Iliac-caval stenting in the obese

Seshadri Raju, MD, Rikki Darcey, BS, and Peter Neglén, MD, PhD, *Jackson, Miss*

Background: Chronic venous insufficiency (CVI) in the obese, often with severe clinical manifestations, is increasingly encountered in clinical practice. The association has drawn special interest as the pathophysiology may be different from that seen in the non-obese. The disease poses special management problems in the obese as traditional conservative measures are seldom effective. Iliac-caval venous stenting has been effective in CVI management but applicability in the obese raises concerns regarding stent compression, morbidity and efficacy.

Methods: Results of iliac-caval stenting in 101 limbs of 87 patients are presented. Clinical features, venous test results, and outcome after stenting are reported with comparison to select relevant features in the non-obese limbs (n = 1513) that were stented over the same 11 year period.

Results: Bilateral clinical manifestations CVI were twice as common in the obese subset compared with the non-obese (28% vs 14% respectively, $P = .0007$), the incidence increasing with BMI. Resting and exercise femoral vein pressures were similar to the non-obese. Obstructive lesions of primary or postthrombotic origin similar to those seen in non-obese limbs were detected by intravascular ultrasound examination in 89% of limbs. Compression by increased intra-abdominal pressure associated with obesity was likely the mechanism of obstruction in 11% of the limbs. Iliac-caval venous stenting was found to be safe with no mortality (<30 days), low morbidity (deep venous thrombosis in 3%), high patency (86% cumulative at five years), and satisfactory clinical outcome. Sixty-eight percent and 46% of limbs showed cumulative improvement in pain and swelling respectively at 5 1/2 years after stenting, with 65% and 31% limbs achieving complete relief of these respective symptoms. Thirty of 45 limbs (58% cumulative) were free of dermatitis/ulcer at four years.

Conclusion: The mechanism of venous obstruction in the obese is substantially similar to those in the non-obese. Primary or post-thrombotic lesions as seen in non-obese CVI cases are present in 89% of cases per IVUS examination. Compression of the venous outflow by adiposity/abdominal pressure may be a factor in 11%. Iliac-caval venous stenting is a satisfactory clinical option in the obese with severe CVI manifestations requiring speedy relief. (J Vasc Surg 2009;50: 1114-20.)

Chronic venous insufficiency (CVI) associated with obesity is increasingly encountered in clinical practice. Obesity associated increased abdominal pressure has been implicated in the genesis of venous symptoms,[1] and unique calf pump abnormalities are present in this subset.[2] CVI manifestations in the obese pose special management challenges. Orthostatic limb pain and swelling retards exercise and activity – essential components in weight reduction programs. Body habitus, local skin condition, and limb adiposity may prohibit or inhibit compression treatment as stockings are difficult to fit, apply without assistance, and maintain wear. Many obese patients cannot physically reach their feet.[3] Non-compliance with prescribed regimes, a frequent factor in the onset of obesity, extends to compression use as well; non-compliance with compression is widely prevalent even in 'ulcer clinics' under medical supervision.[4] Ideally, a program of rigorous weight reduction can be expected to reduce severity of CVI manifestations. These programs are more often unsuccessful than not in clinical experience, and generally do not accomplish satisfactory weight reduction within a reasonable time frame relevant for the relief of severe venous symptoms. Bariatric surgery, which has recently become minimally invasive and safer than before, is a desirable option, but many patients are unable (procedure not covered by many insurance programs) or unwilling to avail themselves of this option. Open surgical procedures to correct specific CVI pathology carry higher risks than usual because of obesity and associated co-morbidities. The advent of minimally invasive techniques such as percutaneous saphenous ablation provides alternative and possibly safer therapeutic options. Iliac venous stenting also falls under this category of modern treatment avenues but stent compression/occlusion by intra-abdominal obesity and associated increased abdominal pressure[5] along with the overall safety of these procedures in this morbidity-prone subset are major concerns. This manuscript describes the outcome of iliac vein stent placement combined with saphenous ablation when indicated in the obese. Venous parameters and other data relevant to the increased abdominal pressure hypothesis in the obese are also presented.

METHODS

A total of 1640 limbs underwent iliac-caval venous stenting from 1997 to 2008 for severe symptoms of chronic venous insufficiency. Of these, 101 limbs (6%) were performed in 87 patients who were obese (World Health Organization [WHO] classification)[6] and had a Body Mass Index (BMI) of ≥30. The current report is based on analysis of this subset.

From the University of Mississippi Medical Center and River Oaks Hospital.

Drs Raju and Neglen have patent applications (pending) related to intravascular ultrasound and venous stenting.

Presented at the 21st Annual Meeting of the American Venous Forum, February 11-14, 2009, Phoenix, Ariz.

Reprint requests: Seshadri Raju, MD, 1020 River Oaks Dr., #420, Flowood, MS 39232 (e-mail: rajumd@earthlink.net).

