Phlebolymphology

ISSN 1286-0107

Vol 32 | No. 3 | 2025 | P79-123

No. 118

Phlebolymphology

Aims and Scope

Phlebolymphology **is an international scientific journal entirely devoted to venous and lymphatic diseases.**

The aim of *Phlebolymphology* is to provide doctors with updated information on phlebology and lymphology written by well-known international specialists.

Phlebolymphology **is scientifically supported by a prestigious editorial board.**

Phlebolymphology **has been published four times per year since 1994, and, thanks to its high scientific level, is included in several databases.**

Phlebolymphology **comprises an editorial, articles on phlebology and lymphology, reviews, and news.**

Correspondence

Editorial Manager
Hurrem Pelin YALTIRIK
Servier Affaires Médicales
35, rue de Verdun,
92284 Suresnes Cedex, France
Tel: +33 (1) 55 72 38 98
Email: hurrem-pelin.yaltirik@servier.com

Publication Director
Christophe CHARPENTIER
Suresnes, France

Publisher
Les Laboratoires Servier
50, rue Carnot,
92284 Suresnes Cedex, France
Tel: +33 (1) 55 72 60 00

Indexed in EMBASE, Index Copernicus, and Scopus.

ISSN 1286-0107

Content

Vol 32, No. 3, 2025 No. 118

Editorial

Dear Readers,

This issue explores an important topic: therapeutic interventions targeting deep venous involvement that affects the overall condition of patients with oncological disease and may shorten their life expectancy.
Unlike conventional procedures, the goal is not to achieve long-term technical success but primarily to relieve disabling symptoms.

Significant venous involvement impacting overall health in cancer patients can mainly present in 2 forms: acute and chronic. The first involves episodes of acute or subacute venous thrombosis, with or without pulmonary embolism. The second results from obstructed venous outflow, which may be caused by intrinsic factors or external compression.

M. J. MULLINS and **G. O'SULLIVAN** ***(Ireland)*** have extensive experience in treating the first condition: acute and subacute venous thrombosis.
The authors note that cancer-related thrombosis increases morbidity and mortality. Early thrombus removal is a vital treatment option for these patients. However, there is debate about when to treat due to limited data on life expectancy and potential complications, such as bleeding risks. Without specific trials in these cases, treatment decisions depend on individual patient factors.

The other scenario involves obstructed venous outflow.
A. RODRÍGUEZ-MORATA and **colleagues** ***(Spain)*** describe obstruction caused by an endovenous expansion process and potential correction via open surgery. The high surgical complexity of these procedures is evident, especially when tumors extend into the heart.

When flow obstruction is due to external compression, endoluminal techniques can be employed. **O. HARTUNG** ***(France)*** describes how minimally invasive procedures can improve quality of life by resolving inferior vena cava obstructions hemodynamically. A recent meta-analysis reports excellent technical results in primary and cumulative patency rates, along with immediate symptom relief after stenting.

Similar to inferior vena cava obstruction, treatments for superior vena cava obstructions, with endovascular stenting, are discussed by **S. M. HABIB** and **colleagues** ***(Saudi Arabia)***. The authors present findings from a meta-analysis of over 2249 patients, showing favorable outcomes and manageable complications.

Enjoy reading!

Editor in chief
Dr Oscar Maleti

Phlebolymphology Vol 32 | No. 3 | 2025

Early thrombus removal in the oncology patient

Matthew J. Mullins, MD, PhD

Consultant Interventional Radiologist, Galway University Hospital, Galway, Ireland

Gerard O'Sullivan, FRCR, FRCPI, F CIRSE, F SIR

Professor of Interventional Radiology, Galway University Hospital, Galway, Ireland

ABSTRACT

In this review article, we will explore the treatment options and clinical considerations in the setting of hospital presentations with acute/subacute thrombosis in the oncology patient. This a complex disease spectrum that leads to significant morbidity, health care costs, and increased hospital visits and admissions over time. Cancer-associated thrombosis has become a significant cause of morbidity and potential mortality in the oncology patient. Along with challenging treatment regimens and clinical dysfunction caused by the primary underlying disease, thrombosis poses a significant threat to life, functional status, and long-term health. Early thrombus removal in the cancer patient is emerging as an important strategy in managing this complex problem and attempting to limit the immediate consequences such as life-threatening pulmonary embolus and long-term consequences such as postthrombotic syndrome.

We discuss and critique the current literature supporting early thrombus removal and the strategies we employ at our institution to manage this challenging patient cohort. Controversies exist including side effect profiles of various treatment options, theoretical increased bleeding risk, long-term anticoagulation suitability, and overall survival benefit depending on life expectancy. Overall, we advocate for establishing a robust hospital policy detailing the interventional management of acute thrombosis that is readily accessible and regularly advertised to clinicians. Notwithstanding an established pathway, there is still significant nuance to the decision-making algorithm that must ultimately put the patient at the center of all decisions and interventions. The future of managing oncology patients with acute thrombosis is to inform our decision-making by having dedicated trials aimed at comparing interventional management options in randomized controlled trials with a specific focus on this patient cohort.

Keywords

cancer-associated thrombosis | mechanical thrombectomy | oncology | postthrombotic syndrome | venous thromboembolism

Phlebolymphology. 2025;32(3):82-91.

www.phlebolymphology.org

Introduction

Cancer-associated thrombosis is a common occurrence in the oncology patient population with an incidence of between 5% and 20% or put differently it occurs at a rate 4 to 7 times higher in patients with malignancy compared with the general population.[1-3] Some papers have reported up to a 15-fold increased risk.[4] Thrombosis results in significant associated morbidity and mortality. Cancer has been found in up to 5% of patients with an unprovoked venous thromboembolism (VTE).[1,5,6]

VTE has been shown to be the second leading cause of death in cancer patients secondary to disease progression and in close proximity to infection as a cause of mortality other than the cancer itself.[7,8]

Trousseau first described the link between cancer and venous thrombosis. This initial discovered link has been consistently proven for over 100 years.[9] The incidence is up to 100 cases per 100 000 people in the general population of which approximately 33% of patients will meet diagnostic criteria for pulmonary embolism (PE).

The incidence of acute VTE has been proven to be significantly higher in the cancer patient cohort. Clinically significant VTE can be seen in up to 15% of patients. This represents a significant health care burden.

Specific risk factors for oncology patients include metastatic disease and treatment-induced risk, including immunotherapies, and this increased risk stems from a complex interplay of tumor biology, prothrombotic factors, therapeutic interventions, and patient-related variables.

In 2008 in *Lancet Oncology*, Noble et al published an important systematic review and meta-analysis assessing the current practice guidelines for patients with acute VTE in high-risk cancer patients.[10] Since this paper, there have been a number of further studies reviewing management of this vulnerable patient cohort.[11-13]

Traditionally, the cornerstone of thrombus management in cancer has been anticoagulation. However, certain clinical scenarios demand a more aggressive approach. Early thrombus removal—through catheter-directed thrombolysis (CDT), mechanical thrombectomy, or surgical intervention—is increasingly considered in patients with severe thrombotic complications, such as massive PE, phlegmasia cerulea dolens, or critical limb ischemia.[14]

Whereas the benefits of early thrombus removal are well-recognized in the general population, their application in oncology is more nuanced. The inherent bleeding risk, chemotherapy-induced cytopenias, and overall prognosis must be carefully balanced against the potential gains of thrombus extraction. Yet, with improving technology and multidisciplinary collaboration, selected cancer patients may experience significant clinical benefit from early thrombus removal strategies.

This article explores the pathophysiology, diagnostic approach, indications, and techniques of early thrombus removal in oncology patients. It also highlights evidence-based recommendations, emerging data, and clinical considerations unique to the cancer population. A multidisciplinary framework is emphasized to ensure optimal, individualized patient care.

Pathophysiology of thrombosis in cancer

Thrombosis in the context of cancer *(Figure 1)* is a multifactorial process driven by the intricate interaction between malignancy, host response, and therapeutic interventions. The phenomenon was first described by Armand Trousseau in the 19th century as mentioned previously and is now recognized as a hallmark of cancer progression and a key contributor to cancer-related mortality.[9]

Hypercoagulability and the cancer state

Cancer induces a hypercoagulable state through several direct and indirect mechanisms. Tumor cells can express and release procoagulant substances such as tissue factor (TF), cancer procoagulant, and inflammatory cytokines (eg, interleukin-6, tumor necrosis factor-α), which activate the coagulation cascade. TF, in particular, plays a central role in initiating thrombin generation, leading to fibrin clot formation.

Moreover, tumor cells interact with platelets, leukocytes, and endothelial cells, contributing to a prothrombotic microenvironment. This interaction can lead to platelet activation, endothelial injury, and the release of neutrophil extracellular traps (NETs), all of which promote thrombogenesis.[15-17]

Tumor-specific factors

Certain cancers are more thrombogenic than others. Pancreatic, gastric, brain, and lung cancers, along with hematologic malignancies like lymphoma and multiple myeloma, are associated with a particularly high risk of VTE.[18] The stage and burden of disease also correlate strongly with thrombotic risk—advanced and metastatic cancers carry a significantly higher risk.[19]

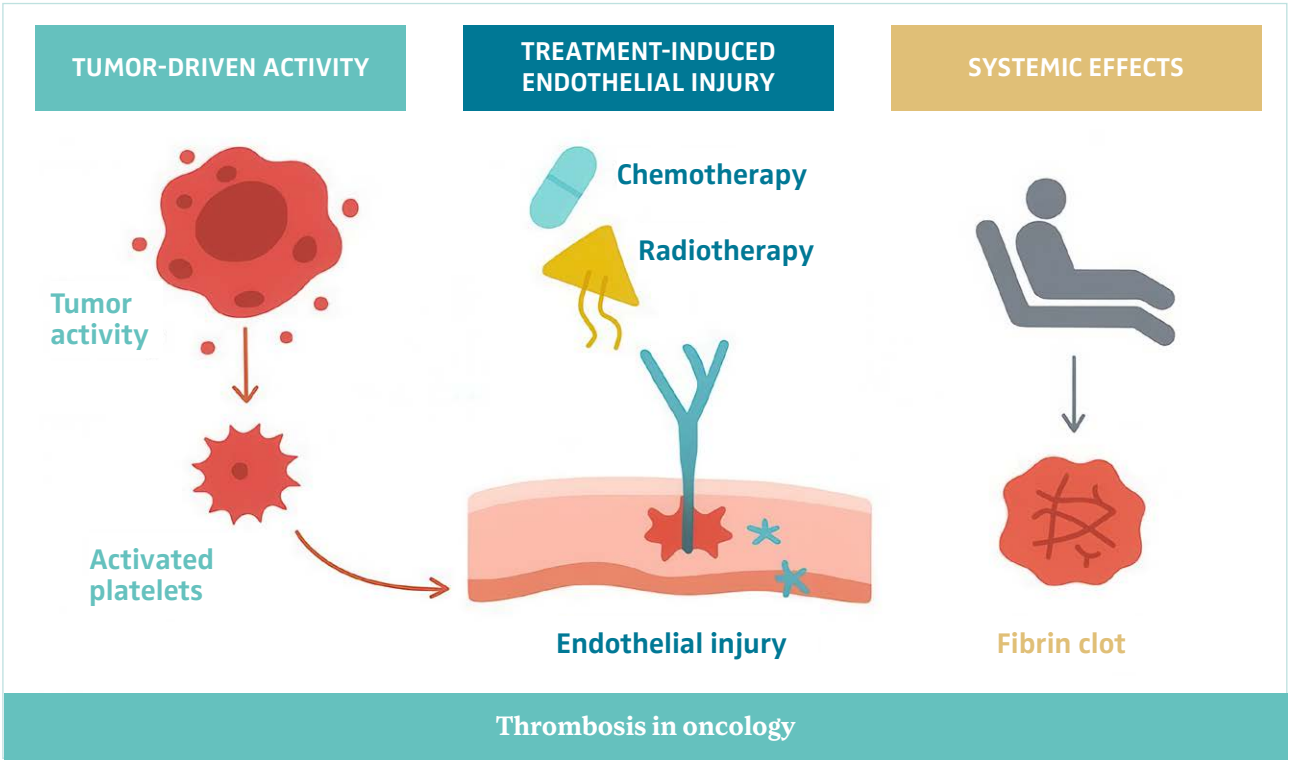

Figure 1. An illustration of the pathophysiology of thrombosis in oncology.
Abbreviations: IL-1β, interleukin 1-beta; TF, tissue factor; TNF-α, tumor necrosis factor alpha; VEGF, vascular endothelial growth factor.

Therapy-related factors

Cancer treatments further exacerbate thrombosis risk. Chemotherapy induces endothelial damage and systemic inflammation. Agents such as cisplatin, thalidomide, lenalidomide, and vascular endothelial growth factor (VEGF) inhibitors are well-known to increase thrombotic risk. Hormonal therapies, including tamoxifen and aromatase inhibitors, also contribute to clot formation.

Radiation therapy can lead to local vascular injury and inflammation, particularly when involving the thoracic or pelvic vasculature. Central venous catheters (CVCs), frequently used in oncology, are another key contributor to thrombus formation, particularly in the upper extremities or central veins.[17]

Patient-related factors

Beyond cancer and its treatments, patient-specific factors—such as immobility, recent surgery, infection, obesity, and inherited thrombophilia—also contribute. Age and performance status further modulate individual risk.[19]

The resulting hypercoagulable state in cancer patients is thus a composite outcome of tumor biology, host response, and therapeutic exposure. This complex pathophysiology underscores the need for a tailored approach to both prevention and intervention, particularly when considering early thrombus removal.[17]

Clinical presentation and diagnosis

Timely recognition of thrombotic events in oncology patients is crucial, given the high morbidity and potential for rapid deterioration. However, the clinical presentation is often subtle, atypical, or masked by underlying malignancy or its treatment *(Table I)*. Clinicians must maintain a high index of suspicion, especially in high-risk patients.

Early diagnosis is critical, particularly when considering advanced interventions like thrombus removal. Delay in recognition can lead to clot propagation, embolization, or postthrombotic complications—outcomes that are particularly dangerous in oncology patients with limited physiological reserve. *Tables II and III* describe diagnostic approaches to cancer-associated thrombosis, along with laboratory and risk assessment tools.

Condition	Common site	Key clinical features	Additional notes
Deep vein thrombosis (DVT)	Lower limbs	Limb swelling Pain/tenderness Erythema, warmth Unilateral calf discomfort	May be asymptomatic or subtle in immunocompromised/debilitated patients
Upper extremity DVT	Arm/neck	Arm swelling Discomfort Jugular distension Visible superficial veins	Common with central venous catheters or mediastinal masses
Pulmonary embolism	Pulmonary arteries	Dyspnea Pleuritic/vague chest pain Tachypnea Hypoxia, syncope	Often clinically silent; symptoms may mimic cancer progression or infection
Catheter-related thrombosis	Catheterized vein	Swelling/discomfort near catheter Poor flow Arm/neck swelling Fever (if infected)	Associated with long-term venous access devices

Table I. Common clinical presentations of thromboembolism in oncology patients.

