

Left iliac vein compression in Leriche syndrome patients — a potential link between aorto-iliac occlusive disease and DVT occurrence?

Ucisk żyły biodrowej lewej u chorych z zespołem Leriche'a — potencjalny czynnik ryzyka proksymalnej zakrzepicy żył głębokich?

Tomasz Urbanek, Bartosz Kończyk, Monika Kasperczyk, Wacław Kuczmik

Department of General Surgery Vascular Surgery, Angiology and Phlebology, Medical University of Silesia, Katowice

Abstract

Introduction: One of the important anatomical conditions, which can potentially limit the lower leg venous outflow, is the compression of the left iliac vein in its proximal segment by the right iliac artery. Several studies suggest the relatively high occurrence of this pathology, also in the asymptomatic patients, with a much higher prevalence in the patients diagnosed with proximal left leg DVT episode. The anatomical locations of the aortic bifurcation and inferior vena confluence suggest the possibility of other vascular pathologies causing venous outflow disturbances in this region such as an aneurysm or atherosclerotic plaques. In the study, the possible compression of the left iliac vein in patients with Leriche syndrome was investigated.

Material and methods: 40 patients with Leriche syndrome and symptomatic peripheral atherosclerosis obliterative disease were evaluated. The median age of the patients was 65 yrs. (from 53 to 76) and all the patients underwent CT-angio of the aorta and iliac arteries. The analysis assessed the diameter of the left and right iliac artery, as well as the diameter of the common left and right iliac vein. The left common iliac vein (CIV) stenosis was evaluated by comparing the CIV vein diameter at the place of the possible stenosis with the contralateral common iliac vein diameter at the same level as well as with the ipsilateral common iliac vein diameter distally to the stenosis.

Results: The median diameter of the left common iliac vein at the pressure site in the study population was 7.42 mm (range 3.9–9.69 mm), with a mean of 12.11 ± 2.35 mm (12.03 ± 2.7 for men and 11.8 ± 2.99 females) on the right side. The mean degree of stenosis of the left iliac vein was 46% for the entire population and the stenosis above 50% was diagnosed in 45% of the studied population (with the prevalence of the stenosis of 70% or more in 5% only). The analysis of the relationship between the presence of stenotic changes or obstruction in the right common iliac artery and the presence of significant stenosis or obstruction of the left iliac vein showed no significant correlation between the degree of left CIV stenosis and the severity of changes in the right common iliac artery. There was no significant difference in the incidence of stenosis of above 50% between the male and female population with atherosclerotic lesions.

Conclusion: The asymptomatic left common iliac vein stenosis can be found in a significant number of patients with atherosclerotic changes in the aortic bifurcation and iliac arteries. Due to the complex process of arterial wall remodelling in patients with atherosclerosis including plaque growth, possible vessel widening, and elongation, the clinical importance of these observations should be investigated in further studies.

Key words: atherosclerosis, deep vein thrombosis, compression syndrome, iliac veins

Chirurgia Polska 2019, 21, 1–2, 20–26

Introduction

Venous thromboembolism, as well as deep vein thrombosis (DVT), remains one of the most important problems related to the major vessel pathology. Among the known DVT risk factors, the factors potentially related to venostasis should also be mentioned [1, 2]. Patient immobilization, calf muscle pump function impairment, as well as venous stenosis or obstruction, can lead to venous outflow disturbances and consequently to the higher risk of DVT occurrence [3, 4].

One of the most important anatomical conditions, which can potentially limit the lower leg venous outflow, is the compression of the left iliac vein in its proximal segment by the right iliac artery [5, 6]. The higher prevalence of the left lower leg thrombosis was noticed by Rudolf Virchow and subsequently confirmed by other authors. The anatomical condition concerning thickening of the lumen of the proximal left iliac vein at the point where the left iliac vein is crossed and compressed against the lumbar vertebra by overlying the right iliac artery was subsequently described and currently known under the name of May-Thurner syndrome (MTS) [7]. Several studies suggest the relatively high occurrence of this pathology, also in the asymptomatic patients, with a much higher prevalence in the patients diagnosed with proximal left leg DVT episode. According to some autopsy studies, as well as an observation based on CT/MRI scan analysis or IVUS, a significant iliac vein compression can be found in up to 22–37% of the overall adult population [8–10]. As suggested in many reports, the prevalence of MTS in the patients with proximal lower leg DVT and iliac vein involvement seems to be much higher with a reported incidence of 22–76% [11]. Overall, MTS is estimated to be the cause of 2% to 5% of all DVT [12].

Besides May-Thurner syndrome, external compression on iliac veins can occur in case of tumour mass presence or retroperitoneal space lymphadenopathy. The anatomical locations of the aortic bifurcation and inferior vena confluence suggest the possibility of other vascular pathologies causing venous outflow disturbances in this region.

One of the most common pathologies in this location is the abdominal aortic aneurysm (AAA). Surprisingly, there are very few reports available that confirm the possibility of the iliac vein compression in patients with isolated AAA. Most of the available reported cases suggest inferior vena cava thrombosis rather than typical iliac vein compression [13, 14].

