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REVIEW

Comprehensive Current Overview of Pelvic Congestion Syndrome: Symptoms, Pathogenesis, Diagnosis, Treatment

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ABSTRACT

Pelvic congestion syndrome (PCS), which is one of the most common causes of chronic pelvic pain (CPP), is a frequently encountered disease in women that generally receives inadequate diagnosis. It is commonly seen in multiparous women. PCS is defined by CPP persisting for at least six months without evidence of inflammatory disease. Patients with complaints of CPP may be associated with PCS in 30% of cases; however, diagnosing it can be challenging due to overlap with other causes of CPP in the pelvic region. Therefore, exclusion of other causes of CPP is necessary. Recent studies indicate a trend towards preferring endovascular treatment over surgical intervention due to its high success rate and low complication risk. The strong evidence about the diagnosis and treatment of PCS is still incomplete. There are few randomized controlled trials available. It should be discussed and evaluated in a multidisciplinary setting involving gynecologists, GI specialists, pain management experts, physical therapists, and interventional radiologists. The aim of this review is to comprehensively examine current information on the symptoms, pathogenesis, diagnosis, and treatment of PCS.

Keywords: Coils, computed tomography, magnetic resonance imaging, nutcracker syndrome, pelvic congestion syndrome, pelvic varicose vein, trans-catheter embolotherapy, vascular stent

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INTRODUCTION

PCS is characterized by chronic pelvic pain that worsens when standing or sitting upright during the day, resulting from enlarged and dysfunctional pelvic veins in the lower abdominal area.[1] PCS occurs due to reflux or obstruction of gonadal or periuterine veins. It is also associated with the presence of varicose veins in the perineal or vulvar region. Compression of the left renal vein (LRV) between the superior mesenteric artery and the aorta, known as nutcracker syndrome, can also result in PCS.[2] Although 30% of patients with CPP complaints are associated with PCS, diagnosing PCS can be challenging due to the similarity of its symptoms with other pelvic pain conditions.

Pathogenesis

The pathology of PCS is not fully understood but is believed to result from valve incompetence in pelvic veins and changes in vessel walls due to multiple pregnancies or high estrogen levels. Estrogen's hormonal effect is known to cause venous dilation. The absence of reported cases of PCS in postmenopausal women supports this view.[2] Additionally, mechanical factors are considered influential. Ultimately, these factors lead to venous stasis, inadequate vessels, and retrograde blood flow in the pelvic venous system.[1–3]

Mechanical causes may cause vessel wall damage (such as valve insufficiency, agenesis or malformation), vessel compression (May-Thurner syndrome, nutcracker syndrome, etc.) or external compression (endometriosis or tumors, etc.).

One proposed mechanism for pain symptoms is that local stasis and stretching of venous walls release nociceptive factors contributing to inflammation and pain. Another theory suggests that dilated vessels can irritate adjacent nerves, leading to irritation.[4]

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Epidemiology

PCS is commonly observed in women of childbearing age. Pelvic varices are reported in up to 30% of CPP patients without other significant pathology; however, dilated veins have also been found in asymptomatic women.[4,5] PCS is generally associated with multiple pregnancies; however, the syndrome has been observed in nulliparous women as well.[5]

Physical Findings and Clinical Presentation

PCS accounts for 30% of cases among patients with chronic pelvic pain. There is no universally accepted or well-defined clinical criterion for PCS diagnosis. Chronic pelvic pain lasting at least 6 months is typically associated with evidence of pelvic venous insufficiency/dilation unless imaging identifies another cause for the pain.It usually develops after the first pregnancy and worsens during subsequent pregnancies. Common symptoms include pain in the abdomen and pelvis that worsens with prolonged standing or walking, and a dull ache or feeling of heaviness that resolves when lying on one's back. Typically worsens by the end of the day. In addition, it is also seen with symptoms such as dysmenorrhea, dysuria and dyspareunia. While vulvar and pelvic varicose veins are seen in women, varicocele is seen in men. PCS also contributes to psychological problems such as anxiety, stress, and depression.[6] A comprehensive history and physical examination should be performed. Careful evaluation should also include assessment of gluteal, vulvar, and/or thigh varices when present.[7]

