

are minimal data characterizing the presence of a concomitant ipsilateral central venous stenosis. Consequently, central venous stenosis may be underdiagnosed and undertreated in this cohort of patients with active or healed venous stasis ulcerations. This study sought to evaluate whether the presence of a pathologic perforator is predictive of the presence of an ipsilateral central venous stenosis.

Methods: This was a retrospective review of a prospectively maintained institutional Vascular Quality Initiative database from May 2016 to April 2018. Consecutive patients were identified who underwent laser ablation of a pathologic perforator per American Venous Forum guidelines, and most had not undergone dedicated central venous imaging. Patients were identified who underwent incidental abdominal imaging that allowed evaluation of the central veins (eg, magnetic resonance, computed tomography, venography or intravascular ultrasound [IVUS]). The patients were divided into those who had imaging (group A) and those who did not (group B). Of those patients who underwent imaging, the primary outcome was the presence of an ipsilateral central venous stenosis as measured by orthogonal diameter reduction of >50% on axial imaging or by cross-sectional surface area reduction of >50% on IVUS.

Results: There were 63 patient limbs that underwent ablation of a pathologic perforator (group A, n = 18; group B, n = 45). Of the total cohort, 47.6% were men; average Venous Clinical Severity Score was 8.8 ± 5.4 , right-sided treatments occurred in 44.4%, and average perforator diameter was 5.7 ± 3.3 mm. Demographic variables did not differ significantly between groups. Right-sided procedures trended more in group A compared with group B (group A, 61.1%; group B, 37.8%; $P = .09$). Imaging was as follows: computed tomography, n = 10; magnetic resonance, n = 6; and venography or IVUS, n = 2. Limbs with pathologic perforators demonstrated ipsilateral central venous stenoses in 83.3% vs the contralateral limbs, which demonstrated central venous stenoses in 44.4% ($P = .04$). In perforator-treated limbs, the average iliac vein diameter decreased from 15.5 ± 4.1 mm to 4.5 ± 1.6 mm ($P < .01$). This represented an average stenosis of $70.2\% \pm 11.0\%$ in the perforator-treated limb vs $45.4\% \pm 31.1\%$ in the unaffected limb ($P = .009$). Concomitant central venous stenting was performed rarely (group A, 2; group B, 0; $P = .07$), presumably because of the lack of imaging and diagnosis.

Conclusions: This study suggests that the majority of patients who undergo treatment for a pathologic perforator have ipsilateral central venous stenosis. The identification of central venous stenosis in limbs with a treated pathologic perforator supports the concept that multilevel disease may underlie severe venous insufficiency and that this is often underdiagnosed and undertreated. Ongoing evaluation will demonstrate whether identification and treatment of central venous stenosis in patients with a pathologic perforator will result in improved outcomes.

Author Disclosures: **M. Sadek:** Nothing to disclose; **L. Kabnick:** stock options, contractor Bard and Veniti; **T. Maldonado:** Nothing to disclose; **C. Rockman:** Nothing to disclose; **N. Cayne:** Nothing to disclose; **T. Berland:** Nothing to disclose; **G. Jacobowitz:** Nothing to disclose.

Spine Stabilization Is a Risk Factor for the Development of Pelvic Iliac Vein Lesions



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Objective: Open lumbar spine stabilization surgery often requires mobilization of the left and right common iliac veins (CIVs) and the placement of plates and screws that can impinge on iliac veins. We reviewed our venography experience during the past 3 years to determine whether there is an association between spine stabilization surgery and the development of iliac vein lesions and symptomatic iliac vein outflow obstruction.

Methods: A retrospective chart review was performed to identify patients who underwent venography with or without venous stenting and who had a history of previous lumbar spine stabilization.