One of the possible explanations of lack of the iliac vein compression (also in the presence of the large size AAA) is the possible elongation of the aorta and iliac arteries in the course of the abdominal aortic aneurysm development as well as ageing. In the study of Moreland and coworkers based on the CT scans analysis of patients with AAA, mean compression of the left common iliac vein for non-AAA patients was 37.8% and only 27.3% for the AAA population ($p < 0.0006$) [15]. The authors of this research suggest that patients with AAA were found to

have more tortuous iliac arteries that led to less anatomic compression of the LCIV compared to non-aneurysmal patients. The more obvious potential cause related to the proximal lower leg DVT development seems to be the presence of common iliac, internal iliac, or external iliac aneurysms, which can directly compress common or external iliac veins at the various levels. Several cases of the coincidence of arterial and venous pathology concerning both non-ruptured and ruptured iliac artery aneurysm were described and correspond with the clinical observations in vascular centres [16–19].

Another potentially valid pathology in the region of the aortic bifurcation and proximal iliac arteries is the presence of atherosclerotic pathology, which can lead to the Leriche syndrome development or isolated iliac artery occlusion. To the authors' knowledge, up to now, there are no studies, concerning the potential influence of the local atherosclerotic plaque development and growth in the aorta and iliac artery on the potential proximal left iliac compression.

As suggested in the studies on atherosclerosis plaque development and vessel morphology, two potential models of the plaque growth leading to the vein lumen remodelling can occur. In the first situation, the plaque grows into the vein lumen leading to stenosis and further occlusion. In the second scenario, the enlargement of the vein lumen compensates the plaque growth allowing preservation of patent vein lumen — in these cases widening of the diameter of the artery can be observed. The question of whether the presence of the atherosclerotic plaque development in the region of the anatomical coincidence of the right iliac artery and left common iliac vein can result in a higher rate of the iliac vein compression, remains to be answered. In this study, the anatomical conditions of the aortic bifurcation with possible compression of the left iliac vein by the stenotic or occluded iliac artery in the patients with Leriche syndrome were investigated.

Material and methods

In the study, 40 patients with Leriche syndrome and symptomatic peripheral atherosclerosis obliterative disease (PAOD) were evaluated. All the patients were admitted to the Department of General Surgery, Vascular Surgery, Angiology and Phlebology Medical University of Silesia and underwent a CT angiogram of the abdominal aorta and lower leg arteries before surgical or endovascular treatment due to aortoiliac occlusion. The median age of the patients was 65 (from 53 to 76). The group consisted of 21 female patients and 19 male patients.

In 5 cases the critical leg ischemia was present, while in other patients short distance of claudication was reported. The following exclusion criteria were applied: previous or current DVT or PE episode, previous arterial or venous reconstruction at the aortoiliac level (open or endovascular), pelvic or vertebral trauma as well as vertebral column operation at the lumbosacral level, presence of the aortic and/or iliac aneurysm, previous diagnosis of the May Thurner syndrome.

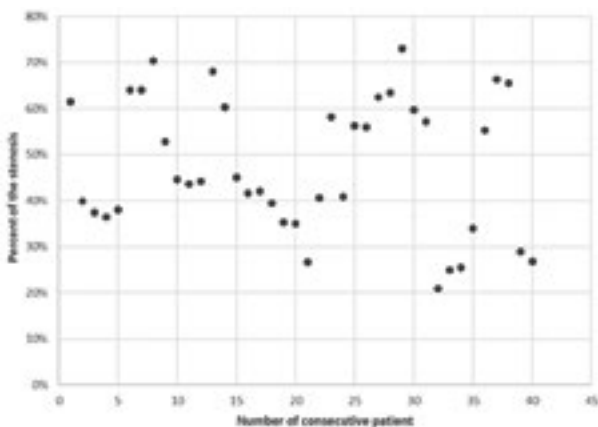


Figure 1. Percent of the left iliac vein stenosis in the study patients

The analysis assessed the diameter of the left and right iliac artery, as well as the diameter of the common left and right iliac vein. Then, the grade of stenosis of the iliac arteries was evaluated. The left common iliac vein (CIV) stenosis was evaluated by comparing the CIV vein diameter at the place of the stenosis with the contralateral common iliac vein diameter at the same level as well as with the ipsilateral common iliac vein diameter distally to the stenosis.

Results

The mean size of the left CIA was 10.13 ± 2.48 mm (10.16 ± 2.98 mm in the male population and 9.69 ± 2.81 mm in the female population). The mean diameter of the right CIA was 11.23 ± 3.08 (11.81 ± 3.4 in men and 10.46 ± 3.51 in females) and did not differ significantly from the left CIA ($p = 0.56$). Analysis of the diameter of

the left iliac vein at the crossing with the right iliac artery showed a significant reduction in the average dimension of the left iliac vein in relation to the diameter of the common iliac vein of the contralateral side. The median diameter of the left common iliac vein at the pressure site in the study population was 7.42 mm (range 3.9–9.69 mm), with a mean of 12.11 ± 2.35 mm (12.03 ± 2.7 for men and 11.8 ± 2.99 females) on the right side.

The diameter of the left CIV peripherally from the stenosis site was comparable to the diameter of the right CIV (left CIV peripherally from the stenosis 12.13 ± 2.27 mm vs. right CIV 12.11 ± 2.35 mm; $p > 0.05$). The mean degree of stenosis of the left iliac vein with its diameter peripherally from stenosis was 46% for the entire population, 45.9% for women, and 50.5% for men. When compared with the diameter of the common iliac vein on the contralateral side (right CIV), the mean degree of stenosis was 46.4% for the entire population, 44.7% for women, and 48.5% for men. Comparing the diameter at the site of left CIV stenosis with the diameter of the contralateral CIV stenosis, stenosis above 50% was diagnosed in 45% of the studied population. In the comparison of the diameter at the site of stenosis with the diameter of the peripheral segment of the left CIV, stenosis of the left CIV exceeding 50% was found in 42.5%. The distribution of the observed stenosis in the entire study population is shown in Figure 1.