IMAGING

Ultrasonography

Ultrasonography (US) should primarily be preferred to evaluate pelvic pain due to its accessibility, ability to rule out other diagnoses, and role in selecting patients for further examination. Transabdominal US can be useful for evaluating anatomical structures such as renal veins or main iliac veins that cannot be adequately visualized with transvaginal ultrasound. Color Doppler US, combined with Valsalva maneuver, is important for assessing valve incompetence.[8] Specific findings identified in ultrasound examination for investigating pelvic pain in non-pregnant patients are listed below.[9]

- Curved parametrial/adnexal pelvic vessels greater than 4 mm in diameter.
- Decreased blood flow velocity (less than 3 cm/s).
- Detection of retrograde flow in the left ovarian vein
- Polycystic ovarian morphology in the absence of amenorrhea or hirsutism history.

Since pelvic varices are estimated to be present in about 9.9% of the general population, isolated findings on imaging are not sufficient for diagnosing PCS. Asymptomatic patients should not receive treatment for PCS.

Computed Tomography and Magnetic Resonance Imaging

Computed tomography (CT) and magnetic resonance imaging (MRI) provide detailed information by allowing cross-sectional imaging of pelvic vascular structures, surrounding tissues, and organs (such as Nutcracker syndrome, tumors causing compression, cysts, or aneurysms).[8] CT and MRI are superior to ultrasound in identifying curved, dilated pelvic and ovarian vessels, as well as detecting vessel occlusion and compression (Fig. 1).[10]

Catheter Venography

Less invasive imaging is typically conducted before resorting to catheter venography, which is regarded as the gold standard for diagnosing PCS. Catheter venography also allows for simultaneous treatments such as transarterial embolization or sclerotherapy.[11] Findings on catheter venography include:

- Ovarian vein reflux flow.
- Diameter enlargement of 5–10 mm in pelvic venous structures.
- Occlusion of the ovarian venous plexus along the middle path, parauterine, uterovaginal, vulvovaginal, or proximal thigh vessels.
- Slowing or stagnation of contrast material in thigh or vulvar varices and pelvic vessels.

Simply discovering dilated pelvic veins incidentally on imaging in asymptomatic patients does not warrant treatment, as isolated findings are not diagnostic for PCS.

Diagnostic Laparoscopy

CPP is an important reason for performing diagnostic laparoscopy. Performing laparoscopy in a supine position can lead to the decompression of varices, CO_2 insufflation into the peritoneal cavity, and brid, which may result in missing 80–90% of PCS cases. For this reason, laparoscopy in conjunction with imaging can aid in ruling out other causes of CPP.[12]

DIFFERENTIAL DIAGNOSIS

The potential causes of PCS are diverse, ranging from urinary tract disorders and gastrointestinal diseases to musculoskeletal issues, neurological conditions, gynecological concerns, and even psychiatric disorders. Common reasons for CPP include painful bladder syndrome, pelvic inflamma-