Results: From January 2014 to April 2018, venography was performed in 1713 limbs in 1245 patients at the Center for Vascular Medicine. Of the 1245 patients, 17 patients had a history of lumbar spine stabilization procedures (4 anterior-posterior and 13 posterior). Nine had single-level and eight had two- or three-level fusions. All 17 patients demonstrated pelvic lesions, which included the following: one left CIV aneurysm, five left CIV stenoses, three bilateral CIV stenoses, two left CIV and inferior vena cava occlusions, and two external iliac vein stenoses. The aneurysm patient was treated

with anticoagulation. Eight patients underwent stenting, and one patient refused stenting because of relocation to another country. One inferior vena cava-CIV occlusion could not be crossed. Fear of dislodging a thrombus and the proximity to a protruding posteriorly placed screw prevented stenting in two patients. Four patients had a venoplasty alone because of undersizing of a stenosis or missed lesions with intravascular ultrasound after review by a blinded reviewer. Lesions in anterior-posterior patients were extremely stenotic, required predilation, and resulted in a residual stenosis requiring venoplasty at a second setting in one patient.

Conclusions: Lumbar spine stabilization surgery may be a risk factor for development of symptomatic venous outflow obstruction lesions. During venography and stenting in patients with anterior-posterior approaches, significant scarring may be encountered, resulting in a residual stenosis after stent placement. Predilation venoplasty, before stent deployment, is recommended to prevent stent migration. Furthermore, a history of spine stabilization surgery in patients presenting with pelvic or lower extremity pain or swelling should prompt consideration of a pelvic venous duplex ultrasound examination to assess for the presence of an iliac venous outflow lesion.

Author Disclosures: **G. Rasouli:** Nothing to disclose; **M. Tran:** Nothing to disclose; **V. Satwah:** honorarium Tactile Medical; **S. Lakhanpal:** Nothing to disclose; **P. Pappas:** Nothing to disclose.

Outcomes of Left Renal Vein Stenting in Patients with Nutcracker Syndrome



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Objective: Nutcracker syndrome (NCS) is a rare condition that can present with hematuria, flank pain, pelvic varicosities, or chronic pelvic congestion related to left renal vein (LRV) compression. Open surgery, specifically LRV transposition, has been the mainstay of treatment, but during the past few years, LRV stenting has emerged as a valid alternative without sufficient evidence to support it. This study aimed to assess outcomes of renal vein stenting in the treatment of NCS.

Methods: A retrospective chart review of patients with NCS who underwent LRV stenting between 2010 and 2018 was performed. End points were perioperative adverse outcomes, symptom relief, and stent patency. Symptom resolution was classified as complete, partial, and none on the basis of the interpretation of medical records on clinical follow-up. Standard descriptive statistics and survival analysis were used.

Results: Seventeen patients (16 female; mean age, 35.8 ± 14.6 years; mean body mass index, 21.3 ± 4.1 kg/m²) diagnosed with NCS and treated with LRV stenting were identified. Five of these had a prior LRV transposition that had failed within a mean of 7.0 ± 4.9 months. Ten patients had coexisting pelvic congestion syndrome treated with gonadal vein embolization. The most frequent sign and symptom were hematuria (9/17 patients) and flank pain (14/17 patients), respectively. All patients received self-expanding stents (mean diameter, 12.7 ± 1.6 mm), the smaller ones typically placed in the previously transposed LRVs. No perioperative complications occurred. Eight patients were discharged on the same day; the remaining stayed longer for pain control (mean hospital stay, 1.0 ± 1.3 days). At an average follow-up of 33 ± 25 months, 13 (76.5%) patients had symptom relief (9 complete, 4 partial). Three of the four patients whose symptoms persisted had previous LRV transposition surgery. Five of nine patients who presented with hematuria had it resolved.

Three patients underwent a reintervention. Two of these had successful balloon venoplasty for restenosis. The third patient had persistent debilitating pain despite a patent stent and eventually underwent renal auto transplantation with no symptom relief. Two-year primary and primary assisted patencies were 81.8% and 90%, respectively. No stent migration occurred.

Conclusions: Endovascular treatment with renal vein stenting is safe and effective, providing good midterm patency rates and symptom relief. Minimally invasive approaches may have a potential role in the treatment of NCS. Larger series and longer follow-up are needed to better assess its comparative performance against LRV transposition.