In the studied population of patients with atherosclerotic lesions, three patients had complete obstruction of the distal aorta. Obstruction of the right iliac artery was found in twelve patients. The remaining patients had hemodynamically significant stenotic changes in the proximal segment of the right iliac artery (Fig. 2).

The analysis of the relationship between the presence of stenotic changes or obstruction in the right common iliac artery and the presence of significant stenosis

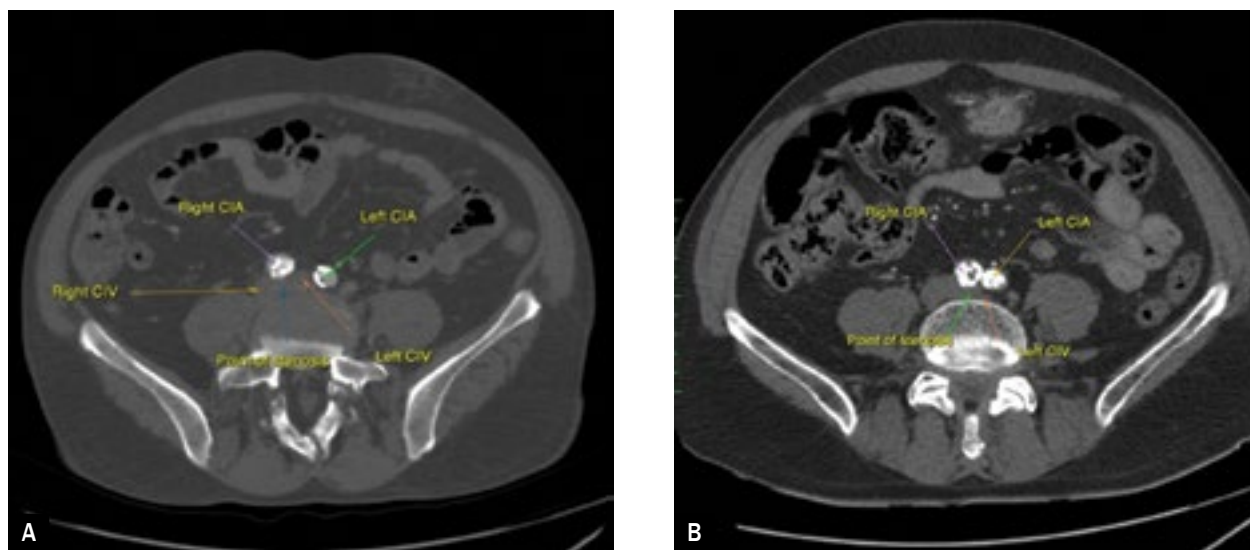


Figure 2. A, B. Examples of the left iliac vein compression in the patients with atherosclerotic changes of the proximal iliac arteries and aortic bifurcation (CIA — common iliac artery, CIV — common iliac vein)

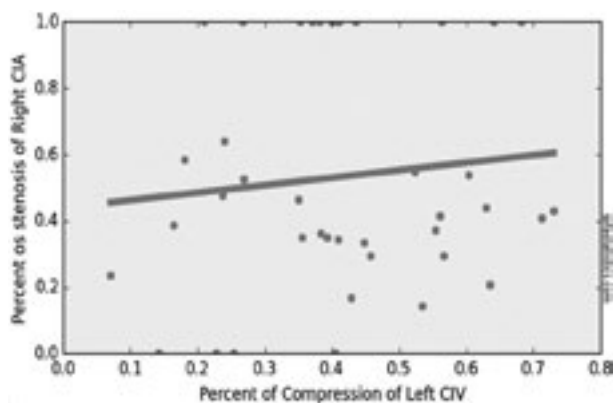


Figure 3. Relationship between the degree of stenosis or the presence of an obstruction of the right common iliac artery and the degree of compression of the left vein/ /Spearman's statistics for the entire study population, $r = 0.081$ ($p = 0.6$)

or obstruction of the left iliac vein showed no significant correlation between the degree of left CIV stenosis and the severity of changes in the right common iliac artery (Fig. 3).

Stenosis of over 50% of the left CIV was found in 18 patients (45% of the study population), including ten men and eight women, aged 53–70 (median 63.5). The degree of stenosis above 50% ranged from 53–72%: stenosis of 50–59% was observed in 7 patients (17.5%), stenosis of 60–69% in 9 patients (22.5%), and a stenosis $\geq 70\%$ concerned 2 patients (5%). According to the adopted exclusion criteria, none of these patients had a previous episode of deep vein thrombosis. In this group of patients, there was no correlation between the degree of stenosis or the presence of obstruction of the right iliac artery and the degree of compression of the left common iliac vein (in 3 patients with stenosis of the left iliac vein of above 50%, obstruction of the right common iliac artery was observed, while the remaining ones showed stenotic changes). Also, there was no statistically significant correlation between the diameter of the right iliac artery and the degree of stenosis of the left common iliac vein. Furthermore, there was no significant difference in the incidence of stenosis of above 50% between the male and female population with atherosclerotic lesions.

