Abdominal and pubic collateral veins as indicators of deep venous obstruction

Ralph L. M. Kurstjens, MD, Timme M. A. J. van Vuuren, MD, Mark A. F. de Wolf, MD, Rick de Graaf, MD, PhD, Carsten W. K. P. Arnoldussen, MD, and Cees H. A. Wittens, MD
Maastricht and Venlo, The Netherlands; and Aachen, Germany

Objective: Chronic deep venous obstruction can cause a significant loss of quality of life, although it can be treated successfully by stenting. A clear referral pattern for additional imaging is warranted in patients with lower limb complaints. The aim of this study was to determine the value of clinically visible abdominal wall collateral veins in the diagnosis of a potentially treatable deep venous obstruction.

Methods: A total of 295 patients referred for evaluation at a tertiary venous clinic with a collateral vein on the abdominal wall or pubic bone, visible on physical examination, were prospectively analyzed and compared with a randomly selected control group of 365 patients without such a collateral vein. Duplex ultrasound, magnetic resonance venography, computed tomography venography, and conventional venography were used to determine the presence or absence of deep venous obstruction.

Results: 

- Mean age of the group with a positive collateral was 43.5 ± 13.7 (6-76) years compared with 44.7 ± 14.2 (16-89) years in the control group. 
- In the control group, 66.1% were female compared with 63.3% in the control group.
- Sensitivity of the abdominal wall collateral vein for any obstruction at the level of the groin or more proximal was 5% (95% confidence interval [CI], 48-57); specificity, 86% (95% CI, 79-91); positive predictive value, 93% (95% CI, 90-96); and negative predictive value, 32% (95% CI, 28-37).
- Sensitivity was 68% (95% CI, 62-73) for higher degrees of post-thrombotic obstruction and 27% (95% CI, 19-36) in iliac vein compression.

Conclusions: A collateral vein on the abdominal wall or across the pubic bone in patients with complaints of the lower limb has an excellent positive predictive value for deep venous obstructive disease at the level of the groin or higher. Such collateral veins should therefore not be removed, and symptomatic patients could be offered further diagnostics and treatment. (J Vasc Surg: Venous and Lym Dis 2016;4:426-33.)

Chronic deep venous obstruction can lead to various complaints. This is often accompanied by a significant reduction in quality of life similar to that with diabetes mellitus, congestive heart failure, and chronic obstructive pulmonary disease. The main reason for development of such a chronic obstruction is inadequate recanalization after deep venous thrombosis (DVT), resulting in the post-thrombotic syndrome (PTS). Annually, 1% to 2% of the Western population will develop DVT, 25% to 56% of whom develop PTS. In patients with a lower extremity thrombosis classification 3 or 4, an iliocaval or common femoral vein (CFV) DVT impairing the central venous outflow from the CFV up to the heart, the recanalization rate is even worse and more patients suffer from PTS afterward. The other main cause of chronic deep vein obstruction causing a central venous outflow obstruction is nonthrombotic iliac vein compression, often referred to as May-Thurner syndrome. This describes compression of the left common iliac vein by the overriding right common iliac artery against the lumbar vertebral column, yet different anatomic compression variants exist.

The prevalence of such an obstruction is unclear as these compressions are often not clinically relevant (eg, compression of >50% has been found in 24% of the general population).

In cases of iliac vein or CFV obstruction for which conservative therapy by compression stockings and mobilization does not yield sufficient results, percutaneous transluminal angioplasty and stenting can be performed with good clinical success. However, adequate identification of possibly treatable disease is necessary. Therefore, it is essential to establish a clear referral pattern. Whereas a history of DVT and venous claudication can be indicators of deep vein obstruction and mandate additional imaging analysis, these signs do not indicate the level of obstruction and therefore possible treatability. Another parameter that might be useful in selection of patients is the presence of a collateral vein visible on the abdominal wall or over the pubic bone.
during physical examination. Such a collateral vein develops as an alternative outflow tract for central venous outflow obstruction and underlines the potential importance of collateral circulation in deep vein obstruction. Post-thrombotic obstruction or iliac vein compression can be diagnosed using duplex ultrasound (DUS), computed tomography venography (CTV), magnetic resonance venography (MRV), intravascular ultrasound, or venography. Because these techniques can be costly and sometimes invasive, it is vital to be as selective as possible in deciding to perform imaging studies to evaluate the deep veins.