0741-5214/$36.00

doi:10.1016/j.jvs.2009.06.055

BMI was calculated for all stented patients from height and weight measurements routinely entered in venous laboratory records. Obese severity was based on standard classification[6]: Class I (BMI 30-35), Class II (BMI 36-40), and Class III (BMI >40).

Relevant data was extracted from electronic medical records that were contemporaneously entered and later analyzed. For comparison purposes, the non-obese limbs in the data set (n = 1513) that were otherwise similar to the obese group were used as controls for most parameters. Because of missing data (test not performed because of technical difficulty, no shows, or other reasons), n values for comparisons vary, which are shown in context.

Indications for intravascular ultrasound (IVUS)/venous stenting in the obese

Patients were selected based on persistent severity of symptoms unresponsive to conservative measures: limb pain, swelling, dermatitis/ulcer, or combination. Pain assessment was based on visual analogue scale (0-10)[7] and swelling on venous severity scoring system (0, none; 1, afternoon onset; 2, morning onset; 3, permanent).[8]

Preoperative investigations. A thrombophilia work-up (Protein C&S, antithrombin 3, anticardiolipin antibody, lupus anticoagulant, homocystine levels Factor V&10 gene mutations) was routinely carried out. Other routine investigations included assessment in the venous laboratory duplex examination, arm/foot pressure differential and reactive hyperemia pressure measurements,[9] ambulatory venous pressure measurement, airplethysmography, nucleotide lymphoscintography[10] (in limbs with swelling for diagnostic and prognostic value), and transfemoral venography with exercise femoral pressure measurement as previously described.[11,12] An exercise pressure increase of ≥4 mm Hg was considered significant. A resting pressure differential of ≥3 mm Hg over the contra lateral limb was also considered significant. Many of the listed tests can be combined into smaller number of procedures or performed from the same venipuncture rendering the assessment process less onerous than it appears. With some coordination in data collection, investigative assessment is well tolerated. One or more of these investigations were not possible in some patients because of technical difficulties with venipuncture, large limb size (air plethysmography [APG], pressure cuff), or radiographic table weight limitations. A diagnostic intravascular ultrasound examination (IVUS)[13] was the definitive test for iliac venous obstruction and carried out in all symptomatic patients considered for stenting even if foregoing tests were inconclusive or non-revealing of iliac vein pathology. Venous stenting was carried out concurrently with IVUS examination when a lesion was found.

IVUS allows area measurements of the venous lumen by an electronic planimetry software that comes with the device. *Focal* stenosis is calculated by comparing area of the stenosis with normal area beyond the stenosis. *Diffuse* stenosis, where the entire iliac vein segments are uniformly narrowed, is approximated by comparing the luminal area at the narrowest point to nominal maximal area expected in 'normal' adults: 254 sq mm (based on 18 mm diameter, which is often even larger in the obese) for the common iliac vein.

Technique. The procedure was performed under general anesthesia for better cardio-pulmonary and pain control in a dedicated endovascular suite in patients ≤450 lb weight. A standard operating room table with C arm was used for those exceeding the weight limit. Endovenous ablation of the great saphenous vein was concurrently carried out with the stent procedure if saphenous reflux was present. IVUS examination was carried out via a mid-thigh ante grade femoral vein access with the patient in the supine position, which enhances procedural ease and anesthetic safety in obese patients. Saphenous access (used for ablation) for stent placement also is unsatisfactory because the distal end of the stent may land in the upper spahenous vein when infrainguinal stent extension is required, and the common femoral vein segment below the saphenous entry will be inaccessible to stent placement if needed. IVUS-identified lesions were balloon dilated to normal adult lumen size (16 mm-18 mm for common iliac, 14 mm-16 mm for external iliac, 12 mm-14 mm for common femoral vein) and stented per standard technique.[14] Recanalization of totally occluded vein segments was carried out if needed per technique previously described.[15] Bilateral venous stenting was staged with several weeks in between procedures for technical ease and because contralateral symptoms often improve or abate following stenting of the most symptomatic limb. When required, bilateral staged iliac vein stenting requires fenestration of the initial stent; the technique has been described in detail elsewhere.[16] Large caliber (16 mm-20 mm) braided stents (Wallstents; Boston Scientific, Natick, Mass) were oversized by at least 2 mm beyond balloon dilated size. This allows for potential overdilatation later in case of instent restenosis or stent compression. Stents were routinely extended into the inferior vena cava for 3 cm-5 cm to prevent recurrence of stenosis at the iliac-caval junction. Stents were extended higher into the infrarenal vena cava if caval disease was present. The entire vein segment(s) (vena cava, common iliac, external iliac, common femoral) bearing the IVUS-detected lesions were covered in continuity with the stent assembly without skip areas and with generous overlap (3 cm-5 cm) between stents. Stents were extended across the inguinal ligament into the common femoral vein if retroinguinal lesions/compression were present or the common femoral vein was itself involved in the disease process.