Discussion

Despite numerous researches dedicated to the hemodynamically significant stenosis of the venous system, we are still far from the proper definition of this pathological condition. The lack of the proven and validated hemodynamical test dedicated to the evaluation of venous stenosis implicates the use of the morphological rather than hemodynamical criteria in the patient evaluation. Traditionally, stenosis greater than 50% of the iliac artery has been considered significant [20–23].

In the study material, the left CIV stenosis exceeding 50% was observed in 45% of the study population. In none of these cases, current or previous, left leg DVT was noticed. The presence of stenosis $\geq 60\%$ was diagnosed in 27.5% and only in 2 out of 40 patients stenosis of 70% or more was found (in one case 70%, in another 72%). The reported rate of the left iliac vein over 50% compression in this study cohort (the patients with atherosclerotic obstructive changes of the distal aorta and/or proximal iliac arteries) is relatively significant and corresponds at least with some of the historical observations as well as data available in the current literature concerning the general population [11]. Nazal and coworkers, in the study based on CT scan evaluation, determined the frequency of left CIV compression in the group of 300 patients with the mean age of 51.8 and found the presence of the significant stenosis ($\geq 70\%$) in 30.6%, with the higher incidence in the female population (in this study, the presence of stenosis was not associated with the presence of clinical symptoms) [24]. Kibbe et al. in the asymptomatic patient population aged from 19 to 85 years (mean 40) described the left CIV compression as a common finding with the mean level of compression 35.5% (range: 5.6–74.8%). In this study, there was no strong correlation between the patient's age or common iliac artery size and compression of the left CIV iliac vein. The authors suggest that the compression of the left CIV may represent a normal anatomic pattern that thus far has been thought of as a pathologic condition [25].

The pathology described by May and Thurner results not only in the mechanical compression of the left CIV vein by the right common iliac artery compressing the vein against the lower lumbar vertebrae. The continuous tension, as well as pulsation on the iliac artery on the vein wall, leads to the formation of "venous spurs" [7, 20, 21]. Their presence, together with the mechanical vein compression and other potential DVT risk factors can result in a higher rate of venous thrombosis in this population. In the available literature, there is no direct confirmation of the higher coincidence of the May-Thurner syndrome-related DVT with the patient's age or atherosclerosis development. According to the clinical observation young women are at a higher risk of developing DVT compared to men especially if some additional transient risk factors are present in patients with CIV compression, which precipitates the thrombotic event (such as hormonal treatment or pregnancy) [26].

During atherogenesis, the artery tends to remodel so that the vessel lumen is often not compromised until plaques become very large (expansive remodelling). This process can lead to the widening of the vessel cross-section diameter. On the other hand, artery stenosis formation can also occur through continued plaque growth into the vein lumen (constrictive remodelling) or as a combination of these two processes. Despite the higher artery stiffness as well as the growth of the plaques within the artery wall and artery lumen leading to the artery remodelling and finally, to the significant stenosis or occlusion,

the authors could not confirm in the presented study the correlation of this process with the grade of the left CIV stenosis. In the analysis, there was also no statistically significant correlation between the right common iliac artery diameter and grade of CIV stenosis. Looking for the explanation of these findings not only the individual characteristics of the artery remodelling but also other possible factors should be taken into consideration. Among them, possible elongation of the iliac arteries related to age and atherosclerosis, as well as changes in the spine corresponding to the patient's ageing can be considered [27].

Although the association between venous and arterial thrombotic disorders remains unclear, several links between deep venous thrombosis and arterial events have been proposed [28]. Prandoni and coworkers evaluated an association between residual vein thrombosis and subclinical atherosclerosis in patients with unprovoked proximal DVT over 40 years and free from atherosclerotic disorders. In the study group, the odds ratio of subclinical atherosclerosis (increased Intima Media Complex thickness and/or presence of carotid plaques) was 2.8 (95% CI, 1.6 to 4.7) [29]. In another study by the Prandoni group, the patients with unprovoked VTE had a 60% higher risk of developing the symptomatic atherosclerotic disease than the patients with secondary venous thrombosis [30]. Yuhong and coworkers assessed the risk factors for atherosclerosis occurrence and VTE development in the meta-analysis of 33 studies and a population of almost 200 000 patients [31]. In the study, several atherosclerosis risk factors have been identified and evaluated. According to this study results, the patients with BMI ≥ 30 kg/m had a significantly higher prevalence of VTE than those with BMI < 30 kg/m. A higher rate of VTE occurrence was also confirmed in patients with arterial hypertension, diabetes as well as smoking. Another finding was the presence of higher total cholesterol, triglyceride concentration as well as high-density lipoprotein concentration in the patients with VTE than without VTE. The role of the commonly known and identified cardiovascular risk factors in increased risk and severity of the unprovoked venous thromboembolism development was also confirmed in a prospective cohort of 515 patients from the REMOTEV registry which included consecutively hospitalized patients with acute symptomatic VTE [32].

The evidence confirms that individuals with venous thrombosis may be at greater risk not only of atherosclerosis development but also of arterial event occurrence [33]. Bilora in the 14 years follow-up study documented that the risk of subsequent symptomatic atherosclerosis among patients with unprovoked DVT is approximately three times as high as that of patients with secondary events [34]. In the study of Pasha the incidence of atherothrombotic events in patients with unprovoked and provoked proximal DVT was investigated [35]. Also in this study, a higher prevalence of arterial events was found in the patients with unprovoked DVT than in provoked ones. The higher incidence of arterial events in patients

with unprovoked VTE episodes was also confirmed by other researchers [36].

