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DIAGNOSIS AND TREATMENT OF PELVIC VENOUS CONGESTION IN WOMEN

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Abstract: Introduction: Pelvic venous congestion syndrome (PVCS) can be defined as a disorder of the pelvic venous system, i.e. the presence of pelvic venous insufficiency (PVI) that manifests itself with a wide range of symptoms and signs. PVCS affects women of reproductive age and often presents with chronic pelvic pain. Other symptoms include pelvic heaviness, dyspareunia, dysmenorrhea, low back pain, frequent and urgent urination, and signs of dilated vulvar, perineal, gluteal superficial veins or varicose veins of the lower extremities and hemorrhoids. PVCS can be caused by a combination of several factors: genetic predisposition, anatomical abnormalities, hormonal factors, dysfunctional valves, obstruction of venous flow, and damage to the vein walls. Diagnostics: The diagnosis of PVCS remains a major challenge, given the lack of universally accepted criteria in diagnostic imaging modalities. The following imaging methods can be used for diagnosis: ultrasound (US), computed tomography (CT), magnetic resonance imaging (MRI) as non-invasive methods and venography (VG) as an invasive method. Pelvic ultrasound is usually the first-line method. Transcatheter venography remains the gold standard for the diagnosis of PVCS. Treatment: Conservative medical treatment for PVCS is limited due to the lack of data on long-term efficacy. Compression is one of the therapeutic options for conservative treatment. Embolization is recommended for the treatment of PVCS. Clinical improvement after embolization ranges from 47 to 100% in different studies, but future randomized trials are needed to determine clear protocols for the management of embolization. Conclusion: PVCS is a common cause of chronic pelvic pain in women, but due to lack of knowledge, it is often not recognized and remains undiagnosed. In the absence of other causes of PVCS, PVI should also be considered. Additional education of gynecologists for the use of ultrasound in the diagnosis of PVCS is needed, given that ultrasound is the initial imaging method.

Key words: pelvic congestive syndrome, pelvic venous insufficiency, chronic pelvic pain, embolization.

INTRODUCTION

Pelvic venous congestive syndrome (PVCS) can be defined as a disorder of the pelvic venous system, namely the presence of pelvic venous insufficiency (PVI), which manifests through a wide spectrum of symptoms and signs. By definition, the primary cause of this syndrome is pelvic venous insufficiency, indicated by dilation and dysfunction of the ovarian veins, internal iliac veins with their tributaries, as well as venous plexuses.

PVCS affects women of reproductive age and is often associated with chronic pelvic pain lasting at least six months [1]. Pain is frequently described as a typical symptom of PVCS, characterized as chronic, dull, unilateral or bilateral [2]. Other symptoms include a feeling of pelvic

heaviness, dyspareunia, dysmenorrhea, lumbar pain, frequent and urgent urination, and signs of dilated vulvar, perineal, gluteal superficial veins or varicosities of the lower extremities and hemorrhoids [1]. Chronic pelvic pain is not necessary for diagnosis, as in many patients the predominant symptom may be atypical superficial varicosities. In some women, superficial varicose veins may be the only sign of PVI. The prevalence of vulvar varicosities in patients with PVCS ranges from 24–40%. Up to 80% of patients with pelvic venous dilation may exhibit varying degrees of associated venous insufficiency of the lower extremities [2].

The complexity of the problem lies in the fact that different symptoms can occur at the same degree of PVI, just as the same symptoms may appear at different degrees of PVI. Chronic pelvic pain in women can arise from various causes, including endometriosis, adhesions, fibroids, adenomyosis, genital organ prolapse, malignancies, and many other causes; very often, in the absence of an explanation for the pain, chronic pain is attributed to psychosomatic disorders.

Due to the nonspecific symptomatology, PVCS is often unrecognized and underdiagnosed. Factors that exacerbate pelvic pain, as described in the literature, include prolonged periods of standing, walking, or sitting, as well as factors that increase intra-abdominal pressure, such as lifting and pregnancy. Pain generally worsens during the day, as well as before and in the first days of menstruation, and decreases when lying down.

Pain also increases during and after sexual intercourse. Osman et al. reported that dyspareunia due to endometriosis is typically associated with deep penetration, whereas pain caused by PVCS usually worsens with sexual activity, producing a pulsating pain after intercourse [3].