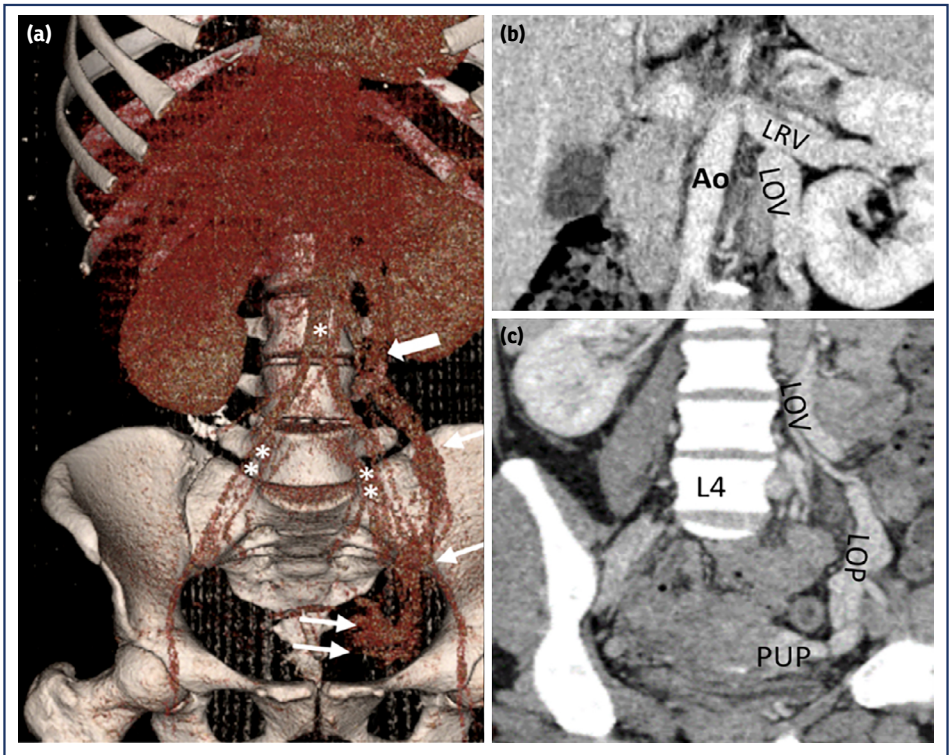

Figure 1. (a) Volume-rendered computed tomography. Left ovarian vein (arrow), ovarian plexus (thin arrows), parauterine plexus (short aroows), inferior vena cava (*), Iliac vessels (**). Curved **(b, c)** coronal curved MPR computed tomography

LOV: Left ovarian vein; LOP: Left ovarian plexus (pampiniform plexus); LRV: Left renal vein; PUP: Parauterine plexus; L4: Lumbar vertebra

tory disease, interstitial cystitis, endometriosis, pelvic neuralgia, irritable bowel syndrome, myofascial pain syndrome, and pelvic floor myalgia. Therefore, accurately diagnosing the underlying cause of CPP can be challenging even with laparoscopic and diagnostic radiological tests.[12]

ANATOMY

The pelvis has a complex venous drainage system with multiple venous plexuses, which can vary among individuals (Fig. 2). Both the internal iliac veins (IIV) and gonadal veins are responsible for draining the ovaries and uterus. The ovarian veins typically emerge from the pampiniform plexus, join the uterine plexus, and eventually empty into the inferior vena cava (IVC) on the right side and into the renal vein on the left side. The IIV runs slightly behind and toward the midline of the internal iliac artery, joining the external iliac vein to form the common iliac vein. It divides into parietal and visceral branches. The parietal branches consist of the superior and inferior gluteal, sacral, sciatic, ascending lumbar, and obturator veins. The visceral branches give rise to the middle hemorrhoidal vein, internal pudendal vein, vesicoprostatic plexuses in males, uterine vein, gonadal vein, and vesicovaginal plexuses in females. The IIV seldom drains directly into the IVC.[13]

The ovarian veins provide drainage for the parametrium, cervix, mesosalpinx, pampiniform plexus, forming anastomotic venous plexuses with paraovarian, uterine, bladder, vulvar and rectal plexuses (Fig. 1). At the level of L4, two or three branches come together to form a single ovarian vein. In most women, the left ovarian vein (LOV) typically drains directly into the left renal vein (LRV), while the ovarian vein (ROV) usually drains directly into the ICV. However, in approximately 10% of women, the ROV drains into the right renal vein (RRV).

A study has demonstrated that the diameter of normal ovarian veins is less than 5 mm.[13] These veins primarily have valves in the distal one-third. In one study, it was found that ovarian vein valves were absent in 15% of cases on the left side and 6% on the right side. Of those with valves present, 40% exhibited insufficient valves on the left side and 35% on the right side. Up to 47% of asymptomatic women who

