# Obstructive lesions of the inferior vena cava: Clinical features and endovenous treatment

**Seshadri Raju, MD, Kathryn Hollis, BA,** and **Peter Neglen, MD, PhD,** *Flowood, Miss*

*Objective:* **Chronic obstructions of the inferior vena cava (IVC) are associated with many odd features. Even total occlusions may remain entirely silent or present late with acute symptoms. Renal dysfunction is rare. Many have chronic symptoms, but often only one limb is affected. We describe the clinical features in a series of 120 patients seen over a 10-year period and the results of successful stent placement in 99 limbs.**

*Methods:* **Patients with acute onset of symptoms due to distal thromboses underwent catheter-directed thrombolysis. Patients with significant chronic symptoms were investigated by duplex, venous function tests, transfemoral venography and finally intravascular ultrasound (IVUS). Stenotic segments were balloon dilated and occluded segments were recannalized when feasible; stents were placed under IVUS control.**

*Results:* **In the asymptomatic group, 10 patients with total occlusions had transient or no occlusive symptoms. In the acute symptom group, four patients with chronic IVC occlusions presented with acute onset of deep venous thrombosis distal to the occlusion but became asymptomatic when the clot was lysed. In the chronic symptom group, 97 patients (99 limbs) had symptoms of chronic venous disease of variable distribution and intensity. In two-thirds, limb symptoms were unilateral. Pathology was total occlusion in 14%, and the rest were stenoses. The lesion extended above the renal vein in 18%. Common iliac obstruction was concurrent in 93%. Distal reflux was present in 66%. Modifications of the basic stent technique were required in recanalization of total occlusions (four extending up to the atrium), two bilateral stent deployments, and nine IVC filter cases. Stent deployment across the renal and hepatic veins or the contralateral iliac vein had no adverse sequelae. Stent patency (cumulative) at 2 years was 82%. Complete relief (cumulative) of pain and swelling at 3.5 years was 74% and 51%, respectively. The cumulative rate of complete ulcer healing at 2 years was 63%. Overall clinical outcome was rated as good or excellent in 70%.**

*Conclusions:* **The unusual clinical features of IVC obstructions seem related to the rich collateralization, which has an embryonic basis. Common iliac vein patency seems to be a crucial link in collateral function, and its concurrent occlusion produces symptoms. Percutaneous stent placement has an emerging role in the treatment of IVC obstructive lesions, with good mid-term stent patency and clinical results. (J Vasc Surg 2006;44:820-7.)**

Chronic obstructive lesions of the inferior vena cava (IVC) are associated with many unusual features: many have chronic symptoms, but some total occlusions may be entirely silent.[1-3] Some, having remained silent, present acutely with the onset of distal thrombosis.[4-9] Renal and, less often, hepatic impairment are usually absent. Obstructive symptoms in the extremities may be surprisingly variable in type and intensity, and often only one extremity is affected. In this article, we describe the clinical features, technique of stent placement, and mid-term results in a group of cases with IVC stenosis or occlusions seen over a 10-year period.

## MATERIALS AND METHODS

A total of 120 patients with IVC obstructions were encountered from 1997 to 2005, representing 2.8% of patients with chronic venous disease (n = 4217) seen during the same period. Stent placement was not offered in 14 patients because symptoms were mild, transient, or absent to begin with (n = 10) or abated (n = 4) after successful catheter-directed lysis of acute distal thrombus below the chronic IVC obstruction. Stent placement was recommended in the remaining 106 patients. Two patients declined, and placement was successful in 97 patients (99 limbs: 2 bilateral, 14 occlusions, and 85 stenoses) and unsuccessful in 7 (all occlusions). IVC stent placement (n = 99) constituted 6% of all venous stent placements for chronic venous disease during this period.

The female-male ratio was 2:1, and the left-right ratio was also 2:1. The median age was 51 years (range, 14-80 years). Among patients treated with stent placement, limb symptoms were bilateral in only 32 (33%) of 97 and were unilateral in the remaining 65 (67%). Among all four patients who developed acute symptoms with distal thrombosis below a chronically occluded IVC, symptoms were unilateral. The CEAP clinical classification[10] in stented limbs (n = 99) was as follows: C1, 2%; C2, 9%; C3, 37%; C4, 26%; C5, 7%; and C6, 19%.

### Pathology

Eighty-two percent (81/97) of the IVC lesions (n = 97) were infrarenal, 14% (14/97) were suprarenal below the diaphragm, and 4% (4/97) extended further into the thoracic IVC segment. The disease was limited to the IVC proper only in 7% of patients. Distal extension into the

From the University of Mississippi Medical Center and River Oaks Hospital.
Competition of interest: none.
Presented at the Eighteenth Annual Meeting of the American Venous Forum, Miami, Fla, Feb 22-26, 2006.
Reprint requests: Seshadri Raju, MD, 1020 River Oaks Dr, Suite 420, Flowood, MS 39232 (e-mail: rajumd@earthlink.net).
**CME article**
0741-5214/$32.00

doi:10.1016/j.jvs.2006.05.054

common iliac vein or beyond occurred in 93% of the limbs, into the distal common iliac vein in 22%, into the external iliac vein in 37%, into the common femoral vein in 31%, and further distally in 3%. IVC lesions were partial (>60% stenosis) in 86% and were total occlusions in 14%. Collaterals were uniformly present. In nine cases, an IVC filter was present.