The same compression regimen (use or non-use) practiced by the patient preoperatively was continued after stenting. Patients were maintained on aspirin 81 mg daily on an empiric basis after stent placement unless thrombophilia was present or patients were already on anticoagulation, in which cases warfarin anticoagulation was started/continued. Patients were discharged after overnight stay.

Follow-up. Patients were clinically examined at six weeks, three months, and at six-monthly intervals thereafter. Stent patency was routinely examined by transfemoral venography at three to six months and yearly thereafter. In

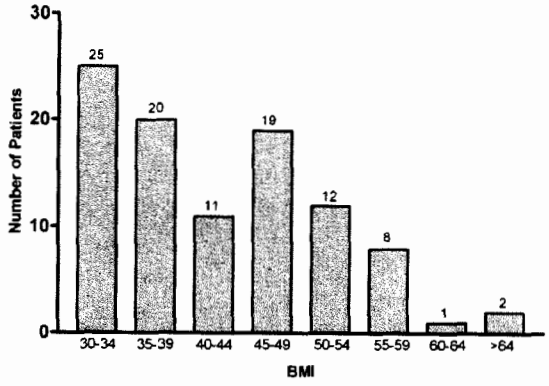

Fig 1. Body mass index (BMI) distribution in 87 obese patients who underwent iliac vein stenting for severe venous symptoms.

the last two years of the study, duplex examination has been used at more frequent intervals for stent surveillance. The duplex technique described by Labropoulos[17] has been found to be reliable in establishing stent patency (flow) even in the obese population (unpublished data).

Data analysis

Continuous and categorical variables were analyzed by paired non-parametric Wilcoxon-Rank test and Chi square test, respectively. Primary, assisted-primary, and secondary patency rates and clinical outcome were calculated using survival analysis with the Kaplan-Meier method. Cumulative survival analysis was used to compare clinical outcome after stenting with present status. Survival curves were pruned when standard error of the mean (SEM) exceeded 10%. A commercially available statistical program (Graph Pad Prism for Windows [version 3.0; GraphPad Software Inc, La Jolla, Calif]) was used for analysis. A *P* value of less than .05 was considered significant.

RESULTS

Iliac-femoral venous stenting was performed in 87 obese patients (101 limbs, 14 bilateral); five patients required recanalization of an occlusion, one bilaterally. The cephalad landing site of the stent assembly was in the distal inferior vena cava in all limbs for the aforementioned technical reasons; in seven limbs a higher landing site below the renal veins was chosen because of disease involvement of the infrarenal vena cava. The distal landing site was in the common iliac vein in 23 (23%) limbs and in the external iliac vein in 21 (21%) limbs; in 57 limbs (56%) the stent was extended below the inguinal ligament into the common femoral vein. Concurrent percutaneous saphenous ablation was performed in 22 limbs (20 laser, two radiofrequency). Five additional limbs underwent traditional open stripping concurrent with stenting.

The median weight was 273 lbs (range, 179 to ≥500 lbs [scale limit]), and BMI 42 (range, 30-83) (Fig 1). Twenty-eight percent of patients were moderately obese

Table I. CEAP classification of 101 obese limbs

	No. of limbs
Clinical classification	
C_2: varicose veins	3*
C_3: edema	52
C_{4a}: pigmentation or eczema	16
C_{4b}: lipodermatosclerosis or white scar	7
C_5: healed venous ulcer	7
C_6: active venous ulcer	16
Etiologic classification	
E_p: primary	57
E_s: secondary	44
Anatomic classification	
A_d: deep veins	51
$A_{s,d}$: superficial and deep veins	45
$A_{d,p}$: deep and perforator veins	1
$A_{s,d,p}$: superficial, deep, and perforator veins	4
Pathophysiologic	
P_o: obstruction	34
$P_{r,o}$: reflux and obstruction	67

*All had severe limb pain.