Modality	Role / utility	Comments
Compression ultrasonography	First-line for suspected DVT	High sensitivity/specificity for lower limb DVT
Doppler ultrasound	Evaluation of upper limb or catheter-related thrombosis	Useful for assessing venous flow
CT Venography/ MR venography	If CDUS is negative and there is high clinical suspicion	CDUS often negative with intra-abdominal malignancy
CT Pulmonary angiography (CTPA)	Gold standard for PE diagnosis	Preferred when contrast is not contraindicated
Ventilation–perfusion (V/Q) scan	Alternative for PE	For patients with renal impairment or contrast allergy
Echocardiography	Assessment of right heart strain or massive PE	Adjunctive tool in unstable patients

CDUS, color Doppler ultrasound; CT, computed tomography; DVT, deep vein thrombosis; PE, pulmonary embolism.

Table II. Diagnostic approach to cancer-associated thrombosis.

Category	Test / tool	Key points
Laboratory testing	D-dimer	Elevated in malignancy; useful for ruling out VTE in low-risk patients only
	Coagulation profile & platelet count	Essential before anticoagulation or thrombus removal
Risk stratification	Khorana score	Predicts VTE risk in ambulatory cancer patients on chemotherapy (based on cancer type, platelet count, hemoglobin, leukocytes, BMI)
	Vienna CATS / COMPASS-CAT	Emerging models; less validated in clinical practice

BMI, body mass index; Vienna CATS, Vienna Cancer and Thrombosis Study; COMPASS-CAT, Prospective Comparison of Methods for Thromboembolic Risk Assessment with Clinical Perceptions and Awareness in Real Life Patients–Cancer Associated Thrombosis study; VTE, venous thromboembolism.

Table III. Laboratory and risk assessment tools.

Clinical significance of acute VTE in oncology

VTE represents a major cause of morbidity and mortality among cancer patients, second only to progression of the underlying malignancy as a cause of death. The incidence of VTE varies by tumor type, stage, and treatment modality, but can exceed 20% in high-risk cohorts, particularly those with pancreatic, gastric, brain, or lung cancer.[17] The consequences extend far beyond the acute event, influencing both short-term survival and long-term functional outcomes.

Acute morbidity is often substantial. Deep vein thrombosis (DVT), particularly when involving the iliofemoral or caval segments, can cause severe limb swelling, pain, and functional impairment. These symptoms can delay or interrupt chemotherapy, radiotherapy, or surgical interventions, thereby compromising oncologic control. PE carries a significant risk of sudden death and is frequently underdiagnosed in this population due to overlapping respiratory symptoms and the presence of comorbid conditions such as infection or tumor emboli. Cancer patients with VTE are at greater risk of recurrent events despite adequate anticoagulation, and they experience higher in-hospital mortality compared with non-cancer counterparts.

Chronic sequelae are equally important. Persistent venous obstruction and valvular damage lead to venous hypertension and the development of postthrombotic syndrome (PTS), characterized by chronic pain, edema, and skin changes, which may progress to ulceration. PTS affects up to 50% of patients following proximal DVT and significantly diminishes quality of life. In oncology, this morbidity compounds the physical and psychological burden of cancer and its treatments.[20] Reduced mobility and chronic limb symptoms can contribute to deconditioning, increased dependency, and reduced ability to tolerate ongoing therapy.

Catheter-associated thrombosis, particularly of the upper extremity or central veins, poses unique challenges. Central venous access is vital for chemotherapy, parenteral nutrition, and blood sampling. Thrombosis can necessitate catheter removal, interrupting essential treatment pathways, or lead to superior vena cava syndrome with significant symptomatic distress. Early thrombus removal strategies in this context may help maintain venous access and avoid treatment delays.

Moreover, VTE carries a prognostic implication in oncology. The occurrence of thrombosis is often a marker of biologically aggressive disease, correlating with higher tumor burden and poorer survival. This clinical reality provides a compelling rationale for considering early thrombus removal in selected patients to preserve function, enhance comfort, and sustain the delivery of cancer therapy.

Rationale for early thrombus removal

The concept of early thrombus removal arises from the recognition that anticoagulation alone, though effective at preventing thrombus extension and recurrence, does not actively lyse established clot or restore venous patency. In the oncology population, where thrombotic events often coincide with ongoing treatment, the persistence of obstructive thrombus can translate to prolonged pain, swelling, functional impairment, and interruption of cancer therapy. Early intervention aims to address these issues by physically or pharmacologically removing clot burden before irreversible venous wall and valve injury occurs.[14,21]

Timing is crucial. Experimental and clinical data indicate that thrombus organization begins within days of formation, leading to fibrosis and adhesion to the venous endothelium. When this has occurred, the success of thrombus dissolution or extraction declines markedly. The term early thrombus removal typically refers to intervention within 10–14 days of symptom onset, a window during which the thrombus remains friable and amenable to mechanical or pharmacologic clearance. Early restoration of venous flow can mitigate inflammation and prevent valvular reflux, thereby reducing the risk of PTS.

The potential benefits of early thrombus removal in oncology are multifaceted. From a symptomatic perspective, rapid decompression of venous obstruction provides prompt relief of pain and swelling, often restoring mobility and improving quality of life. Functionally, it may enable continuation of systemic cancer therapy without delay, preserve venous access routes, and prevent long-term complications that contribute to morbidity and hospital readmission. In younger or fitter oncology patients with good performance status, these benefits can be particularly meaningful, preserving independence and treatment tolerance.

Anticoagulation limitations are well-recognized. Despite appropriate therapy, up to half of patients with proximal DVT develop PTS within 2 years, and residual thrombus is a strong predictor of recurrence. Moreover, in cancer, persistent thrombosis may compromise limb function or necessitate cessation of indwelling catheters essential for chemotherapy. Thus, anticoagulation alone may be insufficient in high-burden or anatomically critical thrombi.

Patient selection is fundamental. Candidates for early thrombus removal typically present with extensive

iliofemoral or caval DVT, severe symptoms, or limb-threatening venous congestion, and have a reasonable life expectancy (>6–12 months) with manageable bleeding risk. Conversely, patients with advanced metastatic disease, thrombocytopenia, or recent major surgery may be unsuitable. A careful multidisciplinary assessment—including oncology, interventional radiology, vascular medicine, and hematology—is essential to balance procedural benefits against hemorrhagic risk.

In summary, early thrombus removal seeks not merely to reduce clot burden but to preserve function, maintain quality of life, and sustain cancer treatment continuity. When judiciously applied, it represents an important adjunct to anticoagulation in selected oncology patients with acute, extensive venous thrombosis.

Evidence base

The evidence supporting early thrombus removal has evolved over the past two decades through a series of randomized controlled trials and observational studies—though, notably, patients with active malignancy have been largely underrepresented in these datasets. Consequently, most current recommendations for oncology patients rely on extrapolation from broader VTE populations.

Trials in the general population

The CaVenT trial (Catheter-Directed Venous Thrombolysis in Acute Iliofemoral Vein Thrombosis; 2012) was among the first randomized studies to demonstrate a potential benefit of CDT. Involving 209 patients with acute iliofemoral DVT, the study found that CDT, in addition to anticoagulation, reduced the incidence of PTS at 2 years (41% vs 56%) and improved iliofemoral patency. However, major bleeding occurred in 3% of patients. Importantly, those with active cancer were excluded, limiting direct applicability to oncology populations.[22]

The ATTRACT trial (Acute Venous Thrombosis: Thrombus Removal with Adjunctive Catheter-Directed Thrombolysis; 2017), the largest to date, randomized 692 patients with proximal DVT to pharmacomechanical CDT (PCDT) or standard anticoagulation alone. Overall, PCDT did not significantly reduce the rate of PTS at 24 months, although it was associated with faster symptom relief and improved quality-of-life measures in the iliofemoral DVT subgroup. The trial confirmed a higher bleeding risk (1.7% vs 0.3%) with intervention but again excluded patients with active malignancy, citing their heightened hemorrhagic risk and limited life expectancy.[23]

The CAVA trial (Ultrasound-Accelerated Catheter-Directed Thrombolysis versus Anticoagulation for the Prevention of Postthrombotic Syndrome; 2020) evaluated ultrasound-accelerated CDT versus anticoagulation alone, showing a trend toward lower PTS rates but no statistically significant benefit. Bleeding risk remained a concern, and as with previous trials, patients with cancer were excluded. Collectively, these trials demonstrate that while early thrombus removal can restore patency and improve short-term symptoms, its role in preventing long-term complications remains debated.[24]

Observational and subgroup data in oncology

Oncology-specific evidence consists mainly of retrospective case series, institutional experiences, and registry data. These reports suggest that thrombectomy and low-dose CDT can be performed safely in selected cancer patients, particularly with modern devices and careful monitoring. Major bleeding rates range from 0% to 5%, lower than historically expected, and technical success rates often exceed 90%.

A retrospective study by Bækgaard et al (published in 2014) included 22 cancer patients undergoing CDT for iliofemoral DVT, reporting significant symptom improvement and acceptable bleeding risk.[25] Similarly, small institutional series have described successful pharmacomechanical thrombectomy using devices such as AngioJet and Lightning 12/16, often achieving rapid recanalization without thrombolytic infusion.[26,27] Although promising, these studies are limited by small sample sizes, heterogeneity in cancer types, and absence of long-term follow-up.

Guideline perspectives

Contemporary guidelines reflect the limited oncology data. The Society of Interventional Radiology (SIR), European Society for Vascular Surgery (ESVS), and American Society of Hematology (ASH) all recommend considering early thrombus removal in selected patients with acute iliofemoral DVT, severe symptoms, and low bleeding risk—but none issue specific guidance for cancer patients. The consensus emphasizes multidisciplinary evaluation and individualized care, acknowledging the need for dedicated studies in this population.[28-30]

Evidence summary

In summary, whereas high-level evidence supports early thrombus removal in well-selected non-cancer patients, data specific to oncology remain scarce. The available retrospective experience suggests that with modern techniques and careful selection, intervention can be safe and effective. However, the absence of prospective oncology-focused trials continues to limit definitive recommendations, underscoring the need for targeted research in this complex and high-risk population.

Risks and challenges

Whereas early thrombus removal offers the potential for rapid symptom relief and prevention of postthrombotic complications, its application in oncology is constrained by unique risks and clinical complexities. The decision to intervene requires careful balancing of the potential benefits of venous patency restoration against the heightened susceptibility to bleeding, infection, and procedural complications inherent to this population.

Bleeding risk represents the most significant challenge. Cancer patients frequently exhibit multiple hemostatic abnormalities—thrombocytopenia from chemotherapy, disseminated intravascular coagulation, hepatic dysfunction, and mucosal tumor involvement—all of which amplify the risk of hemorrhage. Systemic thrombolysis carries prohibitive bleeding rates in this context, and even CDT and pharmacomechanical thrombectomy may precipitate clinically relevant bleeding, particularly when thrombolytics are used. The risk is accentuated in patients with gastrointestinal or genitourinary malignancies, brain metastases, or recent surgery. Consequently, purely mechanical thrombectomy techniques are increasingly preferred where feasible, as they minimize or eliminate lytic exposure.

Procedure-related complications also merit consideration. Vascular injury, distal embolization, and access-site hematomas may occur, particularly in patients with friable or irradiated vessels. Catheter-directed therapies often require prolonged infusion times and intensive monitoring, which can be poorly tolerated by debilitated or immunocompromised patients. Moreover, oncology patients are at higher risk of infection and line sepsis, especially when central venous access is required for both thrombolysis and chemotherapy administration.

Another critical factor is patient prognosis. For individuals with advanced or metastatic disease, the survival benefit of thrombus removal may be marginal, and the procedural burden disproportionate. In such cases, palliative symptom control through compression therapy and anticoagulation may be more appropriate. Conversely, for patients with limited disease burden, good performance status, and longer expected survival, maintaining limb function and treatment continuity may justify a more aggressive interventional approach.

Resource and logistical considerations further complicate decision-making. Thrombectomy procedures require specialized expertise, imaging, and postprocedural surveillance, which may not be available in all centers. The need for multidisciplinary coordination between interventional radiology, oncology, hematology, and vascular medicine adds complexity but is essential for safe and effective care.[31,32]

Ultimately, the key challenge lies in individualizing therapy. No single algorithm can accommodate the heterogeneity of cancer types, patient comorbidities, and treatment goals. Decisions must be guided by tumor biology, anticipated bleeding risk, symptom burden, and patient preference. Meticulous selection, procedural planning, and periprocedural management are critical to minimizing complications while achieving meaningful clinical benefit.

Clinical decision-making framework

Early thrombus removal in oncology patients *(Table IV)* requires a multidisciplinary, patient-focused approach. Balancing thrombotic and bleeding risk, prognosis, and treatment goals mandates collaboration between interventional radiology, oncology, hematology, and vascular medicine.

Patient selection

Careful selection ensures safety and efficacy. Ideal candidates have: i) acute (<14 days) symptomatic iliofemoral or caval DVT with limb-threatening congestion or severe pain; ii) good performance status (ECOG [Eastern Cooperative Oncology Group performance status scale], 0-2) and expected survival >6-12 months; iii) limited metastatic disease where mobility and venous access preservation are priorities; and iv) acceptable bleeding risk (platelets >75 x 10^9/L, no recent major surgery or intracranial metastases). Patients with advanced disease, poor function, or high bleeding risk are best managed conservatively with anticoagulation and compression therapy.

Procedural strategy

Technique selection should match the individual's risk profile. Mechanical or pharmacomechanical thrombectomy is preferred in higher bleeding risk cases, whereas CDT may suit those with stable counts and low-risk tumor types. Key procedural principles include correction of coagulopathy, platelet optimization, ultrasound guidance, and minimal contrast use.

Multidisciplinary coordination

Close coordination between oncology, hematology, and interventional teams ensures timing avoids chemotherapy nadirs and optimizes anticoagulation management.

Shared decision-making

Given variable prognosis, patient preferences and goals of care must guide intervention. Discussions should address

expected symptom relief, procedural risk, and recurrence potential.

At our institution, integrated oncology–interventional radiology (IR) referral pathways support timely intervention. Further education of medical and surgical teams could enhance awareness of the urgency of clot removal in cancer patients.

