

Suprapubic varicose vein formation during pregnancy following pre-pregnancy pelvic vein embolisation with coils, without any residual pelvic venous reflux or obstruction

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Abstract

Suprapubic varicose veins are usually indicative of unilateral iliac vein occlusion and venous collateralisation. We report two cases of suprapubic varicose veins following pelvic vein embolisation and subsequent pregnancy; both presented without residual pelvic venous reflux or pelvic venous obstruction. In both cases, there was no significant flow in the suprapubic veins indicating that they were not acting as a collateral post-pregnancy. One patient had this venous abnormality treated successfully with TRansluminal Occlusion of Perforators, followed by foam sclerotherapy to the main part of the suprapubic vein. This patient has since completed the remainder of her lower limb varicose vein treatment. We suggest that pregnancy may have caused prolonged intermittent compression of the left common iliac vein, and that this, together with the physiological impact of previous embolisation procedures, obstructed venous drainage from the left leg resulting in collateral vein formation within the 9-month gestation period.

Keywords

Suprapubic varicosity, pelvic embolisation, pregnancy

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Introduction

Varicose veins are typically characterised as dilated tortuous venous tributaries arising from incompetent truncal veins in the lower limb. However, lower abdominal and suprapubic varicose veins may develop, and they are commonly caused by venous compression syndromes.^{1,2} Deep venous obstruction is commonly caused by inadequate recanalisation following a deep vein thrombosis (DVT), manifesting as post-thrombotic syndrome, with the second most common cause being non-thrombotic iliac venous compression.³ A common indication of both is venous collateral formation, which develops as a compensation to aid central venous drainage.³

Here, we present two case studies of multiparous females who had pelvic vein embolisation (PVE) prior to pregnancy, during which they both developed a suprapubic varicose vein. At follow-up examination, transvaginal ultrasound (TVUS) showed no pelvic venous reflux (PVR), and further evaluation ruled out any venous obstruction disorders, thereby exhibiting an unknown aetiology.

Patients

Case 1

The first case is a female patient who first represented to our specialist vein unit in 2008, aged 32 years. Previously, she had presented with bilateral recurrent leg varicose veins following stripping elsewhere and was found to have a major pelvic component, which was indicated with a full-leg duplex ultrasound scan (DUS) and confirmed with a TVUS. This was then treated by percutaneous coil embolisation of the left internal iliac and bilateral ovarian veins. On subsequent presentation, TVUS confirmed success and the absence

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of any new PVR. DUS confirmed severe bilateral neovascularisation arising from the sites of previous stripping.

Consequently, she had two endovenous thermoablations for complex recurrent patterns (one for each leg) under local anaesthetic and four additional foam sclerotherapy procedures for profuse neovascular tissue. However, the perforator vein treatment, phlebectomies, and follow-up ultrasound-guided foam sclerotherapy (UGFS) treatment were interrupted by her fifth pregnancy, during which she developed a large varicose vein across the pubic symphysis. All of her deliveries were vaginal and she had not had any previous abdominal or pelvic surgery.

In 2013, she returned to our clinic for a follow-up duplex ultrasound, which confirmed persistent elimination of PVR. However, the scan showed that the visible suprapubic varicose vein appeared to arise from a patent incompetent section at the top of the great saphenous vein (GSV) and project into the left pelvis, across the pubic symphysis, and connect onto the right saphenofemoral junction (SFJ) and communicate with multiple incompetent varicose veins running down the length of her right leg.

From January 2014, she continued her remaining venous treatment. The suprapubic vein was treated with ablation of the ends of the varicose veins where they joined with the normal leg veins, using a percutaneous TRansluminal Occlusion of Perforators (TRLOP)-type technique, followed by foam sclerotherapy of the suprapubic veins under tumescence. TRLOP describes a technique using an endovenous thermoablation device for the treatment of incompetent perforator veins. In this case, the laser was percutaneously inserted into the ends of the suprapubic vein, delivering thermal energy in short bursts to successfully ablate these sections and so that a denaturing sclerosant could be flushed through without spreading into other veins. This achieved excellent result. Her lower limb treatment in both legs encompassed endovenous laser ablation (EVLA) on sections of the GSV, small saphenous vein (SSV) and anterior accessory saphenous vein (AASV), TRLOP, phlebectomies, and UGFS. Patient 1 successfully completed all of her varicose vein treatments in November 2015.