Reflux was present in 66 of 99 limbs, with axial reflux to below the knee in 27 limbs. In 49 limbs, reflux involved two or more venous segments. Among 19 limbs with ulcers, reflux was present in 17, all involving both the superficial and deep systems (axial in 10) except in one limb, for which only the superficial system was involved.

### Clinical features

Patients in this series fell into three clinical groups: (1) asymptomatic, (2) acutely symptomatic, and (3) chronically symptomatic.

**Asymptomatic group.** Ten patients in this series were either totally asymptomatic or had only transient or cosmetic symptoms with little functional impairment of the limb. Several (n = 7) sought clinical advice because of limb, scrotal, or abdominal varices. Superficial thrombosis of the abdominal varix was the presenting complaint in two of these cases. Mild transient leg swelling, transient groin pain, and a transient ulcer were the mode of presentation in one patient each. All were treated conservatively for fear of producing overt limb pain and edema by unwarranted surgical trauma in the context of trivial or absent symptoms.

**Acute symptom group.** Four patients initially presented with acute deep venous thrombosis of the lower extremity without prior leg symptoms. All four underwent catheter-directed thrombolysis during which a chronically occluded vena cava was discovered. After successful lysis of the acute thrombus below the occluded IVC, all four patients became asymptomatic again. Three remained asymptomatic, and the fourth patient developed limb symptoms after recurrent distal thrombosis 5 years later.

**Chronic symptom group.** Symptoms were chronic in 99 limbs of 97 patients.

### Assessment

The degree of swelling was assessed by physical examination (grade 0, none; grade 1, pitting, not obvious; grade 2, ankle edema; and grade 3, obvious swelling involving the limb). The level of pain was measured by using the visual analogue scale.[11]

### Investigations

Patients underwent venous laboratory tests including duplex scan, ambulatory venous pressure measurement (percentage decrease; venous filling time), arm/foot pressure test,[12] air plethysmography ($VFI_{90}$),[13] and contrast studies. A valve closure time of longer than 0.5 seconds on duplex scan was defined as reflux.

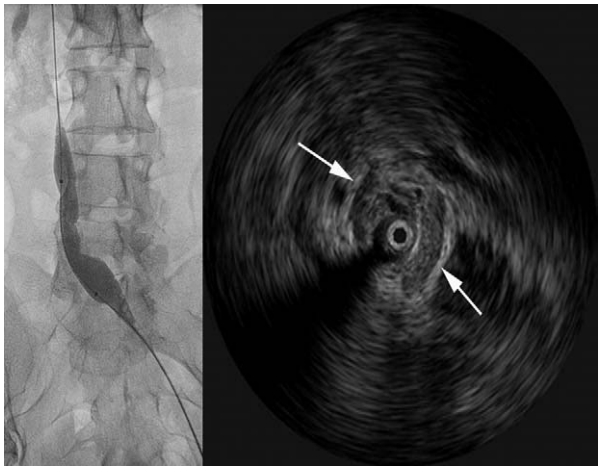

**Fig 1.** The stenosis/occlusion should be dilated to accommodate a stent of approximately the same size as normal anatomy for proper function *(left)*. In rigid lesions and occlusions, overdilation by 1 or 2 mm will allow for recoil. The Glidewire seldom strays outside the occluded vein, and this can be confirmed by intravenous ultrasonography after predilation *(right)*. Dilation of up to 24 mm in the inferior vena cava has not resulted in clinical hemorrhage.

### Stent placement

Stent placement was offered to patients according to symptom severity. Most belonged to CEAP class 3 or higher. Five patients in class C1 and 2 received a stent because of severe associated pain and six others to relieve recurrent cellulitis (4) or recurrent superficial phlebitis (2). Nineteen patients received stents because of active ulceration. In two patients, bilateral iliac vein stents were required in addition to IVC stenting.

**Technique.** Venous stent placement and recanalization procedures have been described in detail elsewhere.[14,15] Some modifications for special situations in IVC were required.

Access was obtained through the femoral vein at the midthigh level under ultrasound guidance with the patient in the supine position. Sheaths ranging from 9F to 14F were used as necessary. Traversing stenotic lesions is straightforward. For recanalizations, Glidewires (Terumo Medical Corporation, Somerset, NJ) of varying sizes, tip angles, and stiffnesses were used in conjunction with supporting catheters (angled or straight tips) as necessary to traverse the occlusions. Because the Glidewire tip is floppy, it invariably remains within the occluded venous structure. The tough adventitia and perivenous fibrosis seem to provide natural resistance against perforations. Limited extravasations of contrast can be safely ignored. Larger dye extravasations (rare with experience) call for cessation of the procedure, which can be reattempted a few weeks later. After the lesion is crossed, further easy passage of the Glidewire signifies proper IVC re-entry, which should be confirmed by venography/intravenous ultrasonography (IVUS). Predilation up to 6 to 8 atm is generally adequate

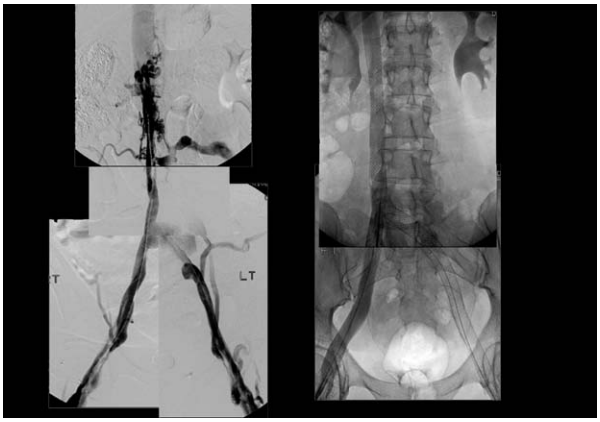


**Fig 2.** A case in which there was segmental occlusion of the inferior vena cava (IVC) at the renal level with additional diffuse stenotic lesions involving infrarenal IVC and both iliofemoral veins *(left)*. The stents were extended into the thoracic IVC and distally into the femoral veins bilaterally *(right)*.