(Class 1), 18% severely obese (Class II), and 54 % were morbidly obese (Class III) per standard classification.[6] Except for higher BMI and increased incidence of bilateral disease, the obese subset was similar to the non-obese cohort in demographics, distribution across CEAP categories, and distribution of reflux. Median age was 49 years (range, 23-84 years) versus 53 years in the non-obese, Male/female ratio was 3:5 (2:5 in the non-obese). CEAP classification of treated limbs is shown in Table I. The incidence of dermatitis/ulcer was 46% (47/101 limbs) in the entire obese subset versus 40% (602/1513 limbs) in the non-obese ($P = .29$, NS.), and 53% (29/55 limbs) in those with BMI ≥40, (vs. 40% in non-obese, $P = .07$). Ratio of primary and postthrombotic etiologies was 1.3:1, ratio of obstruction alone vs obstruction with reflux 1:2, and ratio of deep reflux alone vs combined superficial and deep reflux 1:1; these parameters were not different from those seen in non-obese limbs ($P = .68$, 0.91, 0.90 respectively). Mean multisegment reflux score was 1 (range, 0-6) in the obese and also 1 (range, 0-7) in the non-obese ($P = .85$). Twenty-eight of 101 (28%) obese patients had bilateral clinical symptoms, significantly higher than 212/1513 (14%) in the non-obese patients ($P = .0007$). The incidence of bilateral disease was even higher, 21/55 (38%), in those with a BMI ≥40 ($P = .0001$), significant compared with the non-obese. Twenty-seven of 77 (35%) patients tested had thrombophilia (37% in the non-obese): antithrombin III (1), protein C (2), protein S (3), lupus anticoagulant (3), prothombin gene (1), Leyden gene (2), homocysteine gene (2), multiple (13). Sixty-two patients were maintained on aspirin and 25 on warfarin after the stent procedure.

Significant co-morbidities were present in many patients: non-insulin dependent diabetes in 7%; insulin dependent diabetes in 6%; hypertension in 49%; sleep apnea in 6%; and history of congestive failure in 3%. Anesthetic risk categories (American Society of Anesthesiologists [ASA])

Table II. Femoral vein pressures in obese limbs compared with non-obese limbs

Femoral vein parameter	*Obese limbs* $N = 73$	*Non-obese limbs* $N = 826$	P *value*
Resting pressure (45% tilt) Mm Hg median (range)	24 (14-39)	24 (14-50)	.85
Pressure increase with exercise median (range)	3 mm Hg (0-26)	2.9 mm Hg (0-28)	.48
Exercise pressure increase ≥4 mm Hg (% limbs)	34%	37%	.84
% patients with bilateral ≥4 mm Hg pressure increase	28%	25%	.16
Resting pressure differential ≥3 mm Hg over opposite limb (% patients)	17%	9%	.13

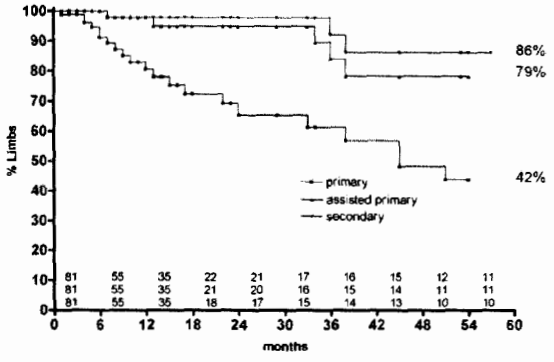

Fig 2. Cumulative patency of stents placed in limbs of obese patients. Limbs at risk at each time interval are shown in the bottom panel.

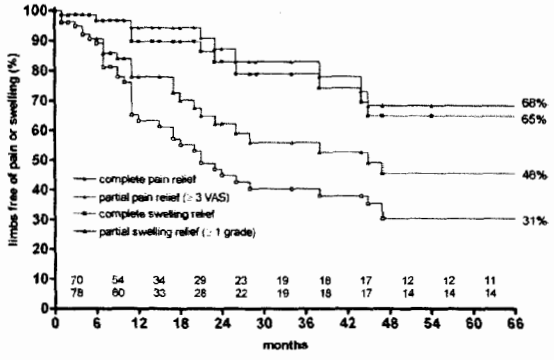

Fig 3. Cumulative relief of pain and swelling in limbs after stenting in the obese. Partial relief of pain is defined as an improvement in pain of at least 3/10 on visual analog scale (VAS) after stent placement. Partial improvement in swelling is defined as an improvement of at least one swelling grade on venous severity scoring system after stent placement. Complete relief of pain or swelling indicates that no residual pain or swelling was present after stent placement. The cumulative curves were generated by censoring limbs that did not meet the stated level of symptom thresholds at each time interval. Limbs at risk at each time interval are shown in the bottom panel.

as recorded by the anesthetist were: Class 1: 1%; Class 2: 43%; Class 3: 52%; and Class 4: 4%.

Femoral vein pressures data was available in 73 obese limbs and in 826 non-obese stented controls; comparison is shown in Table II. Incidence of obstruction based on these hemodynamic criteria was similar.

Nucleotide lymphoscintography showed normal drainage (<15 minute node opacification) in 64 limbs, delayed drainage (15-60 minutes node opacification) in four limbs, and absent drainage (no node opacification at 60 minutes) in one limb. Prevalence of lymphatic dysfunction (7%) was significantly less than in the stented non-obese cohort (18% or 173/964 limbs; $P = .005$).