Case 2

Patient 2 presented to this clinic in 2013, aged 29 years, with primary varicose veins of her legs. Full-leg DUS indicated reflux of pelvic origin, which was confirmed with TVUS. In 2014, with a total of 10 platinum coils (Boston Scientific, Boston, MA, USA), the left ovarian vein and bilateral internal iliac veins were embolised. She had EVLA of her left GSV and bilateral AASV. Subsequent UGFS was interrupted by her third pregnancy, during which she developed a suprapubic varicose vein (see Figure 1). All three deliveries were by caesarean section.

In 2016, 7 months after giving birth, a follow-up TVUS ruled out any pelvic reflux, and further investigation again



Figure 1. Clinical presentation of a suprapubic varicose vein post-pregnancy in a patient with pre-pregnancy pelvic vein embolisation with coils.

demonstrated a collateral vein spanning across the pelvis from left to right. Indeed, the vein projected into the right lower limb, bypassing the SFJ, and establishing direct connection with refluxing vein tributaries of the lower limb, which progressed down its entire length.

To rule out a non-thrombotic iliac vein lesion causing left outflow obstruction, she received air plethysmography, which confirmed no evidence of this. She can now go ahead with her lower limb varicose vein treatment. She is due to complete EVLA, TRLOP, phlebectomies, and UGFS.

Discussion

The development of a suprapubic varicose vein is commonly due to stenosis of the common iliac vein; however, it is rare for this to develop and rarer still without any apparent persistent venous compression.¹ Generally, obstruction indicates thrombotic stenosis or compression caused by angiectopia, for example, May–Thurner syndrome (MTS), which presents as compression of the left common iliac vein anteriorly by the left common iliac artery and posteriorly by the lumbar vertebra.² However, many different compression syndromes exist, including congenital agenesis disorders and compression by structural distention.^{4,5}

Research has demonstrated PVE to be a safe and robust procedure, with minimal consequences following pregnancy.⁶ However, in a previous case study published by this unit, the formation of a suprapubic vein post-embolisation and post-pregnancy complete with a patent common iliac vein was reported.⁶ The authors assumed that this was caused by bilateral common iliac vein compression by the gravidic

uterus.⁶ Further research has also identified similar clinical presentations in patients with venous compression caused by structural distention; one study reported on dilated tortuous epigastric veins developing from bilateral common iliac vein compression following bladder distention, secondary to a urethral calculus.⁵

In 1967, research characterised venous collaterals into groups relating to obstruction in specific pelvic veins.³ From this, it is interesting to note that out of the 11 cases of common iliac vein obstruction reviewed, the majority had a collateral vein emerging from the ipsilateral internal iliac vein, through the parametrial and pre-sacral plexus, through to the contralateral internal iliac vein, and one alternative route was through the ovarian vein.³ In both the cases presented here, at least one of these drainage pathways has been blocked off by coil embolisation, providing a possible explanation for blood diversion across the suprapubic route.

It is important to take into consideration the significant impact pregnancy imposes onto women's vascular system. There is a significant increase in blood plasma together with enhanced venous dilation, which are both non-desirable features in a patient with existing chronic venous disease. Additionally, it is also interesting to note that both collateral veins developed on the left-hand side and projected across the pelvis into refluxing veins in the right lower limb. This may indicate a common cause in both patients, which we suggest is likely to be compression of the left common iliac vein. Enhanced blood volume, venous dilation, and blood pooling together with embolisation and the weight of a pregnant uterus may have all contributed to the development of this collateral vein following prolonged venous compression during pregnancy.

So there does appear to be some risk of intermittent venous obstruction during pregnancy post-embolisation. Therefore, it might be wise to advise PVE for female patients with no plans for future pregnancy.

In conclusion, this case study outlines the clinical manifestation of an abnormal suprapubic varicose vein that developed, probably due to the synergistic influence of coil embolisation, pregnancy and existing venous disease. However, the exact

causal conclusion cannot be pinpointed in this study due to lack of evidence and sample size limitations. Therefore, further research is warranted into collateral blood flow during pregnancy following PVE for better understanding of this clinical abnormality.

Declaration of conflicting interests

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Ethical approval

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Informed consent

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