# Current View on the Problem of Varicose Veins of the Pelvis (Review)

DOI: 10.17691/stm2018.10.2.20
Received April 4, 2017

© **E.E. Fomina,** MD, PhD, Tutor, Department of Ultrasound Diagnosis[1];
**R.V. Akhmetzyanov,** MD, PhD, Tutor, Course of Cardiovascular Surgery[2];
**R.A. Bredikhin,** MD, DSc, Associate Professor, Course of Cardiovascular Surgery[2];
Head of the Vascular Surgery Unit[1];
**M.G. Tukhbatullin,** MD, DSc, Professor, Head of the Department of Ultrasound Diagnosis[1]

[1]Kazan State Medical Academy, Branch of the Russian Medical Academy of Continuing Professional Education, 36 Butlerov St., Kazan, 420012, Tatarstan, Russia;
[2]Kazan State Medical University, 49 Butlerov St., Kazan, 420012, Tatarstan, Russia

Etiopathogenesis and diagnosis of varicose veins of the pelvis or pelvic varicose veins have been considered from the present-day perspective. Despite a high prevalence of this pathology it is still insufficiently explored.

One of the main symptoms of the disease is a chronic pain in the lower abdomen lasting over six months and suffered by women of various ages. In the foreign literature, it is commonly called pelvic congestion syndrome, in the Russian literature — varicose veins of the pelvis.

Varicose veins of the pelvis can arise due to aplasia or venous valve incompetence, the genetic origin of the illness is confirmed by numerous scientific investigations. The second main cause is venous obstruction. The emergence of the disease is also promoted by the specific anatomy of the venous outflow from the small pelvis: the right ovarian vein opens into the inferior vena cava, and the left ovarian vein to the left renal vein. The normal angle between the aorta and the superior mesenteric artery is about 90°, and in case of the angle reduction aorto-mesenteric compression of the renal artery takes place.

One of the disease complications is thrombosis of the intrapelvic venous plexuses and the ovarian veins which occurs most commonly in the period of pregnancy or after delivery, and less rarely after various surgical interventions in the small pelvic organs.

The knowledge of etiopathogenesis principles will facilitate differential diagnosis of chronic pelvic pain in patients with varicose veins of the pelvis.

**Key words:** varicose veins of the pelvis; ultrasound angioscanning; left renal vein; aorto-mesenteric compression; ovarian veins.

## Introduction

Since the 90s of the last century, the problem of chronic pelvic pains (CPP) started to be linked with varicose veins of the small pelvis. This pathology, more common in women, is of low significance for clinicians due to the absence of manual criteria of the disease, however, it is the cause of chronic abdominal pain syndrome which may evoke physical and moral sufferings, lead to disability, menstrual disorders, vein thrombosis, and, probably, to thromboembolism. Nonspecificity of clinical manifestations, vague diagnostic boundaries result in inadequate observation and treatment on the part of neurologists, urologists, gynecologists, psychiatrists. The review presented here provides an opportunity to gain a deeper understanding of the problem.

Varicose veins of the small pelvis are one of the forms of a varicose vein disease, which is considered now more commonly as the main cause of CPP determined as non-cyclic pains in the small pelvis and persisting for more than 6 months [1].

According to the Russian clinical recommendations on diagnosis and treatment of chronic vein diseases (2013), this pathology is designated as varicose veins of the pelvis (VVP) and has the following definition: the disease characterized by dilation and valvular incompetence of the ovarian veins and intrapelvic venous plexuses [2]. Different terms are used in the literature for this disorder: ‘pelvic congestion syndrome (PCS)’, ‘pelvic varices’, ‘pelvic venous incompetence’, ‘pelvic venous disorders’, rarely ‘female varicocele’.

PCS study began long ago: there are data that Gooch described this disease for the first time in 1831. Taylor Jr. (1949) was the first to indicate that symptomatology pertaining to PCS is caused by the extension of the pelvic venous system and, to the less extent, to the arterial and lymphatic systems of the pelvis [3]. According to other reports [4], PCS was first described by Richet in 1857 and then by Aran in 1858. The pathology is being

**Corresponding author:** Elena E. Fomina, e-mail: eefomina@mail.ru

also investigated nowadays, but the problem of its timely revealing and treatment has not been completely solved.

Varicose veins of the pelvis can occur in girls, young women of reproductive age as well as in women of menopausal age. The disease does not disappear with age. It is reported [5] that in the USA 15% of women at the age of 18–50 years suffered from CPP, the cause of this pain syndrome remaining unclear in 60% of them [6, 7]. Soysal et al. [8] found the prevalence of the disease in 31% of cases in the female population. At present, according to the data of Triolo et al. [9], about 3.8% of women at any age and 12% of reproductive age complain of chronic pain sensations in the area of the small pelvis. In other reports [10, 11], pelvic venous pathology occurs in 10% of women in population, and the probability of PCS development in women with pelvic varicose veins may reach 60%.

## Etiopathogenesis

VVP onset is known to be promoted by the following causes: valvular incompetence, venous obstruction, and hormonal changes.

Pelvic congestion syndrome can develop due to congenital absence or incompetence of the venous valves found by anatomical investigations as early as the last century and the current data confirm these findings [12–14].

Also in the last century, it was established that the varicose disease is of genetic origin in 50% of patients [15]. *FOXC2* became one of the first identified genes playing a key role in VVP development [16]. Presently, association between the disease development and gene mutation (*TIE2, NOTCH3*), the level of thrombomodulin, and type 2 transforming growth factor β has been defined. These factors contribute to the structural alterations of the valve itself or the venous wall resulting in the incompetence of the valvular structure, vein dilation which impairs the valve function, to the progressive reflux, and, ultimately, to varicose vein [17].