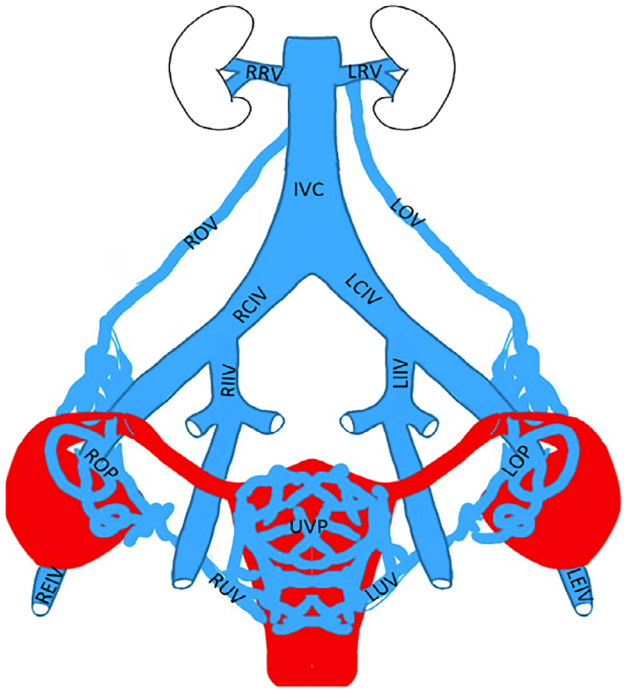

Figure 2. Pelvic venous anatomy. The ovaries and uterus are drained by both IIV and gonadal veins (LOV, ROV). The ovarian veins tpically arise from the pampiniform plexus (LOP, ROP) connect with the uterine plexus, and eventually drain into IVC on the rıght side and on the left side. The IIV runs slighty medial and posterior to the IIA, joining with the EIV to from the CIV

ROV: Right ovarian vein; LOV: Left ovarian vein; LOP: Left ovarian plexus (pampiniform plexus); ROP: Right ovarian plexus (pampiniform plexus); RUV: Right uterine vein; LUV: Left uterine vein; REIV: Right external iliac vein; LEIV: Left external iliac vein; LIIV: Left internal iliac vein; RIIV: Rigth internal iliac vein; UVP: Uterine venous plexus; LCIV: Left common iliac vein; RCIV: Right common iliac vein; IVC: Inferior vena cava; LRV: Left renal vein; RRV: Right renal vein; Figure 2 has been modified from reference number 39

have given birth may exhibit left ovarian reflux, with ovarian diameters ranging from 7 to 12 mm on imaging.[14]

Based on clinical and hemodynamic pathophysiological findings, researchers have identified four primary types of pelvic venous circulation disorders.

- Other types include vulvar varices that occur without accompanying pelvic congestion symptoms, which can present in various forms of pelvic congestion.
- Isolated insufficiency of the hypogastric vein.
- Gonadal vein reflux is the most common pelvic venous circulation disorder type. Nutcracker syndrome, caused by compression of the LRV, is another cause of PCS.[15]

TREATMENT

There is no standard approach to treatment, and therapy is personalized based on symptoms. Referral to multidisciplinary teams including gynecologists, gastroenterologists, pain specialists, physical therapists, and interventional radiologists may be indicated when diagnosis of pelvic pain suggests evaluation for other causes.

Medical Treatment

In medical management, the main objective is to suppress ovarian function or promote vasoconstriction in dilated vessels as part of the treatment approach. Medroxyprogesterone acetate (MPA) and goserelin, a synthetic analog of gonadotropin-releasing hormone (GnRH), have been utilized with limited efficacy. MPA is typically administered orally at a daily dose of 30 mg for 6 months, whereas goserelin acetate is given as a monthly 3.6 mg injection over a 6-month course.[16] Due to numerous side effects associated with chemical ovarian ablation, estrogen replacement therapy is often necessary. While these treatments provide some relief, the effects, especially with MPA, are typically temporary. Studies using MPA in daily oral doses have shown improvement in pain scores as assessed by visual analog scale during treatment; however, sustained results at 9 months post-treatment were achieved only when MPA was combined with psychotherapy.[17] These findings reinforce the strong relationship between the psychological and somatic symptoms of PCS. Both GnRH agonists and MPA are effective treatments for PCS; however, GnRH agonists have shown greater improvement in pain symptoms, sexual function and depressive symptoms 12 months after completing treatment compared to MPA. The side effects of progestins are generally seen as bloating and weight gain, while GnRH agonists are associated with hot flashes, night sweats, vaginal dryness, and mood swings.[18] GnRH agonists are not the preferred choice for long-term treatment of PCS due to their limited effectiveness and associated side effects. Medical treatment is a reasonable option for patients with mild to moderate symptoms who do not wish to undergo endovascular or surgical treatment, as it reduces fertility.

