Přehledový článek | Review article

Pelvic venous disease

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Panvové venózne ochorenie je po endometrióze druhou najčastejšou príčinou chronickej panvovej bolesti a predstavuje príčinu 10 – 20 % gynekologických vyšetrení. U žien je jeho hlavnou príčinou reflux a inkompetencia ľavej ovariálnej žily. U mužov sa ochorenie panvových žíl môže prejaviť ako varikokéla a často je dôsledkom venóznej obštrukcie v iliokaválnej oblasti. Tento článok poskytuje prehľad symptómov, prejavov, diagnostiky a liečby najčastejších príčin panvového venózneho ochorenia.

ABSTRACT

Pelvic venous disease is the second most common cause of chronic pelvic pain in women after endometriosis and accounts for 10–20% of gynecological consultations. Its main cause in women is reflux and incompetence of the left ovarian vein. In men, pelvic venous disease can manifest as varicocele and is often the result of venous obstruction in the iliocaval region. This article provides an overview of the symptoms, signs, diagnosis, and treatment of the most common causes of pelvic venous disease.

Introduction

Pelvic venous disease is a spectrum of symptoms and signs arising from the pelvic veins, namely, gonadal veins, internal iliac veins, and their tributaries, pelvic venous plexuses, and their drainage pathways including the left renal vein, common iliac veins, inferior vena cava, and pelvic escape points.[1–4]

The disease spectrum includes symptoms arising from reflux, most commonly from the gonadal and internal iliac veins, and compression, usually of the left renal or left common iliac vein.[4] Other causes of pelvic venous disease include inferior vena cava thrombosis/aplasia or iliac vein obstruction. The hemodynamic consequence is venous hypertension, which has various clinical manifestations, such as left flank/abdominal pain and hematuria (symptoms typical for uncollateralized left renal vein compression), chronic pelvic pain (typically associated with primary reflux/incompetence of the ovarian/internal iliac veins), venous claudication (symptoms typical for obstruction of the common iliac veins or inferior vena cava), and symptomatic vulvar, perineal and lower extremity varices in atypical or typical distribution.[4] Similar symptoms may arise from different causes (e.g., chronic pelvic pain may arise from primary reflux in the left ovarian vein, left common iliac vein compression, or left renal vein compression), and a single anatomic abnormality may lead to different symptoms (e.g., left renal vein compression may be asymptomatic, associated with left flank/abdominal pain and hematuria, or chronic pelvic pain). This can lead to diagnostic and therapeutic errors and suboptimal therapeutic outcomes.[4]

The American Vein and Lymphatic Society International Working Group on Pelvic Venous Disorders has proposed a classification of the umbrella term "pelvic venous disease".[4] This classification is based on dominant symptoms, area of varicose veins, its pathophysiology and considers the anatomical zones of the abdomen and pelvis. The anatomical zones of the abdomen and pelvis are arranged in

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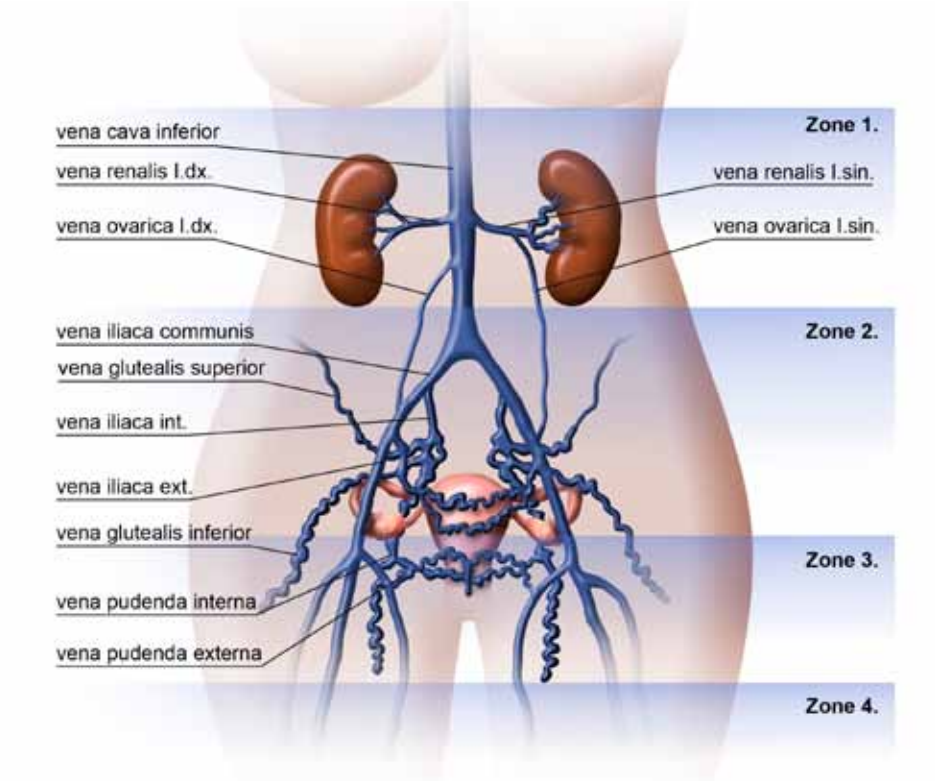


Fig. 1 – Anatomical zones of the abdomen and pelvis in relation to the symptoms–varices–pathophysiology (SVP) classification. It includes symptoms and varices associated with zones 1 (left renal vein), 2 (gonadal vein, internal iliac veins, and venous plexuses), 3 (extrapelvic veins of pelvic origin arising in the pelvis and refluxing through the pelvic escape points to the genitalia, perineum area and lower extremity veins), and 4 (superficial and deep veins of the lower limbs classified according to CEAP classification). Ext. – externa; int. – interna. Modified from 4 (an open access article published under the CC BY-NC-ND license) and drawn by Ivan Hořejší.

