Iliac vein compression syndrome

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Abstract

Iliac vein compression syndrome (IVCS) is a clinical situation in which the right common iliac artery extrinsically compresses the left common iliac vein. Patients with these anatomical changes are predisposed to the formation of deep venous thrombosis on the left side with signs of venous hypertension in the left lower limb (LLL), simulating diseases of similar evolution. In IVCS, due to the existence of an anatomical change, vein decompression is required, either by conventional open surgery or endovascular procedure with stent placement. This study reports the case of a 48-year-old male patient who sought a specialist in vascular surgery due to an increase in varicose veins, venous claudication, and worsening of pain and edema in the LLL, for a long period of time.

Keywords: Iliac Vein. Iliac Artery. Varicose Veins. Edema. Phlebography.

Introduction

Iliac vein compression syndrome (IVCS), also called May-Thurner or Cockett syndrome, is a clinical situation in which the right common iliac artery extrinsically compresses the left common iliac vein, manifesting clinically as pain and edema of the left lower limb (LLL), or even as left iliofemoral deep venous thrombosis (DVT).[1,2]

Such compression can lead to obstruction of the iliac vein by two mechanisms. The first involves the mechanical compression of the vein itself against the vertebral column, while the second is related to intimal hypertrophy of the vein caused by repeated dynamic compression.[1]

Patients with this anatomical change are predisposed to the formation of left iliofemoral DVT, with surgery, pregnancy and prolonged bed rest being the main factors that precipitate acute episodes.[3]

Invasive treatment of IVCS is necessary when the patient is symptomatic, and its main objective is to treat the symptoms related to venous hypertension and to prevent the sequelae of venous occlusive disease.[4]

Because of the complexity and morbidity of open surgery, the endovascular approach has been becoming the main therapeutic choice, as it shows lower surgical risk and greater effectiveness.[5] To treat chronic symptoms, a stent is used for venous decompression; for acute symptoms, such as DVT, cath-

eter-directed thrombolysis is used.[6] O´Sullivan et al. reported that endovascular treatment of IVCS is safe and effective; however, further studies are needed to evaluate the long-term patency of these stents.[7] In a way, the complexity and risks of open surgery naturally limited the indiscriminate performance of the treatment, with florid symptoms being required for surgery to be justified; however, with the advent of endovascular treatment,[8] it can be seen that oligosymptomatic and sometimes asymptomatic patients are undergoing invasive procedures to treat an imaging phenomenon.[9] The present article aims to show the importance of clinical suspicion in the diagnosis of IVCS and the correct indication for invasive treatment.

Case report

A 48-year-old male farmer sought a specialist with a complaint of pain and edema in the left lower limb for six years. He reported waking up with edema that worsened during the day, associated with pain triggered by intense walking or prolonged standing. The pain improved with rest and use of an elastic stocking. He denied other comorbidities or previous surgeries, trauma or fractures of the lower limbs, and had no personal or family history of DVT of the lower limbs.

His first consultation with a vascular surgeon was in 2004, with a complaint of varicose veins. On that occasion, he underwent color Doppler ultrasonography, which showed absence of reflux in the deep and superficial veins and only superficial venous ectasias on the medial aspect of the left leg. He did not undergo any specific venous treatment during that period.

In July 2017, he sought the vascular surgeon because of an increase in the number of varicose veins, venous claudication in the LLL and frequent symptoms of pain and edema in the LLL. Another Doppler ultrasound mapping was then performed, which again showed absence of reflux in the deep and superficial veins of the LLL, with insufficiency of superficial veins on the medial aspect of the left leg and the presence of ectatic veins in that region. Because of a marked asymmetry in calf circumference, the left being 8 cm larger than the right (Figure 1), and the presence of plantar cyanosis in the left foot, CT angiography of the pelvic and abdominal veins was performed in August 2017, which showed signs of extrinsic compression of the left common iliac vein by the right common iliac artery, associated with an extensive venous collateral network on the left side, characterizing IVCS (Figure 2).



Figure 1. Photograph showing the left calf with a circumference 8 cm greater than the right calf.