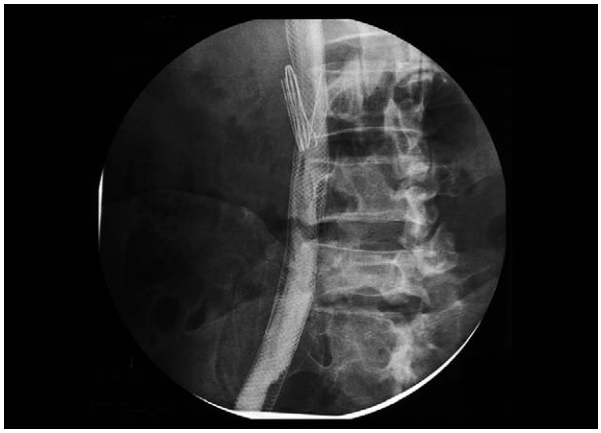

**Fig 3.** A case in which an inferior vena cava filter was pushed aside and a stent was placed past the compacted stent. Nine filters of various makes in common use, including types with prongs, were successfully stented across (despite balloon punctures).

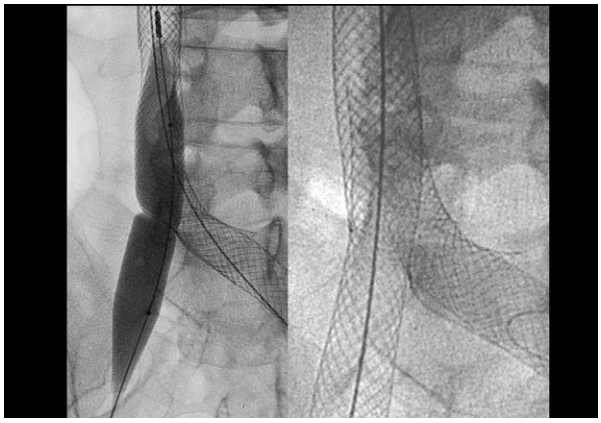

**Fig 4.** Stent placement at the inferior vena cava (IVC) bifurcation: after the IVC stent is extended into one of the iliac veins, a fenestrum is created by balloon dilation over a guidewire. The guidewire is introduced through the opposite side and manipulated through the stent. A generous overlap of 5 cm between the stents is recommended to prevent the second stent from shrinking back through the fenestrum during postdilation. No restriction of either iliac flow has been observed when this technique has been used.

to dilate stenoses, but up to 16 atm can be used to relieve rigid lesions and total occlusions. Progressive dilation is recommended in the latter case (Fig 1).

Wallstents (Boston Scientific, Natick, Mass) ranging from 14 mm in the external iliac vein to 24 mm in the IVC were used as appropriate to match the normal lumen size. Generous overlap of at least 3 to 4 cm is necessary to avoid "shelving" of the stent ends at curvature points and the pelvic brim and to allow for stent shortening with postdeployment dilation. Intravenous ultrasonography is essential to select the optimal proximal and distal landing sites for the stent because disease often extends beyond that visible on venography.[16,17] All diseased segments should be covered by the stent to ensure adequate inflow into and outflow from the stent (Fig 2).

Filters, when present, were treated like rigid lesions and pushed aside by balloon dilation up to 16 atm (Fig 3). When both limbs are symptomatic with bilateral iliac vein/IVC disease, Wallstents can be configured in the form of an inverted Y (Fig 4).

A completion venogram and intravenous ultrasonography are performed to assess proper positioning of the stents and to ensure the absence of intrastent stenoses, thrombi, or uncorrected proximal and distal lesions, all of which should be corrected without hesitation. Prompt deployment of the stents and establishment of flow, once the recanalized channel is predilated, is the key to avoid significant channel or intrastent thrombus formation. When encountered, the thrombus can usually be squeezed out by postdeployment balloon dilation. Rapid clearance of contrast through the stent and disappearance of collaterals is a reliable indication of adequate stent function. Poor inflow, when encountered, should be reassessed with a smaller (6F or 7F) sheath, because larger sheaths can impede inflow. Adjunctive A-V fistulas to increase inflow were not used. We have routinely used sealing devices at the access site (Vasoseal, Datascope Corp, Montvale, NJ) because of the mid-thigh access and the large sheath size needed.