The aim of this study was to determine the value of clinically visible abdominal wall collateral veins in the diagnosis of a potentially treatable deep venous obstruction, to underline the importance of these collaterals, and to investigate whether they should be used as a sign of referral for further diagnostics and potential treatment.

METHODS

Study design and participants

In this retrospective observational study, we evaluated the clinical value of the presence of collateral veins on the abdominal wall or over the pubic bone (Fig 1), visible on physical examination. This was assessed during the patient’s first visit at our tertiary, specialized venous outpatient clinic. Clinical value was based on the presence of deep venous obstruction on imaging in patients with and without these collaterals.

Between May 2009 and November 2015, 1099 patients were referred to our tertiary venous outpatient clinic.

Fig 1. Examples of collateral veins over the pubic bone or abdominal wall.
Patients who presented with a collateral vein on the abdominal wall or pubic bone (from here on referred to as abdominal wall collateral), as determined by physical examination during their first visit, were included in this study. Patients also needed to have undergone DUS, CTV, MRV, or conventional venography to diagnose presence of deep venous obstruction. Those patients who presented with an abdominal wall collateral during a later visit or who suffered a DVT <1 year before their visit were excluded. To create a control group, we randomly selected 403 patients from our database of patients referred to our outpatient clinic. These patients were generally referred because of suspicion of deep venous obstruction based on complaints, a history of DVT, or quickly recurrent varicosities. We first identified patients who did not present with an abdominal wall collateral, after which a researcher blinded for patient data randomly sampled 403 cases. Patients with insufficient imaging data of the deep venous system or a DVT within a year before their visit were excluded from analysis in this control group. For both groups of patients, the following information was collected: complaints; venous claudication; history of DVT; previous venous interventions; C class of the Clinical, Etiologic, Anatomic, and Pathophysiologic (CEAP) classification for chronic venous disorders; Venous Clinical Severity Score; and Vilalta scale score. Venous claudication was defined as the occurrence of heaviness or pain during walking that subsides when the patient assumes a sitting or supine position with the leg elevated.

This study was approved by the Maastricht University Medical Centre Institutional Review Board (METC 15-4-243). Individual patient consent was not obtained as this is not required under Dutch law for retrospective studies.

**Procedures**

Imaging protocols described in the following were the same for both groups of patients.

**DUS.** Patients underwent DUS during their first visit to our outpatient clinic. All DUS examinations were performed using a MyLab Alpha (Esaote, Genoa, Italy) or a ProSound Alpha 7 Premier (Hitachi Aloka, Tokyo, Japan) machine. The deep venous system was visualized from the suprarenal inferior vena cava (IVC) to the CFV in the supine position, using a convex probe (frequency range, 1-8 MHz). Evaluation of the groin downward was performed using a linear probe (frequency range, 3-13 MHz) with the patient standing upright. However, for the purpose of this study, only the evaluation of the CFV was included from investigations in the erect position. Throughout the examination, available color modalities were used to determine flow in both transverse and longitudinal planes. Flow division and intraluminal synchiae were considered signs for post-thrombotic obstruction. Iliac vein compression was defined as >50% lumen reduction compared with the diameter and transverse surface area of a normal contralateral common iliac vein or a healthy vessel segment more distal to the supposed compression.

**MRV.** Patients referred to our dedicated venous clinic with a suspicion of outflow obstruction of the lower limb are routinely analyzed with MRV. All magnetic resonance examinations were performed on a 1.5-T magnetic resonance imaging system (Intera; Philips Healthcare, Best, The Netherlands). A dedicated 12-element phased array peripheral vascular coil with a craniocaudal coverage of 128 cm (Philips) was used for signal reception. Patients were imaged in the supine position. Before delivery of contrast material, all patients underwent a standard two-dimensional noncontrast-enhanced balanced turbo field echo sequence to visualize the abdominal and pelvic veins. Then, a gadolinium-based contrast agent (gadobutrol [Gadavist]; Bayer Healthcare, Berlin, Germany) was administered intravenously at 1.0 mL/s (0.2 mL per kg body weight, equal to 0.2 mmol/kg) in the median cubital vein. Consequently, a 20-mL saline flush was injected at the same flow rate, using a remote-controlled dual-head injector (Spectris; Bayer Healthcare). Acquisition of the first scan volume was started 30 seconds after administration of the contrast agent. A three-dimensional ultrafast gradient echo sequence (Ultrafast GE, THRIVE, Philips Healthcare) with fat suppression (spectral presaturation with inversion recovery) was used for high-resolution steady-state imaging of the venous vasculature, ensuring coverage of at least the popliteal veins up to the suprarenal IVC. Compression of a vein was defined as >50% lumen reduction with the presence of collateral veins. Post-thrombotic obstruction was identified as the presence of intraluminal synchiae, which has been described and successfully performed before.