An important role can be played by connective tissue dysplasia, the morphological basis of which is the reduction in the content of various kinds of collagen or changes in the ratio between them leading to the decrease of the venous strength [18–20].

The incidence of VVP development directly correlates with the quantity of hormonal changes which are especially intensive during pregnancy. In pregnant women, the capacity of the pelvic veins increases by 60% owing to mechanical compression of the small pelvic vessels by the gravid uterus and vasodilatative effect of progesterone [21]. This venous dilation persists for a month following delivery and may cause incompetence of the venous valves. Besides, during pregnancy, the uterus mass increases, its position changes inducing distention of the ovarian veins with the subsequent venous congestion. The risk factors also include endometriosis and other inflammatory diseases of the female reproductive system, estrogen therapy, unfavorable working conditions for the pregnant women, i.e. a hard physical work and long-term forced position (sitting or standing) during a working day [22].

Anatomy-related outflow from the veins of the small pelvis also contributes to the formation of varicose veins in it. The ovarian vein diameter is usually equal to 3–4 mm. The long and thin ovarian vein on the left enters into the left renal vein and on the right into the inferior vena cava [23]. Normally, the left renal vein is located in front of the aorta and behind the superior mesenteric artery. A physiological angle between the aorta and superior mesenteric artery is the angle equal approximately to 90°. Such normal anatomical arrangement prevents compression of the left renal vein [24–27]. On average, the angle between the aorta and superior mesenteric artery in adults is 51±25º, in boys 45.8±18.2º, and in girls 45.3±21.6º [28]. If the angle narrows from 39.3±4.3° [29, 30] to 14.5° [31], aorto-mesenteric compression or a nutcracker syndrome may occur. It is so-called anterior or true nutcracker syndrome which is of the greatest clinical significance. Posterior nutcracker syndrome occurs in rare cases in patients with retroaortic disposition or annular structure of the distal part of the left renal vein [32–34]. Obstruction of the proximal venous bed causes high blood pressure in the renal vein resulting in the formation of reno-ovarian blood reflux along the left ovarian vein with the following development of chronic pelvic venous insufficiency.

May–Thurner syndrome is the compression of the left common iliac vein by the right common iliac artery, is also one of the etiological factors of varicose transformation of the pelvic veins. It occurs in about 3% of cases mainly in women. Currently, this pathology is revealed more often owing to the radiologic and endovascular imaging techniques [35–37].

## Clinical picture

The main nonspecific symptoms of PCS are low abdomen pains (68%), pulsation (47%), a feeling of heaviness in this zone (35%). Patients with venous pathology in this area are younger and more slender than those suffering from varicose veins of lower extremities [38]. The pain syndrome in the small pelvis has been now established to arise due to selective activation of pain receptors of the venous walls with the subsequent development of diffused pains. In a horizontal position with the extremities raised up varicose vein compression on the inner organs is noted to decrease with the following reduction of pain intensity [39–44].

In VVP, there may be no correlation between the pain syndrome and the extent of gonadal and intrapelvic vein dilation leading to PSC. The so-called painless form of this disease due to the difference in pain perception, regulation, and formation of this sensation is often registered. In this case, patients describe similar but much less intensive pains or their absence [14, 45].

One of the common VVP symptoms, which is paid no attention to by the physicians or not associated with this pathology, is atypical varicosity, expressed by varicose veins of perineum, vulva, inner thigh and buttock area. Once these altered veins are detected, their connection with each other, the great saphenous vein, and its branches must be compulsorily revealed as isolated pelvic varicose veins are found in 35–40% of women, in other 60–65% of cases the disease usually occurs in the combination with varicose veins of low extremities [46–51].

One more frequently encountered symptom is dyspareunia (painful sexual intercourse) which results in impairment and fear of sexual relations, family conflicts leading to divorces [40, 42, 52].

VVP picture may be complicated with various pathologies.

Thrombosis of gonadal veins and intrapelvic venous plexuses is a complication which occurs most commonly in the period of pregnancy or after delivery, sometimes after surgical interventions, often intercurrently with varicose veins of the pelvis. When vessel endothelium is damaged, its surface changes from antithrombotic to prothrombotic. Once proadhesive surface of subendothelial matrix appeared, its components, adhesive proteins (von Willebrand factor, collagen, fibrinogen, and so on), are immediately involved in the process of forming a primary (cardio-thrombocytic) thrombus followed by hemocoagulation [53]. In the period of pregnancy, the following factors serve as the basis for phlebothrombosis development: physiological hypercoagulation, increase of blood viscosity, formation of blood stasis in the veins of the pelvis and low extremities together with the changes of hormonal status and topographoanatomical interrelations of the pelvic vessels and organs [54–60].

Ovarian vein thrombosis is a rare pathology which, causing acute abdominal pains, mimics a “surgical abdomen”. Ovarian thrombosis develops during delivery or in the first seven days after it with the incidence of 1/600 and 1/2000 cases, respectively. Investigations showed that in 80% of cases the right ovarian vein is thrombosed, damage of both ovarian veins is registered in 14%, and isolated thrombosis of the left ovarian vein in 6% of cases. The occurrence of pulmonary artery embolism with underlying ovarian vein thrombosis varies within 0.15–0.33% with fatal outcomes up to 4% in patients with pulmonary thromboembolism [59–63].

## Classification of varicose vein disease of the pelvis

One of the first classifications was proposed by Volkov in 2000: mainstream (magistral) variant: a retort-shaped widening of the uterine venous plexus; scattered: multiple unechogenic varicose structures of various sizes; combined (total): dilation of all venous communication systems of the small pelvis [64].