The procedure was performed with patients under general anesthesia on an outpatient basis (23-hour admission). Patients were administered 5000 U of dalteparin before surgery. The procedure was performed under intermittent minimal heparin (1000-2000 U every 90 minutes). More recently, bivalirudin (75-mg intravenous single dose) has been satisfactorily substituted for heparin to further minimize platelet activation. After surgery, patients received dalteparin 5000 U subcutaneously every 12 hours for a maximum of three doses or longer (4 days) if follow-up

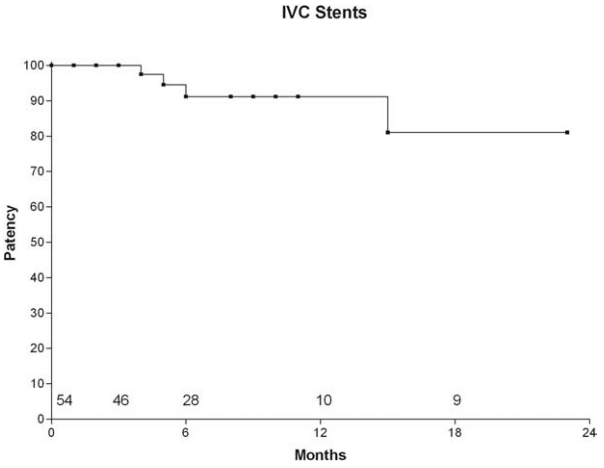


**Fig 5.** Cumulative inferior vena cava *(IVC)* stent patency (primary assisted) confirmed by venography.

warfarin (started the same day after stenting) was required. Warfarin was instituted in cases of thrombophilia and suprarenal stent extension and if patients were taking chronic anticoagulation before surgery. Otherwise, patients were placed on daily aspirin (81 mg).

### Concurrent procedures

Twelve patients underwent saphenous ablation (radiofrequency or laser) concurrent with the stent procedure. Saphenous reflux in five patients was ignored either because the vein was small (<5 mm) in caliber or for technical reasons (prolonged or complex stent procedure).

### Reinterventions

There were 15 primary assisted (patent stent with residual symptoms) interventions in the follow-up period. Nine patients underwent balloon dilation to relieve focal stenoses compressing the stent; these seemed to be the result of inadequate predilation during the original procedure. In six others, extension of the stent proximally or distally was required to correct missed lesions during the original procedure.

### Follow-up and outcome assessment

Patients were examined at 6 weeks and 3, 6, and 9 months after stent placement and at yearly intervals thereafter. Venography and venous laboratory tests were performed during the first 6 months after stent placement and then annually. Outcome was graded as recommended by reporting standards.[18]

### Data analysis

Clinical data were entered prospectively into a time-stamped electronic medical records program for retrospective analysis. A commercially available statistical program (GraphPad Prism for Windows, version 3.0; GraphPad Software, San Diego, Calif) was used for statistical analysis. Actuarial survival curves were constructed according to the Kaplan-Meier method. Nonparametric Wilcoxon rank tests for unpaired data and $\chi^2$ analysis were used to compare groups as appropriate. A $P$ value of less than .05 was considered significant.

**Table.** Venous function tests before and after stent placement in inferior vena cava obstruction

| *Variable* | *Before surgery, median (range)* | *After surgery, median (range)** |
|---|---|---|
| Ambulatory venous pressure | | |
| Pressure drop (%) | n = 58<br>62.5 (1-100) | n = 16<br>51.5 (28-90)† |
| Venous filling time (s) | n = 58<br>19 (3-120) | n = 15<br>33 (6-160)† |
| Air plethysmography | | |
| $VFI_{90}$ (mL/s) | n = 73<br>1.9 (0.1-11.9) | n = 37<br>1.3 (0.2-5.4)† |
| Hand/foot—venous pressure test | | |
| Hand/foot pressure differential (mm Hg) | n = 54<br>1 (0-15) | n = 20<br>0 (0-7)† |
| Reactive hyperemia pressure elevation (mm Hg) | n = 58<br>7 (0-29) | n = 21<br>5 (2-23)† |

*Latest available test.
†$P \leq .0008$.

## RESULTS

Procedure success was 100% in stenotic lesions and 66% (14/21) in occlusions; the latter includes 2 patients in whom recanalization was successful only on the second attempt. In five of seven patients in whom the procedure failed, a minimum of two attempts were made before stent placement was abandoned.

Periprocedure (30-day) mortality and late stent-related mortality during the follow-up period were nil. There were no pulmonary emboli. There was a transient postprocedure creatinine increase (probably contrast related) in one patient, in whom recanalization had failed. There was no instance of clinically apparent hemorrhage that necessitated transfusions or volume replacement. There was no detectable renal or hepatic dysfunction in cases of suprarenal or thoracic stent extensions, respectively. There were no access site complications in this series. There were no malsequelae in patients in whom the procedure was unsuccessful or aborted as a result of major contrast extravasations. Postoperative mild to moderate back pain was a common complaint in many patients (approximately 20%) and was easily controlled by nonsteroidal analgesics; none necessitated admission.

Follow-up was available in 92% (91/99) after stent placement. The median follow-up was 11 months (range, 3-59 months).