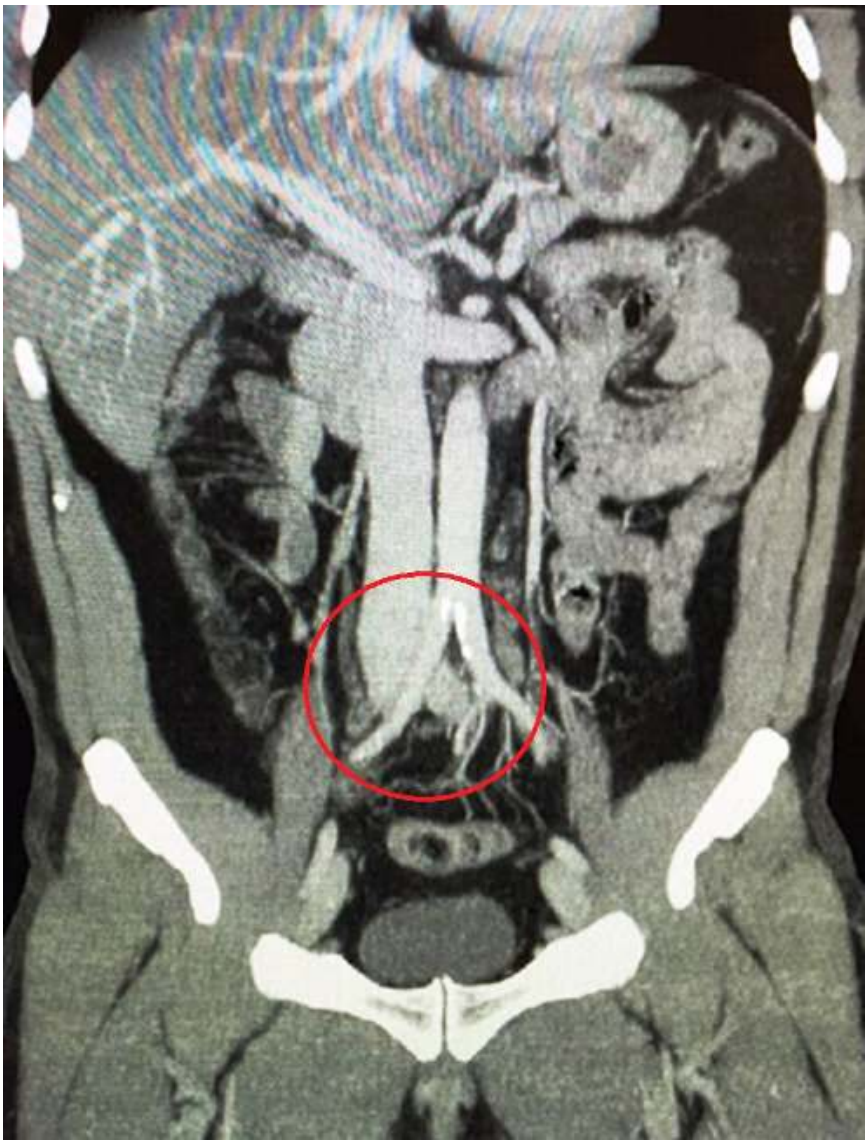


Figure 2. CT venography of the pelvic and abdominal veins showing the right common iliac artery compressing the left common iliac vein.

Venography was also performed, which showed a filling defect in the left common iliac vein and a florid venous collateral circulation (Figure 3). In addition to clinical treatment with elastic compression, en - dovascular surgical treatment was proposed to the patient to eliminate or reduce the stenosis of the left common iliac vein and thereby relieve the patient's venous hypertension (Figure 4).

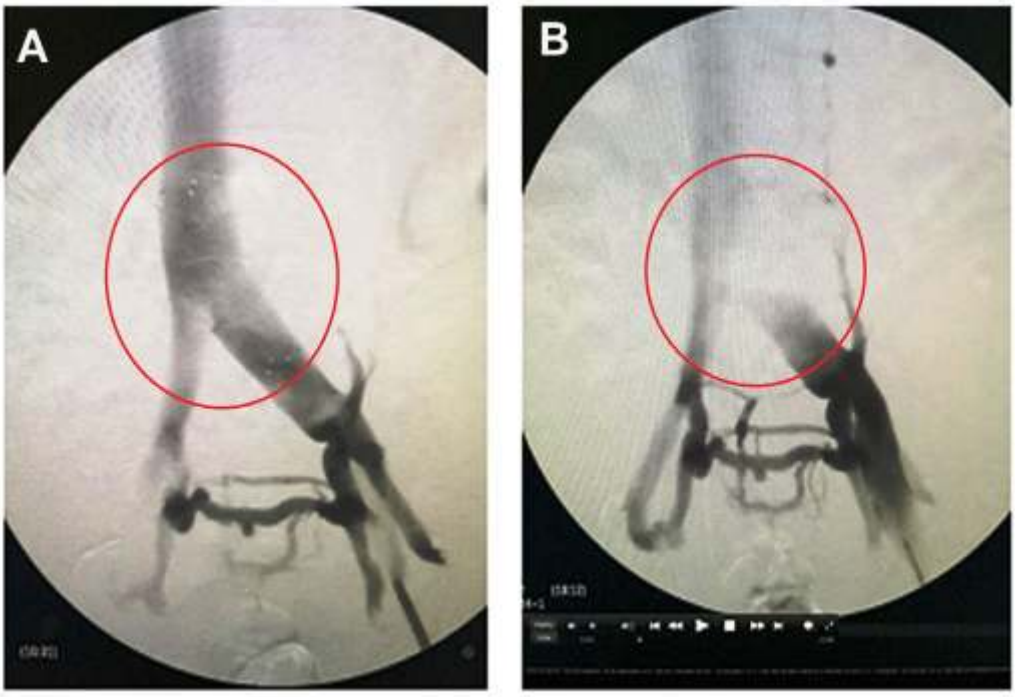


Figure 3. Venography showing a filling defect in the left common iliac vein and florid venous collateral circulation (A), and a filling defect with venous collateral circulation (B).