At present, the most commonly used classification of VVP is one recommended by the Russian Association of Phlebologists (2013) [2]. This pathology is characterized by the following signs:

clinical manifestations: pelvic venous congestion syndrome; varicose veins of genitals;

course: painful form; painless form; latent form;

extension of pelvic vein involvement: isolated dilation of the pelvic venous plexuses; combined dilation of gonadal veins and pelvic venous plexuses; uni- or bilateral dilation of gonadal veins; dilation of the trunk or branches of the internal iliac veins.

## Instrumental diagnosis of varicose veins of the pelvis

Currently, the number of detected cases of VVP has grown owing to new technologies. Several stages are used to examine patients with CPP.

The first stage is a routine examination by a gynecologist: history-taking, manual examination, pelvic ultrasound (to exclude other pathology). Depending on the results patients are additionally referred to a proctologist, urologist, neurologist, and other related specialists [44, 65, 66].

If a diagnosis is not clear but VVP is suspected, ultrasound angioscanning (USAS) of pelvic veins is carried out at the second stage. This is a non-invasive highly informative technique of a screening diagnosis which is used for all women with suspected VVP. If previously a mere pelvic organs examination was considered quite sufficient (inspection of veins was believed to be an inaccessible and non-obligatory procedure), now USAS of the pelvic veins is a compulsory investigation. This method helps to establish presence of varicose veins in the small pelvis by measuring the diameters, blood flow rate in the veins, and to determine preliminarily the leading pathogenetic mechanism: incompetence of the ovarian veins or venous obstruction. This method can also be used for dynamic assessment of conservative and surgical treatment of VVP [22, 38, 40, 67–73].

Investigations are performed transvaginally and transabdominally. Veins of parametrium, pampiniform plexus, uterine veins are visualized treansvaginally. According to various reports, the vessel diameter of the mentioned localizations varies from 2.0 to 5.0 mm (3.9±0.5 mm, on average), i.e. not more than 5 mm [14, 40, 74], and the average diameter of the arcuate veins is 1.1±0.4 mm [74]. Veins with the diameter exceeding 5 mm are considered dilated [22, 51, 67, 68].

Inferior vena cava, iliac veins, left renal vein, and ovarian veins are examined transabdominally in order to exclude thrombotic masses and extravasal compression. The left renal vein is 6–10 mm long and 4–5 mm wide, on average. Normally, the left renal vein is a bit thicker at the site above the aorta but 2–2.5-fold reduction of its diameter does not influence significantly blood flow

acceleration providing normal outflow without pressure increase in the prestenotic zone. In case of vein stenosis, in presence of pathological compression the diameter decreases essentially, 3.5–4 times, the blood flow acceleration exceeds100 cm/s. The sensitivity and specificity of this method is 78 and 100%, respectively [32, 75–80].

Investigation of the ovarian veins is included into the obligatory examination of the pelvic veins. They are located along the anterior abdominal wall and abdominal rectus muscle slightly lateral to the iliac veins and arteries [78]. The diameter over 5 mm and presence of retrograde blood flow are considered the sign of ovarian vein incompetence at USAS [2, 51, 69]. USAS of the veins of low extremities, perineum, vulva, inner femoral surface, and buttocks area is mandatory for the adequate examination, prevention of recurrences, and correct treatment tactics.

Advances in medical technologies provide the opportunity of using new diagnostic methods. At the third stage, after ultrasound verification of the diagnosis, radiological methods are used for its confirmation. The most informative and non-invasive methods are magnetic resonance tomography and multispiral computed tomography-assisted angiography. The value of these techniques lies in the possibility of using them in outpatient settings and their general availability. Up-to-date tomographic scanners perform 3D image reconstruction improving significantly the diagnostic quality and enable physicians to reveal anatomical characteristics and dilation of pelvic veins, and organ pathology, to assess the condition of iliac, renal, ovarian veins, detect aorto-mesenteric compression, compression of the left iliac vein, abnormally high location of the left renal vein, abnormally low origin or atypical branching of the superior mesenteric artery. Sensitivity and specificity of these diagnostic methods are 91.7 and 88.9%, respectively [24, 81–92].

Pelvic phlebology with selective bilateral radiocontrast ovaricography is one of the radiological invasive diagnostic methods which is performed only in the in-patient settings. This method has been long considered the diagnostic gold standard for assessing dilation and detecting valvular incompetence in the veins of the small pelvis. This method consists in the fluoroscopy-guided injection of a contrast agent via the catheter inserted in one of the main veins (jugular, brachial, or femoral) to the iliac, renal, and ovarian veins. In this way, it is possible to identify the anatomical variants of ovarian veins, measure the diameters of the gonadal and pelvic veins. Retrograde contrasting of gonadal veins at the height of Valsalva test serves as pathognomonic angiographic sign of their valvular incompetence with visualization of drastic dilation and tortuosity, respectively [22, 40–41]. This is the most accurate method of revealing May–Thurner syndrome, post-thrombophlebitic changes of iliac veins and inferior vena cava. In case of the left renal vein compression, paranephric venous collaterals with retrograde blood flow into gonadal veins and contrast media congestion in the renal vein are determined. This technique makes it possible to measure a pressure gradient between the left renal vein and inferior vena cava. Normally it is equal to 1 mm Hg; the gradient of 2 mm Hg can suggest a slight compression; at the gradient of >3 mm Hg the diagnosis of aorto-mesenterial compression with hypertension in the left renal vein may be established, and the gradient of >5 mm Hg is regarded as hemodynamically significant stenosis of the left renal vein. Determination of the pressure gradient is an important element of diagnosis as different operative interventions in the small pelvic veins are planned depending on its values which is of great importance in the current conditions. Presently, this examination (at a normal pressure gradient) can be used for therapeutic purposes: in ovarian vein embolization [2, 14, 32, 83, 85, 86].