Author Disclosures: **E. D. Avgerinos:** Nothing to disclose; **Z. Saadeddin:** Nothing to disclose; **R. Humar:** Nothing to disclose; **K. Salem:** Nothing

to disclose; **M. Singh:** Nothing to disclose; **E. S. Hager:** Nothing to disclose; **M. Makaroun:** Nothing to disclose; **R. A. Chaer:** Nothing to disclose.

Medical Compression Stockings Significantly Increase Local Tissue Factor Levels in Advanced Chronic Venous Insufficiency Patients and Healthy Volunteers



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Objective: Evidence that medical compression stockings (MCS) prevent deep venous thrombosis (DVT) is weak. Furthermore, the body position that predisposes to DVT is not fully known. It is assumed that standing is protective through involuntary leg muscle contractions and that lying stationary may provoke DVT. Previous work using ultrasound has shown the presence of venous sludge in the popliteal veins, in both positions. The aim was to investigate the effect of standing, lying, and compression on thrombogenicity. This was achieved by taking local venous blood samples and measuring an array of factors considered relevant in thrombogenesis.

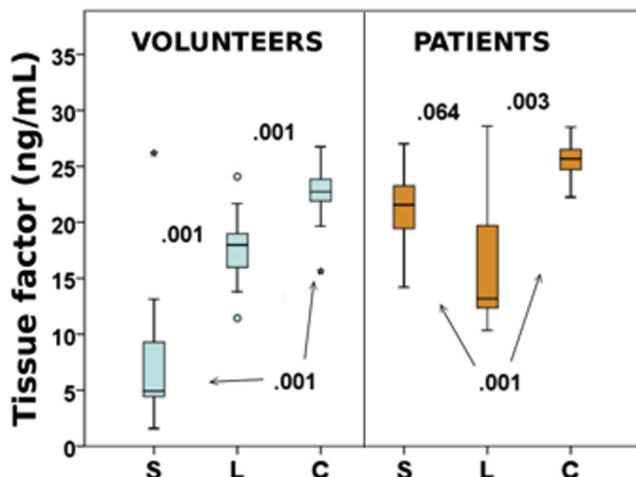


Fig. Local tissue factor concentrations in response to standing (S), lying (L), and compression (C) in volunteers and patients. Significance levels are shown (Wilcoxon).

Methods: Patients with advanced chronic venous insufficiency awaiting endothermal ablation (C4a, C4b) and healthy volunteers (n = 14 legs in each group, 1 leg per patient) had local leg blood samples taken after 1 hour of standing, lying, and standing with compression on separate days. Knee-length MCS of 23 to 32 mm Hg were administered. Platelet-poor plasma samples were analyzed for procoagulant phospholipids, tissue factor, D-dimer, fibrin monomer, and factor VIIa-antithrombin complexes. This was in addition to a thrombin generation test using the PPP-reagent, which measured lag time, endogenous thrombin potential, peak, time to peak, and mean rate index.

Results: The most responsive was tissue factor, with significant increases after an MCS was worn in standing compared with lying and standing with compression in both volunteers and patients (Fig 1). Standing and compression made no difference to D-dimer or factor VIIa-antithrombin levels in either group (Table). Thrombin generation testing revealed no differences in the volunteer group, but in the patients, compression appeared to have a favorable significant effect in four of five measurements compared with lying (Table).

Conclusions: Local tissue factor concentrations were elevated significantly with MCS. This was on a background of unaffected D-dimer and factor VIIa-antithrombin levels, thereby questioning the thrombogenic significance of the elevated tissue factor. However, compression reduced thrombin generation parameters, but only in patients with chronic venous insufficiency. Patients also demonstrated significantly reduced thrombin generation when standing compared with lying. This research supports our hypothesis that standing and compression may offer additional protection from thrombosis, but only in patients.