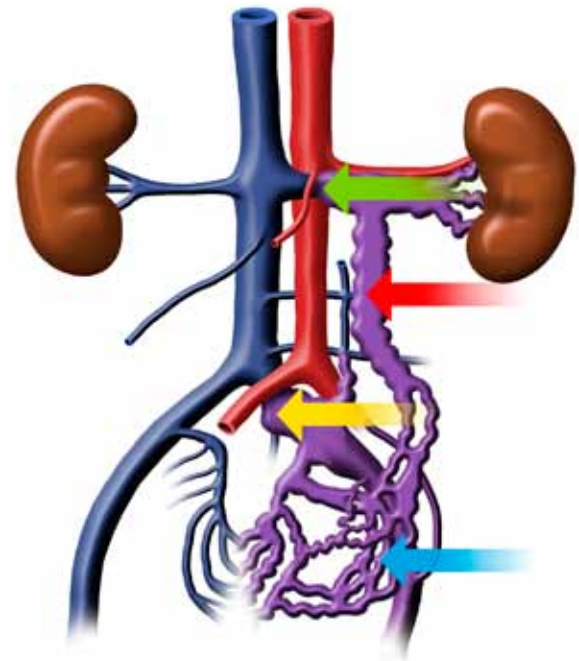


Fig. 2 – Main causes of pelvic venous disease. Purple color shows areas of venous hypertension. Green arrow: left renal vein compression between the aorta and the superior mesenteric artery; red arrow: reflux and incompetence of the left ovarian vein; yellow arrow: left common iliac vein compression by overlying right common iliac artery; blue arrow: reflux in left internal iliac vein. Modified according to 7, 9 and drawn by Ivan Hořejší.

descending order and include symptoms and varices associated with the left renal vein (zone 1); gonadal vein, internal iliac vein, and pelvic venous plexuses (zone 2) and extrapelvic veins of pelvic origin (zone 3) (**Fig. 1**). A schematic image of the causes of pelvic venous disease is shown in **Figure 2** and **Table 1** shows the anatomical zones included in the symptoms–varices–pathophysiology (SVP) classification.

Anatomy and clinical presentation of pelvic venous disease

The pelvic venous system is complex. The pelvic venous system parietal tributaries run alongside the arteries and could have various anatomical variations.[5,6] The visceral tributaries are variable and are formed by avalvular venous plexuses that drain partly into the avalvular internal iliac veins and partly into the gonadal veins.[2] The venous plexuses are interconnected, which results in a variability of symptoms. They consist of the rectal plexus (partially draining into the portal vein), the vesical plexus, the prostatic plexus in men, and the uterine and vaginal plexus in women, often collectively called the uterovaginal plexus.[7] The anatomy of the pelvic veins and ovarian veins is shown in **Figure 3**.

Most patients with pelvic venous disease are asymptomatic or have minor pelvic symptoms.[3] Asymptomatic pelvic varices have been reported in 38–47% of patients

Table 1 – Classification of pelvic venous disease according to the American Vein and Lymphatic Society International Working Group on Pelvic Venous Disorders[4]

(S) Symptoms	(V) Varices	(P) Pathophysiology
S0: no symptoms	**V0**: nonabdominal, pelvic, or extrapelvic varices of pelvic origin	**Anatomy** Inferior vena cava (IVC) Left renal vein (LRV) Gonadal vein[1] (GV) Common iliac vein[1] (CIV) External iliac vein[1] (EIC) Internal iliac vein[1] (IIV) Pelvic escape vein[2] (PELV)
S1: renal symptoms of venous origin	**V1**: renal hilar varices	
S2: chronic pelvic pain of venous origin	**V2**: pelvic varices	
S3: extrapelvic symptoms of venous origin a. Localized symptoms associated with external genitalia veins (vulva and scrotum) b. Localized symptoms associated with pelvic origin veins of the leg c. Venous claudications	**V3**: extrapelvic varices of pelvic origin a. Genital varices (vulvar varices and varicocele) b. Varicose veins of pelvic origin extending to the thigh	**Hemodynamics:** Obstruction: thrombotic/non-thrombotic (O) Reflux: thrombotic/non-thrombotic (R) **Etiology** Thrombotic (T) Non-thrombotic: reflux, proximal obstruction, or external compression (NT) Congenital: vascular malformations (C)

[1] Further subdivision into left, right, and bilateral. [2] Includes inguinal, obturator, pudendal, and/or gluteal points.