The next radiological method is emission computed tomography of pelvic veins with *in vitro* labeled erythrocytes. It is characterized by depositing labeled erythrocytes in the pelvic veins and visualization of gonadal veins. This technique provides also the possibility to detect varicosely changed plexuses of the small pelvis and dilated ovarian veins in various positions, the degree of pelvic venous congestion, reflux of blood from the pelvic veins to the saphenous veins of legs and perineum. Normal ovarian veins do not look contrast, accumulation of radiopharmaceutical in venous plexuses is not noted. For objective evaluation of venous congestion degree in the small pelvis a coefficient of pelvic venous congestion is calculated. But this method has some drawbacks: invasiveness, relatively low spatial resolution, inability to precisely measure vein diameter — these are the reasons for its rare use in clinics nowadays [14, 93].

Videolaparoscopic examination is a valuable tool in the assessment of the overlooked pathology. In complex with other methods, it may help to determine causes of pains and administer proper treatment. In varicose veins of the small pelvis in the area of ovaries, veins in the form of cyanotic dilated vessels with a thinned and tense wall along the round and broad ligaments of uterus can be visualized. The following factors restrict the application of this method: presence of retroperitoneal adipose tissue, assessment of vein dilation on the limited area only, inability to define reflux by veins. At present, the application of this method is diagnostically justified when multifocal character of pains is suspected. Laparoscopy allows phlebologists to visualize causes of CPP, e.g. foci of endometriosis or adhesive process, in 66% of cases [82, 84, 94–97].

## Conclusion

Current diagnostic methods provide the opportunity to obtain exhaustive information about condition of

veins and organs of the small pelvis, to choose optimal treatment on the basis of the investigation findings avoiding recurrence of the disease and complications.

**Study funding and conflicts of interest.** The work was not funded by any sources, and there are no conflicts of interest related to the present study.

**References**

**1.** Zondervan K.T., Yudkin P.L., Vessey M.P., Jenkinson C.P., Dawes M.G., Barlow D.H., Kennedy S.H. The community prevalence of chronic pelvic pain in women and associated illness behaviour. *Br J Gen Pract* 2001; 51(468): 541–547.

**2.** *Rossiyskie klinicheskie rekomendatsii po diagnostike i lecheniyu khronicheskikh zabolevaniy ven* [Russian clinical guidelines for diagnosis and treatment of chronic venous diseases]. *Flebologiya* 2013; 7(2–2): 1–47.

**3.** Beck R.P. The pelvic congestion syndrome. *Can Fam Physician* 1969; 15(5): 46–50.

**4.** Belenky A., Bartal G., Atar E., Cohen M., Bachar G. Ovarian varices in healthy female kidney donors: incidence, morbidity, and clinical outcome. *AJR Am J Roentgenol* 2002; 179(3): 625–627, https://doi.org/10.2214/ajr.179.3.1790625.

**5.** Mathias S., Kuppermann M., Liberman R., Lipschutz R., Steege J. Chronic pelvic pain: prevalence, health-related quality of life, and economic correlates. *Obstet Gynecol* 1996; 87(3): 321–327, https://doi.org/10.1016/0029-7844(95)00458-0.

**6.** Hansrani V., Morris J., Caress A., Payne K., Seif M., McCollum C.N. Is pelvic vein incompetence associated with symptoms of chronic pelvic pain in women? A pilot study. *Eur J Obstet Gynecol Reprod Biol* 2016; 196: 21–25, https://doi.org/10.1016/j.ejogrb.2015.10.023.

**7.** Farquhar C.M., Rogers V., Franks S., Pearce S., Wadsworth J., Beard R.W. A randomized controlled trial of medroxyprogesterone acetate and psychotherapy for the treatment of pelvic congestion. *Br J Obstet Gynaecol* 1989; 96(10): 1153–1162, https://doi.org/10.1111/j.1471-0528.1989.tb03190.x.

**8.** Soysal S., Vicdan K., Ozer S. A randomized controlled trial of goserelin and medroxyprogesterone acetate in the treatment of pelvic congestion. *Hum Reprod* 2001; 16(5): 931–939, https://doi.org/10.1093/humrep/16.5.931.

**9.** Triolo O., Lagana A., Sturlese E. Chronic pelvic pain in endometriosis: an overview. *J Clin Med Res* 2013; 5(3): 153–163, https://doi.org/10.4021/jocmr1288w.

**10.** Karcaaltincaba M., Karcaaltincaba D., Dogra V. Pelvic congestion syndrome. *Ultrasound Clinics* 2008; 3(3): 415–425, https://doi.org/10.1016/j.cult.2008.08.002.

**11.** Cicchiello L.A., Hamper U.M., Scoutt L.M. Ultrasound evaluation of gynecologic causes of pelvic pain. *Ultrasound Clinics* 2010; 5(2): 209–231, https://doi.org/10.1016/j.cult.2010.03.005.

**12.** Ahlberg N.E., Bartley O., Chidekel N. Right and left gonadal veins. An anatomical and statistical study. *Acta Radiol Diagn (Stockh)* 1966; 4(6): 593–601, https://doi.org/10.1177/028418516600400601.