IVUS area measurements. Varying degrees of stenosis were present in all patients who underwent IVUS examination; the stenosis was focal in 79 limbs (81%) and diffuse in 19 limbs (19%) (data missing in three limbs). Among the diffuse stenosis group (n = 19), the etiology was judged (prior thrombotic history and/or IVUS appearance) to be postthrombotic (Rokitanski stenosis) in eight limbs and from transmural compression in the abdominal compartment in 11 limbs, the latter representing 11% of the entire stented obese subset. Mean area stenosis of focal lesions by IVUS measurements was 74.9 % and diffuse lesions was 48.0%.

Patency. Cumulative secondary stent patency at five years was 86%, as shown in Fig 2. Six stents occluded and two were opened up with catheter-directed lysis.

Clinical outcome. Median (range) and mean (±SD) follow up in the obese series were eight months (range, 1-97 months) and 22 months (±27), respectively. There was no mortality (30 day). Deep venous thrombosis occurred in three limbs (3%) involving the stent in one limb, below the stent in one limb, and in the contralateral limb in one.

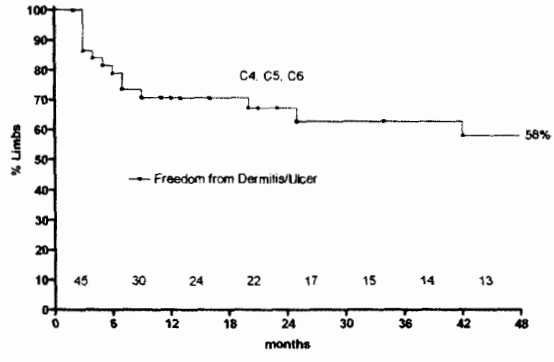

Fig 4. Cumulative freedom from dermatitis/ulcer in CEAP C 4-6 limbs after iliac vein stenting in the obese. Limbs at risk at each time interval are shown in the bottom panel.

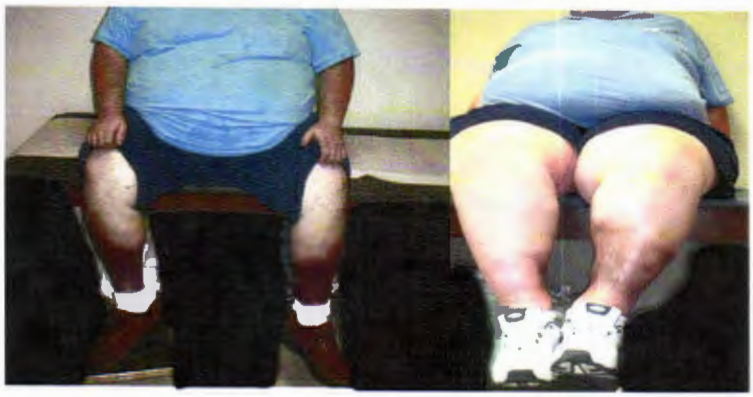

Fig 5. Obese patient with bilateral severe dermatitis, hyperpigmentation, and many small ulcers before (left) and five months after staged bilateral iliac vein stent placement (right).

Table III. Hemodynamic parameters in obese and non-obese limbs

	Non-obese		*Obese*				*Obese vs non-obese pre-stent*	*Obese pre-stent vs post-stent*
Test	*Pre-stent*	*N*	*Pre-stent*	*N*	*Post-stent*	*N*	P	P
AVP post-exercise pressure	68 (0-100)	1084	66 (26-97)	68	68 (35-96)	18	.2924	.1099
VFT seconds	25 (0-248)	1073	24 (2-117)	67	21 (2-120)	19	.4928	.1929
APG VFI_{90} ml	1.9 (0-23)	1343	2.7 (0.3-15.3)	73	2 (0.5-4.2)	34	.0066*	.7609
Venous volume (vv)	85 (5-388)	924	96 (29-226)	49	88 (33-185)	34	.0325*	.0480*
Ejection fraction (EF)	50 (0-107)	923	59 (12-118)	49	67 (15-100)	34	.0437*	.3084
Residual volume fraction (RVF)	32 (0-146)	921	29 (2-88)	49	28 (2-70)	34	.4016	.5998
Hand/foot venous pressure differential	1 (−3-15)	948	1 (0-15)	55	0 (0-3)	18	.1137	.1934
Reactive hyperemia foot pressure rise	5 (−14-48)	995	6 (2-23)	45	4 (2-14)	15	.1848	.0625

APG VFI_{90}, Air plethysmography vein filling index; *AVP*, ambulatory venous pressure; *VFP*, venous filling time.
*Significant.

Cumulative percentage of stented limbs with significant pain relief (>3/10 visual analog scale) at 5.5 years was 68%, with 65% of stented limbs achieving complete pain relief as shown in Fig 3. Significant swelling improvement (≥ Gr. 1) occurred in 46% (cumulative) of limbs at 5.5 years after stenting with complete resolution of swelling in 42% (cumulative) of stented limbs (Fig 3). Fifty-eight percent of limbs (cumulative) were free of ulcer/dermatitis at four years, as shown in Fig 4; eight of 15 active ulcers healed and remained healed at six years. A clinical example is shown in Fig 5.