Prandoni explaining the links between venous and arterial thrombosis emphasized that these two important vascular complications share several risk factors including age, obesity, smoking, diabetes mellitus, metabolic syndrome, or dyslipidemia — at least some of them have been identified in many VTE patient populations [37]. Looking for the additional factors related to them both, arterial and venous thrombosis development, endothelial dysfunction as well as inflammation can be mentioned [38–40]. Ageno, studying the patients with the first episode of idiopathic DVT and control patients suggests that the presence of the metabolic syndrome may play a role in the pathogenesis of idiopathic DVT and may act as the link between venous thrombosis and atherosclerosis [41].

According to the clinical observations, in some of the patients, acute arterial events such as ischemic stroke or myocardial infarction are likely to predict the subsequent development of venous thromboembolic (VTE) complications [40, 42]. Piazza and coworkers documented a high risk of venous thromboembolism in atherosclerosis patients in the population-based Worcester VTE [43]. Of the 818 included patients with VTE, 26% had a history of symptomatic atherosclerosis defined as a history of ischemic heart disease, positive cardiac catheterization, percutaneous or surgical coronary intervention, or history of peripheral artery disease. In further analysis, the patients with atherosclerosis-related events were significantly older, more likely to have immobility, prior heart failure, chronic lung disease, or cerebrovascular disease compared with non-atherosclerosis patients. Prasad investigated if the presence of coronary endothelial dysfunction predicts the development of VTE in 502 patients presenting with coronary atherosclerosis without critical stenoses [44]. Based on the follow-up analysis, coronary endothelial dysfunction was associated with an increased risk of VTE development.

Our study focused on asymptomatic patients without current or previous VTE episodes with simultaneous advanced and symptomatic atherosclerotic changes in the region of the aortic bifurcation. Based on the analysis, the left iliac vein compression is a common finding in this study population, corresponding to the data from some other studies based on the image study analysis in the general population. As documented, the severity of the left iliac vein stenosis is not related to the severity of the arterial stenosis or the complete iliac artery occlusion related to the atherosclerotic plaque growth in this particular location. The potential widening of the iliac arteries related to the excentric atherosclerotic plaque enlargement seems to be only the part of the artery remodelling process in this particular location and does not result in a higher rate of significant left iliac vein stenosis. Taking into account the risk of the DVT development in the surgical and medical patients with distal aortic and/or iliac artery occlusion other factors should also be taken into consideration (including advanced age, obesity, immo-

bilization as well as the presence of the comorbidities). As mentioned above, the presence of the left iliac vein stenosis in this patient cohort corresponds with the data from some other reports and should be evaluated as just one more potential DVT occurrence risk factor. In this study, only 5% of the patients' stenosis equal to or higher than 70% was reported but the overall rate of the stenosis over 50% was 45%.

Some limitations of this study should be mentioned, including the lack of the control group as well as the limited size of the patient cohort. There was also no follow-up available except the hospitalization time when none of the patients developed proximal DVT potentially related to the left iliac vein stenosis.

Conclusion

The asymptomatic left common iliac vein stenosis can be found in a significant number of patients with atherosclerotic changes in the aortic bifurcation and iliac arteries. Due to the complex process of arterial wall remodelling in patients with atherosclerosis including plaque growth, possible vessel widening, and elongation, the clinical importance of these observations should be investigated in further studies.