Technique	Key devices	Indications	Lytic use	Bleeding risk	Advantages	Limitations
Systemic thrombolysis	Alteplase, Urokinase	Life- or limb-threatening thrombosis when catheter access unavailable	High systemic dose	High (especially CNS or GI malignancies)	Rapid clot lysis	High bleeding, rarely used in cancer
Catheter-directed thrombolysis (CDT)	Multi-side-hole infusion catheters	Acute iliofemoral/caval DVT, severe symptoms	Low-dose local infusion	Moderate	Targeted therapy, improved patency	Prolonged infusion, ICU monitoring, bleeding risk
Pharmaco-mechanical thrombectomy (PMT)	AngioJet[a], Cleaner[b]	Acute proximal DVT with high clot burden	Low-dose adjunctive	Low–moderate	Rapid clot removal, reduced lytic dose	Requires expertise, device-specific learning curve
Mechanical thrombectomy (MT)	Lightning 12/16[c], FlowTriever[d], ClotTriever[d], Aspirex[e]	Acute proximal DVT, high bleeding risk	None	Low	Single-session, lytic-free, fast recovery	Limited evidence in oncology, may not remove chronic thrombus
Surgical thrombectomy	Open venous thrombectomy	Rare cases: caval obstruction, tumor invading vein	N/A	Moderate–high	Definitive removal	Invasive, high morbidity, rarely needed

CNS, central nervous system; DVT, deep vein thrombosis; GI, gastrointestinal; ICU, intensive care unit.
[a] Manufactured by Boston Scientific; [b] Manufactured by Argon Medical Devices Inc.; [c] Manufactured by Penumbra; [d] Manufactured by INARI; [e] Manufactured by BD

Table IV. Summary of early thrombus removal techniques in oncology patients.

Future directions and ongoing trials

The management of thromboembolic disease in oncology is entering a new era, driven by technological innovation and a growing emphasis on individualized care. The future of early thrombus removal in cancer patients will depend on refining patient selection, minimizing procedural risk, and validating long-term outcomes through robust clinical trials.

Parallel developments in intravascular imaging and artificial intelligence–assisted venous flow modeling may allow real-time assessment of thrombus composition and optimize device selection. Integration of imaging biomarkers could also improve prediction of thrombus chronicity and response to therapy in oncology-specific cohorts.

Technological advancements

Recent progress in mechanical and aspiration thrombectomy devices—such as large-bore aspiration catheters (eg, FlowTriever, Lightning 12/16, Aspirex)—has transformed the interventional landscape. These systems enable efficient clot extraction without the need for thrombolytics, thereby reducing bleeding risk in patients with fragile hemostasis. Next-generation devices now incorporate ultrasound-assisted and rotational mechanisms to enhance efficacy in organized or chronic thrombus, potentially expanding the window for intervention beyond the traditional 14-day limit.

Research priorities

Despite technological gains, the evidence base for early thrombus removal in cancer remains limited. Existing trials, such as CaVenT, ATTRACT, and CAVA, largely excluded patients with malignancy. Dedicated studies are now underway to address this gap, including observational registries exploring outcomes of mechanical thrombectomy in cancer-associated iliofemoral DVT. Key end points include symptom resolution, bleeding complications, recurrence rates, and quality-of-life measures.

Precision thrombectomy

Ultimately, future care will likely adopt a precision-medicine approach, combining clinical, radiologic, and molecular data to guide individualized decisions. Interventional radiology is poised to play a central role in this evolution, bridging oncologic care and vascular medicine to improve survival, preserve limb function, and enhance quality of life.

Conclusion

VTE represents a significant source of morbidity and mortality in oncology patients. Oral anticoagulation plays an essential role but often fails to address the immediate symptom burden or prevent long-term venous complications. Early thrombus removal using CDT, pharmacomechanical thrombectomy, or purely mechanical thrombectomy offers the potential to restore venous patency, relieve symptoms, and preserve limb function.

The rationale for intervention is strongest in patients with acute, extensive proximal DVT, good performance status, and manageable bleeding risk, where procedural benefits can translate into maintained mobility, uninterrupted cancer therapy, and improved quality of life. However, oncology patients do present unique challenges including elevated hemorrhagic risk, comorbidities, and variability in prognosis. This necessitates an individualized multidisciplinary approach. Current evidence supporting early thrombus removal in cancer is limited, largely extrapolated from trials in the general population, highlighting the urgent need for oncology-specific prospective studies.

Advances in device technology and imaging-guided intervention have improved the safety and efficacy of thrombus removal, particularly for patients at high bleeding risk, and emerging research may soon enable precision-guided thrombectomy tailored to tumor biology and thrombus characteristics.

Our final thoughts are that early thrombus removal represents a promising adjunct to anticoagulation for selected oncology patients. Its judicious application, guided by patient goals, multidisciplinary collaboration, and evolving evidence can optimize clinical outcomes and enhance quality of life. This is all taken in the context of underscoring the importance of ongoing research to define best practices in this high-risk population.

CORRESPONDING AUTHOR

Dr Matthew J. Mullins

Radiology Department, University Hospital Galway, Ireland, H91 YR71

EMAIL: *mullinmj@tcd.ie*

Professor Gerard O'Sullivan received an honorarium from Servier for the writing of this article.

References

1. Guntupalli SR, Spinosa D, Wethington S, Eskander R, Khorana AA. Prevention of venous thromboembolism in patients with cancer. *BMJ*. 2023;381:e072715.
2. Lip GY, Chin BS, Blann AD. Cancer and the prothrombotic state. *Lancet Oncol*. 2002;3(1):27-34.
3. Stein PD, Beemath A, Meyers FA, Skaf E, Sanchez J, Olson RE. Incidence of venous thromboembolism in patients hospitalized with cancer. *Am J Med*. 2006;119(1):60-68.
4. Khan F, Tritschler T, Marx CE, et al. Predictors of recurrent venous thromboembolism and bleeding in patients with cancer: a meta-analysis. *Eur Heart J*. 2025:ehaf453.
5. Bastounis EA, Karayiannakis AJ, Makri GG, Alexiou D, Papalambros EL. The incidence of occult cancer in patients with deep venous thrombosis: a prospective study. *J Intern Med*. 1996;239(2):153-156.
6. Prandoni P, Lensing AW, Büller HR, et al. Deep-vein thrombosis and the incidence of subsequent symptomatic cancer. *N Engl J Med*. 1992;327(16):1128-1133.
7. Khorana AA. Venous thromboembolism and prognosis in cancer. *Thromb Res*. 2010;125(6):490-493.
8. Khorana AA, Francis CW, Culakova E, Kuderer NM, Lyman GH. Thromboembolism is a leading cause of death in cancer patients receiving outpatient chemotherapy. *J Thromb Haemost*. 2007;5(3):632-634.
9. Trousseau A. Phlegmasia alba dolens. Clinique medicule de l'Hotel-Dieu de Paris. 1865;3:94.
10. Noble SI, Shelley MD, Coles B, et al. Management of venous thromboembolism in patients with advanced cancer: a systematic review and meta-analysis. *Lancet Oncol*. 2008;9(6):577-854.
11. Malgor RD, Gasparis AP. Pharmaco-mechanical thrombectomy for early thrombus removal. *Phlebology*. 2012;27(Suppl 1):155-162.
12. Laporte S, Benhamou Y, Bertoletti L, et al. Management of cancer-associated thromboembolism in vulnerable population. *Arch Cardiovasc Dis*. 2024;117(1):45-59.
13. Laporte S, Benhamou Y, Bertoletti L, et al; INNOVTE TAC Working Group; Groupe de travail INNOVTE CAT. [Translation into French and republication of: ''Management of cancer-associated thromboembolism in vulnerable population'']. *Rev Med Interne*. 2024;45(6):366-381.
14. Meissner MH. Rationale and indications for aggressive early thrombus removal. *Phlebology*. 2012;27(Suppl 1):78-84.
15. Caine GJ, Stonelake PS, Lip GY, Kehoe ST. The hypercoagulable state of malignancy: pathogenesis and current debate. *Neoplasia*. 2002;4(6):465-473.
16. Zhang Y, Zeng J, Bao S, et al. Cancer progression and tumor hypercoagulability: a platelet perspective. *J Thromb Thrombolysis*. 2024;57(6):959-972.
17. Wan T, Song J, Zhu D. Cancer-associated venous thromboembolism: a comprehensive review. *Thromb J*. 2025;23(1):35.
18. Moik F, Ay C, Pabinger I. Risk prediction for cancer-associated thrombosis in ambulatory patients with cancer: past, present and future. *Thromb Res*. 2020;191(Suppl 1):S3-S11.
19. Mulder FI, Horváth-Puhó E, van Es N, et al. Venous thromboembolism in cancer patients: a population-based cohort study. *Blood*. 2021;137(14):1959-1969.
20. Nawasrah J, Zydek B, Lucks J, et al. Incidence and severity of postthrombotic syndrome after iliofemoral thrombosis - results of the Iliaca-PTS - Registry. *Vasa*. 2021;50(1):30-37.
21. Plotnik AN, Haber Z, Kee S. Early thrombus removal for acute lower extremity deep vein thrombosis: update on inclusion, technical aspects, and postprocedural management. *Cardiovasc Intervent Radiol*. 2024;47(12):1595-1604.
22. Enden T, Haig Y, Kløw NE, et al. Long-term outcome after additional catheter-directed thrombolysis versus standard treatment for acute iliofemoral deep vein thrombosis (the CaVenT study): a randomised controlled trial. *Lancet*. 2012;379(9810):31-38.
23. Vedantham S, Goldhaber SZ, Julian JA, et al. Pharmacomechanical catheter-directed thrombolysis for deep-vein thrombosis. *N Engl J Med*. 2017;377(23):2240-2252.
24. Notten P, Ten Cate-Hoek AJ, Arnoldussen CWKP, et al. Ultrasound-accelerated catheter-directed thrombolysis versus anticoagulation for the prevention of post-thrombotic syndrome (CAVA): a single-blind, multicentre, randomised trial. *Lancet Haematol*. 2020;7(1):e40-e49.
25. Bækgaard N. Benefit of catheter-directed thrombolysis for acute iliofemoral DVT: myth or reality? *Eur J Vasc Endovasc Surg*. 2014;48(4):361-362.
26. Li JW, Xue J, Guo F, Han L, Ban RB, Wu XL. Clinical comparison of the efficacy of systemic thrombolysis, catheter-directed thrombolysis, and AngioJet percutaneous mechanical thrombectomy in acute lower extremity deep venous thrombosis [Article in Chinese]. *Zhongguo Yi Xue Ke Xue Yuan Xue Bao*. 2023;45(3):410-415.
27. Pandya YK, Tzeng E. Mechanical thrombectomy devices for the management of pulmonary embolism. *JVS Vasc Insights*. 2024:2:100053.
28. Gerhard-Herman MD, Gornik HL, Barrett C, et al. 2016 AHA/ACC Guideline on the management of patients with lower extremity peripheral artery disease: a report of the American College of Cardiology/American Heart Association Task Force on clinical practice guidelines. *J Am Coll Cardiol*. 2017;69(11):e71-e126.
29. Kakkos SK, Gohel M, Baekgaard N, et al. Editor's Choice - European Society for Vascular Surgery (ESVS) 2021 Clinical practice guidelines on the management of venous thrombosis. *Eur J Vasc Endovasc Surg*. 2021;61(1):9-82.
30. Ortel TL, Neumann I, Ageno W, et al. American Society of Hematology 2020 guidelines for management of venous thromboembolism: treatment of deep vein thrombosis and pulmonary embolism. *Blood Adv*. 2020;4(19):4693-4738.
31. O'Sullivan GJ, Waldron D, Mannion E, Keane M, Donnellan PP. Thrombolysis and iliofemoral vein stent placement in cancer patients with lower extremity swelling attributed to lymphedema. *J Vasc Interv Radiol*. 2015;26(1):39-45.
32. Rahmani G, O'Sullivan GJ. Lessons learned with venous stenting: in-flow, outflow, and beyond. *Tech Vasc Interv Radiol*. 2023;26(2):100897.

Phlebolymphology Vol 32 | No. 3 | 2025

Neoplastic obstruction of the inferior vena cava: surgical approach

Alejandro Rodríguez-Morata, PhD, MD

Angiology and Vascular Surgery Department, Quirónsalud Málaga Hospital, Spain

Juan Pedro Reyes-Ortega, MD

Angiology and Vascular Surgery Department, Quirónsalud Málaga Hospital, Spain

María Luisa Robles-Martín, MD

Angiology and Vascular Surgery Department, Quirónsalud Málaga Hospital, Spain

Laura Gallego-Martín, MD

Angiology and Vascular Surgery Department, Quirónsalud Málaga Hospital, Spain

Patricio David Viteri-Estévez, MD

Angiology and Vascular Surgery Department, Quirónsalud Málaga Hospital, Spain

Phlebolymphology. 2025;32(3):92-99.

www.phlebolymphology.org

ABSTRACT

Neoplastic obstruction of the inferior vena cava (IVC) constitutes a rare but formidable challenge in both vascular and oncologic surgery. It may arise from primary tumors originating within the vessel wall, such as leiomyosarcomas, or more frequently as secondary invasion or tumor thrombus from adjacent malignancies, including renal cell carcinoma, hepatocellular carcinoma, or adrenal cortical carcinoma. The anatomic complexity of the IVC, its proximity to vital structures, and the hemodynamic consequences of obstruction demand a thorough understanding of tumor characteristics, precise preoperative imaging, and meticulous surgical planning. This chapter provides a comprehensive review of the classification, diagnostic workup, and detailed surgical techniques used to manage neoplastic IVC obstruction. We explore the roles of segmental resection, reconstruction techniques, and the use of advanced bypass methods, including cardiopulmonary bypass for intracardiac tumor extension. Outcomes, complications, and long-term prognosis are discussed, emphasizing the importance of an aggressive, multidisciplinary approach to optimize patient survival and quality of life.

Keywords

inferior vena cava | IVC obstruction | leiomyosarcoma | oncovascular surgery | surgical resection | tumor thrombus | venous reconstruction

Introduction

The inferior vena cava (IVC) is the major conduit for venous return from the lower extremities, pelvis, and abdominal viscera to the right atrium of the heart. Neoplastic obstruction of the IVC, although an infrequent clinical entity, represents a significant surgical and oncological challenge due to the vessel's critical role and intricate anatomical relationships. Neoplastic involvement may be classified as primary, originating from tumors such as leiomyosarcomas arising from the tunica media of the IVC wall, or secondary, arising from contiguous spread or tumor thrombus from malignancies in adjacent organs, notably the kidneys and liver.[1,2]

The clinical presentation of neoplastic IVC obstruction varies widely depending on the extent and level of involvement, ranging from asymptomatic incidental findings to life-threatening complications such as Budd-Chiari syndrome or massive venous congestion. Surgical management remains the mainstay of treatment for resectable tumors, aimed at restoring venous patency and achieving local oncologic control. However, the complex anatomy, combined with the high risk of intraoperative complications, necessitates a multidisciplinary approach, including vascular surgeons, cardiac surgeons, oncologists, radiologists, and anesthesiologists.[3,4]

Classification of neoplastic IVC obstruction

Accurate classification of IVC tumors is essential to guide surgical planning and prognostication. The most widely accepted classification divides tumors based on their anatomical location relative to key landmarks such as the renal and hepatic veins and the diaphragm.[5]

- Level I: Tumors confined to the infrarenal segment of the IVC, below the renal veins.
- Level II: Tumors involving the IVC between the renal and hepatic veins, often the most common location for primary leiomyosarcomas *(Figure 1)*.
- Level III: Suprahepatic involvement extending up to the right atrium, requiring more extensive surgical access.
- Level IV: Tumors with intracardiac extension into the right atrium, representing the most advanced stage and necessitating complex cardiothoracic procedures.[6,7]

This stratification is pivotal, as higher-level tumors often require cardiopulmonary bypass, hypothermic circulatory arrest, or thoracoabdominal approaches, whereas lower-level tumors may be addressed via abdominal access alone.[8]

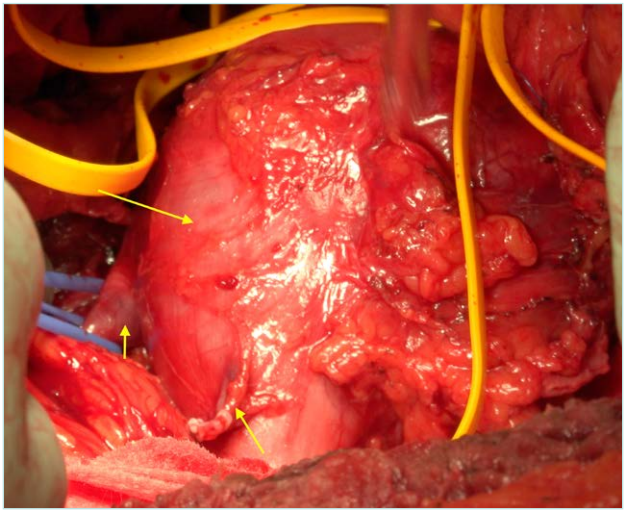

Figure 1. Inferior vena cava (IVC) leiomyosarcoma, ranging from the infrarenal to the adrenal part. Short arrow: right renal vein. Intermediate arrow: right gonadal vein. Long arrow: IVC leiomyosarcoma.