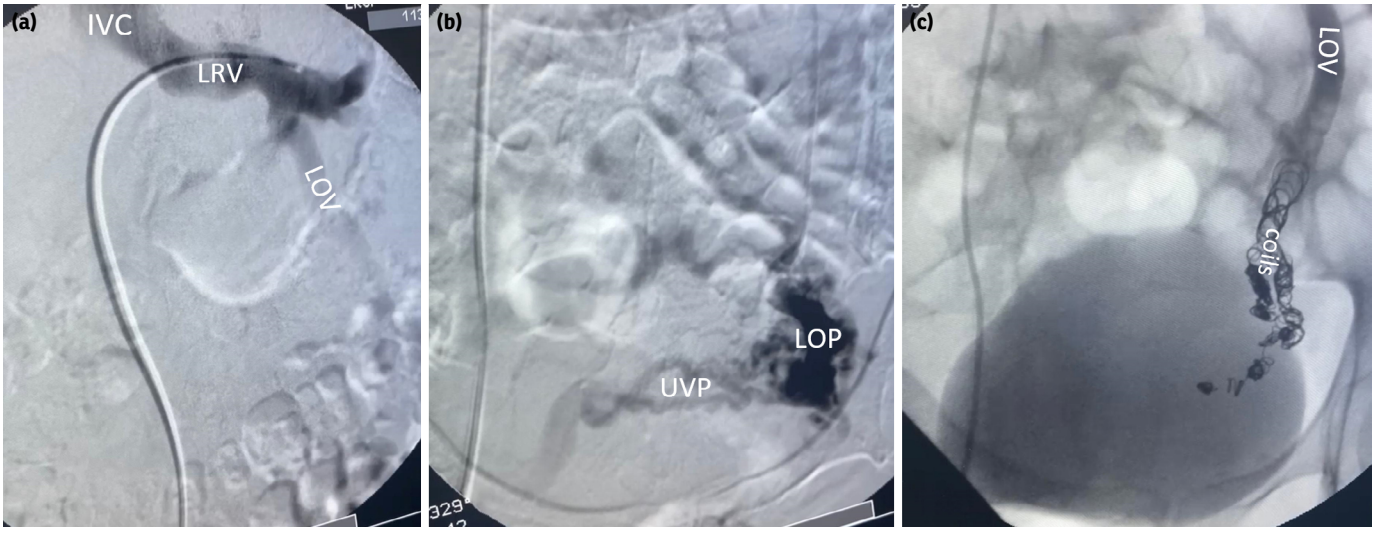

Figure 3. In a patient with pelvic congestion syndrome, catheter angiography showed: **(a)** Enlarged left renal vein (LRV) and left ovarian vein (LOV) in the left renal venogram. **(b)** In a more distal microcatheter LOP angiography, enlarged and rete-forming LOP, pronounced UPV, and reflux into the contralateral ovarian vein were observed. **(c)** After embolization of the LOP level and distal LOV with transcatheter coils of varying sizes, the angiogram showed that the LOP was completely occluded, reflux into the contralateral ovarian vein had ceased, and the UPV was no longer visible

LOP: Left ovarian plexus (pampiniform plexus); UVP: UVP: Uterine venous plexus; IVC: Inferior vena cava

Surgical Treatment

The extraperitoneal resection of the left ovarian vein, first reported by Rundqvist and others, has been shown to provide symptomatic improvement in two-thirds of patients with PCS.[18] Gargiulo and others published a major series on laparoscopic transperitoneal ovarian vein ligation in women in 2003, reporting complete resolution of symptoms with this method. Among the disadvantages of this procedure are higher surgical complications and the risk of developing deep vein thrombosis. Hospital stay and recovery time are more acceptable with surgical ovarian vein ligation. Surgery should be considered in patients with recurrent symptoms despite embolotherapy.[19]

TRANSCATHETER EMBOLOTHERAPY

Since its introduction to the market in 1993, transcatheter ovarian vein embolization (OVE) has been recognized as a cornerstone method in the treatment of PCS.[20] Various methods involving embolic agents such as sclerosing foam,[21] adhesives, Amplatzer plugs,[22] and coils[23] have been described (Fig. 3). There are numerous articles on transcatheter OVE based on small series and retrospective studies, with success rates in reducing CPP ranging from 47% to 94%. The follow-up period in these studies varies between 12 to 36 months.