References

- Malone PC, Agutter PS, Malone PC, et al. The aetiology of deep venous thrombosis. *QJM*. 2006; 99(9): 581–593, doi: [10.1093/qjmed/hcl070](https://doi.org/10.1093/qjmed/hcl070), indexed in Pubmed: [16905749](https://pubmed.ncbi.nlm.nih.gov/16905749/).
- Bulger CM, Jacobs C, Patel NH. Epidemiology of acute deep vein thrombosis. *Tech Vasc Interv Radiol*. 2004; 7(2): 50–54, doi: [10.1053/j.tvir.2004.02.001](https://doi.org/10.1053/j.tvir.2004.02.001), indexed in Pubmed: [15252760](https://pubmed.ncbi.nlm.nih.gov/15252760/).
- Kearon C. Epidemiology of venous thromboembolism. *Semin Vasc Med*. 2001; 1(1): 7–26, doi: [10.1055/s-2001-14668](https://doi.org/10.1055/s-2001-14668), indexed in Pubmed: [15199510](https://pubmed.ncbi.nlm.nih.gov/15199510/).
- Cushman M. Epidemiology and risk factors for venous thrombosis. *Semin Hematol*. 2007; 44(2): 62–69, doi: [10.1053/j.seminhematol.2007.02.004](https://doi.org/10.1053/j.seminhematol.2007.02.004), indexed in Pubmed: [17433897](https://pubmed.ncbi.nlm.nih.gov/17433897/).
- Liddell RP, Evans NS. May-Thurner syndrome. *Vasc Med*. 2018; 23(5): 493–496, doi: [10.1177/1358863X18794276](https://doi.org/10.1177/1358863X18794276), indexed in Pubmed: [30187833](https://pubmed.ncbi.nlm.nih.gov/30187833/).
- Harbin MM, Lutsey PL. May-Thurner syndrome: History of understanding and need for defining population prevalence. *J Thromb Haemost*. 2020; 18(3): 534–542, doi: [10.1111/jth.14707](https://doi.org/10.1111/jth.14707), indexed in Pubmed: [31821707](https://pubmed.ncbi.nlm.nih.gov/31821707/).
- MAY R, THURNER J. The cause of the predominantly sinistral occurrence of thrombosis of the pelvic veins. *Angiology*. 1957; 8(5): 419–427, doi: [10.1177/000331975700800505](https://doi.org/10.1177/000331975700800505), indexed in Pubmed: [13478912](https://pubmed.ncbi.nlm.nih.gov/13478912/).
- Sermathanasawadi N, Pruekprasert K, Pitaksantayothin W, et al. Prevalence, risk factors, and evaluation of ilioacaval obstruction in advanced chronic venous insufficiency. *J Vasc Surg Venous Lymphat Disord*. 2019; 7(3): 441–447, doi: [10.1016/j.jvsv.2018.10.021](https://doi.org/10.1016/j.jvsv.2018.10.021), indexed in Pubmed: [30765330](https://pubmed.ncbi.nlm.nih.gov/30765330/).
- Marston W, Fish D, Unger J, et al. Incidence of and risk factors for ilioacaval venous obstruction in patients with active or healed venous leg ulcers. *J Vasc Surg*. 2011; 53(5): 1303–1308, doi: [10.1016/j.jvs.2010.10.120](https://doi.org/10.1016/j.jvs.2010.10.120), indexed in Pubmed: [21215568](https://pubmed.ncbi.nlm.nih.gov/21215568/).
- Kalu S, Shah P, Natarajan A, et al. May-thurner syndrome: a case report and review of the literature. *Case Rep Vasc Med*. 2013; 2013: 740182, doi: [10.1155/2013/740182](https://doi.org/10.1155/2013/740182), indexed in Pubmed: [23509664](https://pubmed.ncbi.nlm.nih.gov/23509664/).
- Harbin MM, Lutsey PL. May-Thurner syndrome: History of understanding and need for defining population prevalence. *J Thromb Haemost*. 2020; 18(3): 534–542, doi: [10.1111/jth.14707](https://doi.org/10.1111/jth.14707), indexed in Pubmed: [31821707](https://pubmed.ncbi.nlm.nih.gov/31821707/).
- Mousa AY, AbuRahma AF. May-Thurner syndrome: update and review. *Ann Vasc Surg*. 2013; 27(7): 984–995, doi: [10.1016/j.avsg.2013.05.001](https://doi.org/10.1016/j.avsg.2013.05.001), indexed in Pubmed: [23850314](https://pubmed.ncbi.nlm.nih.gov/23850314/).
- Ikeda A. Inferior Vena Cava Compression Caused by a Retroperitoneal Hematoma Following an Abdominal Aortic Aneurysm Rupture. *Ann Vasc Dis*. 2019; 12(1): 87–90, doi: [10.3400/avd.cr.18-00110](https://doi.org/10.3400/avd.cr.18-00110), indexed in Pubmed: [30931067](https://pubmed.ncbi.nlm.nih.gov/30931067/).