Author Disclosures: **C. R. Lattimer:** funding SIG Research Grant; **E. Kalodiki:** Nothing to disclose; **P. Van Dreden:** Nothing to disclose; **A. Rousseau:** Nothing to disclose; **G. T. Gerotziapas:** Nothing to disclose.

Effectiveness of the PowerWire Radiofrequency Guidewire in Recanalizing Chronically Occluded Iliac Venous Stents



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Objective: Iliac venous stenting is a commonly performed procedure in treating post-thrombotic and nonthrombotic iliac venous disease. Whereas overall stent patency rates are high, stent occlusion does occur. Recanalizing chronically occluded stents is technically challenging and often impossible with conventional guidewires and catheters. The PowerWire (Baylis Medical, Burlington, Mass) radiofrequency guidewire is a 0.035-inch guidewire that delivers radiofrequency energy at the end of the guidewire.

Methods: A retrospective chart review was conducted of patients with chronically occluded iliac venous stents who underwent an iliac venous recanalization attempt using the PowerWire from March 2015 to June 2018. A total of 15 patients underwent a recanalization attempt with the PowerWire. All patients had initial unsuccessful attempts at recanalizing the occluded iliac vein stents with conventional guidewires.

Table. Median (interquartile range) values in three different laboratory situations for 1 hour

Parameters	Volunteers			Patients (C4)		
	Standing	Lying	Compression	Standing	Lying	Compression
Procoagulant phospholipids, \$	67.7 (63.5-80)^a	76.7 (66.6-92.6)	71.5 (67-80.7)	82.8 (74.7-91.2)	89.5 (76.3-96.5)	77.1 (67.6-81.8)^a
Tissue factor, ng/mL	4.94 (4.03-10)^a	17 (15.5-19.1)	22.7 (21.7-23.9)^b	21.6 (18.7-23.3)	13.2 (12.2-20.4)	25.7 (24.6-26.7)^b
D-dimer, µg/mL	0.27 (0.27-0.34)	0.27 (0.27-0.32)	0.27 (0.27-0.45)	0.35 (0.27-0.9)	0.34 (0.28-0.63)	0.29 (0.27-0.53)
Fibrin monomer, µg/mL	5 (5-5.8)	5 (5-60.3)	5 (5-50.4)	5 (5-7.32)	5 (5-5.07)	5 (5-5)
Factor VIIa, units/mL	74.7 (55.2-300)	71.9 (54.3-54.5)	17.3 (10.6-241)^a	26.7 (19.7-55.5)	28.1 (19.9-55.9)	32.5 (24.6-67.5)
Lag time, minutes	6.74 (5.49-8.92)	5.91 (4.74-7.16)	5.67 (4.67-6.93)	6.75 (5.04-9.13)	6.46 (5.06-7.42)	7.25 (5.33-8.88)
ETP, µM/min	1.1 (0.75-1.25)	1.1 (0.72-1.72)	1 (0.77-1.54)	0.94 (0.84-1.08)	1.08 (0.68-1.38)	0.96 (0.43-1.11) ^a
Peak, nM	150 (95.5-194)	1.69 (95.2-349)	136 (97.3-291)	150 (101-182)	175 (108-214)^a	150 (62.8-185)^a
ttPeak, minutes	10.8 (9-12.3)	8.83 (7.87-11.2)	9.83 (7.55-10.8)	10.8 (8.51-13.7)	10 (7.9-11.9)^a	10.9 (8.67-12.9)^a
MRI	40.8 (21.7-61.1)	48.6 (24.3-150)	36.7 (24.1-107)	44.9 (21.3-56.6)	51.3 (31.3-69.4)^a	41.9 (18.8-56.7)^a
Factor VIIa-antithrombin, pM	102 (82.5-179)	115 (57.4-227)	82.9 (53.6-194)	118 (91.4-161)	111 (97-133)	118 (101-146)

ETP, Endogenous thrombin potential; MRI, mean rate index; ttPeak, time to peak.

^aP < .05 vs standing.

^bP < .05 vs lying.