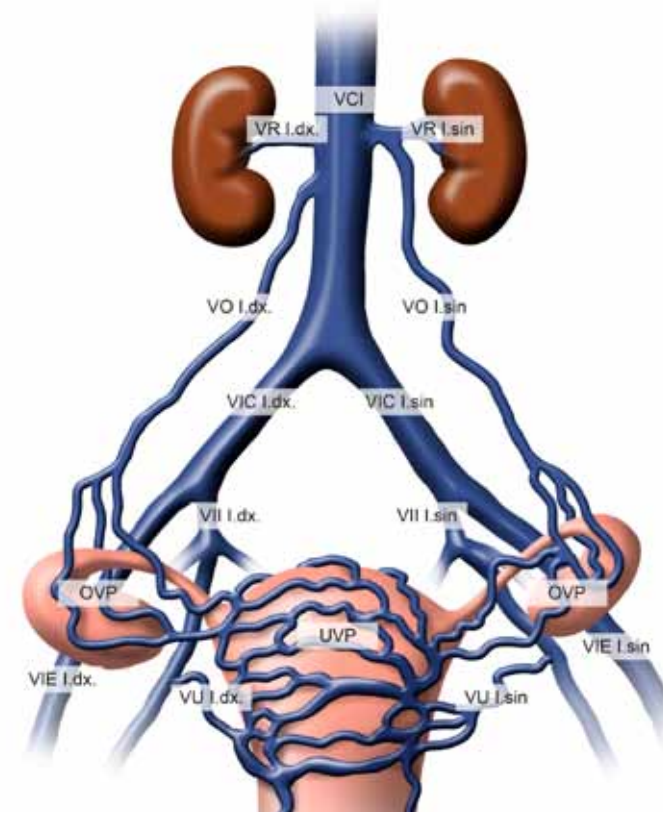


Fig. 3 – Pelvic and ovarian venous anatomy and venous plexuses. The uterine venous plexus (UVP) is drained by the right (VU l.dx.) and left uterine vein (VU l.sin.) into the internal iliac veins (VII), which have anastomoses with the external iliac veins (VIE), and the venous flow is directed to the common iliac veins (VIC). In the upper part, the UVP forms anastomoses with the ovarian venous plexus (OVP), which is drained into the ovarian veins (VO). Blood from the VO l.sin. is drained by the left renal vein (VR l.sin.), whereas blood from the right ovarian vein (VO l.dx) is drained by the inferior vena cava (VCI); however, in 10% of cases, it can drain into the right renal vein. The venous flow from the rectal, and vesicular venous plexuses is drained mainly into the VII. Modified according to 7, 9.

on computed tomography or magnetic resonance imaging.[5] Left iliac vein compression (May-Thurner syndrome) may be present in one-quarter to one-third of the population. Similarly, left renal vein (LRV) compression (nutcracker phenomenon) is common in healthy individuals.[8] Therefore, interpretation of anatomical findings is important in the context of the patient's symptoms and signs.

According to the pathophysiology and dominant clinical picture, pelvic venous disease can be divided into four main types, which can be combined with each other.

Pelvic venous disease associated with left renal vein compression

The LRV is located between the aorta and the superior mesenteric artery (SMA), and the narrow space between these structures leads to compression of the LRV (nutcracker phenomenon/syndrome). Causes of compression include a sharp angle between the SMA and the aorta, often due to loss of retroperitoneal fat during weight loss, compression due to lymphadenopathy, malignancy, pregnancy, severe lordosis, or renal ptosis. Posterior compression (posterior nutcracker phenomenon/syndrome) results from LRV compression between the aorta and the vertebral body. Patients may be asymptomatic (nutcracker phenomenon). Collateralized compression (via the perirenal collaterals, lumbar plexus, and left gonadal vein) causes hypertension in the pelvic venous plexuses. If the compression is not collateralized, symptoms are related to renal venous hypertension (micro- or macrohematuria due to rupture of peripelvic and periureteral varices and/or abdominal/left flank pain). Orthostatic proteinuria is explained by nephron damage due to venous hypertension.

Pelvic venous disease associated with reflux and incompetence of the ovarian vein

According to the American College of Gynecology and Obstetrics, chronic pelvic pain is noncyclical pain lasting at least 6 months, localized in the pelvis, anterior abdomi-