- Moore RD, Rutter ED, Zapko DR, et al. Abdominal aortic aneurysm with inferior vena cava compression in association with deep venous thrombosis. *Am J Med Sci*. 2013; 346(6): 521–522, doi: [10.1097/MAJ.0b013e3182a55a96](https://doi.org/10.1097/MAJ.0b013e3182a55a96), indexed in Pubmed: [24263082](https://pubmed.ncbi.nlm.nih.gov/24263082/).
- Moreland NC, Ujiki M, Matsumura JS, et al. Decreased incidence of left common iliac vein compression in patients with abdominal aortic aneurysms. *J Vasc Surg*. 2006; 44(3): 595–600, doi: [10.1016/j.jvs.2006.05.046](https://doi.org/10.1016/j.jvs.2006.05.046), indexed in Pubmed: [16950440](https://pubmed.ncbi.nlm.nih.gov/16950440/).
- Walsh JJ, Williams LR, Driscoll JL, et al. Vein compression by arterial aneurysms. *J Vasc Surg*. 1988; 8(4): 465–469, indexed in Pubmed: [3172384](https://pubmed.ncbi.nlm.nih.gov/3172384/).
- Secil M, Sarisoy HT, Hazan E, et al. Iliac artery aneurysm presenting with lower extremity deep vein thrombosis. *J Emerg Med*. 2003; 24(1): 65–67, doi: [10.1016/s0736-4679\(02\)00669-8](https://doi.org/10.1016/s0736-4679(02)00669-8), indexed in Pubmed: [12554043](https://pubmed.ncbi.nlm.nih.gov/12554043/).
- Venturini L, Grande R, Sapienza P. A Rare Presentation of an Isolated Internal Iliac Artery Aneurysm: Report of a Case and Literature Review. *Vasc Endovascular Surg*. 2017; 51(5): 320–323, doi: [10.1177/1538574417702778](https://doi.org/10.1177/1538574417702778), indexed in Pubmed: [28399793](https://pubmed.ncbi.nlm.nih.gov/28399793/).
- Khang NC, Hanif H, Zainal Ariffin A. Rare cause of lower limb deep venous thrombosis: a case report. *Med J Malaysia*. 2014; 69(3): 144–145, indexed in Pubmed: [25326359](https://pubmed.ncbi.nlm.nih.gov/25326359/).
- Neglén P, Thrasher TL, Raju S. Venous outflow obstruction: An underestimated contributor to chronic venous disease. *J Vasc Surg*. 2003; 38(5): 879–885, doi: [10.1016/s0741-5214\(03\)01020-6](https://doi.org/10.1016/s0741-5214(03)01020-6), indexed in Pubmed: [14603188](https://pubmed.ncbi.nlm.nih.gov/14603188/).
- Raju S, Neglen P. High prevalence of nonthrombotic iliac vein lesions in chronic venous disease: a permissive role in pathogenicity. *J Vasc Surg*. 2006; 44(1): 136–43; discussion 144, doi: [10.1016/j.jvs.2006.02.065](https://doi.org/10.1016/j.jvs.2006.02.065), indexed in Pubmed: [16828437](https://pubmed.ncbi.nlm.nih.gov/16828437/).
- Labropoulos N, Borge M, Pierce K, et al. Criteria for defining significant central vein stenosis with duplex ultrasound. *J Vasc Surg*. 2007; 46(1): 101–107, doi: [10.1016/j.jvs.2007.02.062](https://doi.org/10.1016/j.jvs.2007.02.062), indexed in Pubmed: [17540535](https://pubmed.ncbi.nlm.nih.gov/17540535/).
- Gagne PJ, Gasparis A, Black S, et al. Analysis of threshold stenosis by multiplanar venogram and intravascular ultrasound examination for predicting clinical improvement after iliofemoral vein stenting in the VIDIO trial. *J Vasc Surg Venous Lymphat Disord*. 2018; 6(1): 48–56.e1, doi: [10.1016/j.jvsv.2017.07.009](https://doi.org/10.1016/j.jvsv.2017.07.009), indexed in Pubmed: [29033314](https://pubmed.ncbi.nlm.nih.gov/29033314/).
- Nazzal M, El-Fedaly M, Kazan V, et al. Incidence and clinical significance of iliac vein compression. *Vascular*. 2015; 23(4): 337–343, doi: [10.1177/1708538114551194](https://doi.org/10.1177/1708538114551194), indexed in Pubmed: [25398228](https://pubmed.ncbi.nlm.nih.gov/25398228/).
- Kibbe MR, Ujiki M, Goodwin AL, et al. Iliac vein compression in an asymptomatic patient population. *J Vasc Surg*. 2004; 39(5): 937–943, doi: [10.1016/j.jvs.2003.12.032](https://doi.org/10.1016/j.jvs.2003.12.032), indexed in Pubmed: [15111841](https://pubmed.ncbi.nlm.nih.gov/15111841/).
- Brinegar KN, Sheth RA, Khademhosseini A, et al. Iliac vein compression syndrome: Clinical, imaging and pathologic findings. *World J Radiol*. 2015; 7(11): 375–381, doi: [10.4329/wjr.v7.i11.375](https://doi.org/10.4329/wjr.v7.i11.375), indexed in Pubmed: [26644823](https://pubmed.ncbi.nlm.nih.gov/26644823/).

