unaddressed collateral branches can reconstitute retroperitoneal reflux pathways to the deep pelvis and cause recurrent symptoms. The internal iliac veins receive inflow from utero-ovarian, vesicular, hemorrhoidal, and sacral venous plexes.[7] When incompetent, internal iliac venous hypertension results in varicosity formation in the deep pelvis.

Pathophysiology

As with primary varicose veins in the legs, the development of PVI is likely a multifactorial process in which genetic predisposition is implicated and pregnancy has a strong causal association. During pregnancy, the ovarian and pelvic veins are subjected to the strain of increased circulatory volume and flow and to compression by the gravid uterus. The repeated insult from multiple pregnancies results in irreversible dilatation and valve damage resulting in pelvic venous reflux. Although PCS can be bilateral, venous incompetence is often asymmetric, with left ovarian and right internal iliac veins being the most commonly implicated vessels.[5]

As is the case with varicose veins elsewhere, pelvic varicosities can result from primary valvular insufficiency, or secondarily owing to more central outflow obstruction. Syndromes of central venous compression such as nutcracker syndrome and iliac vein compression syndrome can cause or worsen PVI. In nutcracker syndrome, the central left renal vein is compressed between the aorta and superior mesenteric artery resulting in functional obstruction and pelvic venous congestion when the left renal venous outflow decompresses down the left gonadal vein (Fig. 1). Demographically, nutcracker syndrome differs from PCS in that it can affect both genders as well as the pediatric population. In addition to symptoms and physical findings of PVI, symptoms of nutcracker syndrome also include flank pain, hematuria, and varicocele in men. Various treatments exist for repair of nutcracker pathophysiology including left renal vein transposition, external renal vein stenting with ringed polytetrafluoroethylene, gonadal caval bypass, and nephrectomy, and most recently endovenous stenting of the compressed renal vein segment.[8,9] In iliac vein compression syndrome (May-Thurner), the left common iliac vein is compressed by the right common iliac artery with decompression of the left pelvic veins via retrograde flow through the internal iliac veins and cross-pelvic collaterals.[10] Even more rarely, pelvic venous congestion may develop secondarily from regional venous overload from congenital vascular anomalies such as venous and arteriovenous malformations. In cases of secondary pelvic venous congestion resulting from central venous obstruction, correction of the underlying cause generally should precede treatment of the secondary reflux.

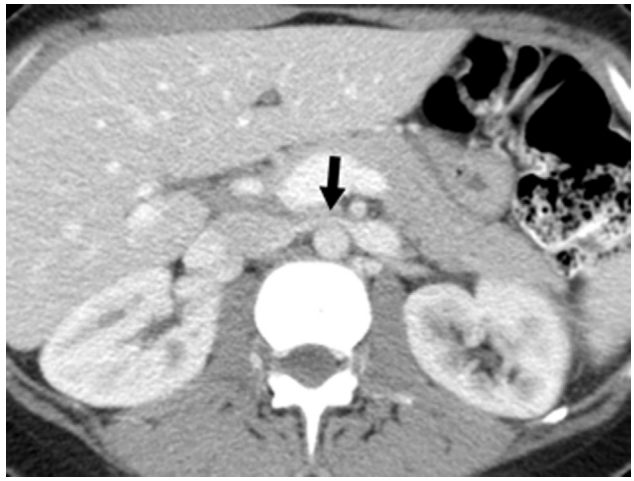

Figure 1 Axial computed tomography angiography image of nutcracker syndrome. Note the compression and narrowing of the central left renal vein where it passes between the superior mesenteric artery and aorta (black arrow). There is increased contrast enhancement in the distal left renal artery owing to delayed emptying of opacified blood from the left kidney.

Evaluation of the Patient

Clinical

Pelvic pain and prominent pelvic veins are often both present in women of child-bearing age, but are not necessarily causally related. Determining which patients have CPP specifically related to venous reflux remains one of the more challenging aspects of treating PVI. Clinical features that support the diagnosis include the following[10]:

- Quality of the pain: typical PCS-related pain is described as an aching discomfort that is reproducibly positional, worse when upright and improved by recumbency. Cyclical pain that most strongly coincides with menses is more suggestive of other pathology such as endometriosis.
- Multiparity: PCS is a disorder of primary venous insufficiency in most cases and therefore can occur in nulliparous women. However, pregnancy appears to be a major predisposing risk factor and a history of multiparity is frequently present.
- The presence or history of gluteal, vulvar, and perineal varicosities supports the diagnosis of PCS.
- Postcoital exacerbation of discomfort that may persist for hours.
- Premenopausal state: the pain of pelvic venous congestion tends to abate after menopause. In older women, positional pelvic pain may be due to other