Venous function studies in the obese limbs before and after stenting are shown in Table III; comparative data in non-obese limbs are also shown. The limb venous volume and ejection fraction measured by airplethysmography was increased in the obese subset (prior to stenting) compared with the non-obese. After stenting, venous volume normalized. Subset analysis indicated that this change in venous volume was entirely due to a substantial reduction in this parameter (97 mL [range, 7-122 mL; n = 16] to 64 mL [range, 33-110 mL; n = 11]; $P = .009$) in obese patients who underwent combined saphenous ablation with stenting. No such changes were noted in venous volume (96 mL pre-stent [n = 33] vs 101 mL post stent [n = 23]; $P = 0.7$ [NS]) in patients after stenting without saphenectomy. Vein filling index (Vfi_{90}) also decreased significantly (3.7 ml [range, 0.8-15.3 mL; n = 24] to 1.8 mL [range, 0.4-12.2 mL; n = 18]; $P = .0025$) after concurrent saphenous ablation but was not reflected however in aggregate in the entire obese subset. Higher ejection fraction noticed in obese limbs was unrelated to co-existing saphenous pathology and trended even higher (not reaching statistical significance) after stent placement; the increase was unrelated to saphenous ablation. No other differences in functional testing from the non-obese or after stenting were noted.

Reinterventions. There were 21 (21%) reinterventions because of residual/recurrent symptoms. In 14 limbs, only balloon dilatation of the existing stent stack was required. In seven others, the stent stack was extended cephalad in one, caudad in five, and in both directions in one limb.

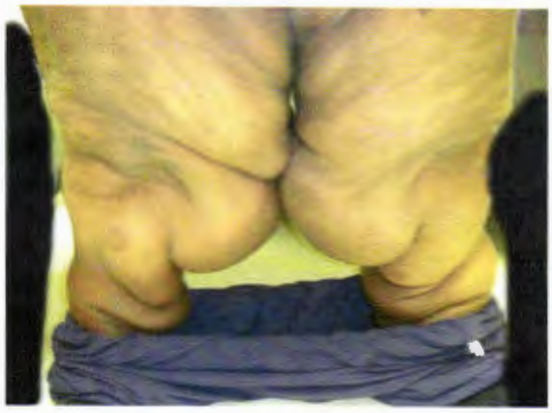

Fig 6. Painful lipedema in obese patient. Pain was completely relieved after iliac vein stenting.

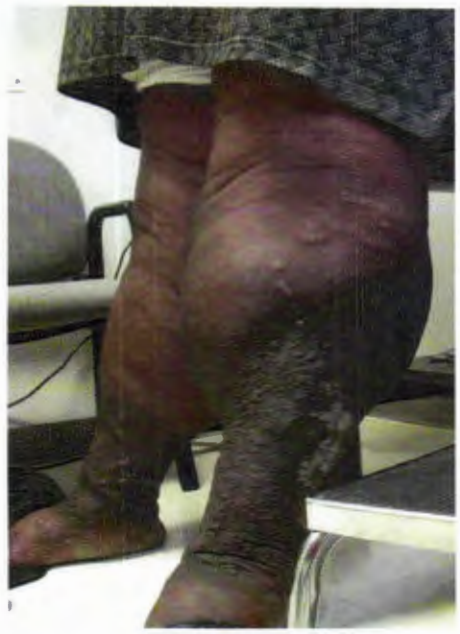

Fig 7. Obese patient with 'inverted bottle' leg. Lipodermatosclerosis and scarring has constricted the lower leg with the upper leg remaining large. Severe dermatitis with ulcerations is present circumferentially around the limb.