Urinary symptoms may occur in PVCS due to perivesical varicosities, such as bladder irritability, urgency, or dysuria. Other manifestations of PVCS can include rectal discomfort, vulvar swelling, vaginal discharge, persistent genital arousal, and nonspecific gastrointestinal symptoms such as bloating and nausea. Chronic pelvic pain and these additional symptoms negatively affect patients' quality of life, leading to a significantly higher incidence of depression, anxiety, and generalized lethargy in this group. [1].

Anatomy

The pelvic venous system is responsible for returning venous blood from the walls and organs of the pelvis back to the central circulation. The external iliac vein (EIV) primarily drains the lower extremities, whereas the internal iliac vein (IIV) drains the pelvic organs, pelvic walls, gluteal region, and perineum. All veins from the pelvis and lower extremities generally converge into the inferior vena cava (IVC) and proceed to the right atrium. Smaller vessels can vary between individuals, but the major vessels are anatomically consistent.

The ovaries and uterus are drained by both the internal iliac and ovarian veins (OV). The IIV runs slightly medial and posterior to the internal iliac artery, joining the EIV to form the common iliac vein (CIV). Its tributaries are divided into parietal and visceral groups. Parietal tributaries include the superior and inferior gluteal, sciatic, sacral, ascending lumbar, and obturator veins. Visceral tributaries include the internal pudendal, middle hemorrhoidal, and vesicoprostatic plexuses in men, and the uterine, gonadal, and vesicovaginal plexuses in women. Valves are rarely present in the internal iliac veins (10% of cases in the main trunk and 9% in its tributaries).

The ovarian veins drain the pampiniform plexus, mesosalpinx, parametrium, and cervix, forming a rich anastomotic venous network with the paraovarian, uterine, vesical, rectal, and vulvar plexuses. Two or three branches form a single ovarian vein at the level of L4, with the left ovarian vein draining into the left renal vein (LRV), and the right ovarian vein in most women draining

directly at an acute angle into the anterolateral wall of the IVC, below the right renal vein (RRV). In up to 10% of women, the right ovarian vein may drain into the RRV instead of the IVC. Studies have shown that normal ovarian veins have an average diameter of less than 5 mm. Valves are present, mainly in the distal third of the vein. Valves are absent in 15% of left OVs and 6% of right OVs. When present, valves are incompetent in 40% of cases on the left and 35% on the right [1].

The left-sided predominance of PVCS can be explained by these anatomical features, as well as by the fact that the left ovarian vein is longer than the right, which impedes drainage in the upright position. Additionally, the left ovarian vein may be compressed by the sigmoid colon during constipation. Nonetheless, it should be noted that pelvic venous drainage is complex and venous anatomy can vary among patients. [5].

Etiology and Pathophysiology

The etiology of Pelvic Venous Congestive Syndrome (PVCS) remains poorly understood, and it is considered that multiple factors contribute to its pathogenesis. Pelvic venous insufficiency (PVI) can result from a combination of factors, including genetic predisposition, anatomical abnormalities, hormonal influences, valve dysfunction, obstruction of venous outflow by adjacent structures, and damage to the vein walls.

Many studies have indicated a connection between varicose veins and genetics, with some reports suggesting that up to 50% of varicose veins may have a genetic component. Congenital abnormalities of the vein wall may also exist, causing dilation and subsequent valve dysfunction.

Hormonal factors play a significant role in the development of PVCS. Estrogen increases nitric oxide production, resulting in venous dilation and weakening, which increases stress on the valves. Progesterone also contributes to weakening venous valves in the pelvic veins. Pregnancy is considered one of the major risk factors for PVCS due to increased circulatory volume in the pelvic veins, elevated flow through the ovarian veins (up to 60-fold), and increased intra-abdominal pressure caused by the gravid uterus, which further exacerbates ovarian vein reflux. Estradiol-induced venous dilation during pregnancy increases valve stress, ultimately leading to chronic venous insufficiency. The therapeutic use of vasoconstrictors has shown some efficacy in alleviating PVCS symptoms by increasing venous flow through compression, supporting the hormonal theory. Additionally, symptoms typically resolve completely after menopause.