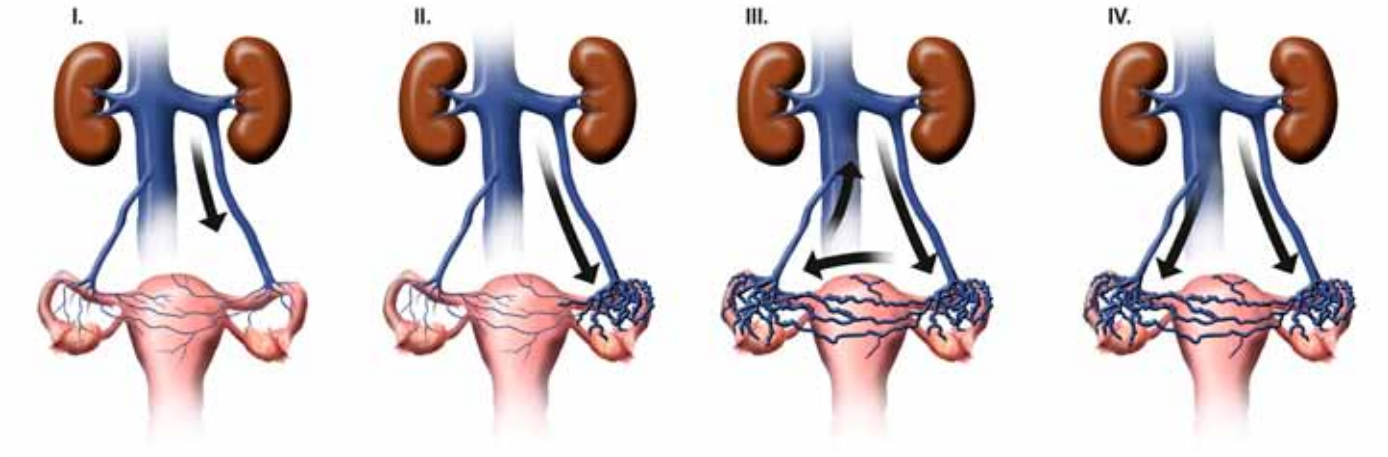


Fig. 4 – Retrograde flow in the vena ovarian vein (modified from 16).
I. Retrograde flow in the left ovarian vein does not extend into the parauterine veins.
II. Retrograde flow in the left ovarian vein with extension into the ipsilateral parauterine veins.
III. Retrograde flow in the left ovarian vein with extension into the contralateral parauterine veins.
IV. Retrograde flow in both ovarian veins.

nal wall, umbilical region, or disabling pain in the lumbosacral region requiring medical assistance.[10] Its most common vascular cause in women is reflux and incompetence of the ovarian vein spreading to the pelvic venous plexuses. Chronic pelvic pain, a sensation of dull heaviness or fullness, has variable intensity and is usually localized asymmetrically.[10] Acute severe pain may also occur.[1] Pain typically worsens during menstruation, pregnancy, with prolonged standing, and at the end of the day.[1] Postcoital pain radiates to the anus and takes several hours to resolve.[5,12] Dysmenorrhea, urological problems (dysuria, pollakisuria), rectal discomfort, and ovarian tenderness may also be present. Associated symptoms are nonspecific and include headache, back pain, bloating, nausea, and abdominal cramping.[9,11] Symptoms are often accompanied by depression and anxiety.[5] The incidence of pelvic pain increases with the number of pregnancies, and after menopause symptoms weaken or even disappear.[9]

Predisposing factors include uterine retroversion, multiple pregnancies and family history of varices. More frequent reflux in the left ovarian vein is a consequence of its greater length and perpendicular flow into the LRV, in contrast to the right ovarian vein which is shorter, and enters the inferior vena cava at an oblique angle.[1,2] Absent valves have been documented in 13–15% of women in the upper part of the left ovarian vein and in 6% of women in the upper part of the right ovarian vein.[13–15] The degrees of the retrograde blood flow extension originating from the left ovarian vein into pelvic veins are shown in **Figure 4**. Estrogens lead to the dilation of ovarian veins, and a negative effect of progesterone on venous valves has been documented. During menstruation, the ovarian veins are exposed to significantly higher estrogen concentrations compared with the estrogen concentration in the peripheral blood.[12] The increased risk of chronic pelvic pain in multiparous women may be attributed to hormonal changes during pregnancy, increased capacity, dilation, and incompetence of the pelvic veins, as well as impaired venous drainage due to uterine pressure.

Pelvic venous disease associated with proximal venous compression and obstruction

Compression of the left common iliac vein by overlying right common iliac artery against the lumbar vertebrae (May-Thurner syndrome) cause reflux and retrograde blood flow in the ipsilateral left internal iliac vein with venous drainage into the pelvic venous plexuses and contralateral iliac veins. There are also variants of compression of the right common iliac vein or compression of the inferior vena cava (in the case of a high aortic bifurcation).[1,13,17,18] In addition to chronic pelvic pain, symptoms of compression/obstruction in iliocaval region also include venous claudication, limb swelling, varicose veins, and leg ulcers.[16]

Pelvic venous disease associated with extrapelvic varices of pelvic origin

Pelvic venous hypertension may cause vulvar and perineal varices and primary or recurrent varices of the lower limbs, mainly in the gluteal and posteromedial region of the thigh (**Fig. 5**).[3] About one third of women with pelvic venous disease has vulvar varices, and up to 90% of women have leg varices in a typical or atypical location.[19] These varices are the result of pelvic venous reflux propagation through pelvic escape points. Franceschi et al. described six pelvic escape points including the perineal point: reflux through the pudendal vein; obturator point: reflux through the obturator vein; upper gluteal point: reflux through the superior gluteal vein; lower gluteal point: reflux through the lower gluteal vein; inguinal point: reflux through the veins of the round ligament of the uterus and clitoral point: reflux through the dorsal vein of the clitoris.[20] Vulvar varicosities are mostly caused by the reflux through the pudendal veins, while perineal, gluteal and posterior thigh varices are mostly caused by internal iliac vein reflux.[3] There is a strong association between hemorrhoids and internal iliac vein reflux.[3]

Table 2 – Findings supporting the left renal vein compression at the aortomesenteric angle[8,11,21,22]

Modality	Finding
Ultrasonography	Ratio between the PSV in the aortomesenteric angle and PSV in the renal hilum >4–5. (The ratio is higher in the standing position.)
CT-venography MR-venography	Ratio between renal vein diameter in the renal hilum and its diameter in the aortomesenteric area ≥4.9. Beak sign on axial images (sudden narrowing or interruption of the LRV in the SMA area). Left renal vein beak angle (angle of widening after narrowing at the aortomesenteric angle) >32°. Aortomesenteric angle <35°[21], <41°.[22] Dorsolateral torsion of the left kidney. Signs of pelvic congestion and dilatation of the left gonadal vein.
Catheter venography and IVUS	Renocaval gradient >3 mmHg. Renocaval gradient >1 and ≤3 mmHg with collateral veins.

