Isolated External Iliac Vein Occlusion Presenting as Primary Chronic Venous Disease (CVD) - a Diagnostic Pitfall

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Case Report

Abstract: Secondary varicose veins/secondary CVD is most often due to deep venous occlusion. The causes for deep venous occlusion can be thrombotic or non-thrombotic. Irrespective of the cause, the manifestations are similar in secondary CVD and the management is usually conservative or reconstructive venous surgery. In contrast, primary varicose veins are treated by ablative surgery. Applying ablative venous surgery to secondary varicose veins will prove disastrous. Here we present a case of isolated iliac vein occlusion manifesting as primary CVD on color Doppler study and discuss how to overcome this diagnostic pitfall and avoid disastrous consequences of mistreatment.

Keywords: iliac vein occlusion, secondary CVD, venous ulcer.

Introduction

Chronic venous disease (CVD) is a common disorder affecting the lower limb veins. Manifestations of CVD include leg heaviness, aching and varicose veins, edema, lipodermatosclerosis, eczema and venous ulcers. Edema, lipodermatosclerosis and ulcers are thought to represent severe CVD. People with severe symptoms are said to have chronic venous insufficiency (CVI). According to the CEAP classification[1], primary chronic venous disorders are those not associated with an identifiable of venous pathology or dysfunction. The natural history and treatment are different for primary and secondary CVD. Primary CVD is usually due to degenerative changes in venous wall and valves. It is usually mild, involves the superficial and perforator veins. Primary CVD is a slowly progressive disorder that may progress to C4 – C6 manifestations over a period of many years. Venotensive changes such as edema, lipodermatosclerosis, and active ulceration develop in only about 0.5% of patients with primary CVD. Surgical management usually involves varicose vein surgery. Where as, in secondary CVD, there is obstruction of deep veins and consequently venotensive changes are prominent. Superficial varicosities develop within two years after thrombosis or obstruction. Management of secondary CVD is usually conservative. Surgical management involves venous reconstructive surgery or stenting of the occluded vein. Removal of varicose veins will usually worsen the condition of the limb. Here we present a case of iliac vein occlusion (secondary CVD) presenting as primary CVD and suggest how to overcome this diagnostic pitfall.

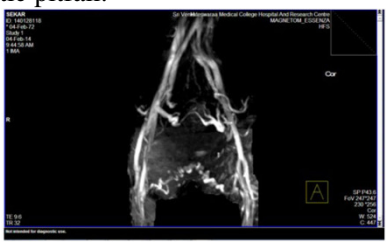

Figure 1: 2D MR venogram (TOF) showing no flow in the left external iliac vein. Terminal portion of the common femoral vein is seen and also the common iliac vein

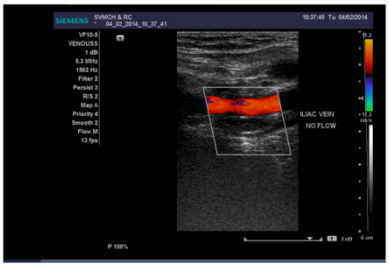

Figure 2: Color Doppler study showing flow in the left external iliac artery and no flow in the accompanying external iliac vein

Case Report

A 42 year old male patient was admitted in our hospital with the complaints of swelling of the leg of one week duration and multiple ulcers in the dorsum of left foot for one week duration. The swelling was insidious onset and was aggravated by walking and on long travel. The ulcers on the dorsum of left foot are small and have no discharge. There was no co-morbid illness and no previous history of surgery. There was no history of contact with tuberculous patients or chronic drug intake. He is a tobacco chewer. Family history was not contributory. On clinical examination, mild edema was