Cumulative primary and primary assisted stent patency were 58% and 82%, respectively, at 2 years (Fig 5). Four stents had become occluded during the follow-up period: two for correction of stenoses and two others for recanalizing totally occluded segments. There was no worsening in

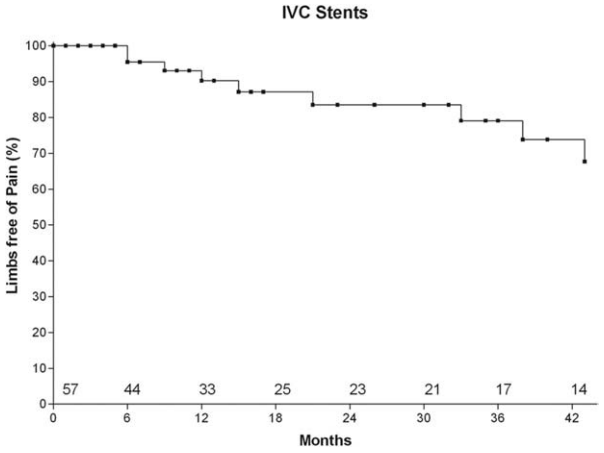


**Fig 6.** Cumulative complete relief of pain after inferior vena cava *(IVC)* stent placement; partial pain relief was marked as failure and censored. Only those who had preoperative pain were included.

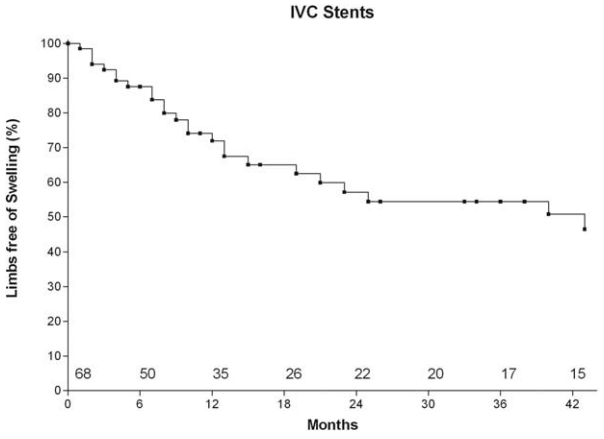


**Fig 7.** Cumulative complete relief of swelling after inferior vena cava *(IVC)* stent placement; partial swelling relief was marked as failure and censored. Only those who had preoperative swelling were included.

reflux or global venous function parameters after stent placement (Table); reflux and obstructive indices actually showed a slight improvement after surgery.

Preoperative pain was present in only 61 (61%) of 99 limbs, and the remainder were pain-free. Among limbs that had limb pain before, 74% (cumulative) were completely pain-free at 3.5 years after stent placement (Fig 6).

Preoperative swelling was present in 75 of 99 patients and absent in the remainder. Among the limbs that had swelling before, 51% (cumulative) were completely free of swelling at 3.5 years after stent placement (Fig 7).

Among 19 with active ulcers, 12 (63%; cumulative) healed and remained healed with complete epithelialization at 2 years. Among the 12 healed ulcers, only 2 had concurrent saphenous ablation, meaning that ulcer healing in the rest could be directly attributed to the stent procedure, because the associated reflux remained uncorrected. Nine limbs with ulcers that healed after stent placement had untreated residual deep reflux (4/9 axial). All seven limbs with unhealed ulcers had residual untreated reflux (six axial).

The overall clinical outcome was graded as follows according to the reporting standards (+3, excellent; +2, good; +1, mild improvement; 0, no improvement; and −1, worse): +3, 43%; +2, 27%; +1, 16%; 0, 13%; and −1, 1%. Thus, 70% of the limbs had an excellent or good clinical outcome at the end of their follow-up period.

## DISCUSSION

The fully developed IVC is a complex structure derived from different segments of an array of multiple paired longitudinal embryonic veins (at least four pairs) and the interconnections between them.[19,20] The development of the IVC near the liver and diaphragm is particularly complex, because new outgrowths from the hepatic veins and the infrarenal IVC have to meet and establish a connection. This area seems to be prone to developmental anomalies such as webs and strictures. Parts of the formative venous network disappear or remain as embryonic rudiments; other parts are recognizable in the adult as named structures, such as the azygos, hemiazygos, accessory hemiazygos, and thoracolumbar veins. All interconnect with each other to freely form a potentially rich collateral network. The thoracolumbar vein receives drainage directly from the common iliac vein through a large connection that is prominent in cases of IVC obstruction (Fig 8). As embryonic alternates to the IVC, growth of the retained named and unnamed putative collateral veins can be expected to be vigorous if the IVC failed to develop properly before birth or occluded even later during the growth period. The normal flow direction in these veins is the same as potential collateral flow. These attributes probably provide for very efficient collateral function, and the collaterals can be appropriately termed *natural collaterals.* These may be so dilated as to appear as a left-sided vena cava or double venae cavae on venograms (Fig 9). Given the fact that the collateral network in IVC obstructions receives drainage directly or indirectly from the common iliac veins, the patency of the latter vein seems to be crucial for the collateral network to function effectively. Natural collaterals elsewhere with an embryonic basis and natural flow direction also seem to function efficiently.[21] Tributary collaterals—main alternate pathways in isolated iliac vein obstructions—seem to function less efficiently; collateral flow direction is opposite normal. Many or most iliac vein occlusions seem to remain symptomatic despite the presence of tributary collaterals on venography.[12] In cases of combined iliac/IVC obstruction, collateral compensation is likely to be poor.