IVUS – intravascular ultrasonography; LRV – left renal vein; PSV – peak systolic velocity; SMA – superior mesenteric artery.

Diagnosis of pelvic venous disease

Before diagnosing pelvic venous disease as a cause of chronic pelvic pain, all alternative pathologies should be ruled out. Differential diagnosis includes diseases of the urinary tract, digestive tract, musculoskeletal system, neurological disorders, gynecological problems, and mental health disorders. Ultrasonography (US) has limited sensitivity, lacks a method for imaging pelvic venous reflux, and cannot visualize venous valves. In addition, definitive diagnostic criteria for subtypes of pelvic venous disease are lacking, and radiological findings in symptomatic and asymptomatic patients often overlap.

Diagnosis of pelvic venous disease arising in zone 1 (renal region)

The diagnostic options are shown in **Table 2**. Patients should be adequately hydrated, as insufficient hydration may lead to overestimation of the degree of LRV compression.[6] US determines the ratio between the peak systolic velocity (PSV) in the LRV at the aortomesenteric angle and the PSV in the LRV at the renal hilum. The angle between the aorta and the SMA is determined from CT venography (CTV) and MR venography (MRV) (normal value 45–90°).[21] As the absolute diameters of the LRV vary, the ratio between the renal hilum LRV diameter and its diameter in the aortomesenteric segment is used.[1] Retrograde venography with/without intravascular ultrasonography (IVUS) provides information about the pressure gradient, collaterals veins, flow pattern and left gonadal vein. Normal pressure gradient between the LRV and inferior vena cava is <1 mmHg.[16] However, even asymptomatic patients may have renocaval pressure gradients ≥ 3 mmHg, and symptomatic patients may have a pressure gradient < 3 mmHg after collateral circulation is established. All examinations should be conducted in the left lateral position, as the supine position may lead to false-positive results. Patients examined with IVUS had a 55% significant LRV stenosis when examination was performed in the supine position compared with a 10% stenosis when examination was carried out in the left lateral position.[2] Findings supporting LRV compression are shown in **Figures 6, 7** and **8**. CTV findings of the posterior LRV vein compression between the aorta and the vertebral body shown in **Figure 9**. **Figures 10** and **11** show US findings of LRV vein

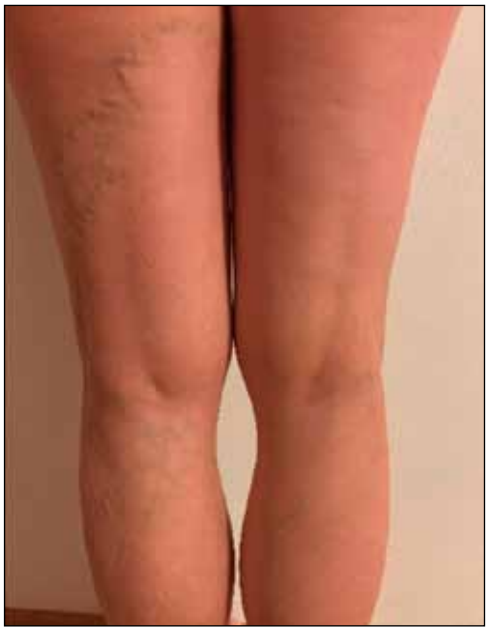


Fig. 5 – Atypical left posterior thigh varices due to pelvic reflux propagation through pelvic escape points.

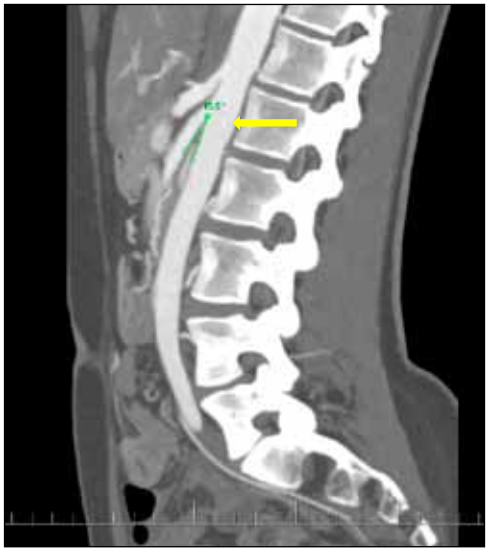


Fig. 6 – CT-venography findings of the left renal vein compression. Aortomesenteric angle = 15° (archive of the VÚSCH).