present in the left lower limb. Hyperpigmentation over the lower third of left leg was present. Multiple healing ulcers were present in the dorsum of the left foot. Small to medium sized varicose veins were present in the leg, both on the medial and posterior aspect. In the thigh, even though the GSV was not grossly enlarged on inspection, palpation revealed minor dilatation of the GSV also. All the arterial pulsations in the left lower limb are normal. On conservative management, the edema lessened ad the ulcers healed. Color Doppler examination of the left lower limb veins showed common femoral, popliteal, anterior and posterior tibial veins are normal in their course and caliber. No evidence of any deep vein thrombosis. Saphenofemoral and saphenopopliteal junctions are incompetent. Varicosities of GSV and LSV of the left lower limb with incompetent perforators at the following levels: medial side of the leg - upper 1/3 and middle 1/3; posterior - middle 1/3; lateral – middle 1/3. Based on these investigations a diagnosis of Primary CVD was made and it was decided to perform surgery for the varicose veins. But during preoperative planning, the presence of extensive venotensive changes (edema, hyperpigmentation and ulcers) with comparatively small varicose veins which did not correlate well with the extensive reflux suggested by the color Doppler made us to ask for a repeat color Doppler with the surgeon present during the study. During the repeat study, spontaneous reflux was seen in SFJ even in supine position, indicating probable proximal obstruction. Hence a MR Venogram of pelvic veins was done. 2D MR venogram showed no flow in the distal left common iliac vein and external iliac vein. Left external iliac vein appears smaller in caliber and cord like with no flow in the lumen. Pelvic collateral veins are seen. Left common femoral vein is patent and IVC appears normal (Fig.1). Color Doppler of the iliac veins also showed evidence of vein wall thickening and no flow in the external iliac vein (Fig.2). Based on these studies, the diagnosis was revised to left external iliac venous occlusion and secondary varicose veins with healed ulcers (CEAP class5) and the patient put on conservative management. We presume that the cause of the occlusion may be due to previous thrombotic episode which went unrecognized.

Discussion

Isolated iliac vein occlusion is rare. It is difficult to diagnose because compression ultrasonography of lower limb veins is usually normal and pelvic veins are not imaged as a routine procedure. More over, imaging of pelvic vein by duplex has its limitations. Both thrombotic and nonthrombotic conditions contribute to iliac vein occlusion. The syndrome of non-thrombotic iliac vein occlusion has been variously known as May-Thurner syndrome[2], Cockett syndrome[3], or iliac vein compression syndrome. This syndrome was thought to be rare. But recent investigations such as MR Venogram and intravascular ultrasound (IVUS) have shown that presence of such lesions are much more common in highly symptomatic CVD patients (around 90%)[5].Isolated acute iliac vein thrombosis is rare and has been reported mainly as a complication in the post-partum period. Occlusion of the iliac vein is it due to thrombotic or non-thrombotic causes, leads to venous stasis and venous hypertension in the affected leg. This venous hypertension leads to development of varicose veins as well as venotensive changes such as edema, lipodermatosclerosis, eczema and venous ulceration. Cross pelvic collaterals and ascending pelvic collaterals develops to carry blood towards the heart. The long term outcome of thrombosis may be postphlebitic syndrome which is a chronic condition characterized by leg pain, edema, varicose veins, lipodermatosclerosis and ulceraton. Studies have shown that it usually manifests itself within the first 2 years after an episode of DVT[4]. Proximal venous occlusion due to nonthrombotic causes also have similar long term outcome, because the underlying pathophysiology in both conditions is similar (i.e. venous hypertension) Clinical diagnosis of iliac vein occlusion is difficult and it should be suspected in the presence of venotensive changes (edema, lipodermatosclerosis and ulceration) with a normal duplex ultrasound study of the lower limb veins. It has been estimated that 0.5% of patients with primary CVD will have lipodematosclerosis and ulceration[6]. It is worthwhile to submit those patients to additional investigations to detect iliac vein occlusion which will prevent the catastrophic outcome following varicose vein surgery in the presence of proximal occlusion. The investigation of choice to image pelvic veins is intravascular ultrasound (IVUS)[5]. But this is not widely available and hence MR Venogram, CT venogram or ultrasound study of pelvic veins, as done in this patient, can be done to evaluate the iliac veins.

Conclusion

Limiting the diagnostic imaging, to veins below the inguinal ligament in patients with venotensive changes (severe CVD), will lead to this diagnostic pitfall of missing deep venous obstruction in the pelvic veins. In patients with severe CVD, iliac veins also should be imaged to prevent misdiagnosis.

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