**CTV.** CTV was performed in patients who had a contraindication to MRV. Each examination was performed on a Siemens Flash scanner (Siemens Healthcare, Erlangen, Germany). An intravenous injection of 120 mL of iodine contrast medium (Ultravist 300; Bayer Healthcare) was administered at 3.5 mL/s through an 18-gauge antecubital infusion catheter, followed by a saline flush of 40 mL at 3.5 mL/s. Computed tomography images were obtained and reconstructed with a slice thickness of 2.0 mm from the dome of the diaphragm to the toes 180 seconds after the start of the injection. Images were routinely interpreted by a subspecialty-trained cardiovascular radiologist on a regular picture archiving and communication system workstation using IMPAX 6.5 (Agfa Healthcare, Mortsel, Belgium). The primary criteria for deep venous obstruction were segmental nonvisualization or lack of opacification of the vein lumen, luminal narrowing with or without external compression, and identification of collateralization. Compression was defined as >50% lumen reduction compared with a normal vessel segment.

**Conventional venography.** Venography was performed in all cases in which treatment was indicated on the basis of noninvasive imaging. In some cases, no pathologic change was found on DUS or MRV or CTV, although deep venous obstruction was suspected on the basis of the patient’s signs or symptoms. In the latter instance, anteroposterior venography was performed to identify typical signs of nonthrombotic iliac vein lesions (eg, contrast translucency appearing
as a filling defect; broadening/pancaking of the vein; and axial, transpelvic, or ascending lumbar collaterals). Ultrasound guidance was used to puncture the femoral vein, about 10 cm below the main deep femoral vein branch. This was done to identify any collateral veins above the level of the saphenofemoral junction, if present. Venography was performed during inspiration through a 6F sheath (PreludePRO; Merit Medical Systems, South Jordan, Utah) with 10 mL of iodinated contrast material (Ultravist 300, Bayer Healthcare) at a flow velocity of 5 mL/s. In the presence of the aforementioned classic nonthrombotic iliac vein lesion signs, we determined a hemodynamically relevant downstream obstruction (ie, significant iliac vein compression). The presence of collaterals, specifically, has been suggested to be of hemodynamic importance. No efforts were made to estimate iliac vein stenosis grade because venography has never been validated to determine iliac vein lumen reduction accurately.

Outcomes

Primary outcome was the presence of deep venous obstruction of the CFV or more proximal. Obstruction was also divided into post-thrombotic disease and non-thrombotic iliac vein compression. Subanalysis for patients with >50% lumen reduction was performed in the post-thrombotic group. Obstruction was scored as positive if DUS or MRV or CTV identified the obstruction. Obstruction was scored as negative if both MRV or CTV and DUS did not show obstruction. If a segment was negative on one modality and not evaluated or not visualized on another modality, it was also scored as negative. When no obstruction was identified on DUS, MRV, or CTV and conventional venography was performed, obstruction was scored as positive if one was identified on venography.

Statistical analysis

Continuous data were presented as mean with standard deviation or median with interquartile range (IQR), depending on normality of distribution, unless otherwise specified. An independent t-test or Mann-Whitney U test was performed to assess for differences between the two patient groups (presence and absence of abdominal wall collateral), depending on normality of distribution. Categorical data were presented using percentages, and a Pearson χ² test was used to test for differences in patient characteristics. Fisher exact test was used in cases in which expected counts were <5. Sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) were calculated for the ability of abdominal wall collaterals to identify deep venous obstruction. Obstructions were stratified according to etiology, and a sub-analysis was performed for patients with >50% lumen reduction based on DUS. Diagnostic value analyses were performed using GraphPad Prism version 5.04 (GraphPad Software, San Diego, Calif).
RESULTS

Of the 1099 patients who were referred, 308 presented with an abdominal wall collateral. Eleven patients were excluded as determined by our exclusion criteria, and an additional two patients were excluded because of insufficient imaging data. In the randomly selected control group of 403 patients, 30 patients had to be excluded according to our exclusion criteria and 8 did not have full visualization of the deep venous system. Thus, a total of 660 patients were analyzed: 295 patients with an abdominal wall collateral and 365 patients without (Fig 2).