Primary tumors of the IVC

Primary neoplasms of the IVC are rare, with leiomyosarcoma being the most frequently reported histological type. These tumors derive from the smooth muscle cells of the venous wall and show a predilection for the middle segment of the IVC (Level II).[9] The aggressive nature of leiomyosarcomas manifests in rapid local progression, a high rate of local recurrence after resection, and the potential for distant metastases.[7]

Clinically, patients may report nonspecific symptoms such as vague abdominal or flank pain, lower limb edema due to venous obstruction, or symptoms consistent with Budd-Chiari syndrome when the suprahepatic IVC is involved.[10] Preoperative diagnosis relies on imaging and tissue biopsy; however, biopsy must be approached with caution due to the risk of tumor seeding and hemorrhage.[11]

Secondary involvement of the IVC

More commonly than primary tumors, the IVC is involved secondarily through direct invasion or tumor thrombus propagation from neighboring malignancies. Renal cell carcinoma (RCC) is the most frequent culprit, with tumor thrombus extending into the IVC in up to 10% of cases, occasionally reaching the right atrium.[12,13] Hepatocellular carcinoma (HCC) may invade the hepatic veins and the suprahepatic IVC, whereas adrenal cortical carcinomas and retroperitoneal sarcomas are less common sources.[14]

Surgical management in these cases often involves en bloc resection of the tumor thrombus together with nephrectomy or hepatectomy depending on the primary tumor site. This complex surgery requires preoperative planning and intraoperative strategies tailored to the extent of vascular involvement.[15]

Preoperative imaging and planning

Accurate preoperative imaging is indispensable for delineating tumor extent, vascular involvement, and surgical feasibility. Contrast-enhanced computed tomography (CT) remains the cornerstone modality, providing detailed anatomic information on tumor size, location, and involvement of adjacent structures. Magnetic resonance imaging (MRI) offers superior soft tissue contrast and is especially valuable in assessing Level III and IV tumors with possible intracardiac extension.[16]

Transesophageal echocardiography is routinely employed for real-time evaluation of intracardiac tumor thrombus, crucial for surgical planning.[17] Additionally, venography and intravascular ultrasound (IVUS) can provide valuable insights into intraluminal tumor characteristics and venous flow dynamics.[18] A comprehensive assessment including renal function and cardiopulmonary status is mandatory, and all findings should be reviewed in a multidisciplinary tumor board to optimize surgical strategy.[19]

Techniques of IVC resection

The overarching surgical objective is complete oncologic resection with restoration of adequate venous return. Complete excision with negative margins is essential to minimize local recurrence and improve survival outcomes. Preservation of contralateral renal vein drainage is critical to maintain renal function. Surgeons must take great care to avoid tumor fragmentation that can precipitate pulmonary embolism or systemic dissemination.[2,20]

Venous reconstruction is often necessary after tumor resection to maintain hemodynamic stability and prevent venous hypertension. Patient selection is guided by functional status, presence of distant metastases, and extent of tumor invasion; unresectable metastases and poor performance status contraindicate aggressive surgery.[21]

The surgical management of neoplastic obstruction of the IVC requires tailored approaches based on the tumor's location, extent, and involvement of adjacent structures. The techniques can be broadly categorized as follows:

Segmental cavectomy

Segmental cavectomy involves resecting the affected segment of the IVC en bloc with the tumor (*Figure 2*). This technique is the standard for localized tumors, especially those confined to the infrarenal or juxtarenal portions of the IVC, where vascular control is more accessible. Surgical exposure is achieved via laparotomy or thoracoabdominal approaches depending on tumor level.[7,9]

The procedure begins with careful proximal and distal vascular control to prevent hemorrhage and embolization. This is followed by meticulous dissection to separate the tumor-bearing segment from adjacent organs, such as the duodenum, pancreas, or renal structures, preserving vital anatomy. The segment is then excised en bloc with oncologic principles of negative margins.[8]

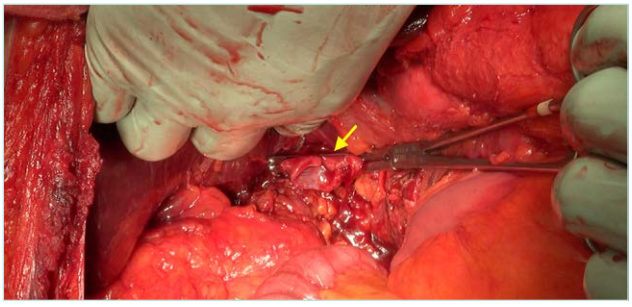

Figure 2. Cavectomy and inferior vena cava (IVC) clamping at the infrahepatic level, ready for vascular reconstruction. Arrow: clamped edge of the IVC.

Reconstruction depends on the length and location of the resected segment. Short resections may allow primary anastomosis, whereas longer segments require patch repair or graft interposition. Segmental cavectomy offers the advantage of complete tumor removal and is associated with favorable oncologic outcomes in selected patients.[22]

Primary closure and patch reconstruction

After tumor excision, small defects of the IVC wall may be closed primarily if the luminal diameter and venous flow can be preserved without significant stenosis. This approach is feasible in cases where the resection does not involve extensive circumferential vessel wall loss.[10]

When larger defects exist, primary closure risks narrowing the lumen and consequent venous hypertension, increasing the risk of thrombosis. In such cases, patch reconstruction is preferred. Autologous materials such as pericardium (either harvested from the patient or bovine derived) offer biocompatibility and durability, reducing infection risk. Synthetic patches like polytetrafluoroethylene (PTFE) are alternatives when autologous tissue is unavailable *(Figure 3)*.[4,14]

Patch cavoplasty restores venous continuity and preserves flow dynamics, minimizing postoperative venous congestion. The choice of material depends on surgeon preference, availability, and patient-specific factors.[7]

Interposition grafts

When resection results in a full circumferential loss of a segment of the IVC, interposition grafting becomes necessary to reestablish venous return. Prosthetic grafts such as PTFE or Dacron are widely used due to their availability and ease of handling *(Figure 4)*. These grafts provide reliable patency but carry a risk of infection, particularly in contaminated fields or prolonged surgeries.[12]

Alternatively, autologous vein grafts, typically harvested from the femoral vein or superficial femoral vein, may reduce infection risk and offer better biocompatibility. However, vein harvesting adds complexity, increases operative time, and introduces donor site morbidity.[23]

Graft sizing is critical to maintain laminar flow and avoid turbulence, which predisposes to thrombosis. Oversized grafts may cause stasis, whereas undersized grafts can cause outflow obstruction. Postoperative anticoagulation protocols should be adapted accordingly to enhance graft patency.[6]

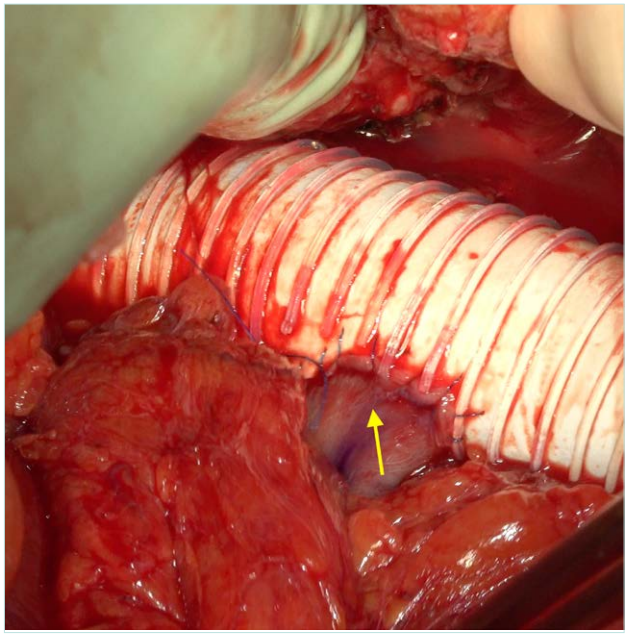

Figure 3. Reimplantation of the right renal vein in the polytetrafluoroethylene (PTFE) body. Arrow: right renal vein.

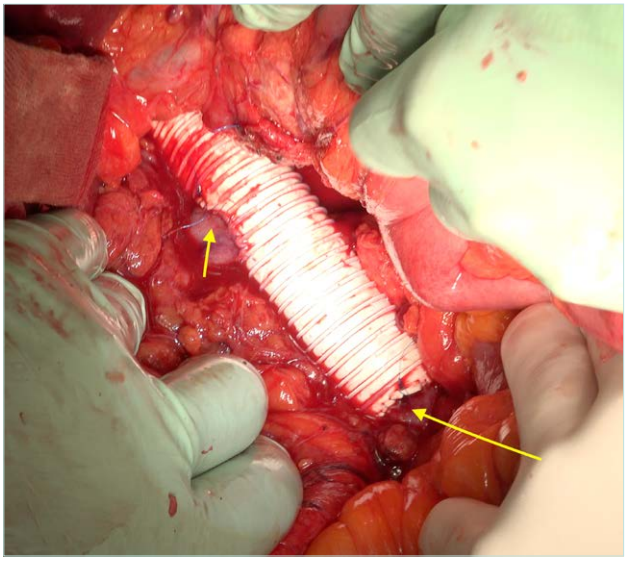

Figure 4. End-to-end bypass reconstruction between the inferior vena cava (IVC) immediately proximal to its iliocaval bifurcation and the retrohepatic zone. Short arrow: right renal vein reimplanted. Long arrow: distal anastomosis, close to the bifurcation.

Cavocaval and atriocaval bypass

In cases of extensive IVC involvement or when prolonged cross-clamping threatens venous return and hemodynamic stability, bypass techniques are employed. Cavocaval bypass entails creating an alternative venous route around the resection site using prosthetic grafts, allowing continuous drainage of lower extremity and abdominal venous blood.[11]

For tumors with intracardiac extension, atriocaval bypass is performed, involving cannulation of the right atrium and the IVC to maintain systemic venous return during complex resections. These bypasses provide a bloodless surgical field and prevent venous congestion, enabling safe tumor excision even in challenging cases.[20]

Bypass procedures require careful coordination between vascular and cardiothoracic teams and meticulous intraoperative management to avoid complications such as air embolism or hemodynamic instability.[19]

Techniques with cardiopulmonary bypass

Level III and IV tumors extending into the right atrium necessitate the use of cardiopulmonary bypass (CPB) to safely resect intracardiac tumor thrombi. CPB provides circulatory and respiratory support, allowing the surgeon to open the right atrium (atriotomy) under controlled conditions.[4,24]

Hypothermic circulatory arrest may be employed in complex cases to reduce metabolic demands and provide a bloodless field, facilitating complete tumor removal. CPB reduces intraoperative blood loss, supports systemic circulation, and improves surgical visualization.[8]

Post-CPB management involves careful hemodynamic support and monitoring for coagulopathy, given the effects of extracorporeal circulation on platelet function and coagulation cascades.[2]

Management of suprahepatic and intracardiac extension

Tumors that involve the suprahepatic IVC and extend into the right atrium present formidable surgical challenges requiring combined thoracoabdominal approaches. A median sternotomy combined with laparotomy is often necessary to provide adequate exposure of the heart, IVC, and liver.[22]

Hypothermic circulatory arrest during CPB facilitates tumor excision under optimal conditions by minimizing blood flow and reducing tissue oxygen demand. This technique allows safe removal of tumor thrombi extending into cardiac chambers and reduces the risk of embolization.[17]

Following tumor removal, reconstruction of the IVC and atrial walls is performed, often requiring patch angioplasty or graft interposition to restore venous continuity. Hepatic mobilization may be necessary to access the suprahepatic IVC fully. Additionally, hepatic vein reconstruction may be required to maintain adequate liver drainage and function.[6]

These complex procedures demand careful perioperative planning, multidisciplinary expertise, and advanced surgical skills to balance oncologic radicality with preservation of vital functions.[7]

Postoperative care and complications

The postoperative management of patients undergoing surgical treatment for neoplastic obstruction of the IVC is complex and demands a multidisciplinary and highly vigilant approach. Given the extensive nature of the surgery, the involvement of major vascular structures, and the potential for significant hemodynamic alterations, meticulous postoperative care is essential to optimize outcomes and reduce morbidity and mortality.[20]

Hemodynamic monitoring: Close hemodynamic monitoring in an intensive care setting is mandatory immediately after surgery. Due to the manipulation, resection, and reconstruction of the IVC, patients are at high risk for fluctuations in venous return and cardiac output. Continuous monitoring of central venous pressure, arterial pressure, urine output, and cardiac function allows early detection of hemodynamic instability, fluid imbalances, or cardiac complications. Advanced monitoring techniques, including pulmonary artery catheterization or transesophageal echocardiography, may be indicated in select cases to guide fluid management and inotropic support.[11]

Anticoagulation and thromboprophylaxis: Preventing graft thrombosis is a critical component of postoperative care. Patients who undergo IVC resection and reconstruction are inherently at risk for venous thromboembolism due to endothelial injury, altered flow dynamics, and the hypercoagulable state often associated with malignancy. A carefully tailored anticoagulation regimen should be initiated early, balancing the risk of bleeding with thrombosis. Low molecular weight heparin or unfractionated heparin protocols are commonly employed initially, transitioning to oral anticoagulants as clinically appropriate. Regular assessment of coagulation parameters and vigilant monitoring for signs of bleeding or thrombosis is imperative.[14]