Many curious features of IVC obstructions/occlusions can be explained on the basis of the aforementioned collateral development. Approximately 10% in this series had no, mild, or only transient symptoms. With widespread use of imaging studies, the lesion is being detected in asymptomatic patients as an incidental finding during examination for

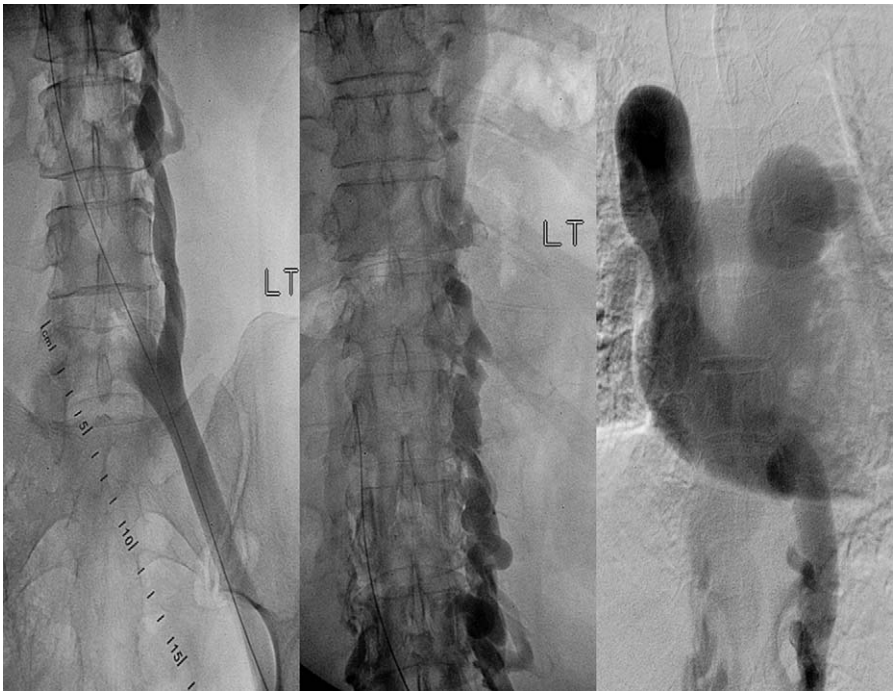


**Fig 8.** Collaterals in a case of inferior vena cava obstruction. Note the large connection between the thoracolumbar vein and the common iliac vein *(left)*. Higher up, the thoracolumbar vein feeds into the azygos network *(center)* to drain into the right atrium *(right)*.

unrelated problems.[1-4,9,22,23] Many are assumed to be congenital agenesis or atresias, even though early occlusions could present a similar picture. The lack of symptoms, including the nearly universal absence of renal or hepatic dysfunction, in this subset is undoubtedly related to excellent collateral development and function. Symptomatic Budd-Chiari syndrome related to segmental IVC obstructions seems to behave differently in this respect; this may in part be related to the concurrent involvement of hepatic veins.[24,25]

There are scores of reports in the literature of previously silent IVC obstructions presenting with symptomatic acute distal deep venous thrombosis. For this reason, chronic anticoagulation has been suggested when such silent lesions are found.[2] There were four patients in this series with such a presentation. All four became symptom-free with lysis of the acute thrombus. This clinical pattern underscores the importance of the common iliac vein as the primary outflow source for the collateral network in IVC obstructions. Ninety-two of 99 symptomatic limbs that received stent placement also had common iliac vein stenosis/obstruction on the stented side. Ipsilateral common iliac vein involvement also may explain why two-thirds of patients in this series had only unilateral symptoms in IVC occlusions. In seven cases in this series, there was no common iliac vein involvement, and the lesion was entirely confined to the IVC. In some of these cases, severe coexisting reflux could be responsible for symptom production. In 3 of 7 cases, stenotic lesions in the major collateral were identified. The critical lesion in the collateral in all three cases was at or near the diaphragm, which could not be traversed with a guidewire (Fig 10).

IVC lesions, particularly occlusions, are lengthy, and there is an intuitive tendency to limit the length of stented segments to minimize thrombosis from stent exposure. Counterintuitively, our experience indicates otherwise. Thrombosis of treated segments has been less related to the metal load of the stents per se than to the violation of established principles of vascular surgery: ensuring proper inflow and outflow (correcting all lesions) and avoiding technical defects related to the conduit (stent). In pursuit of these principles, we have extended the stent above patent renal veins, across contralateral iliac veins, and below the inguinal ligament when necessary.[14-16] There were no thromboses of either the stent or the major tributary outflow in this series. The Wallstent seems to allow free flow of the tributaries across the interstices of the stent. Crossing the inguinal crease has not resulted in fracture or increased stent thrombosis. Stent fracture in arteries at flexion points may be related to metal fatigue from movement related to arterial pulsations. There may be hesitation to dilate the obstructive vein to the size recommended, but fears of hemorrhage with rupture have not borne out in more than 1000 stent deployments (unpublished data). This is probably related to the comparatively low prevailing venous pressure and the constraining influence of perivenous fascia and retroperitoneal cover if rupture indeed occurs in some cases. Imaging studies in the cases of common postopera-