DISCUSSION

It is not clear if there is a true increase in the incidence of CVI because of obesity or if it merely reflects the increasing obesity of the general population in association with a common disease; epidemiological evidence is conflicting.[18-22] It has been reported that disease severity of CVI is higher in the obese with bilateral manifestation and more frequent incidence of dermatitis/ulcer than in the non-obese.[1-3] Painful lipedema is a distinct clinical feature in the obese (Fig 6). Severe skin changes are often circumferential in extent (Fig 7). In the current report, increased incidence of bilateral disease was noticed in the obese subset, but evidence for increased clinical and functional severity was mixed. There was a trend towards greater clinical severity in those with a BMI >40 but a significant association in the entire obese subset was not present. Pathophysiologic mechanisms contributing to CVI may also be different in the obese. Hypotheses of such non-traditional mechanisms include genetic predisposition,[1] cytokine activation associated with obesity that damages microcirculation,[23-25] lymphatic dysfunction,[3,26,27] and increased intra-abdominal pressure.[1-3,23,28,29] In the current series, incidence of lymphatic dysfunction using standard nucleotide technique was actually less than in the non-obese subset and less than generally reported by others in CVI patients.[30,31] More sophisticated lymph transport tests showing a link between obesity and lymphatic dysfunction[26] were not carried out in this series. There is solid evidence that intra abdominal pressure is increased in the obese, which may be reflected in increased femoral vein pressures as reported by others.[1,23] Peripheral venous pressure may also be increased.[2] There is dilatation of the femoral vein with increased severity of reflux though calf muscle pump may be paradoxically increased attributable to bigger calf muscle mass in the obese; lack of activity may, however, nullify this advantage.[2] In the current study, ejection fraction was shown to be abnormally high in the obese; after stenting there was a trend in further improvement in ejection fraction. However, resting and exercise femoral pressures were similar to the non-obese in the current analysis. Vfi_{90} did improve and venous volume normalized because of concurrent saphenous ablation. The mechanistic connection between obesity and venous symptoms is particularly intriguing in patients who have severe venous manifestations without detectable duplex reflux. In a report by Padberg and associates,[3] nearly two thirds of patients fell into this category, leading the authors to suggest that compression of venous outflow by increased abdominal pressure was likely the basis of the venous manifestations. In the obese subset presented herein, the incidence and distribution of reflux was similar but all were found to have iliac vein outflow obstruction on IVUS examination. Focal or diffuse primary or postthrombotic lesions of the traditional variety were identified in all but 11 (11%) limbs. Obesity related compressive mechanism was thought to be the operative mechanism in the latter small group. This finding does not necessarily contradict the increased intra abdominal pressure theory as higher transmural pressure can further amplify existing organic obstructive lesions. We have previously shown that venography even by the transfemoral route is insensitive to iliac vein assessment.[11] IVUS is the preferred diagnostic tool for this purpose; IVUS-detectable lesions are present in >90% of patients with advanced CVI features (CEAP clinical class 3 or greater).[11,32]

Weight reduction surgery improves venous symptoms,[28] and is the ideal first choice to address comprehen-

sively the multi-system abnormalities, including CVI manifestations associated with obesity. The severity of venous symptoms such as pain, recurrent cellulitis, or the extensiveness of skin breakdown in some patients may dictate immediate attention to this problem before considering a slower systemic solution. Iliac venous stenting appears to offer specific relief for such patients with high degree of long term patency, low morbidity, and satisfactory efficacy. The procedure is conveniently combined with percutaneous saphenous ablation with negligible additional morbidity when indicated.[33] While saphenous reflux is a factor in CVI manifestations and may be associated with medial ulcers, saphenous ablation alone is unlikely to relieve the circumferential dermatitis/ulcerations, extensive swelling, and pain that are common in the obese patient. A combined saphenous ablation/vein stent placement in a single stage when saphenous reflux is present is therefore preferred in this subset over an incremental approach to maximize outcome at the outset and minimize the need for additional future procedures with attendant additive risks.

AUTHOR CONTRIBUTIONS

Conception and design: SR, PN
Analysis and interpretation: SR, PN
Data collection: SR, PN, RD
Writing the article: SR, PN
Critical revision of the article: SR, PN
Final approval of the article: SR, PN
Statistical analysis: SR, PN, RD
Obtained funding: N/A
Overall responsibility: SR