27. Smedby O, Bergstrand L. Tortuosity and atherosclerosis in the femoral artery: what is cause and what is effect? *Ann Biomed Eng.* 1996; 24(4): 474–480, doi: [10.1007/BF02648109](https://doi.org/10.1007/BF02648109), indexed in Pubmed: [8841722](https://pubmed.ncbi.nlm.nih.gov/8841722/).
28. Milan M, Vedovetto V, Bilora F, et al. Further evidence in support of the association between venous thrombosis and atherosclerosis: a case-control study. *Thromb Res.* 2014; 134(5): 1028–1031, doi: [10.1016/j.thromres.2014.09.007](https://doi.org/10.1016/j.thromres.2014.09.007), indexed in Pubmed: [25248687](https://pubmed.ncbi.nlm.nih.gov/25248687/).
29. Prandoni P, Ciammaichella M, Mumoli N, et al. Veritas Investigators. An association between residual vein thrombosis and subclinical atherosclerosis: Cross-sectional study. *Thromb Res.* 2017; 157: 16–19, doi: [10.1016/j.thromres.2017.06.036](https://doi.org/10.1016/j.thromres.2017.06.036), indexed in Pubmed: [28679112](https://pubmed.ncbi.nlm.nih.gov/28679112/).
30. Prandoni P, Ghirarduzzi A, Prins MH, et al. Venous thromboembolism and the risk of subsequent symptomatic atherosclerosis. *J Thromb Haemost.* 2006; 4(9): 1891–1896, doi: [10.1111/j.1538-7836.2006.02058.x](https://doi.org/10.1111/j.1538-7836.2006.02058.x), indexed in Pubmed: [16961597](https://pubmed.ncbi.nlm.nih.gov/16961597/).
31. Mi Y, Yan S, Lu Y, et al. Venous thromboembolism has the same risk factors as atherosclerosis: A PRISMA-compliant systemic review and meta-analysis. *Medicine (Baltimore).* 2016; 95(32): e4495, doi: [10.1097/MD.0000000000004495](https://doi.org/10.1097/MD.0000000000004495), indexed in Pubmed: [27512866](https://pubmed.ncbi.nlm.nih.gov/27512866/).
32. Gaertner S, Cordeanu EM, Mirea C, et al. Increased risk and severity of unprovoked venous thromboembolism with clustering cardiovascular risk factors for atherosclerosis: Results of the REMOTEV registry. *Int J Cardiol.* 2018; 252: 169–174, doi: [10.1016/j.ijcard.2017.11.055](https://doi.org/10.1016/j.ijcard.2017.11.055), indexed in Pubmed: [29169908](https://pubmed.ncbi.nlm.nih.gov/29169908/).
33. Green D. Risk of future arterial cardiovascular events in patients with idiopathic venous thromboembolism. *Hematology Am Soc Hematol Educ Program.* 2009: 259–266, doi: [10.1182/asheducation-2009.1.259](https://doi.org/10.1182/asheducation-2009.1.259), indexed in Pubmed: [20008208](https://pubmed.ncbi.nlm.nih.gov/20008208/).
34. Bilora F, Ceresa M, Milan M, et al. The impact of deep vein thrombosis on the risk of subsequent cardiovascular events: a 14-year follow-up study. *Int Angiol.* 2017; 36(2): 156–159, doi: [10.23736/s0392-9590.16.03664-6](https://doi.org/10.23736/s0392-9590.16.03664-6), indexed in Pubmed: [26883440](https://pubmed.ncbi.nlm.nih.gov/26883440/).
35. Pasha SM, Tan M, van Rees Vellinga TFD, et al. Risk of atherothrombotic events in patients after proximal deep-vein thrombosis. *Blood Coagul Fibrinolysis.* 2016; 27(1): 13–18, doi: [10.1097/MBC.0000000000000228](https://doi.org/10.1097/MBC.0000000000000228), indexed in Pubmed: [25402192](https://pubmed.ncbi.nlm.nih.gov/25402192/).
36. Bova C, Marchiori A, Noto A, et al. Incidence of arterial cardiovascular events in patients with idiopathic venous thromboembolism. A retrospective cohort study. *Thromb Haemost.* 2006; 96(2): 132–136, indexed in Pubmed: [16894454](https://pubmed.ncbi.nlm.nih.gov/16894454/).
37. Prandoni P, Piovella C, Pesavento R. Venous thromboembolism and arterial complications. *Semin Respir Crit Care Med.* 2012; 33(2): 205–210, doi: [10.1055/s-0032-1311801](https://doi.org/10.1055/s-0032-1311801), indexed in Pubmed: [22648494](https://pubmed.ncbi.nlm.nih.gov/22648494/).
38. Folsom AR, Lutsey PL, Heckbert SR, et al. Longitudinal increases in blood biomarkers of inflammation or cardiovascular disease and the incidence of venous thromboembolism. *J Thromb Haemost.* 2018; 16(10): 1964–1972, doi: [10.1111/jth.14241](https://doi.org/10.1111/jth.14241), indexed in Pubmed: [30007116](https://pubmed.ncbi.nlm.nih.gov/30007116/).
39. Riva N, Donadini MP, Ageno W. Epidemiology and pathophysiology of venous thromboembolism: similarities with atherothrombosis and the role of inflammation. *Thromb Haemost.* 2015; 113(6): 1176–1183, doi: [10.1160/TH14-06-0563](https://doi.org/10.1160/TH14-06-0563), indexed in Pubmed: [25472800](https://pubmed.ncbi.nlm.nih.gov/25472800/).
40. Prandoni P. Is there a link between venous and arterial thrombosis? A reappraisal. *Intern Emerg Med.* 2020; 15(1): 33–36, doi: [10.1007/s11739-019-02238-6](https://doi.org/10.1007/s11739-019-02238-6), indexed in Pubmed: [31773560](https://pubmed.ncbi.nlm.nih.gov/31773560/).
41. Ageno W, Prandoni P, Romualdi E, et al. The metabolic syndrome and the risk of venous thrombosis: a case-control study. *J Thromb Haemost.* 2006; 4(9): 1914–1918, doi: [10.1111/j.1538-7836.2006.02132.x](https://doi.org/10.1111/j.1538-7836.2006.02132.x), indexed in Pubmed: [16848878](https://pubmed.ncbi.nlm.nih.gov/16848878/).
42. Wattanakit K, Lutsey PL, Bell EJ, et al. Association between cardiovascular disease risk factors and occurrence of venous thromboembolism. A time-dependent analysis. *Thromb Haemost.* 2012; 108(3): 508–515, doi: [10.1160/TH11-10-0726](https://doi.org/10.1160/TH11-10-0726), indexed in Pubmed: [22782466](https://pubmed.ncbi.nlm.nih.gov/22782466/).
43. Piazza G, Goldhaber SZ, Lessard DM, et al. Venous thromboembolism in patients with symptomatic atherosclerosis. *Thromb Haemost.* 2011; 106(6): 1095–1102, doi: [10.1160/TH11-07-0469](https://doi.org/10.1160/TH11-07-0469), indexed in Pubmed: [22012325](https://pubmed.ncbi.nlm.nih.gov/22012325/).
44. Prasad M, McBane R, Reriani M, et al. Coronary endothelial dysfunction is associated with increased risk of venous thromboembolism. *Thromb Res.* 2016; 139: 17–21, doi: [10.1016/j.thromres.2015.12.024](https://doi.org/10.1016/j.thromres.2015.12.024), indexed in Pubmed: [26916291](https://pubmed.ncbi.nlm.nih.gov/26916291/).

Address for correspondence:

Prof. dr hab. n. med. Tomasz Urbanek
Department of General Surgery Vascular Surgery,
Angiology and Phlebology, Medical University of Silesia, Katowice
Ziolowa 45/47, 40–635 Katowice
e-mail: urbanek.tom@interia.pl

Praca wpłynęła do Redakcji: 07.05.2019 r.