Mean age of the group with a visible collateral was 43.5 ± 13.7 (6-76) years compared with 44.7 ± 14.2 (16-89) years in the control group (P = .28). Of all patients with a collateral, 66.1% were female compared with 63.3% in controls (P = .45). History of venous interventions was comparable between the two groups (30.9% vs 35.1%; P = .55). One patient in the control group had an abdominal wall collateral removed for cosmetic reasons in the past. Patients with a visible collateral more often had a history of DVT (79.3% compared with 61.9%; P < .01). Of those patients with a history of DVT or iatrogenic lesions, the group with a visible collateral presented at our outpatient clinic at 9.0 years (IQR, 3.0-18.0) after the event compared with 6.0 years (IQR, 3.0-14.0) for controls (P = .06). Severity of disease, determined by

### Table I. Patient characteristics

<table>
<thead>
<tr>
<th></th>
<th>Abdominal wall collateral (n = 295)</th>
<th>No abdominal wall collateral (n = 365)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years (range)</td>
<td>43.5 ± 13.7 (6-76)</td>
<td>44.7 ± 14.2 (16-89)</td>
<td>.28</td>
</tr>
<tr>
<td>Female sex</td>
<td>195 (66.1)</td>
<td>231 (63.3)</td>
<td>.45</td>
</tr>
<tr>
<td>History of venous intervention</td>
<td></td>
<td></td>
<td>.55</td>
</tr>
<tr>
<td>No</td>
<td>206 (70.1)</td>
<td>237 (64.9)</td>
<td></td>
</tr>
<tr>
<td>Superficial</td>
<td>79 (26.9)</td>
<td>113 (30.9)</td>
<td></td>
</tr>
<tr>
<td>Deep</td>
<td>13 (4.5)</td>
<td>20 (5.4)</td>
<td></td>
</tr>
<tr>
<td>Removal of abdominal wall collateral</td>
<td>1 (0.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of DVT</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>55 (18.6)</td>
<td>137 (37.5)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Yes</td>
<td>234 (79.3)</td>
<td>226 (61.9)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Iatrogenic lesion</td>
<td>6 (2.0)</td>
<td>2 (0.5)</td>
<td>.15</td>
</tr>
<tr>
<td>Years since eventb</td>
<td>9.0 (3.0-18.0)</td>
<td>6.0 (3.0-14.0)</td>
<td>.06</td>
</tr>
<tr>
<td>Location of complaintsc</td>
<td></td>
<td></td>
<td>.29</td>
</tr>
<tr>
<td>No complaints</td>
<td>7 (2.3)</td>
<td>8 (2.2)</td>
<td></td>
</tr>
<tr>
<td>Left leg</td>
<td>163 (55.4)</td>
<td>163 (45.2)</td>
<td></td>
</tr>
<tr>
<td>Right leg</td>
<td>43 (14.6)</td>
<td>73 (20.2)</td>
<td></td>
</tr>
<tr>
<td>Both legs</td>
<td>76 (25.9)</td>
<td>109 (30.2)</td>
<td></td>
</tr>
<tr>
<td>Abdomen/pelvis</td>
<td>27 (9.2)</td>
<td>34 (9.4)</td>
<td></td>
</tr>
<tr>
<td>Venous claudicationd</td>
<td>156 (54.2)</td>
<td>172 (49.0)</td>
<td>.19</td>
</tr>
<tr>
<td>VCSS</td>
<td>7.4 ± 3.8</td>
<td>6.9 ± 3.8</td>
<td>.14</td>
</tr>
<tr>
<td>Highest C class of CEAPf</td>
<td></td>
<td></td>
<td>.19</td>
</tr>
<tr>
<td>C0</td>
<td>21 (7.2)</td>
<td>39 (10.8)</td>
<td></td>
</tr>
<tr>
<td>C1</td>
<td>24 (8.2)</td>
<td>29 (8.0)</td>
<td></td>
</tr>
<tr>
<td>C2</td>
<td>56 (19.2)</td>
<td>51 (14.1)</td>
<td></td>
</tr>
<tr>
<td>C3</td>
<td>96 (33.0)</td>
<td>121 (33.5)</td>
<td></td>
</tr>
<tr>
<td>C4</td>
<td>61 (21.0)</td>
<td>69 (19.1)</td>
<td></td>
</tr>
<tr>
<td>C5</td>
<td>21 (7.2)</td>
<td>24 (6.6)</td>
<td></td>
</tr>
<tr>
<td>C6</td>
<td>12 (4.1)</td>
<td>28 (7.8)</td>
<td></td>
</tr>
<tr>
<td>Villalta scoreg</td>
<td></td>
<td></td>
<td>.34</td>
</tr>
<tr>
<td>No PTS</td>
<td>20 (9.8)</td>
<td>18 (13.0)</td>
<td></td>
</tr>
<tr>
<td>Mild PTS</td>
<td>74 (26.3)</td>
<td>46 (35.5)</td>
<td></td>
</tr>
<tr>
<td>Moderate PTS</td>
<td>69 (33.8)</td>
<td>38 (27.5)</td>
<td></td>
</tr>
<tr>
<td>Severe PTS</td>
<td>44 (20.1)</td>
<td>36 (26.1)</td>
<td></td>
</tr>
</tbody>
</table>