Although valves are generally present in the distal segments of the main ovarian vein trunks (about 85% of cases), they are incompetent in 40% of cases on the left and 35% on the right ovarian vein [6]. The mechanisms by which venous valves become incompetent are not precisely defined. On one hand, there may be a primary change in valve structure leading to leakage, progressive reflux, and subsequent vein dilation. On the other hand, a primary structural abnormality in the vein wall may cause venous dilation, which distorts the valves and renders them nonfunctional [5].

PVCS can also result from obstruction of blood outflow from the ovarian veins. The most common cause of obstruction is compression of the left renal vein between the superior mesenteric artery and the abdominal aorta, known as Nutcracker syndrome. May-Thurner syndrome is another cause of obstruction, where the left common iliac vein is compressed by the right common iliac artery. This compression can sometimes lead to deep vein thrombosis. Abnormal uterine positioning with ovarian torsion can rarely cause obstruction. Additionally, endometriotic lesions, fibroids, postsurgical or infectious adhesions, hypervascular pelvic tumors, gestational trophoblastic neoplasms, ovarian tumors, and mesenteric tumors can also compress veins. Regardless of etiology, the final result of obstruction is the development of numerous refluxing varicosities, cross-venous collaterals, and painful venous congestion. [1].

Prolonged venous dilation in varicose veins in PVI induces inflammation, which further damages the vessel walls, causing additional weakening and dilation of the veins and increasing reflux. Venous hypertension enhances the expression of matrix metalloproteinases, promoting the degradation of collagen, elastin, and endothelium, thereby impairing vascular tone regulation [7]. This process leads to further endothelial damage and inflammation.

Although venous distension generally should not cause pain, congestion and stretching of the ovarian and pelvic veins can activate pain receptors within the venous walls. Venous dilation leads to activation of nociceptors connected to C-afferent fibers, which have slow conduction velocities and mediate the sensation of dull, burning pain [2].

Venous dilation and inflammation also trigger the release of substance P and calcitonin gene-related peptide (CGRP), which further dilate the vessels and increase vascular wall permeability. Simultaneously, cytokines are released, enhancing inflammation and nociceptor activity [8].

Supporting evidence that dilation of pelvic veins activates pain receptors comes from clinical observations that gabapentin and amitriptyline—standard treatments for neuropathic pain—are more effective than opioids or nonsteroidal analgesics in alleviating pelvic pain. [9].

Diagnosis

The diagnosis of Pelvic Venous Congestion Syndrome (PVCS) remains challenging due to the lack of universally accepted criteria in imaging modalities and its heterogeneous presentation. Patients with PVCS typically first consult a general practitioner and/or gynecologist in primary care before being referred for further investigations and specialist consultation. Once more common causes of chronic pelvic pain—including endometriosis, pelvic inflammatory disease, interstitial cystitis, and fibroids—are excluded, the first diagnostic step is usually pelvic ultrasound (US) to visualize the blood vessels [1].

Various nomenclatures have been used to describe the diverse clinical presentation of pelvic venous insufficiency. A step toward better understanding of this condition is the recently established "Symptoms-Varices-Pathophysiology" (SVP) classification for assessing pelvic venous disorders, proposed by the International Working Group convened by the American Vein and Lymphatic Society. Although this classification may seem complex for routine clinical practice, it could help in more precise diagnosis, better selection of patients for therapeutic intervention, and generation of homogeneous samples for future research [10].

Imaging methods for diagnosis include non-invasive techniques such as ultrasound (US), computed tomography (CT), and magnetic resonance imaging (MRI), as well as invasive venography (VG).

Pelvic ultrasound is generally the first-line method for patients suspected of having PVCS. Ultrasound assesses pelvic anatomy and, using Doppler modes, allows visualization of blood vessels and evaluation of blood flow. Ultrasound can be performed transvaginally, transabdominally, or transperineally. Transvaginal ultrasound (TVUS) better excludes other gynecological conditions, provides improved visualization of pelvic venous plexuses, and allows dynamic assessment of blood flow through tortuous pelvic veins. Transabdominal and transperineal ultrasound, on the other hand, allow better visualization of longer vessels, such as the ovarian veins. Ultrasound can also be performed with the patient standing or performing the Valsalva maneuver, which accentuates venous filling and improves visualization of pelvic varices [2,5].