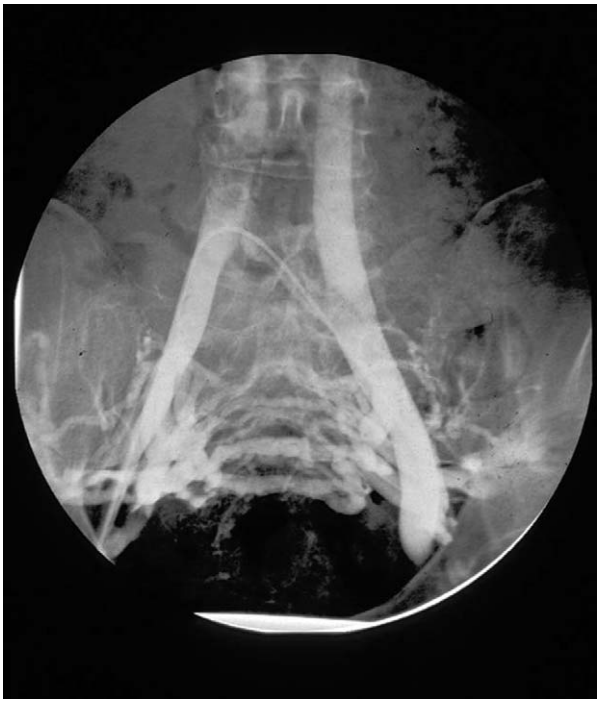

**Fig 9.** A case of a double vena cava with each channel receiving direct drainage from the ipsilateral iliac vein. Anomalous inferior vena cava (IVC) development or later occlusion can give rise to a similar picture. The potentially rich collateral development in IVC occlusions is dependent on the patency of the common iliac vein for proper function.

tive back pain in our early experience ruled out vein rupture as an unlikely etiologic factor. They are no longer performed on a routine basis. Compromising dilation to the required size is likely to result in residual stenosis in the stented segment, stent malfunction, or thrombosis.

We have learned that success or failure in recanalizing total occlusions cannot be predicted by the extent of the lesion or by its venographic appearance. One or more attempts at reopening the occlusion are warranted in all cases. We have been astonished at how even extensive lesions can be traversed with some persistence and patience. Percutaneous stent placement is emerging as an alternative to open surgery[26] to correct IVC obstructions.[27,28] Many of the technical challenges are solvable. Mid-term stent patency with symptom relief is excellent. The healing of stasis ulceration by stent placement alone, even in the presence of significant untreated residual reflux, has been previously reported.[29] Stent placement is an attractive option in treating IVC lesions because it is minimally invasive and safe and seems to be effective at least in the mid term. Also, later open surgery is not precluded.

## AUTHOR CONTRIBUTIONS

Conception and design: SR, PN
Analysis and interpretation: SR, KH, PN

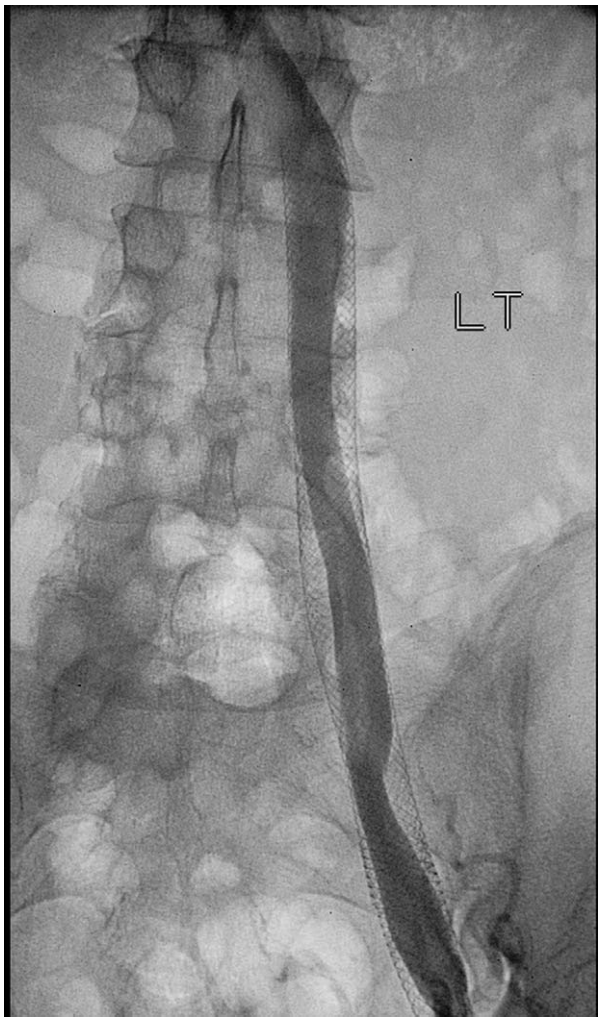


**Fig 10.** A case of inferior vena cava (IVC) occlusion without iliac involvement. The IVC lesion could not be recanalized. The prominent thoracolumbar collateral appeared to harbor multiple stenoses in the lumbar region and also a nonpassable lesion near the diaphragm where it interconnected with the azygos system. The lumbar portion of the collateral was stented, but this failed to relieve the symptoms.

Data collection: SR, KH
Writing the article: SR, PN
Critical revision of the article: SR, PN
Final approval of the article: SR, PN, KH
Statistical analysis: PN, KH
Overall responsibility: SR

## REFERENCES

1. Siegfried MS, Rochester D, Bernstein JR, Miller JW. Diagnosis of inferior vena cava anomalies by computerized tomography. Comput Radiol 1983;7:119-23.
2. Yun SS, Kim JI, Kim KH, Sung GY, Lee do S, Kim JS, et al. Deep venous thrombosis caused by congenital absence of inferior vena cava, combined with hyperhomocysteinemia. Ann Vasc Surg 2004;18:124-9.