Figure 4. Angioplasty and stent deployment in the stenotic area.

Discussion

The presence of symptoms in patients with the iliac vein compression phenomenon is not very com - mon.[10] Studies that evaluated the incidence of IVCS in asymptomatic patients showed that it is very low.[10] As for those who present symptoms related to chronic venous disease of the lower limbs, IVCS affects 2 to 5% of patients with venous disease of the lower limbs and is present in 18 to 49% of patients with DVT of the LLL.[1,11] There is a predominance in women, with a mean age of 34.4 years in the Brazilian population.[12]

The current gold standard for defining hemodynamically significant stenosis is phlebography and in - travascular ultrasound; however, they are invasive and expensive examinations, reserved for the time of surgery.

Doppler ultrasound is a method that allows the presence of significant obstruction to be identified in a less invasive way, showing the degree of compression and the blood flow velocity.

Therefore, the hemodynamic changes and the diagnosis of target events (acute DVT) should initially be based on clinical and ultrasound examination.[10]

Even so, Doppler ultrasound of the lower limbs alone cannot exclude proximal venous compression, owing to numerous factors: it is examiner-dependent; it requires compression of the transducer on the

pelvis; it is a limited examination for visualizing the iliac vessels located deeper in the pelvis; and it does not reveal anatomical features specific to IVCS. Furthermore, if the clinical presentation is highly suggestive of proximal venous compression, the investigation should be continued.[13]

CT angiography of the abdomen and pelvis is useful and reliable in the diagnosis of IVCS, demonstrating the compression of the iliac vein and allowing extrinsic causes of compression to be excluded, but it is expensive and requires radiation, and is indicated for patients with high clinical suspicion.[10]

In a prospective study published in January 2017, a sample of 500 asymptomatic patients who underwent computed tomography was used, divided into groups according to sex and subdivided by age above or below 40 years.[10] Of the 500 initial patients, 367 completed the study, 17 were diagnosed with target events during follow-up and, of these, only 6 (1.6%) had IVCS. It was therefore concluded that, for asymptomatic patients, further investigation with CT angiography is unnecessary.[11] In cases of significant changes on Doppler ultrasound (degree of compression > 50%) associated with signs of asymmetric venous hypertension, CT angiography should be performed.[10]

The diagnosis of IVCS is more associated with acute events such as DVT and the sudden onset of asymmetric edema in the LLL than with non-thrombotic venous hypertension, whose clinical presentation evolves more insidiously and less floridly.[6] In the patient above, the clinical suspicion raised by the signs of venous hypertension (varicose veins, edema, cutaneous trophic changes) in the LLL, together with color Doppler ultrasonography, was decisive for the suspicion of IVCS, which was confirmed by CT angiography.

Therefore, for the diagnosis of this disease, it is essential to be attentive to all clinical changes of venous hypertension, predominantly in the LLL, suggestive of IVCS that require more detailed investigation, reducing the number of underdiagnosed cases and inappropriate management.

The aim of treatment is to improve the symptoms and prevent thromboembolic phenomena. At present, there is no consensus in the literature regarding the ideal treatment for patients with IVCS who have not yet developed DVT. However, symptomatic patients, with impaired quality of life and risks of acute and chronic complications,[1] should be treated.

In IVCS, since there is an anatomical change, decompression of the vein by conventional open or endovascular surgery is necessary, the endovascular technique being the current method of choice both to treat iliofemoral DVT and to correct the anatomical abnormality with stent placement,[1] as it is considered a safe and effective alternative compared with open surgical treatment.[14]

Although stent implantation is routinely performed for peripheral arterial disease, there are few data on the safety and long-term patency of stents implanted for venous disease,[15] which raises some uncertainty regarding the ideal treatment: how to implant a stent in a highly thrombogenic venous system in a relatively young and oligosymptomatic patient, who will need periodic follow-up throughout life and anticoagulation for at least six months after the stent placement procedure.

Another factor is that the stents currently available are much larger than the zone where the artery compresses the iliac vein, increasing the thrombogenic surface, besides not being dedicated to this specific anatomical location, which shows that there is a lack of stents specific to this syndrome.

In conclusion, IVCS may manifest clinically with signs of venous hypertension in the LLL or even with left iliofemoral DVT, simulating diseases of similar evolution. It is therefore underdiagnosed and mismanaged, and it is concluded that suggestive cases require more detailed investigation, to increase diagnostic accuracy and ensure appropriate therapeutic management, reducing morbidity and mortality.

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