Technique

After skin cleansing and covering with sterile drapes, a 6F introducer sheath is placed into the right femoral vein following local anesthesia. The right femoral vein is the most commonly used access route for venography and embolization. Transjugular, transbrachial, and basilic approaches are also options for access. Contrast medium is injected through the sheath to obtain iliocaval venograms. Next, a guidewire and vertebral catheter are used to enter the left renal vein via the external iliac vein and IVC route. After contrast injection through either a Vertebral, Cobra, or Sim 1 catheter, the LRV is visualized. The presence of stenosis, thrombus, or compression in the LRV is determined. Then, the guidewire is advanced into the LRV. Using either a vertebral catheter or microcatheter, depending on the operator's preference and the chosen embolic agent, varicose veins surrounding the ovaries and uterus are investigated and embolized (Fig. 3).

In studies, no significant difference in treatment response has been shown between unilateral and bilateral OVE. Researchers have used the visual analog pain scale to assess the reduction in severity of PCS symptoms before and after embolotherapy. One study reported an 85% success rate in reducing pain. Another study indicated that performing internal iliac vein (IIV) embolization as additional treatment after OVE improves outcomes and prevents re-

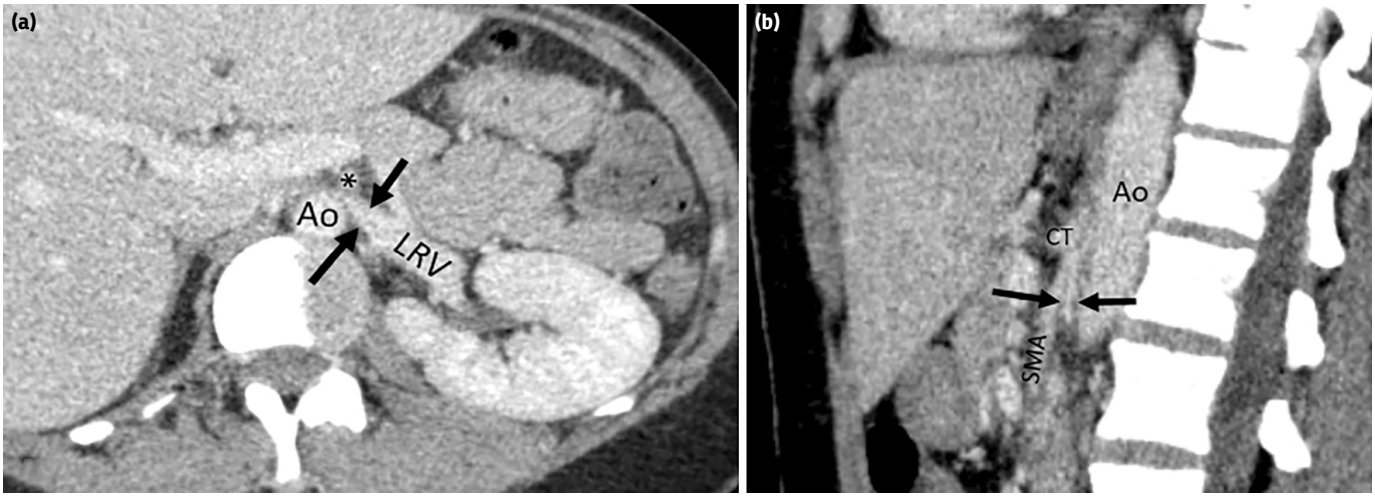

Figure 4. Anterior nutcracker syndrome. Contrast-enhanced axial and **(a)** sagittal **(b)** abdominal computed tomography. In image a, compression of the left renal vein (LRV) between the superior mesenteric artery (SMA) (*) and the abdominal aorta (Ao) is observed, creating a beak-like appearance (double arrows). Image b shows significant narrowing of the LRV secondary to compression

CT: Celiac trunk

currences.[23–26] Isolated ovarian vein reflux has resulted in symptom improvement in patients treated with OVE.