CEAP, Clinical, Etiologic, Anatomic, and Pathophysiologic classification for chronic venous disease; DVT, deep venous thrombosis; PTS, post-thrombotic syndrome; VCSS, Venous Clinical Severity Score.

Categorical variables are presented as number (%). Continuous variables are presented as mean ± standard deviation or median (interquartile range [IQR]).

*One missing.

Six missing.

Five missing.

Twenty-one missing.

One hundred eight missing.

Eight missing.

Only given for patients with post-thrombotic disease (n = 401), 59 missing.

Statistically significant.
venous claudication, C class of CEAP classification, Venous Clinical Severity Score, and Villalta score, was not different between the two groups (Table I).

A total of 522 obstructions on the level of the groin or higher were observed using DUS, CTV, MRV, and venography: 404 (77.4%) were of post-thrombotic nature; 116 (22.2%) were of nonthrombotic nature; and in 2 (0.4%) patients, compression was observed without adequate visualization of all vessel segments. Thus, the last two patients might have post-thrombotic changes in the not visualized vessel segments of the lower limb, leading to exclusion of these patients for subanalysis. Imaging with more than one modality was performed in 612 (92.7%) patients.

More detailed information on identified disease can be found in Fig 3. Of all detected post-thrombotic lesions, 60.4% were indentified in the collateral group and 39.6% in the control group (P < .01). Conversely, 26.7% of all detected nonthrombotic iliac vein compression cases were identified in the collateral group compared with 73.8% in the control group (P = .02).

The presence of an abdominal wall collateral visible during physical examination yielded a sensitivity of 53% (95% confidence interval [CI], 48-57), specificity of 86% (95% CI, 79-91), PPV of 93% (95% CI, 90-96), and NPV of 32% (95% CI, 28-37) for any obstruction found on imaging from the groin up to the right atrium (Table II).

Subanalysis for post-thrombotic obstruction demonstrated similar results with a sensitivity of 60% (95% CI, 55-65), specificity of 86% (95% CI, 79-91), PPV of 92% (95% CI, 89-95), and NPV of 43% (95% CI, 37-49). PPV did not change when degree of obstruction was investigated: 90% (95% CI, 85-94) for post-thrombotic obstructions with >50% lumen reduction. Yet, the sensitivity increased (68% [95% CI, 62-73]), as did NPV (58% [95% CI, 51-65]). In comparing the presence of an abdominal wall collateral to the subgroup of nonthrombotic iliac vein compression, the PPV (61% [95% CI, 46-74]) and sensitivity (27% [95% CI, 19-36]) were considerably lower (Table II).

Those patients with a positive collateral vein sign but no objectified obstruction often demonstrated some degree of venous disease. For example, two patients demonstrated gonadal vein incompetence during venography. In two additional patients, gonadal veins were dilated on MRV, and one patient showed slight compression of the left renal vein by the superior mesenteric artery on DUS, although not significant enough to warrant a nutcracker syndrome diagnosis. Also, eight patients suffered from superficial vein incompetence in the area of the saphenofemoral junction, one from an aneurysmatic dilation of the stump of a previously treated great saphenous vein, one of retrograde flow of the superficial epigastric vein, and one from an incompetent internal pudendal vein. Four patients had no suggestion of venous disease whatsoever.