Ultrasound parameters that can be evaluated include: internal diameter of the largest pelvic vein (right and left), maximum diameter of the largest venous plexus (right and left), dilation and low velocity or reversed flow in the ovarian veins during Valsalva, enlargement of arcuate veins (vv. arcuate) in the myometrium, presence of crossed veins, maximum diameter of crossed veins in the myometrium, uterine volume, volume of the right and left ovary, and presence of polycystic ovaries (PCO).

The threshold for ovarian vein dilatation remains controversial, with different authors defining it between 5 and 8 mm. According to Park et al., the positive predictive value for a threshold diameter of the left ovarian vein was 71.2% at 5 mm, 83.3% at 6 mm, 81.8% at 7 mm, and 75.8% at 8 mm. [11].

Rocio Garcia-Jimenez et al. designed an ultrasound predictive model for identifying PVCS based on the presence of a pelvic vein or venous plexus measuring 8 mm or more, identified via transvaginal ultrasound (TVUS). This model was able to predict 79% of patients with PVCS, with good sensitivity (86.05%) and specificity (66.67%). Given its simplicity, relying on a single parameter, this model appears to be a feasible alternative compared to previously proposed predictive models [12]. Labropoulos et al. (2017) reported on the standardization and technique of ultrasound application in PVCS diagnosis using a transabdominal approach [13].

Computed tomography (CT) allows imaging in cross-sections and precise anatomical visualization. Magnetic resonance imaging (MRI) of the pelvis provides excellent image quality and high resolution, and unlike CT, does not involve radiation, making it safer for women of reproductive age. Diagnostic criteria for CT and MRI proposed by Coakley et al. include the presence of at least four ipsilateral tortuous parauterine veins of varying calibers, at least one vein with a diameter >4 mm, or an ovarian vein diameter >8 mm [14]. Both contrast-enhanced and non-contrast CT and MRI provide good sensitivity in diagnosing venous insufficiency. Osman et al. reported a sensitivity of 94.8% for CT and 96% for MRI [15].

Flow information in the veins can be obtained using MRI techniques such as phase-contrast velocity mapping (Phase Contrast MRI) or Time-Resolved MRA, which provide accurate information on whether flow in the ovarian vein is antegrade or retrograde. Yang et al. compared Time-Resolved MRA with conventional venography, showing that Time-Resolved MRA is an excellent non-invasive diagnostic tool for pelvic venous insufficiency, with no significant difference compared to conventional venography in determining the level of ovarian venous reflux [16].

Laparoscopy is not effective in detecting pelvic varices and is negative in 80–90% of PVCS patients because it requires Trendelenburg positioning and CO_2 insufflation, which increases intra-abdominal pressure and compresses (often masking) pelvic varices. However, laparoscopy allows visualization of other causes of chronic pelvic pain [2].

Transcatheter venography remains the gold standard for PVCS diagnosis. As an invasive procedure, it should be reserved for patients whose non-invasive imaging findings are inconclusive or for those planned for interventional embolization therapy [15]. Catheter-directed venography is performed by inserting a catheter via the jugular, brachial, or femoral vein to the renal, ovarian, common iliac, and internal iliac veins, followed by contrast injection. This technique allows measurement of pressure gradients, providing valuable information about the severity of pelvic venous pathology, as well as morphological assessment of the veins. The procedure is usually performed on an outpatient basis without hospitalization. A key advantage is that treatment can be performed in the same session. The main protocol begins with catheterization of the left renal vein, simultaneously measuring the pressure gradient to assess Nutcracker syndrome. The catheter is then

moved to the left iliac vein to evaluate May-Thurner syndrome. Subsequently, the ovarian veins are assessed, followed by the internal iliac veins [17].

Venographic diagnostic criteria for incompetent pelvic veins include an ovarian vein diameter >10 mm; congestion of ovarian, pelvic, vulvovaginal veins; and retrograde filling. [5].

Treatment of Pelvic Venous Congestion Syndrome (PVCS)

Conservative (Medical) Treatment

Medical management of PVCS is limited, as long-term efficacy data are lacking. Hormonal therapies that inhibit ovarian function, such as medroxyprogesterone acetate (MPA) and gonadotropin-releasing hormone (GnRH) agonists, have shown some efficacy, but their use is associated with multiple side effects. Dihydroergotamine has demonstrated temporary pain relief, but its effects are transient and accompanied by adverse events. Nonsteroidal anti-inflammatory drugs (NSAIDs) may alleviate symptoms but do not address the underlying condition [2].