In patients with PCS, it has been reported that reduction in vulvar varices after OVE is more significant compared to lower extremity varices.[27] On average, six coils per vessel were reported to be used; coils are the most common method for embolization.[28]

To prevent coil migration into the pulmonary artery, the diameter of the coils should be at least 30% larger, especially after IIV embolization.[29]

In Europe, many centers use sclerosants for embolization of the spermatic cord in the treatment of varicocele in men. The effectiveness of this method has also been demonstrated in patients with vulvar varices in women.[30] Commonly used sclerosants include sodium tetradecyl sulfate and polidocanol, typically used in concentrations ranging from 3% to 5%. A catheter is placed into the reflux segment of the spermatic vein, and 3 or 4 mL of sclerosant is injected during the Valsalva maneuver. Subsequently, the catheter is held at the spermatic vein orifice for 2 to 3 minutes to maximize the effect of the sclerosant.[31]

Currently, there is ongoing uncertainty regarding the optimal technique for OVE. No significant difference has been found in terms of symptomatic improvement between unilateral and bilateral OVE.[32] When treating patients with OVE, the severity of symptoms, anatomy of pelvic varices, and degree of reflux in ovarian veins should be taken into consideration. For instance, in patients with bilateral high-grade ovarian vein reflux and complex varicose networks involving the internal iliac veins, bilateral OVE should be considered. On the other hand, unilateral OVE may be suitable for patients with unilateral mild varices. Although transcatheter OVE has replaced surgical and medical treatments, solid multicenter randomized controlled trials are still lacking in terms of diagnosis, treatment, and outcomes.[33]

Nutcracker syndrome

In patients with Nutcracker syndrome, treatment of LOV obstruction secondary to LRV compression should focus on relieving anatomical compression rather than embolization. Symptoms in Nutcracker syndrome patients typically include flank pain and microhematuria. Acute narrowing of the LRV, known as the "beak sign," is a significant diagnostic indicator (Fig. 4). A ratio of the narrowed to the dilated portion of the LRV greater than 4.9 (>4:1 normally) is strongly suggestive of the syndrome.[34] The pressure gradient between the LRV and IVC is generally 1 mmHg or less, although evidence regarding the use of pressure gradients in diagnosis is inconclusive. Different studies have indicated a gradient greater than 3 mmHg as indicative of LRV obstruction.[35] While LRV hypertension may not be diagnostic for Nutcracker syndrome, measuring pressure gradients during venography and stenting provides insight into the technical success of the procedure.

The choice between surgical treatment and stenting for Nutcracker syndrome depends on the type of anatomical

anomaly. In anterior Nutcracker syndrome, when the LRV is compressed between the superior mesenteric artery and the aorta, placing a stent may be sufficient (Fig. 4). However, in posterior Nutcracker syndrome, where the LRV is compressed between the vertebra and the aorta, surgical transposition may be required to relieve the obstruction.[36]

Several methods have been described for the surgical treatment of Nutcracker syndrome. Open surgery can be performed in young women with persistent and severe symptoms. Techniques such as LRV transposition, superior mesenteric artery transposition,[37] abdominal aorta transposition, and inferior mesenteric vein-gonadal vein bypass have been reported as successful.[38,39] However, surgical morbidity and the risk of renal ischemia are undesirable outcomes associated with open surgical treatment.

As a result, since stents are not a permanent solution in young patients, surgery should be preferred.

CONCLUSION

CPP is a common and widespread disease in women. The diagnosis and management of PCS remain areas open to new research. The differential diagnosis of CPP is broad and overlaps with other symptoms of PCS. Due to difficulties in recognizing pelvic varices and ovarian vein reflux, PCS continues to be an unrecognized cause of CPP. Transcatheter pelvic vein embolotherapy has replaced the surgical and medical treatment of PCS; however, the lack of robust, multicenter randomized controlled trials on diagnosis, treatment, and outcomes has hindered complete acceptance of this method by relevant experts. In academic and societal settings, vascular surgeons and interventional radiologists should educate and raise awareness among primary care physicians and general practitioners about the signs, symptoms, and treatment of PCS through training seminars. Coordination among primary clinicians, gynecologists, and interventional radiologists is critical for achieving successful outcomes in the treatment of patients with PCS.

Disclosures

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