**13.** Freedman J., Ganeshan A., Crowe P.M. Pelvic congestion syndrome: the role of interventional radiology in the treatment of chronic pelvic pain. *Postgrad Med J* 2010; 86(1022): 704–710, https://doi.org/10.1136/pgmj.2010.099473.

**14.** Gavrilov S.G., Kirienko A.I. *Varikoznaya bolezn' taza* [Varicose disease of the pelvis]. Moscow: Planida TM; 2015.

**15.** Matoušek V., Přerovský I. A contribution to the problem of the inheritance of primary varicose veins. *Hum Hered* 1974; 24(3): 225–235, https://doi.org/10.1159/000152655.

**16.** Brice G., Mansour S., Bell R., Collin J.R., Child A.H., Brady A.F., Sarfarazi M., Burnand K.G., Jeffery S., Mortimer P., Murday V.A. Analysis of the phenotypic abnormalities in lymphoedema-distichiasis syndrome in 74 patients with FOXC2 mutations or linkage to 16q24. *J Med Genet* 2002; 39(7): 478–483, https://doi.org/10.1136/jmg.39.7.478.

**17.** Oklu R., Habito R., Mayr M., Deipolyi A.R., Albadawi H., Hesketh R., Walker T.G., Linskey K.R., Long C.A., Wicky S., Stoughton J., Watkins M.T. Pathogenesis of varicose veins. *J Vasc Interv Radiol* 2012; 23(1): 33–39, https://doi.org/10.1016/j.jvir.2011.09.010.

**18.** Chilla B.K., Knusel P.R., Zollikofer Ch.L., Huber T., Kubik-Huch R.A. Pelvic congestion syndrome. *Schweiz Rundsch Med Prax* 2006; 95(14): 1583–1588.

**19.** Il'ina I.Iu., Dobrokhotova Iu.E., Titchenko I.P., Grudkin A.A. Female small pelvic varix as one of the manifestations of connective tissue dysplasia. *Rossiyskiy vestnik akushera-ginekologa* 2009; 2: 39–42.

**20.** Veropotvelyan P.N., Veropotvelyan N.P., Avksent'ev O.N. Varikoznaya bolezn' ven malogo taza, obuslovlennaya displaziey soedinitel'noy tkani. *Zhinyuchiy likar* 2011; 5: 15–17.

**21.** Perry C.P. Current concepts of pelvic congestion and chronic pelvic pain. *JSLS* 2001; 5(2): 105–110.

**22.** Phillips D., Deipolyi A.R., Hesketh R.L., Midia M., Oklu R. Pelvic congestion syndrome: etiology of pain, diagnosis, and clinical management. *J Vasc Interv Radiol* 2014; 25(5): 725–33, https://doi.org/10.1016/j.jvir.2014.01.030.

**23.** Kamina P., Chansigaud J.P. Functional anatomy of pelvic veins in women. *Phlebologie* 1989; 42(3): 363–384.

**24.** Ahmed K., Sampath R., Khan M.S. Current trends in the diagnosis and management of renal nutcracker syndrome: a review. *Eur J Vasc Endovasc Surg* 2006; 31(4): 410–416, https://doi.org/10.1016/j.ejvs.2005.05.045.

**25.** O'Brien M.T., Gillespie D.L. Diagnosis and treatment of the pelvic congestion syndrome. *J Vasc Surg Venous Lymphat Disord* 2015; 3(1): 96–106, https://doi.org/10.1016/j.jvsv.2014.05.007.

**26.** Perkov D., Vrkić Kirhmajer M., Novosel L., Popić Ramač J. Transcatheter ovarian vein embolisation without renal vein stenting for pelvic venous congestion and nutcracker anatomy. *Vasa* 2016; 45(4): 337–341, https://doi.org/10.1024/0301-1526/a000547.

**27.** Rolim D., Sampaio S., Teixeira J.F. Pelvic congestion syndrome — a clinical report. *Rev Port Cir Cardiotorac Vasc* 2015; 22(1): 53–56.

**28.** Arthurs O.J., Mehta U., Set P.A. Nutcracker and SMA syndromes: what is the normal SMA angle in children? *Eur J Radiol* 2012; 81(8): e854–e861, https://doi.org/10.1016/j.ejrad.2012.04.010.

**29.** Kim K.W., Cho J.Y., Kim S.H., Yoon J.H., Kim D.S., Chung J.W., Park J.H. Diagnostic value of computed tomographic findings of nutcracker syndrome: correlation with renal venography and renocaval pressure gradients. *Eur J Radiol* 2011; 80(3): 648–654, https://doi.org/10.1016/j.ejrad.2010.08.044.

**30.** Fu W.J., Hong B.F., Gao J.P., Xiao Y.Y., Yang Y., Cai W., Guo G., Wang X.X. Nutcracker phenomenon: a new diagnostic method of multislice computed tomography angiography. *Int J Urol* 2006; 13(7): 870–873, https://doi.org/10.1111/j.1442-2042.2006.01430.x.

**31.** Inal M., Karadeniz Bilgili M.Y., Sahin S. Nutcracker syndrome accompanying pelvic congestion syndrome; color doppler sonography and multislice CT findings: a case report. *Iran J Radiol* 2014; 11(2): e11075, https://doi.org/10.5812/iranjradiol.11075.

**32.** Gulleroglu K., Gulleroglu B., Baskin E. Nutcracker syndrome. *World J Nephrol* 2014; 3(4): 277–281, https://doi.org/10.5527/wjn.v3.i4.277.

**33.** Hartung O. Syndrome of the left renal vein compression in aorto-mesentric clamp (nutcracker syndrome). *Flebolimfologiya* 2010; 36: 10–15.