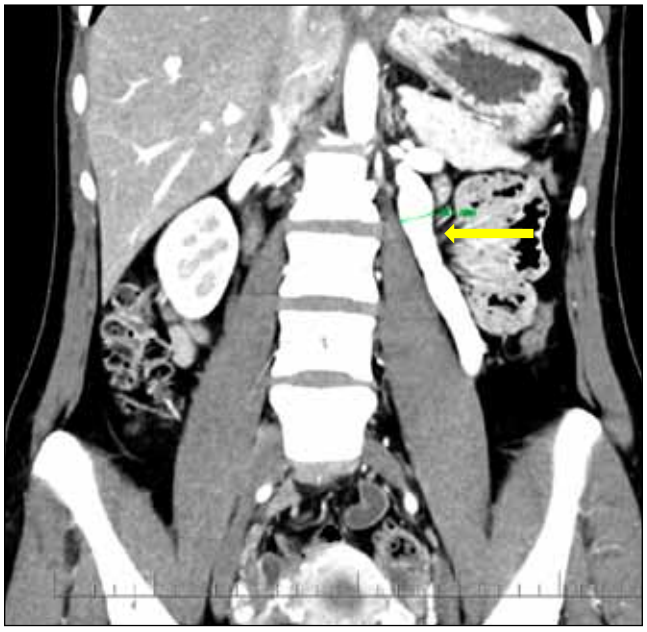


Fig. 7 – CT-venography findings of the left renal vein compression with diameter of dilated left ovarian vein = 16.1 mm (archive of the VÚSCH).

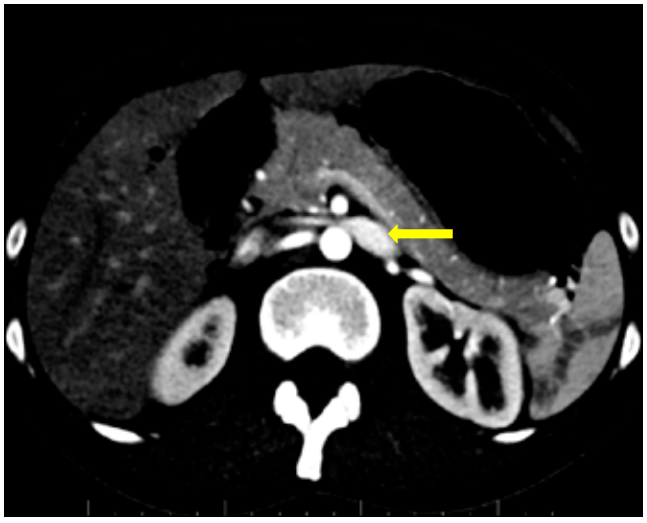
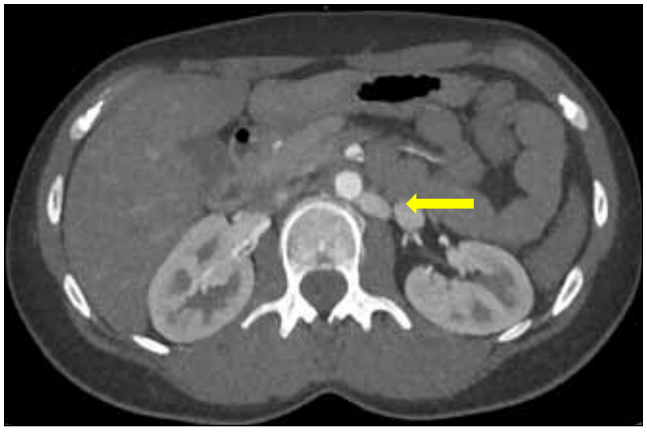


Fig. 8 – CT-venography findings of the left renal vein compression. Beak sign of the left renal vein (archive of the VÚSCH).



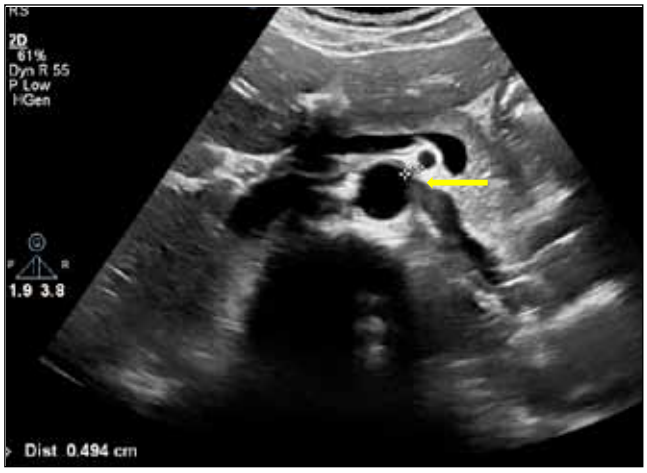


Fig. 10 – US findings of suspected left renal vein compression in supine examination (yellow arrow indicates suspected beak sign of the left renal vein). The distance between the aorta and the superior mesenteric artery is marked (archive of the VÚSCH).