Surveillance imaging: Early postoperative imaging plays a vital role in identifying complications and assessing graft patency. Doppler ultrasound is frequently used for bedside evaluation of venous flow within the reconstructed IVC or grafts. CT or MRI may be indicated to evaluate suspected thrombosis, graft infection, or other complications. Scheduled imaging during follow-up enables timely intervention in cases of graft stenosis or occlusion.[23]

Renal function and hepatic congestion: Given the central role of the IVC in venous drainage from the kidneys and liver, postoperative renal function requires close surveillance. Venous congestion resulting from impaired outflow can precipitate acute kidney injury. Serial measurement of serum creatinine, urine output, and electrolytes is essential. Similarly, hepatic congestion secondary to impaired IVC drainage may lead to liver dysfunction, manifesting as elevated liver enzymes, coagulopathy, or ascites. Early recognition and management, including optimization of volume status and hemodynamics, are critical to prevent progression.[18]

Common postoperative complications:

- *Bleeding:* The extensive vascular dissection and anticoagulation increase the risk of postoperative hemorrhage, which may necessitate re-exploration.[2]
- *Graft thrombosis:* Thrombosis of venous grafts or patches can lead to acute venous congestion and limb swelling, requiring urgent anticoagulation or thrombectomy.[4]
- *Pulmonary embolism:* Embolization of thrombus fragments or deep vein thrombosis remains a significant risk despite prophylaxis. Vigilance for respiratory symptoms and prompt diagnostic workup is mandatory.[22]
- *Renal failure:* Both acute tubular necrosis secondary to hypoperfusion and venous congestion contribute to renal impairment postoperatively. Renal replacement therapy may be necessary in severe cases.[13]
- *Surgical site infections:* Given the complexity and length of the surgery, patients are at risk for wound infections, which can be compounded by prosthetic grafts and immunosuppression. Strict aseptic technique and early antibiotic therapy are essential.[9]

Outcomes and prognosis

The long-term outcomes following surgical management of neoplastic obstruction of the IVC are influenced by several critical factors, including tumor histology, the completeness of surgical resection, and the presence or absence of metastatic disease at the time of surgery.[19,20]

Tumor histology plays a pivotal role in prognosis. Primary leiomyosarcomas of the IVC, despite being the most common primary tumor in this location, generally carry a guarded prognosis. These tumors are characterized by aggressive local behavior, high rates of local recurrence, and a significant propensity for distant metastases, often to the lungs or liver.[10] The inherently infiltrative nature and biological aggressiveness of leiomyosarcomas limit long-term survival, with reported 5-year survival rates varying widely but generally remaining low. Nevertheless, selected patients who undergo complete surgical resection with negative margins followed by adjuvant therapies, such as radiotherapy or chemotherapy, may experience improved survival outcomes.[12] However, the role of systemic therapy remains adjunctive and is not yet standardized,[14] underscoring the importance of aggressive surgical management as the cornerstone of treatment.

In contrast, **secondary involvement of the IVC**, most notably by RCC, presents a different prognostic landscape. RCC with tumor thrombus extending into the IVC can be associated with relatively better postoperative outcomes, especially when the tumor thrombus is confined to the venous system without distant metastases. Surgical removal of the renal tumor combined with thrombectomy of the IVC thrombus can provide significant survival benefit and symptom relief. The extension level of the tumor thrombus, although a technical challenge, does not independently dictate prognosis if complete resection is achieved. Patients with nonmetastatic disease who undergo radical surgery can achieve 5-year survival rates that are markedly higher compared with patients with metastatic spread.[12]

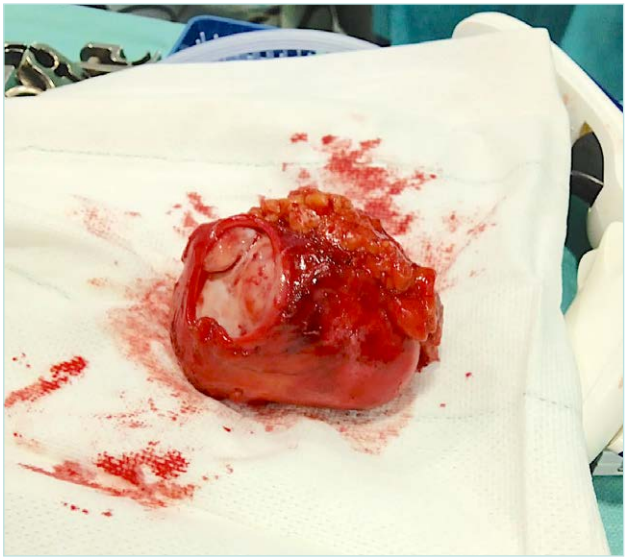

Figure 5. Inferior vena cava (IVC) leiomyosarcoma. Complete resection with free margins (posterior pathological anatomy).

Completeness of resection is arguably the most critical surgical factor influencing outcomes. Achieving an R0 resection, defined as complete tumor removal with microscopically negative margins, is strongly correlated with improved local control and overall survival.[15] Incomplete resections or positive margins predispose patients to rapid local recurrence, which is frequently associated with worsening symptoms and reduced survival. Given the

complexity of the IVC anatomy and tumor involvement, meticulous surgical technique, and thorough preoperative planning are essential to maximize the likelihood of achieving complete resection. The involvement of multidisciplinary teams including vascular, urologic, and cardiothoracic surgeons is often necessary for optimal oncologic clearance.[21]

The choice and success of **venous reconstruction techniques** after tumor resection also impact long-term patency and clinical outcomes. Reconstruction strategies must ensure durable venous return while minimizing complications such as thrombosis or graft infection. Use of autologous vein grafts or prosthetic materials like PTFE has shown favorable patency rates when performed under optimal conditions *(Figure 5)*. Postoperative anticoagulation protocols tailored to the patient's risk profile further contribute to graft longevity.[23]

Finally, **patient selection and perioperative management** are critical determinants of survival. Factors such as patient comorbidities, performance status, and absence of distant metastases influence both surgical candidacy and postoperative recovery.[8] Meticulous perioperative care, including vigilant hemodynamic monitoring, early detection and management of complications, and rehabilitation, enhances functional outcomes and survival.

Conclusions

Neoplastic obstruction of the IVC is a rare but complex surgical condition requiring a tailored and multidisciplinary approach. Mastery of tumor classification, detailed preoperative imaging, and advanced surgical techniques are essential to optimize patient outcomes. Innovations in reconstruction and extracorporeal support have expanded resectability and improved survival for selected patients. Vascular surgeons must continue leading the management of these challenging cases to deliver effective, evidence-based care.

CORRESPONDING AUTHOR

Dr Alejandro Rodríguez-Morata

Hospital Quirónsalud Málaga, Avenida Imperio Argentina, 1; 29004, Málaga, España

EMAIL: *vascular@rodriguezmorata.es*

Dr Alejandro Rodríguez-Morata received an honorarium from Servier for the writing of this article.

References

1. Herr HW, Schlossberg RE, Whitmore WF. Leiomyosarcoma of the inferior vena cava: a rare and difficult diagnosis. *J Urol*. 2005;173(4):1220-1221.
2. Neves RJ, Zincke H. Surgical treatment of renal cancer with vena cava extension. *Br J Urol*. 1987;59(5):390-395.
3. Langan EM, Ferreira M, Sundaram C, et al. Multidisciplinary management of inferior vena cava tumors: a systematic review. *J Surg Oncol*. 2021;124(4):583-593.
4. Tsukuda H, Shimizu T, Yasui N, et al. Vascular reconstruction using bovine pericardial patch for inferior vena cava leiomyosarcoma. *Ann Vasc Surg*. 2017;44:261.e7-261.e10.
5. Mussi C, Casali PG, Lo Vullo S, et al. Inferior vena cava leiomyosarcoma: clinical experience with 15 cases. *Ann Surg Oncol*. 2007;14(1):353-360.
6. Thorson CM, Turaga KK, Reith JD, et al. Surgical management of inferior vena cava leiomyosarcoma: a single-institution experience. *Ann Vasc Surg*. 2013;27(8):1083-1090.
7. Wachtel H, Neuwirth MG, Cai L, et al. Surgical outcomes of resection of inferior vena cava leiomyosarcoma. *Ann Vasc Surg*. 2019;61:176-185.
8. Samaan S, Al-Haddad M, Aoun H, et al. Surgical techniques for tumors involving the inferior vena cava: review and classification. *J Cardiothorac Surg*. 2017;12(1):61.
9. Mingoli A, Cavallaro A, Sapienza P, et al. Primary leiomyosarcoma of the inferior vena cava: results of surgical treatment and prognosis. *J Vasc Surg*. 1997;26(5):768-775.
10. National Comprehensive Cancer Network (NCCN). Soft tissue sarcoma guidelines, version 3.2023. [Internet] 2023. https://www.nccn.org
11. Kim SJ, Cho YP, Kim HY, et al. Role of transesophageal echocardiography in evaluation of tumor thrombus in renal cell carcinoma involving the inferior vena cava. *J Cardiothorac Vasc Anesth*. 2014;28(2):342-346.
12. Herr HW, Schlossberg RE, Whitmore WF. Surgical management of renal cell carcinoma with vena cava thrombus. *J Urol*. 2005;173(4):1220-1221.
13. Blute ML, Leibovich BC, Lohse CM, et al. The Mayo Clinic experience with surgical management, complications and outcome for patients with renal cell carcinoma and venous tumour thrombus. *BJU Int*. 2004;94(1):33-41.
14. Minami T, Chikamoto A, Arii S. Hepatocellular carcinoma with tumor thrombus in the inferior vena cava and right atrium. *J Hepatobiliary Pancreat Surg*. 2004;11(6):376-379.
15. Iannelli A, Scardina L, Pellegrino L, et al. Inferior vena cava leiomyosarcoma: surgical management and outcome. *Int J Surg Case Rep*. 2018;51:101-106.
16. Padua RA, Belli AM, Curry A, et al. MRI in the evaluation of inferior vena cava tumour thrombus in renal cell carcinoma. *Clin Radiol*. 1995;50(8):517-520.
17. Abutaka A, Morikawa M, Miyamoto K, et al. Surgical resection of a right atrial tumor thrombus arising from renal cell carcinoma using hypothermic circulatory arrest. *Ann Thorac Cardiovasc Surg*. 2011;17(5):469-472.
18. Groshar D, Gaitini D, Epelman M, et al. Role of color Doppler sonography and renal function tests in the evaluation of Budd-Chiari syndrome. *AJR Am J Roentgenol*. 1993;161(2):377-381.
19. Schelzig H, Thelen S, Rehders TC, et al. Surgical management of leiomyosarcoma of the inferior vena cava: results of a multicenter study. *Ann Surg*. 2020;271(3):567-575.
20. Lenz CJ, Krutman M, Tseng JF, et al. Multidisciplinary surgical treatment of tumors involving the inferior vena cava. *Ann Surg Oncol*. 2018;25(11):3401-3410.
21. Wang S, Zhao W, Ding J, et al. Oncovascular surgical approach for inferior vena cava tumors. *J Vasc Surg*. 2019;69(1):130-139.
22. Wong HH, Karam SD, Schmid RA, et al. Surgical management of primary and secondary tumors of the inferior vena cava. *Ann Thorac Surg*. 2015;100(1):331-337.
23. Yao X, Xiao Y, Ma K, et al. Venous reconstruction with autologous femoral vein graft after resection of inferior vena cava leiomyosarcoma. *Ann Vasc Surg*. 2019;60:364.e15-364.e18.
24. Goltz JP, Lehnert T, Kröger JR, et al. MR imaging of inferior vena cava leiomyosarcoma: report of 13 cases. *Eur Radiol*. 2010;20(9):2221-2228.

Phlebolymphology Vol 32 | No. 3 | 2025

Endovascular approach to IVC obstruction in oncological patients

Olivier Hartung,
MD, MSc, FEBVS

Department of Vascular Surgery, North University Hospital of Marseille, France

ABSTRACT

Cancer can cause inferior vena cava (IVC) obstruction. Usually, an oncologic resection is not possible and palliative treatment is needed. An endovascular procedure can be performed in most cases under local anesthesia plus sedation through a percutaneous approach. Stenting is a safe and the most effective technique and provides excellent technical and clinical success rates with immediate improvement of symptoms. Despite the short life expectancy of these patients, the technique allows them a far better quality of life. On the other hand, treatment of venous sequalae of carcinological treatment can give good long-term results.

Keywords

complications of cancer treatment | endovascular | inferior vena cava obstruction | oncology patients | palliative care | stent | venous sequalae

Phlebolymphology. 2025;32(3):100-108.

www.phlebolymphology.org

Introduction

Inferior vena cava (IVC) obstruction is mainly due to postthrombotic disease. It can also have other causes such as retroperitoneal fibrosis, IVC atresia or hypoplasia, and benign tumors. It can also represent a complication of cancers. In such cases, all kinds of histology can be found from renal cell carcinoma to sarcoma (IVC leiomyosarcoma but also other kinds of sarcoma), lymphoma, adenocarcinoma, and others.

All intra-abdominal primary or secondary locations of cancers can compress or even invade the IVC, even more so when these lesions are located in the retroperitoneum. But IVC obstruction can also be linked with the treatment of cancers, whether it was surgery (fibrosis, lymphocele, restenosis after grafting), radiotherapy (fibrosis) and chemotherapy (mainly after central lines insertion) or even with insertion of IVC filters. These iatrogenic complications can occur during the treatment but can also occur later.

Signs and symptoms

Signs and symptoms can present as an acute deep venous thrombosis; however, the present article focuses on chronic cases.

IVC obstruction can be asymptomatic, poorly symptomatic, or very symptomatic, depending on the severity, location, and extent of the obstruction, the speed of evolution of the tumor, and the development of collateral pathways.

The most common sign is lower-extremity edema that can be limited to the limbs but can also include thighs and even the pelvis. It can be associated with venous claudication, but all signs of the C class of CEAP (clinical-etiological-anatomical-pathophysiological classification) can be found.[1] All cases of lower-limb edema in oncologic patients should have venous imaging, as emphasized by O'Sullivan.[2] Wang found that 80% of women treated for uterine cervical cancer with lower-limb swelling had deep vein lesions and only 20% had lymphoedema.[3]

Signs are typically bilateral with or without the presence of abdominal collateral pathways *(Figure 1)*.

In case of bilateral renal veins or suprarenal IVC involvement, renal insufficiency can develop, mainly if there is a rapid progression of the disease. Indeed, low-expansion-rate tumors leave time for the development of collateral pathways through the azygos system. Hepatic insufficiency and even Budd-Chiari syndrome can also be present, mainly when suprahepatic veins are involved.

Of course, these lesions can be associated with acute deep venous thrombosis (and in this case, a dedicated treatment associating a clot removal strategy then stenting should be proposed). Cardiac function can also be affected. In all, most patients' quality of life is severely impaired.