**34.** Skeik N., Gloviczki P., Macedo T.A. Posterior nutcracker syndrome. *Vasc Endovascular Surg* 2011; 45(8): 749–755, https://doi.org/10.1177/1538574411419376.

**35.** Gavrilov S.G., Shipovskii V.N., Karalkin A.V., Maksimova M.A., Beliaeva E.S. A case of successful treatment of pelvic venous congestion caused by May–Turner syndrome. *Flebologiya* 2010; 1: 68–71.

**36.** Rastogi N., Kabutey N.K., Kim D. Incapacitating pelvic congestion syndrome in a patient with a history of May–Thurner syndrome and left ovarian vein embolization. *Ann Vasc Surg* 2012; 26(5): 732e–711e, https://doi.org/10.1016/j.avsg.2011.08.029.

**37.** Ahmed O., Ng J., Patel M., Ward T.J., Wang D.S., Shah R., Hofmann L.V. Endovascular stent placement for May–Thurner syndrome in the absence of acute deep vein thrombosis. *J Vasc Interv Radiol* 2016; 27(2): 167–173.

**38.** Shcheglov E.A., Alontseva N.N. Congress of the American West Coast venous forum "Challenges and polemic in treatment of venous pathology" (30 April–2 May, 2015, Napa, the USA). *Novosti khirurgii* 2015; 23(5): 582–587.

**39.** Huang C., Shelkey J., Singh H., Silvis M. Chronic hip pain as a presenting symptom in pelvic congestion syndrome. *J Vasc Interv Radiol* 2013; 24(5): 753–755, https://doi.org/10.1016/j.jvir.2013.01.004.

**40.** Bredikhin R.A., Ignatiev I.M., Fomina E.E., Volodyukhin M.Yu., Gaptravanov A.G., Mikhailov M.K. Diagnosis and treatment of varicose disease of small pelvic veins. *Angiologiia i sosudistaia khirurgiia* 2012; 18(1): 63–69.

**41.** Sokolov A.A. *Varikoznaya bolezn' ven malogo taza* [Varicousity of small pelvis veins]. URL: http://www.medcentre.com.ua/articles/Varikoznaya-bolezn-ven-malogo-71928.

**42.** Savel'ev V.S., Gologorskiy V.A., Kirienko A.I. *Flebologiya* [Phlebology]. Moscow: Meditsina; 2001; 664 p.

**43.** Sator-Katzenschlager S.M., Scharbert G., Kress H.G., Frickey N., Ellend A., Gleiss A., Kozek-Langenecker S.A. Chronic pelvic pain treated with gabapentin and amitriptyline: a randomized controlled pilot study. *Wien Klin Wochenschr* 2005; 117(21–22): 761–768, https://doi.org/10.1007/s00508-005-0464-2.

**44.** Ferrero S., Ragni N., Remorgida V. Deep dyspareunia: causes, treatments, and results. *Curr Opin Obstet Gynecol* 2008; 20(4): 394–399, https://doi.org/10.1097/gco.0b013e328305b9ca.

**45.** Gavrilov S.G. Varicosity of small pelvis veins: whom and how to treat. *Flebologiya* 2007; 1(1): 48–54.

**46.** Kim A.S., Greyling L.A., Davis L.S. Vulvar varicosities: a review. *Dermatol Surg* 2017; 43(3): 351–356, https://doi.org/10.1097/dss.0000000000001008.

**47.** Knuttinen M.G., Xie K., Jani A., Palumbo A., Carrillo T., Mar W. Pelvic venous insufficiency: imaging diagnosis, treatment approaches, and therapeutic issues. *AJR Am J Roentgenol* 2015; 204(2): 448–458, https://doi.org/10.2214/ajr.14.12709.

**48.** Koo S., Fan C.M. Pelvic congestion syndrome and pelvic varicosities. *Tech Vasc Interv Radiol* 2014; 17(2): 90–95, https://doi.org/10.1053/j.tvir.2014.02.005.

**49.** Sukovatykh B.S., Belikov L.N., Rodionov O.A., Rodionova I.G., Gorbachev Yu.I., Sukovatykh M.B. The mechanisms of the natural history of small pelvis varicosis. *Angiologiia i sosudistaia khirurgiia* 2004; 10(3): 73–80.

**50.** Fomina E.E. *Ul'trazvukovoe dupleksnoe skanirovanie v diagnostike i otsenke rezul'tatov khirurgicheskogo lecheniya varikoznoy bolezni ven malogo taza.* Avtoref. dis. … kand. med. nauk [Duplex US scanning in the diagnosis and assessment of surgical treatment of small pelvic varicose veins. PhD Thesis]. Kazan; 2012.

**51.** Champaneria R., Shah L., Moss J., Gupta J.K., Birch J., Middleton L.J., Daniels J.P. The relationship between pelvic vein incompetence and chronic pelvic pain in women: systematic reviews of diagnosis and treatment effectiveness. *Health Technol Assess* 2016; 20(5), 1–108, https://doi.org/10.3310/hta20050.

**52.** Bogachev V.Yu. Varicosity of small pelvis veins. *Ginekologiya* 2006; 8(4): 64–67.

**53.** Bolevich C.B., Voynov V.A. *Molekulyarnye mekhanizmy v patologii cheloveka* [Molecular mechanisms in human pathology]. Moscow: Meditsinskoe informatsionnoe agentstvo; 2012.

**54.** Zlatovratskii A.G., Kapranov S.A., Kurtser M.A., Kuznetsova V.F. Endovascular prevention of pulmonary thromboembolism in patients with embologenic thrombosis of gonadal veins. *Flebologiya* 2008; 3: 54–58.