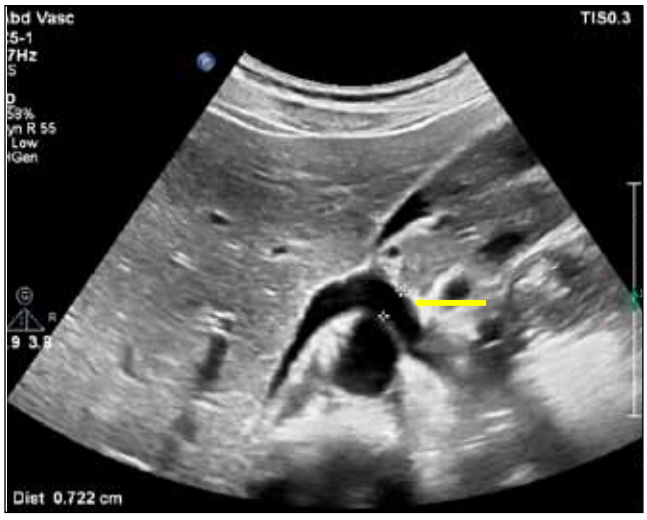


Fig. 11 – US findings of suspected left renal vein compression when the examination is performed in the left lateral position (yellow arrow indicates the absence of left renal vein compression). The diameter of the left renal vein is marked (archive of the VÚSCH).

compression when the examination is performed in the supine and in the left lateral position.

Diagnosis of pelvic venous disease arising in zone 2 (gonadal veins, internal iliac veins, and pelvic venous plexuses)

Diagnostic findings supporting pelvic venous disease arising in zone 2 are listed in **Table 3**. Transvaginal US allows better visualization of the pelvic veins and is not affected by obesity and flatulence.[3] Reflux in the pelvic and ovarian veins during the Valsalva maneuver is examined in the reverse Trendelenburg and standing position.[12] An ovarian vein diameter of 6 mm on transabdominal US is associated with a 96% positive predictive value for pelvic varices.[23,24]

Fig. 9 – CT-venography findings of the posterior left renal vein compression between the aorta and the vertebral body (posterior nutcracker phenomenon/syndrome) (archive of the VÚSCH).

Table 3 – Findings supporting the diagnosis of pelvic venous disease arising in zone 2[1,3,13,16,29,30]

Modality	Finding
Ultrasonography	Ovarian vein diameter >6 mm. Diameter of tortuous pelvic veins >5 mm. Slow pelvic vein/ovarian vein blood flow ≤3 cm/s or reversed caudal flow. Dilated arcuate veins in the myometrium communicating between the bilateral pelvic varices.
CT-venography **MR-venography**	At least four ipsilateral tortuous parauterine veins of different calibers, at least one with a diameter >4 mm or an ovarian vein diameter >8 mm.
Catheter venography	Ovarian vein diameter >6 mm with proven reflux. Contrast retention >20 s. Engorgement of the pelvic venous plexuses and/or opacification of the ipsilateral (or contralateral) internal iliac vein. Filling of vulvovaginal and femoral varices.

The sensitivity of MRV, preferred for its absence of ionizing radiation, is higher than US, but lower than catheter venography.[12] MRV and CTV should be performed in the reverse Trendelenburg position, as the results may be underestimated when examined in the supine position. CTV is not the primary imaging modality in women of reproductive potential; but it can exclude other pelvic pathologies and compressive syndromes (including LRV compression and left common iliac vein compression).[12]

Catheter venography (selective ovarian and iliac venography) during the Valsalva maneuver and in the reverse Trendelenburg position is performed in patients with a strong clinical history, inconclusive results of noninvasive investigations, and before endovascular treatment.[25] Beard's criteria include maximum ovarian vein diameter (normal < 5 mm; mildly dilated = 5–8 mm; severely dilated > 8 mm); time to resolution of contrast (0, 20, and 40 s) and degree of perfusion (normal = veins are small and straight; moderate = veins are tortuous; severe = veins are very tortuous and wide). Each criterion is scored from 1 to 3 points. A final score > 5 points determines the diagnosis of pelvic venous disease with 91% sensitivity and 89% specificity.[26]

If left common iliac vein compression by the right common iliac artery is suspected, a sufficiently hydrated patient should be examined in a semi-sitting position at 45°.[17,18] Suspicion arises when the ratio of PSV at the site of the stenosis and proximal to the compression is > 2.5 and in the absence of phasic venous flow distal to the compression.[27] Other examinations include CTV, MTV, IVUS, and catheter venography with a pressure gradient determination between the right and left common iliac veins and between inferior cava vein and left common iliac vein.[18,19,28]

Diagnosis of perineal varices and lower limb varices of pelvic origin

In the case of vulvar and perineal varices, the pelvic escape points and varices of the lower limbs should be examined by US. Further diagnostics is directed toward determining the etiology of the varices (inferior vena cava obstruction, left common iliac vein compression, LRV compression, ovarian vein incompetence, etc.).