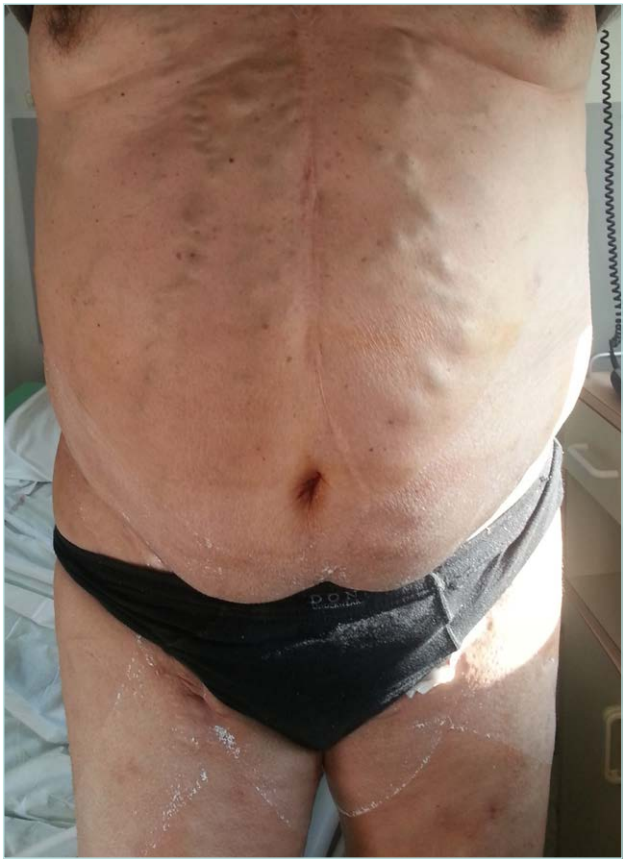

Figure 1. Subcutaneous collateral pathways due to inferior vena cava occlusion.

Imaging

The ideal imaging technique should provide identification of the primary tumor and depict the lesion, its extension in the IVC and in the surrounding structures, and differentiate blood thrombus from tumoral thrombus. It should also look for thromboembolic complications (pulmonary embolism [PE]) and secondary localizations.

Duplex scan

A duplex scan can find signs of central obstruction (decreased flow and absence of phasicity) on the common femoral and iliac veins. IVC exploration should look for obstruction or occlusion, an abdominal mass compressing or invading the IVC and collateral pathways.

CT scan and magnetic resonance imaging

Computed tomography (CT) venography is an easily available and the most commonly used imaging technique as it can be performed through peripheral venous access with injection of 150 mL of iodinated contrast agent with acquisition at 90 and 120 seconds[4] *(Figures 2 and 3)*. According to a recent Delphi consensus, it should be the first-line imaging technique.[5]

Magnetic resonance venography (MRV) is far less available but helps to differentiate neoplasic thrombus obstruction from clot[5]. It is performed with gadolinium enhancement, but time of flight (TOF) and phase techniques can evaluate the IVC without contrast.

Both techniques can depict the tumoral and vascular lesion(s). CT angiography and CT venography (or MRV) are mandatory in order to look for tumor vascularization, IVC status (obstruction or occlusion *[Figures 2 and 3]*) and collateral pathways. According to O'Sullivan, CT venography should be proposed in all cancer patients with lower-limb edema.[2]

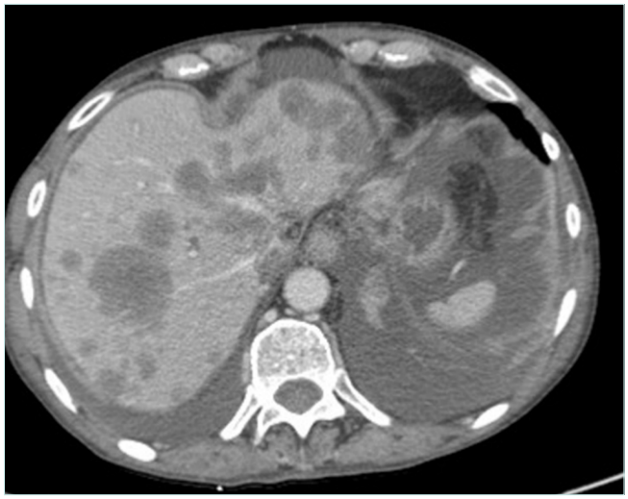

Figure 2. Computed tomography (CT) scan in a patient with metastatic liver and ascites with inferior vena cava occlusion.

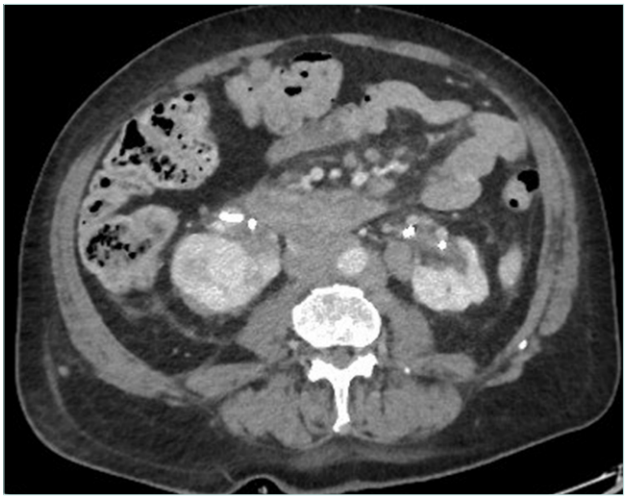

Figure 3. Complete occlusion of the infrarenal inferior vena cava due to lymph nodes from bladder cancer; presence of bilateral nephrostomy due to bilateral ureteral compression.

Positron emission tomography scan

Positron emission tomography (PET) can identify the tumor and look for extension (lymph node, metastasis). It should be performed before any procedure in order to evaluate the prognosis.

Transesophageal echocardiography

This technique can identify the superior extension of the thrombus, mainly in case of suspected supradiaphragmatic involvement.[5]

Iliocavography and intravascular ultrasound

These techniques have poor usefulness preoperatively but should be used during endovascular treatment. Intravascular ultrasound (IVUS) can be used after catheterization of the lesions (after recanalization in case of complete occlusion). They help to evaluate lesion extension, neck localization, diameter, and length, and they help localize more precisely important branches such as the renal and suprahepatic veins. Moreover, after stenting, these techniques can be used to confirm the result and that stents are well expanded with a circular shape.[6]

Indications

According to the cause of the obstruction, different treatments can be considered in order to abolish or reduce obstruction. Whereas obstruction can sometimes be improved via radio/chemotherapy by tumor volume reduction, surgery as well as endovascular techniques can be needed.

Surgery of IVC obstruction needs in-bloc tumor resection that can include IVC. In this case, IVC reconstruction can be performed, mainly in symptomatic patients with IVC syndrome. It is indicated as a carcinological procedure but not as a palliative treatment. This surgery is invasive but can provide good results as recently reported by Mac Arthur in 167 cases.[7]

Treatment of venous obstruction by endovascular techniques, ie, stenting, began in the early 90's but has expanded over the last 15 years with good long-term results. A recent review of the literature provides specific results of IVC stenting.[8] In oncologic patients, endovascular techniques do not make a pretense of treating the cancer as the lesions remain in place. It can have 2 main indications: palliative treatment of obstruction or curative treatment of complications of the oncologic treatment.

Preoperative workup

Besides imaging technique, endovascular procedures do not need a specific workup as in most cases they can be performed under local anesthesia plus sedation. Indeed, these approaches and catheterization are not painful unless perforation occurs. Thus, local anesthesia plus sedation provides prevention of complications when recanalization is needed.

Preoperatively, mostly if the patient suffers lower-limb edema, an intermittent compression device should be used in order to reduce the edema and facilitate the approach on the lower limbs.

Procedure

The procedure is performed in prone position to allow multiple access possibilities in an operating or radiologic room equipped with digital subtraction angiography, echography, and IVUS.

Sedation is performed by intravenous injection of 1 mg midazolam before percutaneous approach. Then another 1 mg midazolam and 5-10 µg sufentanil are given before beginning the painful part of the procedure, ie, angioplasty and stenting. If needed, 10 mg ketamine is added. Postoperative prevention of pain is achieved via intravenous injection of 100 mg ketoprofen associated with 1 g paracetamol before the end of the procedure.

In case of IVC lesion, different points of access can be used. Most of the time, it is advisable to have bilateral lower-limb access (through the common femoral vein when lesions are limited to the IVC with or without common iliac veins, but sometimes femoral vein or even popliteal vein access can be needed) and an internal jugular vein access, mainly on the right side. Access via any of these points should be performed percutaneously under echographic guidance.

Then 6F sheaths are inserted. Catheterization can be performed using different catheters and guidewire, usually starting with a 5F vertebral catheter and a 0.035-

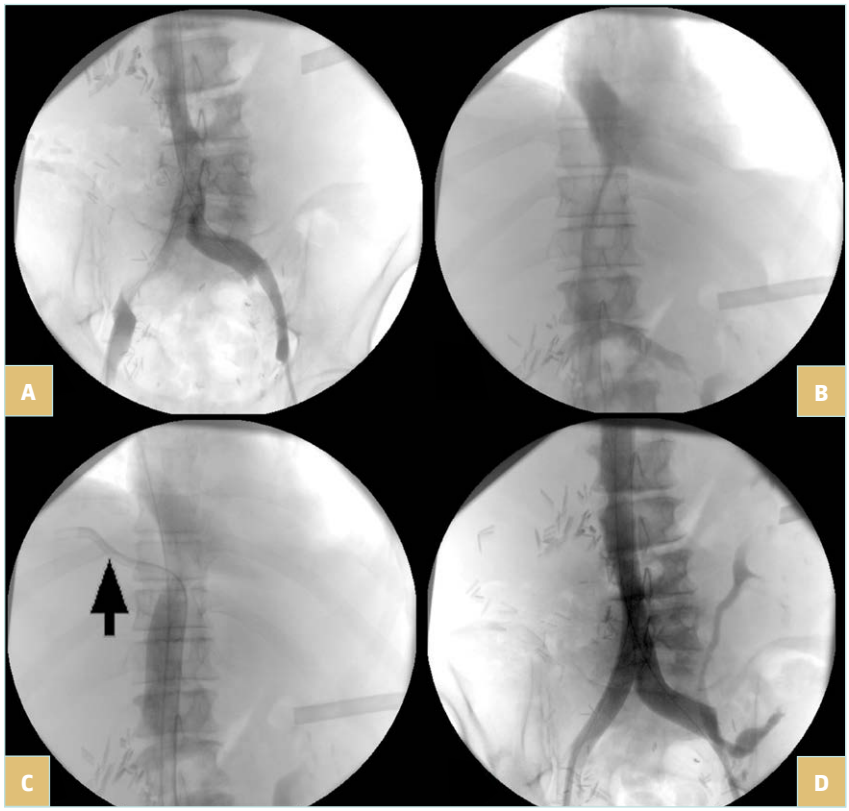

Figure 4. 76-year-old man suffering from right iliac and inferior vena cava (IVC) compression by iliac and infrarenal IVC nodes and liver metastasis from urothelial cancer with ascites (history of cystectomy and right uretero-nephrectomy): A) compression of the right iliac vein and infrarenal IVC; B) compression of the suprarenal IVC; C) after angioplasty and stenting of the suprarenal IVC; a guidewire (arrow) was positioned in the suprahepatic vein before stent deployment; D) after biiliocaval stenting according to the Eiffel tower technique.

Stents	Diameter (mm)	Length (mm)	Sheath size	Cells	Structure
Wallstent (Boston Scientific)	8-24	20-90	7-11F	CC	Braided Elgiloy
Gianturco Z stent (Cook)	15-35	5	14-16	OC	Stainless steel
Sinus XL Flex (Optimed)	14-24	40-160	10F	OC	Nitinol LC
Venovo (Bard)	10-20	40-160	8-10F	OC	Nitinol LC
Abre (Medtronic)	10-20	40-150	9F	OC	Nitinol LC
Viafort (Gore)	10-28	50-150	10-14Fr	H	Nitinol + ePTFE

CC, closed cells; H, hybrid; OC, open cells; ePTFE, expanded polytetrafluorethylene.

Table I. Bare stents available for treatment of inferior vena cava (IVC) obstructive lesions.

inch hydrophilic guidewire. In some cases, mainly when recanalization is needed, smaller devices or even chronic total occlusion (CTO) crossing devices can be used.

Once the lesion is crossed *(Figure 4A and 4B)*, super stiff or extra stiff guidewire and then a larger sheath are inserted (10-12 Fr depending on balloon and stent needs) and intravenous heparin should be given at the dose of 50 UI/kg. At this time, IVUS can be used to ensure a better evaluation of the lesion.[6] Pressure gradient measurements can be performed but have value only if positive (the absence of pressure gradient can be due to collateral pathways, even more in a patient that is lying down). Then predilatation is performed using high-pressure balloons of progressive diameter mainly in case of postradiotherapy lesions (in these cases, downsizing by 2 mm in diameter compared with a standard procedure is recommended to avoid venous rupture).

Stenting is then performed using self-expanding stents *(Figure 4C and 4D)*. Different stents were used in the literature. Until 2010, Wallstent (Boston Scientific, Marlborough, Massachussetts, USA) and Gianturco Z stent (Cook Medical, Bloomington, Indiana, USA) were the 2 most used stents. Since the development of nitinol self-expanding stents, many others have become available, though some of them are designed for femoro-iliac veins (Venovo, Abre). Stent sizing depends not only on the diameter of the adjacent nonpathologic IVC size—that can be evaluated by CT scan or IVUS—but also to the tumor. In most cases 18 to 22 mm in diameter should be used for the IVC as larger stents

can have difficulties with expanding; too-small stents can migrate and also limit flow. Few nitinol stents are available in 20-mm diameter or higher *(see Table I)*. These stents have a more precise deployment than the Wallstent without foreshortening. Regarding stent length, they should cover at least 15 to 20 mm beyond the obstructive lesion at both ends, covering an area that goes not only from healthy-to-healthy segment but also includes a safety margin to avoid restenosis by tumor progression.[9,10] When multiple stents are needed, an overlap of at least 20 mm should be used. In case of biiliocaval lesions, different stent configurations can be used: the Eiffel tower configuration while deploying the IVC stent first, then both iliac stents simultaneously *(Figure 4D)*, or a double-barrel technique (mostly if lesions are limited to the iliocaval confluence).

After postdilatation, to ensure proper expansion of the stent(s), completion IVUS is needed to check the quality of the treatment and the optimal deployment of the stent(s).[6]

At the end of the procedure, sheaths are removed with direct slight compression at the access site, then compressive bandages are applied. There are no indications for closing devices in veins.

Postoperative care

Intermittent compression devices are used to improve the flow and reduce the risk of thrombosis. Anticoagulation can be performed using unfractionated or low-molecular-weight heparin (LMWH) or even oral anticoagulation. Patients should be walking as soon as possible, most of the time when back in the ward. There is no need for a postoperative stay in the intensive care unit (ICU) after uncomplicated procedures.

Patient improvement is usually impressive. Edema persistence would call for an investigation into rethrombosis or persistent obstruction. In some cases, edema persistence can be caused by an associated lymphoedema.