Micronized purified flavonoid fraction (MPFF), a venoactive drug, has been investigated by Simsek et al., Tsukanov et al., and Gavrilov et al. All studies showed that 1000 mg of MPFF daily reduces the severity of pelvic symptoms such as pain, heaviness, and vulvar swelling due to pelvic varices [18,19,20]. Gavrilov et al. also demonstrated that doubling the dose (1000 mg twice daily) in the first month accelerates symptom resolution [21].

Compression garments are another conservative treatment option. In a study by Gavrilov et al., wearing compression shorts for 2 weeks reduced chronic pelvic pain, dyspareunia, and discomfort in 81.3% of patients. They also reduced leg heaviness and swelling. However, there was no effect on clinical symptoms of vulvar varices. Elastic stockings did not show clinical improvement or enhanced venous drainage [22].

Non-conservative treatment includes surgical intervention and minimally invasive endovascular therapy. Earlier surgical approaches, such as left ovarian vein resection or hysterectomy with unilateral or bilateral adnexectomy, were associated with high recurrence rates, residual pain, longer hospital stays, and higher morbidity compared to endovascular approaches.

In a randomized controlled trial by Chung et al., ovarian vein embolization was significantly more effective than hysterectomy with unilateral or bilateral salpingo-oophorectomy 12 months post-treatment [23].

The first report of embolization as a treatment for PVCS was published by Edwards in 1993. According to the Society for Vascular Surgery and the American Venous Forum, embolization is recommended with a 2B level of evidence for PVCS treatment [17]. Embolization is usually performed after unsuccessful medical therapy but is increasingly used as a primary treatment. Indications generally include women with chronic pelvic pain and/or dyspareunia, severe labial or perineal varices, or lower limb varices, with confirmed pelvic venous insufficiency, typically verified by venography.

There is no standardized protocol for endovascular PVCS treatment. Techniques, vascular access sites, and embolic materials (sclerosants, coils, plugs) vary across publications [2]. Clinical improvement after embolization ranges from 47% to 100% in different studies [2]. The debate continues over whether unilateral or bilateral embolization should be performed; some clinicians perform only unilateral ovarian vein embolization, while others perform complete bilateral embolization [2].

If a hemodynamically significant stenosis is present, it should be corrected. This may include stenting the left common iliac vein in May-Thurner syndrome or the left renal vein in Nutcracker syndrome, as well as any other catheter-accessible site of pelvic venous obstruction. Stenting of the left renal vein carries a high risk of migration to the vena cava and heart due to the vein's short length and diameter changes during posture changes or Valsalva maneuvers [24]. The main risk of endovascular stenting failure is stent occlusion. Duration of post-procedural antithrombotic therapy varies between studies [2].

Complications are generally rare and minor, including allergic reactions, puncture site hematoma, local thrombophlebitis, vessel perforation, embolic migration, and recurrence of symptoms. PVCS symptoms may recur after ovarian vein embolization due to reflux from other venous tributaries. Post-embolization syndrome occurs in approximately 20% of patients and is characterized by increased pelvic pain, low-grade fever, and tenderness around the embolized vein, usually managed with NSAIDs. A potentially serious complication is migration of the coil or vascular plug to the pulmonary artery, which is typically successfully retrieved endovascularly. [2].

CONCLUSION

Pelvic venous congestion syndrome (PVCS) is a common cause of chronic pelvic pain in women, but due to insufficient awareness, this syndrome is often unrecognized and remains undiagnosed. The symptoms can be nonspecific and are frequently underestimated. Diagnosing PVCS is very challenging and complex, yet equally important for implementing appropriate and targeted treatment. Globally accepted diagnostic algorithms that allow for an objective diagnosis are still lacking. Considering that most patients with chronic pelvic pain initially consult general practitioners or gynecologists, it is important to always consider this syndrome in the absence of other causes. Additional education of gynecologists in the use of ultrasound for diagnosing pelvic venous insufficiency (PVI) and familiarity with diagnostic criteria would also be beneficial, as ultrasound is the first-line method in PVCS diagnosis. There is also a need for validated imaging diagnostic criteria.

Regarding treatment, endovascular embolization appears to be an effective method; however, future randomized studies are needed to establish clear protocols for managing embolization.

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