Treatment of pelvic venous disease

Treatment of left renal vein compression and left common iliac vein compression

Treatment of LRV compression in the aortomesenteric angle depends on the patient's age, the cause of the compression, and the symptoms. Conservative treatment is recommended for patients younger than 18 years and for patients with mild and tolerable symptoms. In children, symptoms may resolve due to the increase of adipose tissue between the aorta and the SMA and also due to the collateral flow formation. Conservative treatment with an emphasis of weight gain should be the first step in treating all patients with a low body weight (body mass index <18.5 kg/m^2).[25]

In patients with severe hematuria, anemia, and intense pelvic pain (patients aged < 18 years with pain lasting > 24 months and in adults with pain lasting > 6 months), invasive treatment is considered.[14]

The first choice of surgical treatment is LRV transposition with LRV reimplantation more distally into the inferior vena cava.[14,25] Left gonadal vein transposition into the left iliac vein can be an alternative in selected patients who do not have May-Thurner syndrome.[25] In the posterior LRV compression, anterior transposition of the LRV is performed.[14]

Compared to stenting for left common iliac vein compression, the LRV stenting is controversial. It is associated with risk of stent migration (due to the short length of the vein, changes in vein diameter when changing the patient's position or during the Valsalva maneuver).[14,25] Therefore, stenting is not recommended as a primary treatment for LRV compression and open interventions are considered safer options.[25] Left ovarian vein embolization should not be done, as the left ovarian vein serves as a collateral pathway, and symptoms of renal venous hypertension may worsen following the procedure.[2,9,31]

For mild symptoms of left common iliac vein compression by the right common iliac artery, conservative treatment is recommended. For moderate and severe symptoms of compression (CEAP C4–6), angioplasty and stenting are recommended. Symptom relief after endovascular treatment was noted in 68–100% of women; however, 6–32% of women did not report significant pain improvement.[5,9,32,33]

Treatment of pelvic venous disease arising from reflux in ovarian veins, internal iliac veins, and pelvic venous plexuses

Pharmacological treatment with micronized purified flavonoid fraction (MPFF) has protective and tonic effects on the vein wall and a good clinical effect of MPFF treatment was demonstrated.[1,12,34–37] MPFF reduced the severity of pelvic symptoms, and a dose of 1,000 mg twice daily was associated with faster symptom resolution.[35]

The accepted treatment is analgesics, including short-term/intermittent treatment with nonsteroidal anti-inflammatory drugs.[12] The beneficial effects of gabapentin and amitriptyline were documented.[38] Pain relief was greater in patients who were treated with gabapentin alone or in combination with amitriptyline than in patients who received amitriptyline alone.[38] Medoxyprogesterone and the gonadotropin-releasing hormone analog goserelin may provide short-term relief of symptoms, although symptom improvement is often not permanent.[5] Furthermore, a stable effect 9 months after medoxyprogesterone treatment was achieved only in combination with psychotherapy.[39] Long-term use of hormone therapy is not recommended because of undesirable side effects (weight gain, pseudomenopausal symptoms including bone loss).[1,5,9,12,32] In case of pharmacotherapy failure in patients with chronic severe pelvic pain caused by reflux in the gonadal veins, internal iliac veins and their tributaries, endovascular treatment with transcatheter occlusion of the refluxing veins is considered.[3,33] According to the Society for Vascular Surgery and the American Venous Forum, embolization in this case is recommended with level 2B evidence.[40] However, this recommendation is based on empirical data, since the results of many published studies are limited by their heterogeneity and insufficient long-term follow-up.[9] Various agents are used in embolization, such as coils, plugs, liquid adhesives, and sclerotherapeutic agents.[3,5] Although no data support the superiority of one method over another, coils with or without liquid sclerosing agents are commonly used.[13] The entire length of the ovarian vein is embolized. The decision to perform unilateral or bilateral ovarian vein embolization remains a topic of debate.[2,41] Technical success was documented in 96–100% of patients, with complete or partial improvement of symptoms occurring in 68–100% of patients and with recurrence in 32% of patients. Small studies have reported that embolization has no impact on reproductive capacity.[9,42]

Embolization of parts of the internal iliac veins or their insufficient tributaries can be performed together with ovarian vein embolization or in a deferred treatment procedure.[1,9] Complications occur in 3.1–4.4% of patients and include coil migration (into the right ventricle and pulmonary circulation), vein perforation, local phlebitis, deep vein thrombosis, and contrast reactions.[5] Complications rise to 14.8% if self-limiting postembolization abdominal discomfort (pelvic heaviness and pain, low back pain, and fever) is considered a complication.[5] Coil migration is more common during embolization of the internal iliac veins and their tributaries; therefore, some authors prefer sclerotherapy in this area.[5] Surgical treatment, such as retroperitoneal or laparoscopic ovarian vein ligation/resection is not recommended.[2]

Treatment of perineal and lower limb varices of pelvic origin

Compression therapy using compression stockings is advised for lower limb varices of pelvic origin. Direct percutaneous sclerotherapy is used to treat atypical varices in the perineum, vulva, and posterior thigh. Coiling is not suitable in these areas, as it may cause patient discomfort.[13] For patients with lower limb varices of pelvic origin who do not exhibit symptoms of pelvic venous disease, initial treatment with a minimally invasive endovascular procedure or ligation of pelvic escape points is recommended.[3]

Conclusions

Pelvic venous disease encompasses signs and symptoms pathophysiologically caused by various diseases that result in pelvic venous hypertension. All alternative pathologies should be excluded before diagnosing pelvic venous disease as a cause of chronic pelvic pain. Determining the exact cause of pelvic venous disease and subsequent treatment is a challenge in daily practice. There is a need to establish diagnostic criteria and treatment strategies supported by prospective studies for all subunits of pelvic venous disease.

Conflict of interest
None.

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Ethical statement
This review article is based on previously published literature. The authors affirm that this manuscript adheres to the ethical standards of academic publishing.

Authors' contributions
All authors contributed equally to the manuscript, read and approved the final version of the manuscript.

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