Results

Palliative treatment

Sawada was the first in 1992 to publish on the use of stents to treat malignant IVC lesions.[11] Since then, many reports have described the results. Most series report excellent technical (100%) and good clinical success on lower-limb edema and good short-term patency rates *(Table II)*,[2,9,10,12-20] but results on ascites and anasarca are far less impressive, reporting few improvements.[9,14,16] A recent meta-analysis on 194 patients reported technical success, primary patency, and cumulative patency rates of respectively 100% (78%-100%), 78.5% (68%-94%), and 99% (87%-100%) with a median follow-up of 1 month (1-2.9) for malignant patients and did not differ from the whole population.[8] Stenting was shown to be more efficient than chemotherapy or radiotherapy[14]; moreover, symptom relief is immediate after stenting, whereas it takes weeks to improve with classic oncologic treatments. As this treatment is palliative, in most cases, survival is not long with a median length inferior to 6 months in all series but one *(Table II)*. Thus, the goal of this treatment is to improve, usually immediately, the quality of life of the patients until their death. According to Augustin[18], factors of death seem to be the absence of subsequent radiotherapy and/or chemotherapy, the length of stented vein, and the involvement of the intrahepatic segment.

A phase 2 trial (28 patients, 42% IVC) and then a phase 3 randomized controlled trial (RCT; 32 patients, stenting versus medical therapy, 43% IVC) were reported by Takeuchi to treat vena cava syndromes.[21] The phase 2 trial on 19 patients (enrolment stopped based on interim analysis) found technical success in 100%, clinical success in 67.9%, with 14.3% of adverse events. The RCT found a significant superiority of stenting regarding symptoms and the physical summary of an 8-item short-form health survey (SF-8) (for IVC only) without differences regarding adverse events and survival.

Regarding the type of stent, self-expanding stents are most often used. Different stents were used in the literature, from the Wallstent and the Z-stent to dedicated venous nitinol stent, for which deployment is very precise without significant shortening. Closed-cell stents have better radial force and chronic outward force resistance, properties that are of interest in that indication. Covered stents were proposed for the treatment of superior vena cava syndrome (SVCS), and according to Gwon[22] patency rates seem to be superior to those of bare stents. Moreover, in case of intraluminal development of the tumor, covered stents should reduce the risk of emboly of malignant material during the procedure and later tumor progression between the struts.[9,22] Bare stents, on the other hand, preserve flow from the side branches and should be less prone to rethrombosis in the IVC, mainly in patients with contraindication to anticoagulants.

IVC stenting of course is associated with a decrease in IVC pressure caudal to the obstruction[9,16,17]; however,

according to Kishi, IVC stenting is also associated with an increase in diuresis, with edema and ascites improvement an established correlation between these criteria.[16] This can induce hyponatremia. He also noted an improvement in serum creatinine, blood urea nitrogen, serum creatinine, lactate dehydrogenase, fibrinogen and platelets count. Cardiac monitoring should be carried out as the increase in caval return can provoke cardiac overload. According to Brountzos,[14] preoperative elevated total bilirubin is associated with poor improvement but should not contraindicate the procedure.

Prandoni showed that patients with cancer and venous thrombosis have higher risk of rethrombosis but also of bleeding complications during anticoagulant treatment.[23] Despite this, postprocedural anticoagulation is mandatory after stenting in these patients[24]: in a series of 30 nonthrombotic cases, primary patency rates at 12 months were 69.8% in patients with anticoagulants versus 33.3% in those without, and 3.3% of major bleeding occurred in the anticoagulated patients (self-limited retroperitoneal hematoma).

Placement of stents in front of the ostia of the renal veins did not lead to acute renal failure because these patients already have collateral pathways through the azygos system, as already described for nonmalignant lesions by O'Sullivan.[25]

In case of lesion of the suprahepatic IVC, it was proposed to use a bridging technique with stenting from the IVC to the SVC.[26-28]

Reported complications included stent shortening (mainly with the Wallstent), migration or dislocation, vein rupture, loss of patency, and restenosis; however, these are rare, certainly due to the short survival, thus few reinterventions are needed. Evaluation included a hybrid score mixing the imaging description of the lesions and clinical evaluation.

A special mention must be made of the Wu publication[29] that retrospectively compared stenting (38 patients) with stenting that was associated with a linear radioactive I^{125} seeds strand (19 patients) placed between the stent and the venous wall, even if they mixed different anatomic lesions (brachiocephalic vein–SVC in 24 patients and IVC–iliac and femoral veins in 33 patients). Patients receiving stents plus I^{125} had significantly better patency rates and a better Karnofsky Performance Status score at 1 and 6 months but no significant difference in survival even if it was higher in patients receiving I^{125} strands (155 vs 98 days). This technique provides continuous brachytherapy to surrounding tumor tissues thus decreasing the risk of complications due to stent compression by tumor growth or intraluminal development between the struts of the stent.

Author	N	% Chronic symptoms	Technical success	Clinical success (edema)	PP	aPP	SP	Survival (Days)
Furui[12]	23	100%	100%	95%	NS	NS	NS	Mean 87
Fletcher[13]	28	NS	100%	NS	NS	NS	100%	Mean 34
Brountzos[14]	50	NS	100%	86%	59%		100%	Mean 75
McGee[15]	5	20%	100%	80%	80%	80%	80%	Median 65
Kishi[16]	7		100%	100%	100%	NS	NS	Mean 94
O'Sullivan[2]	62	46%	100%	100%	75%	79%	79%	Mean 230
Devcic[9]	57	51%	100%	83%[A]	NS	NS	90%	Median 38
Kuetting[10]	19	NS	95%	79%	NS	NS	NS	NS
Epelboym[17]	17	NS	100%	58%	94%	NS	100%	Median 28
Augustin[18]	21	100%	100%	85,7%	93%	100%	100%	Median 81
Ozawa[19]	54	100%	100%	100%	NS	NS	96%	Median 27[B]
Aly[20]	37	43.3%	100%	78%	69%	NS	NS	Median 141
Hartung	7	100%	100%	100%	100%	100%	100%	Mean 118

[A]Better results with Wallstent. [B]Median survival, 69 days for patients with postoperative prophylactic anticoagulation vs 30 days in patients without.
aPP, assisted-primary patency; PP, primary patency; SP, secondary patency.

Table II. Results of stenting for obstructive lesion of the inferior vena cava (IVC) due to cancer.

Treatment of late complications after oncologic treatment

In these cases, venous obstruction is not due to tumoral compression but to sequelae of the treatment and can have different etiologies (*Table III*). The main issue is to diagnose the presence of venous obstruction as emphasized by O'Sullivan.[2]

In these patients, prognosis mainly relies on the oncologic status and can be excellent when the cancer is cured (*Figure 5*). Therefore, results of stenting in terms of clinical outcome and patency rates should be comparable to nonmalignant lesions.

Literature is very scarce on this specific topic, and reports focus mainly on complications of the iliac veins but some

cases are certainly included in series on venous stenting for active cancer. Brechtel reported 1 case of anastomotic stenosis after surgical bypass on the IVC during reconstruction for hepatic tumor that was successfully treated by stenting.[30] Murakami reported 1 case of retroperitoneal fibrosis after radiotherapy for uterine cervical cancer with good result after IVC stenting.[31] In our experience, we treated 7 patients with late complications of cancer treatment (2 postoperative retroperitoneal fibrosis, 3 had previous lymph node resection and subsequent radiotherapy, 1 radiotherapy, 1 anastomotic stenosis after surgical IVC reconstruction for leiomyosarcoma). All had successful stenting and secondary patency was 100% after a mean 81-month follow-up (26-212 months).

Table III. Causes of venous obstruction secondary to oncologic treatments.

Oncologic therapy	Complications
Surgery	Fibrosis Stenosis / thrombosis after venous reconstruction Lymphocele
Radiotherapy	Fibrosis Stenosis, thrombosis
Indwelling catheter	Stenosis, thrombosis
IVC filter	Stenosis, thrombosis Perforation
Chemotherapy	Thrombosis

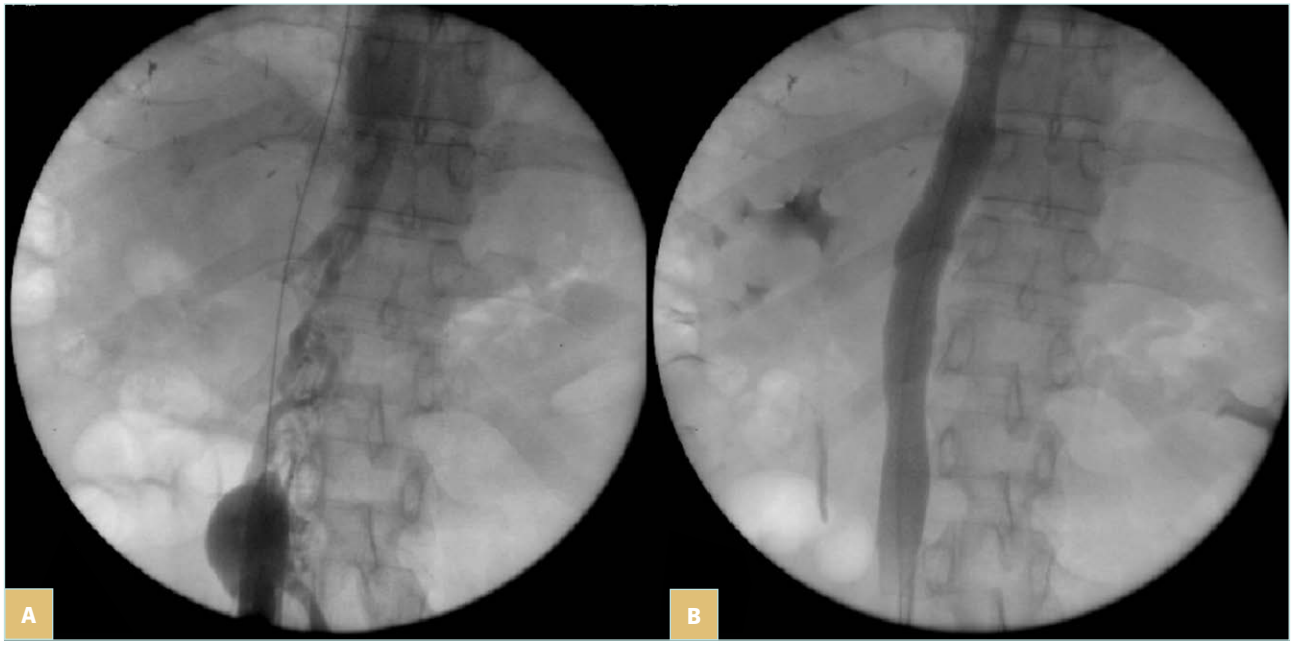

Figure 5. 44-year-old man suffering from symptomatic inferior vena cava (IVC) (lower-limb edema) occlusion due to retroperitoneal fibrosis occurring after liver tumor resection: A) IVC occlusion; B) result after recanalization and stenting with 2 Wallstents of 16 mm in diameter. Patient remained asymptomatic at 108 months of follow-up.

Conclusion

IVC obstruction due to cancer is very often out of the range of efficient carcinological treatment such as surgery. Most of the time, the treatment is palliative: endovascular procedures help these patients to have a better quality of life through a safe minimally invasive endovascular procedure with good technical and patency results and immediate clinical improvement, at least for edema; however, their life expectancy is short. Late complications of the carcinological treatment can occur in these patients and can also be treated by stenting.

CORRESPONDING AUTHOR

Olivier Hartung

Service de Chirurgie Vasculaire, CHU Nord, Chemin des Bourrelly, 13915 Marseille Cedex 20, France.

EMAIL: *olivier.hartung@ap-hm.fr*

Dr Olivier Hartung received an honorarium from Servier for the writing of this article.