**55.** Kudykin M.N., Kletskin A.E., Kachalina T.S., Siubaeva R.I., Pugin V.A., Izmaïlova T.S. Prevention and treatment of venous thromboembolism and chronic venous diseases of the lower extremities in pregnant women. *Flebologiya* 2010; 4(4): 21–24.

**56.** Andriiashkin V.V., Dzhenina O.V., Bychkova T.V., Leont'ev S.G., Zolotukhin I.A., Kirienko A.I. Surgical strategy for pregnant women with deep vein thrombosis in the lower extremities. *Flebologiya* 2010; 4(3): 62–66.

**57.** Iumin S.M., Andriiashkin V.V., Leont'ev S.G., Zolotukhin I.A. Partial occlusion of inferior vena cava for the prevention of pulmonary embolism. *Flebologiya* 2010; 4(1): 41–46.

**58.** Tsoukanov Yu.T., Tsoukanov A.Yu. Varicosis of the lower extremities as a consequence of connective tissue dysplasia. *Angiologiia i sosudistaia khirurgiia* 2004; 10(2): 84–90.

**59.** Salomon O., Dulitzky M., Apter S. New observations in postpartum ovarian vein thrombosis: experience of single center. *Blood Coagul Fibrinolysis* 2010; 21(1): 16–19, https://doi.org/10.1097/mbc.0b013e32832f2ada.

**60.** Jenayah A.A., Saoudi S., Boudaya F., Bouriel I., Sfar E., Chelli D. Ovarian vein thrombosis. *Pan Afr Med J* 2015; 21: 251, https://doi.org/10.11604/pamj.2015.21.251.6908.

**61.** Kearon C. Diagnosis of pulmonary embolism. *CMAJ* 2003; 168(2): 183–194.

**62.** Nagayama M., Watanabe Y., Okumura A., Amoh Y., Nakashita S., Dodo Y. Fast MR imaging in obstetrics.

*Radiographics* 2002; 22(3): 563–580, https://doi.org/10.1148/radiographics.22.3.g02ma03563.

**63.** Labropoulos N., Malgor R.D., Comito M., Gasparis A.P., Pappas P.J., Tassiopoulos A.K. The natural history and treatment outcomes of symptomatic ovarian vein thrombosis. *J Vasc Surg Venous Lymphat Disord* 2015; 3(1): 42–47, https://doi.org/10.1016/j.jvsv.2014.07.008.

**64.** Kharchenko V.P., Zubarev A.R., Kotlyarov P.M. *Ul'trazvukovaya flebologiya* [Ultrasound phlebology]. Moscow: ZAO "Eniki"; 2005.

**65.** Akhmetzyanov R.V., Bredikhin R.A., Gaptravanov A.G., Fomina E.E. Historical aspects of diagnosis and treatment of pelvic varicosity. Review of literature. *Ambulatornaya khirurgiya* 2016; 1–2: 36–43.

**66.** Tu F.F., Hahn D., Steege J.F. Pelvic congestion syndrome-associated pelvic pain: a systematic review of diagnosis and management. *Obstet Gynecol Surv* 2010; 65(5): 332–340, https://doi.org/10.1097/ogx.0b013e3181e0976f.

**67.** Durham J.D., Machan L. Pelvic congestion syndrome. *Semin Intervent Radiol* 2013; 30(4): 372–380, https://doi.org/10.1055/s-0033-1359731.

**68.** Sharma K., Bora M.K., Varghese J., Malik G., Kuruvilla R. Role of trans vaginal ultrasound and Doppler in diagnosis of pelvic congestion syndrome. *J Clin Diagn Res* 2014; 8(7): OD05–OD07, https://doi.org/10.7860/jcdr/2014/8106.4570.

**69.** Ricci S., Moro L., Minotti G.C., Incalzi R.A., De Maeseneer M. Valsalva maneuver in phlebologic practice. *Phlebology* 2017; 33(2): 75–83, https://doi.org/10.1177/0268355516678513.

**70.** Hansrani V., Dhorat Z., McCollum C.N. Diagnosing of pelvic vein incompetence using minimally invasive ultrasound techniques. *Vascular* 2016; 25(3): 253–259, https://doi.org/10.1177/1708538116670499.

**71.** Labropoulos N., Jasinski P.T., Adrahtas D., Gasparis A.P., Meissner M.H. A standardized ultrasound approach to pelvic congestion syndrome. *Phlebology* 2016; 32(9): 608–619, https://doi.org/10.1177/0268355516677135.

**72.** Malinova M., Shopov A. Current echography diagnosis of pelvic congestion syndrome. *Akush Ginekol (Sofiia)* 2012; 51(Suppl 1): 10–15.

**73.** Malgor R.D., Adrahtas D., Spentzouris G., Gasparis A.P., Tassiopoulos A.K., Labropoulos N. The role of duplex ultrasound in the workup of pelvic congestion syndrome. *J Vasc Surg Venous Lymphat Disord* 2014; 2(1): 34–38, https://doi.org/10.1016/j.jvsv.2013.06.004.

**74.** Ozerskaya I.A., Ageeva M.I. *Khronicheskaya tazovaya bol' u zhenshchin reproduktivnogo vozrasta. Ul'trazvukovaya diagnostika* [Chronic pelvic pain in women of reproductive age. Ultrasound diagnosis]. Moscow: Vidar-M; 2009; 299 p.

**75.** Kurklinsky A.K., Rooke T.W. Nutcracker phenomenon and nutcracker syndrome. *Mayo Clin Proc* 2010; 85(6): 552–559, https://doi.org/10.4065/mcp.2009.0586.