3. Senecail B, Lefevre C, Person H, Meriot P. Radiologic anatomy of duplication of the inferior vena cava: a trap in abdominal imaging. A report of 8 cases. Surg Radiol Anat 1987;9:151-7.
4. Debing E, Tielemans Y, Jolie E, Van den Brande P. Congenital absence of inferior vena cava. Eur J Vasc Surg 1993;7:201-3.
5. Chee YL, Culligan DJ, Watson HG. Inferior vena cava malformation as a risk factor for deep venous thrombosis in the young. Br J Haematol 2001;114:878-80.
6. Obernosterer A, Aschauer M, Schnedl W, Lipp RW. Anomalies of the inferior vena cava in patients with iliac venous thrombosis. Ann Intern Med 2002;136:37-41.
7. Lane DA. Congenital hypoplasia of the inferior vena cava: an underappreciated cause of deep venous thromboses among young adults. Mil Med 2005;170:739-42.
8. Gayer G, Luboshitz J, Hertz M, Zissin R, Thaler M, Lubetsky A, et al. Congenital anomalies of the inferior vena cava revealed on CT in patients with deep vein thrombosis. AJR Am J Roentgenol 2003;180:729-32.
9. Alexander ES, Clark RA, Gross BH, Colley DP. CT of congenital anomalies of the inferior vena cava. Comput Radiol 1982;6:219-26.
10. Beebe HG, Bergan JJ, Bergqvist D, Eklof B, Eriksson I, Goldman MP, et al. Classification and grading of chronic venous disease in the lower limbs. A consensus statement. Eur J Vasc Endovasc Surg 1996;12:487-91; discussion 491-2.
11. Scott J, Huskisson EC. Graphic representation of pain. Pain 1976;2:175-84.
12. Raju S, Fredericks R. Venous obstruction: an analysis of one hundred thirty-seven cases with hemodynamic, venographic, and clinical correlations. J Vasc Surg 1991;14:305-13.
13. Christopoulos D, Nicolaides AN. Noninvasive diagnosis and quantitation of popliteal reflux in the swollen and ulcerated leg. J Cardiovasc Surg (Torino) 1988;29:535-9.
14. Raju S, Owen S Jr, Neglen P. The clinical impact of iliac venous stents in the management of chronic venous insufficiency. J Vasc Surg 2002;35:8-15.
15. Raju S, McAllister S, Neglen P. Recanalization of totally occluded iliac and adjacent venous segments. J Vasc Surg 2002;36:903-11.
16. 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.
17. Neglen P, Raju S. Intravascular ultrasound scan evaluation of the obstructed vein. J Vasc Surg 2002;35:694-700.
18. Porter JM, Rutherford RB, Clagett GP, Cranley JJ, O'Donnell TF, Raju S, et al. Reporting standards in venous disease. J Vasc Surg 1988;8:172-81.
19. Warwick R, Williams PL, editors. Gray's anatomy. 35th ed. Philadelphia: WB Saunders; 1973. p. 164-7.
20. McClure CFW, Butler EG. The development of vena cava inferior in man. Am J Anat 1925;35:331-83.
21. Raju S, Fountain T, Neglen P, Devidas M. Axial transformation of the profunda femoris vein. J Vasc Surg 1998;27:651-9.
22. Ruggeri M, Tosetto A, Castaman G, Rodeghiero F. Congenital absence of the inferior vena cava: a rare risk factor for idiopathic deep-vein thrombosis. Lancet 2001;357:441.
23. Schneider JG, Eynatten MV, Dugi KA, Duex M, Nawroth PP. Recurrent deep venous thrombosis caused by congenital interruption of the inferior vena cava and heterozygous factor V Leiden mutation. J Intern Med 2002;252:276-80.
24. Rector WG Jr, Xu YH, Goldstein L, Peters RL, Reynolds TB. Membranous obstruction of the inferior vena cava in the United States. Medicine (Baltimore) 1985;64:134-43.
25. Lee BB, Villavicencio L, Kim YW, Do YS, Koh KC, Lim HK, et al. Primary Budd-Chiari syndrome: outcome of endovascular management for suprahepatic venous obstruction. J Vasc Surg 2006;43:101-8.
26. Jost CJ, Gloviczki P, Cherry KJ Jr, McKusick MA, Harmsen WS, Jenkins GD, et al. Surgical reconstruction of iliofemoral veins and the inferior vena cava for nonmalignant occlusive disease. J Vasc Surg 2001;33:320-7; discussion 327-8.
27. Robbins MR, Assi Z, Comerota AJ. Endovascular stenting to treat chronic long-segment inferior vena cava occlusion. J Vasc Surg 2005;41:136-40.
28. Razavi MK, Hansch EC, Kee ST, Sze DY, Semba CP, Dake MD. Chronically occluded inferior venae cavae: endovascular treatment. Radiology 2000;214:133-8.
29. Neglen PN, Thrasher TL, Raju S. Venous outflow obstruction—an underestimated contributor to chronic venous disease. J Vasc Surg 2003;38:879-85.

Submitted Mar 10, 2006; accepted May 20, 2006.