REFERENCES

1. Danielsson G, Eklof B, Grandinetti A, Kistner RL. The influence of obesity on chronic venous disease. Vasc Endovascular Surg 2002;36: 271-6.
2. van Rij AM, De Alwis CS, Jiang P, Christie RA, Hill GB, Dutton SJ, Thomson IA. Obesity and impaired venous function. Eur J Vasc Endovasc Surg 2008;35:739-44.
3. Padberg F Jr, Cerveira JJ, Lal BK, Pappas PJ, Varma S, Hobson RW 2nd. Does severe venous insufficiency have a different etiology in the morbidly obese? Is it venous? J Vasc Surg 2003;37:79-85.
4. Raju S, Hollis K, Neglen P. Use of compression stockings in chronic venous disease: patient compliance and efficacy. Ann Vasc Surg 2007; 21:790-5.
5. Raju S, Tackett P Jr, Neglen P. Reinterventions for nonocclusive iliofemoral venous stent malfunctions. J Vasc Surg 2009;49:511-8.
6. Buchwald H. Overview of bariatric surgery. J Am Coll Surg 2002;194: 367-75.
7. Scott J, Huskisson EC. Graphic representation of pain. Pain 1976;2: 175-84.
8. Rutherford RB, Padberg FTJ, Comerota AJ, Kistner RL, Meissner MH, Moneta GL. Venous severity scoring: an adjunct to venous outcome assessment. J Vasc Surg 2000;31:1307-12.
9. Neglen P, Raju S. Detection of outflow obstruction in chronic venous insufficiency. J Vasc Surg 1993;17:583-9.
10. Raju S, Owen SJ, Neglen P. Reversal of abnormal lymphoscintigraphy after placement of venous stents for correction of associated venous obstruction. J Vasc Surg 2001;34:779-84.
11. Raju S, Neglen P. High prevalence of nonthrombotic iliac vein lesions in chronic venous disease: a permissive role in pathogenicity. J Vasc Surg 2006;44:136-43; discussion 44.
12. Negus D, Cockett FB. Femoral vein pressures in post-phlebitic iliac vein obstruction. Br J Surg 1967;54:522-5.
13. Neglen P, Raju S. Intravascular ultrasound scan evaluation of the obstructed vein. J Vasc Surg 2002;35:694-700.
14. Neglen P, Berry MA, Raju S. Endovascular surgery in the treatment of chronic primary and post-thrombotic iliac vein obstruction. Eur J Vasc Endovasc Surg 2000;20:560-71.
15. Raju S, McAllister S, Neglen P. Recanalization of totally occluded iliac and adjacent venous segments. J Vasc Surg 2002;36:903-11.
16. Raju S, Owen SJ, Neglen P. The clinical impact of iliac venous stents in the management of chronic venous insufficiency. J Vasc Surg 2002;35: 8-15.
17. Labropoulos N, Borge M, Pierce K, Pappas PJ. Criteria for defining significant central vein stenosis with duplex ultrasound. J Vasc Surg 2007;46:101-7.
18. Abramson JH, Hopp C, Epstein LM. The epidemiology of varicose veins. A survey in western Jerusalem. J Epidemiol Community Health 1981;35:213-7.
19. Callam MJ. Epidemiology of varicose veins. Br J Surg 1994;81:167-73.
20. Hirai M, Naiki K, Nakayama R. Prevalence and risk factors of varicose veins in Japanese women. Angiology 1990;41:228-32.
21. Malhotra SL. An epidemiological study of varicose veins in Indian railroad workers from the South and North of India, with special reference to the causation and prevention of varicose veins. Int J Epidemiol 1972;1:177-83.
22. Stanhope JM. Varicose veins in a population of lowland New Guinea. Int J Epidemiol 1975;4:221-5.
23. Arfvidsson B, Eklof B, Balfour J. Iliofemoral venous pressure correlates with intraabdominal pressure in morbidly obese patients. Vasc Endovascular Surg 2005;39:505-9.
24. Cottam DR, Mattar SG, Barinas-Mitchell E, Eid G, Kuller L, Kelley DE, Schauer PR. The chronic inflammatory hypothesis for the morbidity associated with morbid obesity: implications and effects of weight loss. Obes Surg 2004;14:589-600.
25. Visser M, Bouter LM, McQuillan GM, Wener MH, Harris TB. Elevated C-reactive protein levels in overweight and obese adults. JAMA 1999; 282:2131-5.
26. Brautigam P, Foldi E, Schaiper I, Krause T, Vanscheidt W, Moser E. Analysis of lymphatic drainage in various forms of leg edema using two compartment lymphoscintigraphy. Lymphology 1998;31:43-55.
27. Harvey NL. The link between lymphatic function and adipose biology. Ann N Y Acad Sci 2008;1131:82-8.
28. Melissas J, Christodoulakis M, Spyridakis M, Schoretsanitis G, Michaloudis D, Papavasiliou E, Tsiftsis D. Disorders associated with clinically severe obesity: significant improvement after surgical weight reduction. South Med J 1998;91:1143-8.
29. Sugerman H, Windsor A, Bessos M, Kellum J, Reines H, DeMaria E. Effects of surgically induced weight loss on urinary bladder pressure, sagittal abdominal diameter and obesity co-morbidity. Int J Obes Relat Metab Disord 1998;22:230-5.
30. Collins PS, Villavicencio JL, Abreu SH, Gomez ER, Coffey JA, Connaway C, et al. Abnormalities of lymphatic drainage in lower extremities: a lymphoscintigraphic study. J Vasc Surg 1989;9:145-52.
31. Gloviczki P, Calcagno D, Schirger A, Pairolero PC, Cherry KJ, Hallett JW, Wahner HW. Noninvasive evaluation of the swollen extremity: experiences with 190 lymphoscintigraphic examinations. J Vasc Surg 1989;9:683-9; discussion 90.
32. Raju S. Endovenous treatment of patients with iliac-caval venous obstruction. J Cardiovasc Surg (Torino) 2008;49:27-33.
33. Neglen P, Hollis KC, Raju S. Combined saphenous ablation and iliac stent placement for complex severe chronic venous disease. J Vasc Surg 2006;44:828-33.

Submitted Apr 8, 2009; accepted Jun 24, 2009.