**76.** Hartung O. Nutcracker syndrome. *Phlebolymphology* 2009; 16(2): 246–252.

**77.** He Y., Wu Z., Chen S., Tian L., Li D., Li M., Jin W., Zhang H. Nutcracker syndrome — how well do we know it? *Urology* 2014; 83(1): 12–17.

**78.** Fomina E.E., Akhmetzyanov R.V., Tukhbatullin M.G. Methodology of ultrasound investigation of pelvic varicose disease. *Prakticheskaya meditsina* 2016; 9(101): 53–58.

**79.** Jeanneret C., Beier K., von Weymarn A., Traber J. Pelvic congestion syndrome and left renal compression syndrome — clinical features and therapeutic approaches. *Vasa* 2016; 45(4): 275–282, https://doi.org/10.1024/0301-1526/a000538.

**80.** Juhan V. Chronic pelvic pain: an imaging approach. *Diagn Interv Imaging* 2015; 96(10): 997–1007, https://doi.org/10.1016/j.diii.2015.07.010.

**81.** Bhanji A., Malcolm P., Karim M. Nutcracker syndrome and radiographic evaluation of loin pain and hematuria. *Am J Kidney Dis* 2010; 55(6): 1142–1145, https://doi.org/10.1053/j.ajkd.2009.10.010.

**82.** Wozniak S. Chronic pelvic pain. *Ann Agric Environ Med* 2016; 23(2): 223–226, https://doi.org/10.5604/12321966.1203880.

**83.** Shokeir T., Amr M., Abdelshaheed M. The efficacy of Implanon for the treatment of chronic pelvic pain associated with pelvic congestion: 1-year randomized controlled pilot study. *Arch Gynecol Obstet* 2009; 280(3): 437–443, https://doi.org/10.1007/s00404-009-0951-1.

**84.** Smith P.C. The outcome of treatment for pelvic congestion syndrome. *Phlebology* 2012; 27(Suppl 1): 74–77, https://doi.org/10.1258/phleb.2011.012s01.

**85.** Thors A., Haurani M.J., Gregio T.K., Go M.R. Endovascular intervention for pelvic congestion syndrome is justified for chronic pelvic pain relief and patient satisfaction. *J Vasc Surg Venous Lymphat Disord* 2014; 2(3): 268–273, https://doi.org/10.1016/j.jvsv.2013.12.002.

**86.** Yetkin E., Ileri M. Dilating venous disease: pathophysiology and a systematic aspect to different vascular territories. *Med Hypotheses* 2016; 91: 73–76, https://doi.org/10.1016/j.mehy.2016.04.016.

**87.** Gupta R., Gupta A., Aggarwal N. Variations of gonadal veins: embryological prospective and clinical significance. *J Clin Diagn Res* 2015; 9(2): AC08–AC10, https://doi.org/10.7860/jcdr/2015/9493.5578.

**88.** Leiber L.M., Thouveny F., Bouvier A., Labriffe M., Berthier E., Aubé C., Willoteaux S. MRI and venographic aspects of pelvic venous insufficiency. *Diagn Interv Imaging* 2014; 95(11): 1091–1102, https://doi.org/10.1016/j.diii.2014.01.012.

**89.** Motta-Ramírez G.A., Ruiz-Castro E., Torres-Hernández V., Herrera-Avilés R.A., Rodríguez-Treviño C. The role of the computed tomography in the identification of the syndrome of pelvic congestion. *Ginecol Obstet Mex* 2013; 81(7): 389–402.

**90.** Cimsit C., Yoldemir T., Tureli D., Aribal M.E. Evaluation of sacroiliac joint MRI for pelvic venous congestion signs in women clinically suspected of sacroiliitis. *Acta Radiol* 2016; 58(7): 849–855; https://doi.org/10.1177/0284185116675656.

**91.** Shi W.Y., Gu J.P., Lou W.S., Chen G.P. Left ovarian vein dilation or pelvic congestion syndrome secondary to abdominal aortic dissection: incidental findings on CT angiography. *Clin Imaging* 2015; 39(3): 480–483, https://doi.org/10.1016/j.clinimag.2014.12.003.

**92.** Winer A.G., Chakiryan N.H., Mooney R.P., Verges D., Ghanaat M., Allaei A., Robinson L., Zinn H., Lang E.K. Secondary pelvic congestion syndrome: description and radiographic diagnosis. *Can J Urol* 2014; 21(4): 7365–7368.

**93.** Kiriyenko A.I., Karalkin A.V., Gavrilov S.G., Saitova G.D., Moskalenko Ye.P., Cherkashin M.A. Diagnostic capacities of emission computed tomography in small pelvic varicosity. *Annaly hirurgii* 2004; 1: 50–54.

**94.** Hebbar S., Chawla C. Role of laparoscopy in evaluation

of chronic pelvic pain. *J Minim Access Surg* 2005; 1(3): 116–120, https://doi.org/10.4103/0972-9941.18995.

**95.** Artymuk N.V. Female small pelvic varicose veins. *Rossiyskiy vestnik akushera-ginekologa* 2007; 7(6): 74–78.

**96.** Balica A.C., Nassiri N., Horne J., Egan S., Wang X.K. Pelvic congestion syndrome. *J Minim Invasive Gynecol* 2015; 22(6S): S152, https://doi.org/10.1016/j.jmig.2015.08.561.

**97.** Borghi C., Dell'Atti L. Pelvic congestion syndrome: the current state of the literature. *Arch Gynecol Obstet* 2016; 293(2): 291–301, https://doi.org/10.1007/s00404-015-3895-7.