**DISCUSSION**

This study has shown that an abdominal wall collateral vein visible during physical examination has a PPV of 93% for diagnosis of deep venous obstructive disease at the level of the groin or higher. This clinical sign of venous obstruction has been described in the literature before, as has its diagnostic abilities. The high PPV indicates that an abdominal wall collateral is a sign that patients should be referred for additional imaging. Specificity was fair (86%), indicating few false-negative test results, although NPV was consistently very low (32%), suggesting that no conclusions should be drawn in the absence of such a collateral vein during physical examination.

Post-thrombotic disease appears to have the most influence on the formation of a visible collateral vein, as subanalysis yielded results similar to those for the whole group, whereas nonthrombotic iliac vein compression demonstrated poorer results. This difference is substantiated by the fact that the collateral group more often had a history of DVT. Conversely, iliac vein compression without post-thrombotic abnormalities was more often...
present in patients without a visible abdominal wall collateral. The lower prevalence of abdominal wall collaterals in patients with nonthrombotic iliac vein compression therefore explains its inferior sensitivity in these patients.

An interesting observation is that one of the patients in the control group had a collateral vein over the pubic bone that was removed for cosmetic reasons. This patient also had lower limb complaints that remained stable after removal of the pubic collateral vein, which is certainly not always the case in our experience. We have also seen patients whose complaints deteriorated after removal of such a collateral vein. It is crucial that the physician realizes that these collaterals are an essential part of the outflow of the leg and should never be removed because the complaints related to the impaired outflow will likely increase. Visible collaterals on the abdominal wall or across the pubic bone are only the tip of the iceberg, though. In cases of common iliac vein obstruction, blood flow can be diverted by the internal iliac vein through the presacral and parametrial plexuses, ipsilateral ascending lumbar vein, ovarian veins, or paravertebral plexuses. When obstruction also involves the external iliac vein or CFV, the deep circumflex iliac vein, obturator vein, or deep external pudendal vein can be activated too. In addition, blood flow can be diverted through the superficial external pudendal, pubic, and superficial epigastric veins, which leads to the visible collateral veins on the abdominal wall or across the pubic bone. Involvement of the IVC likely results in a more extensive collateral network, involving the paravertebral plexus and epigastric veins, draining into theazygos system and the superior vena cava. The latter veins can lead to wide-ranging externally visible collateral formation.

Results could have been confounded by the presence of labial or scrotal varicosities. A distinction between such varicosities and collateral veins across the pubic bone could not always be determined because of succinct reporting. Isolated pudendal incompetence or incompetence of the saphenofemoral junction might have led to retrograde flow in the superficial epigastric vein and therefore a visible collateral vein. Finally, it is conceivable that abdominal wall collaterals could also be connected to pelvic congestion syndrome, which is a complex phenomenon that is currently not completely understood and can be difficult to diagnose.

Some limitations should be mentioned. Despite extensive imaging, the lack of intravascular ultrasound use might have influenced diagnosis of nonthrombotic iliac vein compression. Also, selection bias may be present as all patients were referred to our outpatient clinic either with complaints of the abdomen or lower limb, suspected to be of venous origin, or with known obstruction of the deep venous system. Collateral circulation can also be found in a number of different disorders. Ovarian tumors can cause dilated ovarian veins, and gestational trophoblastic neoplasms can lead to a more pronounced uterine plexus, as can uterine arteriovenous malformations. These deep collateral networks could theoretically lead to a collateral sign visible on physical examination. Finally, portal hypertension can cause collateral formation and is known to demonstrate clinically visible abdominal veins. Studies in a general population with complaints of the lower limb could determine whether such a bias was actually present.

CONCLUSIONS

A collateral vein on the abdominal wall or across the pubic bone in patients with complaints of the lower limb has an excellent PPV for potentially treatable deep venous obstructive disease at the level of the groin or higher. This warrants closer inspection of the groin and abdominal area in all patients with lower limb symptoms. Such collateral veins should never be removed for aesthetic purposes, and symptomatic patients should be referred for additional imaging, and possibly treatment, of their obstruction.

AUTHOR CONTRIBUTIONS

Conception and design: RK, MW, CW
Analysis and interpretation: RK, TV, RG, CW
Data collection: RK, TV, CA
Writing the article: RK
Critical revision of the article: RK, TV, MW, RG, CA, CW
Final approval of the article: RK, TV, MW, RG, CA, CW
Obtained funding: Not applicable
Overall responsibility: RK

REFERENCES