References

1. Lurie F, Passman M, Meisner M, et al. The 2020 update of the CEAP classification system and reporting standards. *J Vasc Surg Venous Lymphat Disord*. 2020;8:342-352.
2. O'Sullivan GJ, Waldron D, Mannion E, Keane M, Donnellan PP. Thrombolysis and iliofemoral vein stent placement in cancer patients with lower extremity swelling attributed to lymphedema. *J Vasc Interv Radiol*. 2015;26:39-45.
3. Wang PL, Cheng YB, Kuerban G. The clinical characteristic differences between thrombosis-related edema and lymphedema following radio-therapy or chemoradiotherapy for patients with cervical cancer. *J Radiat Res*. 2012;53:125-129.
4. Chaudhary RK, Nepal P, Kumar S, et al. Imaging of inferior vena cava normal variants, anomalies and pathologies, Part 2: Acquired. *SA J Radiol*. 2023;27:2694.
5. Balaz P, Whitley A, Heinola I, Garguilo M, Vikatmaa P; OVS Collaborative Author Group. Surgical management of tumours invading the inferior vena cava: a Delphi consensus document. *Eur J Vasc Endovasc Surg*. 2025;70:389-399.
6. Gagne PJ, Tahara RW, Fastabend CP, et al. Venography versus intravascular ultrasound for diagnosing and treating iliofemoral vein obstruction. *J Vasc Surg Venous Lymphat Disord*. 2017;5:678-687.
7. MacArthur TA, Mendes BC, Colglazier JJ, et al. Early and late outcomes of patients treated with graft replacement of the inferior vena cava for malignant disease: a single center experience over three decades. *J Vasc Surg Venous Lymphat Disord*. 2025:102326.
8. Morris RL, Jackson N, Smith A, Black SA. A systematic review of the safety and efficacy of inferior vena cava stenting. *Eur J Vasc Endovasc Surg*. 2023;65:298-308.
9. Devcic Z, Techasith T, Banerjee A, Rosenberg JK, Sze DY. Technical and anatomic factors influencing the success of inferior vena caval stent placement for malignant obstruction. *J Vasc Interv Radiol*. 2016;27:1350-1360.
10. Kuetting D, Thomas D, Wilhelm K, Pieper CC, Schild HH, Meyer C. Endovascular management of malignant inferior vena cava syndromes. *Cardiovasc Intervent Radiol*. 2017;40:1873-1881.
11. Sawada S, Fujiwara Y, Koyama T, et al. Application of expandable metallic stents to the venous system. *Acta Radiol*. 1992;33:156-159.
12. Furui S, Sawada S, Kuramoto K, et al. Gianturco stent placement in malignant caval obstruction: analysis of factors for predicting the outcome. *Radiology*. 1995;195:147-152.
13. Fletcher WS, Lakin PC, Pommier RF, Wilmarth T. Results of treatment of inferior vena cava syndrome with expandable metallic stents. *Arch Surg*. 1998;133:935-938.
14. Brountzos EN, Binkert CA, Panagiotou IE, Petersen BD, Timmermans H, Lakin PC. Clinical outcome after intrahepatic venous stent placement for malignant inferior vena cava syndrome. *Cardiovasc Intervent Radiol*. 2004;27:129-136.
15. McGee H, Maudgil D, Tookman A, Kurowska A, Watkinson AF. A case series of inferior vena cava stenting for lower limb oedema in palliative care. *Palliat Med*. 2004;18:573-576.
16. Kishi K, Sonomura T, Fujimoto H, et al. Physiologic effect of stent therapy for inferior vena cava obstruction due to malignant liver tumor. *Cardiovasc Intervent Radiol*. 2006;29:75-83.
17. Epelboym Y, Stecker MS, Fan CM, Killoran TP, Rabkin DJ, Schenker MP. Treatment of malignant inferior vena cava obstruction with Gianturco-Rosch-Z stents: a single center 13-year experience. *Clin Imaging*. 2020;59:95-99.
18. Augustin AM, Lucius LJ, Thurner A, Kickuth R. Malignant obstruction of the inferior vena cava: clinical experience with the self-expanding Sinus-XL stent system. *Abdom Radiol*. 2022;47:3604-3614.
19. Ozawa M, Sone M, Sugawara S, et al. Necessity of prophylactic anticoagulation therapy following inferior vena cava stent placement in patients with cancer. *Interv Radiol*. 2023; 8:70-74.
20. Aly AK, Moussa AM, Chevallier O, et al. Iliocaval and iliofemoral venous stenting for obstruction secondary to tumor compression. *CVIR Endovasc*. 2024;7:33.
21. Takeuchi Y, Arai Y, Sone M, et al. Evaluation of stent placement for vena cava syndrome: phase II trial and phase III randomized controlled trial. *Support Care Cancer*. 2019;27:1081-1088.
22. Gwon DI, Ko GY, Kim JH, et al. Malignant superior vena cava syndrome: a comparative cohort study of treatment with covered stents versus uncovered stents. *Radiology*. 2013;266:979-987.
23. Prandoni P, Lensing AWA, Piccioli A, et al. Recurrent venous thromboembolism and bleeding complications during anticoagulant treatment in patients with cancer and venous thrombosis. *Blood*. 2002;100:3484-3488.
24. Drabkin MJ, Bajwa R, Perez-Johnston R, et al. Anticoagulation reduces iliocaval and iliofemoral stent thrombosis in patients with cancer stented for nonthrombotic venous obstruction. *J Vasc Surg Venous Lymphat Dis*. 2021;9:88-94.
25. O'Sullivan GJ, Lohan DA, Cronin CG, Delappe E, Gough NA. Stent implantation across the ostia of the renal veins does not necessarily cause renal impairment when treating inferior vena cava occlusion. *J Vasc Interv Radiol*. 2007;18:905-908.
26. Taylor JD, Lehmann ED, Belli AM, et al. Strategies for the management of SVC stent migration into the right atrium. *Cardiovasc Intervent Radiol*. 2007;30:1003-1009.
27. Okamoto D, Takeuchi Y, Arai Y, et al. Bridging stent placement through the superior vena cava to the inferior vena cava in a patient with malignant superior vena cava syndrome and an iodinated contrast material allergy. *Jpn J Radiol*. 2014;32:496-499.
28. Zander T, Caro VD, Maynar M, Rabellino M. Bridging stent placement in vena cava syndrome with tumor thrombotic extension in the right atrium. *Vasc Endovascular Surg*. 2021;55:69-72.
29. Wu B, Yin G, He X, et al. Endovascular treatment of cancer-associated venous obstruction: comparison of efficacy between stent alone and stent combined with linear radioactive seeds strand. *Vasc Endovasc Surg*. 2020;54:565-572.
30. Brechtel K, TepeG, Heller S, et al. Endovascular treatment of venous graft stenosis in the inferior vena cava and the left hepatic vein after complex liver tumor resection. *J Vasc Interv Radiol*. 2009;20:264-269.
31. Murakamia N, Araib Y, Takagawaa Y, et al. Inferior vena cava syndrome caused by retroperitoneal fibrosis after pelvic irradiation: a case report. *Gynecol Oncol Rep*. 2019;27:19-21.

Phlebolymphology Vol 32 | No. 3 | 2025

Endovascular approach to neoplastic superior vena cava syndrome: a comprehensive review of stenting outcomes and advancements in management strategies

Syed Mohammad Habib, MBBS
College of Medicine, Sulaiman Al-Rajhi University, Al-Bukayriyah, Qassim, Saudi Arabia

Reem Ali AL-Hussain, MBBS
College of Medicine, Sulaiman Al-Rajhi University, Al-Bukayriyah, Qassim, Saudi Arabia

Abdulmajeed Hassan Alsamsami, MBBS
College of Medicine, Taif University, Taif, Saudi Arabia

Almamoon I. Justaniah, MD, FSIR
Interventional Radiology Section, King Faisal Specialist Hospital & Research Center – Jeddah, Saudi Arabia

ABSTRACT

Superior vena cava syndrome (SVCS) resulting from malignant obstruction presents significant clinical challenges requiring prompt intervention. Endovascular stenting has emerged as the primary therapeutic approach for neoplastic SVCS, demonstrating superior efficacy compared with conventional treatments. This comprehensive review synthesizes current evidence on endovascular management, analyzing technical approaches and clinical outcomes from recent systematic reviews and meta-analyses encompassing over 2249 patients. Endovascular stenting demonstrates consistently high technical success rates of 96.8% and clinical success rates of 92.8% across all-cause SVCS. Primary patency rates reach 81.5% at 1 year, declining to 63.2% at 12 to 24 months, whereas secondary patency remains robust at 76.6% beyond 24 months following salvage procedures. Complication rates remain acceptably low at 5.8%, with reintervention required in 9.1% of cases. Malignant etiology demonstrates superior primary patency compared with benign causes, with symptom relief typically achieved within 24 to 72 hours compared with weeks required for conventional chemotherapy or radiotherapy. Endovascular stenting represents the optimal first-line intervention for neoplastic SVCS, providing rapid, safe, and durable palliation. Whereas primary patency declines over time, excellent secondary patency rates support structured surveillance and reintervention protocols. Future research priorities include standardizing outcome definitions, optimizing anticoagulation strategies, and developing evidence-based surveillance protocols for long-term management of this challenging clinical condition.

Keywords

interventional radiology | superior vena cava syndrome | SVC syndrome | venous obstruction | venous recanalization | venous stenting

Phlebolymphology. 2025;32(3):109-120.

www.phlebolymphology.org

Introduction

Superior vena cava syndrome (SVCS) manifests as a result of partial or complete, intrinsic or extrinsic obstruction/compression of the superior vena cava (SVC) and/or its tributaries.[1,2] The spectrum of clinical manifestations in SVCS varies depending on the underlying etiology and the speed of onset, as well as the development of venous collaterals over time.[3] This progression can range from minor symptoms, such as distension of neck veins, cough, and headache, to more serious manifestations, including cerebral edema, laryngeal edema, acute respiratory compromise, and mortality. Other symptoms include orthopnea, dizziness, swelling and fullness in the head and neck, and blurring of vision, which is more severe in the morning, especially after prolonged periods of recumbency during the night.[4] Additional symptoms may be observed depending on the underlying cause, such as hemoptysis, dysphagia, hoarseness, lethargy, fever, weight loss, night sweats, palpable lymph nodes, in cases of an underlying malignancy.[4,5] Symptoms exacerbate in supine positions, and in worst-case scenarios, the patient may be completely unable to lie flat or bend forward.

Initially, SVCS was considered a medical emergency, but a review of recent data now indicates that SVCS follows a relatively benign course and improves without any active treatment.[6,7] No definitive guidelines have been established yet for the approach to SVCS and its management. Hence, it has not yet been well-defined which patients require immediate intervention and which ones require little specific treatment, although it would greatly depend on the underlying etiology.

Specific recommendations are lacking. However, a general recommendation has been made by the National Comprehensive Cancer Network and American College of Chest Physicians supporting the consideration of stent placement (SP) and/or radiotherapy (RT) for cases of superior vena cava obstruction as a result of lung cancer. The definition of management has broadened with time, now encompassing a wide array of treatment options such as RT, chemotherapy, thrombolytics, and SVC SP, which will be discussed further as part of this review for different etiologies, specifically malignant ones in a comprehensive manner.

Etiology and incidence

Literature has reported the global incidence of SVCS to range from 1 in 650 to 1 in 3100 individuals.[8] A wide spectrum of etiological factors has been found responsible for the development of SVCS. The first ever case of SVCS was discovered in 1757, in a patient with a syphilitic aortic aneurysm. Schecter reviewed 274 well-documented cases as part of a review published in 1954, where about 40% of the cases were identified as a result of tuberculous mediastinitis or syphilitic aneurysm.[8,9] The importance of this study was that it revealed that there were other possible etiologies of SVCS apart from syphilitic aneurysms. It is estimated that the most common etiology is malignant tumors, accounting for 60% of the cases, whereas iatrogenic causes from thrombosis or stenosis caused by central lines or medical devices account for 30% to 40% of the total cases.[10] The incidence of SVCS has been observed to rise continuously due to the increase in use of catheters, pacemakers, parenteral feeding lines, central venous lines, and defibrillators.[3,11] Rice et al found that 28% of all SVCS are associated with placements of intravascular catheters or devices.[3,4] Whereas complications arising from these devices contribute to a significant proportion of SVCS cases, Chee et al observed that it is a rare complication affecting only about 0.1% to 3.3% of all pacemaker patients.[11] Major thrombophilia and Behcet disease are also other common benign causes of spontaneous SVCS. In older adults, malignancy remains the most common cause of SVCS, with lung cancers being the most common, followed by lymphoma.[3,4,12-15] Around 75% of all cases of SVCS were observed to be a result of lung cancer, and right-sided lung cancer was seen to be more prone to causing superior vena cava obstruction in contrast to the left side.[14,16,17] Small cell lung cancer (SCLC) is the most common histologic type of lung cancer related to SVCS.[18] Sculier et al reported that among 643 patients with SCLC, SVCS was present in 8.6% before the treatment was commenced.[19] In a Cochrane review by Rowell and Gleeson, SVCS was seen present in 10% of cases of SCLC and 1.7% of cases of non-SCLC (NSCLC) at the time of diagnosis.[20] Fifteen percent of cases of SVCS are a result of lymphoma.[21] Perez-Soler et al conducted a study, reporting that 36 of 915 patients with non-Hodgkin lymphoma presented with SVCS, and the histologic types associated were a diffuse large cell in 23 patients, lymphoblastic in 12, and follicular large cell in 1 patient.[21] Diffuse large-cell lymphoma and lymphoblastic lymphoma account for the majority of lymphoma-associated SVCS cases. On the contrary, Hodgkin lymphoma rarely causes SVCS.[22] Cancers due to secondary metastases account for around 5% of cases of SVCS and have been observed to have a poorer prognosis.[22] Patients with malignant SVCS have a generally poor prognosis with a median survival period of 101 days from the time of occurrence.[23] However, the survival is not linked to the presence or absence of SVCS but to the tumor stage and subtype.[24] Other notable causes of SVCS include thymomas, thyroid carcinoma, esophageal cancer, germ cell tumors, and breast cancer.[14] SVCS was found in 1 of every 10 cases of breast cancer. More rarely, SVCS can manifest in patients with pleural mesothelioma, thymic carcinoma, or other primary mediastinal germ cell tumors and intrathoracic sarcomas *(Figure 1)*.

Clinical assessment and diagnostic workup

Neoplastic SVCS commonly manifests as progressive shortness of breath, puffiness of the face and neck, venous distention, accompanied by cough and arm edema. In advanced cases, patients may exhibit stridor, laryngeal edema, or neurological manifestations secondary to cerebral venous hypertension.[1,25-27] Variations in clinical presentation are determined by severity and chronicity of the obstruction, in addition to the presence and efficiency of collateral venous routes.[26,28] Structures and tools, such as the Kishi symptoms score, facilitate assessment of severity and inform treatment decisions, with urgent stenting usually recommended when the scores reach or exceed 4.[8,29] Early supportive measures focus on head elevation, oxygen supplementation, and airway protection when needed; the routine use of empiric corticosteroids or diuretics is no longer advised.[1,25] Before intervention, patients should undergo laboratory testing including blood count, coagulation studies, renal function, particularly in patients needing contrast or anticoagulation.[29]

As the first-line modality, enhanced computed tomography (CT) venography is the preferred initial imaging approach, for delineating the obstruction, detecting tumor involvement, collateral circulation, and planning vascular access; for patients with contraindications to iodinated contrast, magnetic resonance (MR) venography is employed, but digital subtraction venography continues to be the gold standard, confirming anatomy and pressure gradients just before stenting.[29] In stable patients, histopathologic confirmation should be obtained before oncology therapy, whereas urgent endovascular stenting may be performed to relieve symptoms prior to tissue sampling.[1,25,29]

Formulating a multidisciplinary management plan is crucial. One should be aware of any planned post stenting radiation, chemotherapy, or surgical debulking, as this may alter the tumor size and the degree of venous compression, which would affect the stent sizing and stability.

The endovascular approach to SVCS

Introductions

Stent implantation remains the primary acute therapeutic option for tumor-related SVCS because they bring fast relief in about 24 to 72 hours. In contrast, chemotherapy or radiation usually need a few weeks to provide benefit.[30,31] More cases of benign conditions, for example catheter-related thrombosis and fibrosing mediastinitis, have been documented due to broader use of vascular devices.[30]

Preprocedural evaluation

Evaluation generally involves the use of contrast-enhanced CT, whereas MR imaging may be employed in selected cases; evaluation aims to assess the degree of obstructions, delineate collateral circulations, detect associated thrombosis, and measure the veins before placing the stent.[31,32] Performing venography during the intervention enables real-time confirmation of the anatomical structures and lesion properties.[31,32]

Access strategy

The preferred method is dual access, usually femoral and internal jugular veins, which helps in crossing chronic total venous occlusions. Although upper extremity routes may also be used, the subclavian route is less often selected because of the higher risk of pneumothorax.[32]

Lesion's crossing

To pass the lesion, a hydrophilic wire and an angled catheter can be used, using both antegrade and retrograde attempts when needed.[32] Long sheaths and guide catheters can be used to provide stability, pushability, and stiffness to optimize the lesion's crossing. Imaging in multiple projections with correlation to the preprocedural CT/MR may increase the success and minimize complications related to extravascular passage of the wire/catheter. If a blood clot is present, therapeutic approaches can include using a catheter or mechanical clot removal.[32] Whereas anticoagulants are standard during stent insertion, long-term therapy is a matter of debate. Evidence suggests that every case must be managed individually, notably when benign and malignant present differently.

Stenting

Nowadays, dedicated venous stents are available in the market. They have different lengths, sizes, and sometimes, characteristics. The benefits of these stents are flexibility and conformity to the venous course, high radial force to maintain the vein open against external compression or recoil, large diameter and long shaft to improve flow and prevent migration. Predilation is advised to enable smooth stent positioning and improve stent expansion. In general, stenting over a stiff wire is advised. It is prudent to stent from flow-to-flow and position the tumor in the middle of the stent. Post stenting angioplasty is indicated to optimize stent expansion. In addition, intravascular ultrasound can be used to assess the stent position, patency, and proper expansion. In cases of bilateral occlusions, Y-shaped (kissing) stents can be employed. Covered stents offer potential protection from tumor ingrowth; but on the other hand, they may carry a risk of migration especially when they

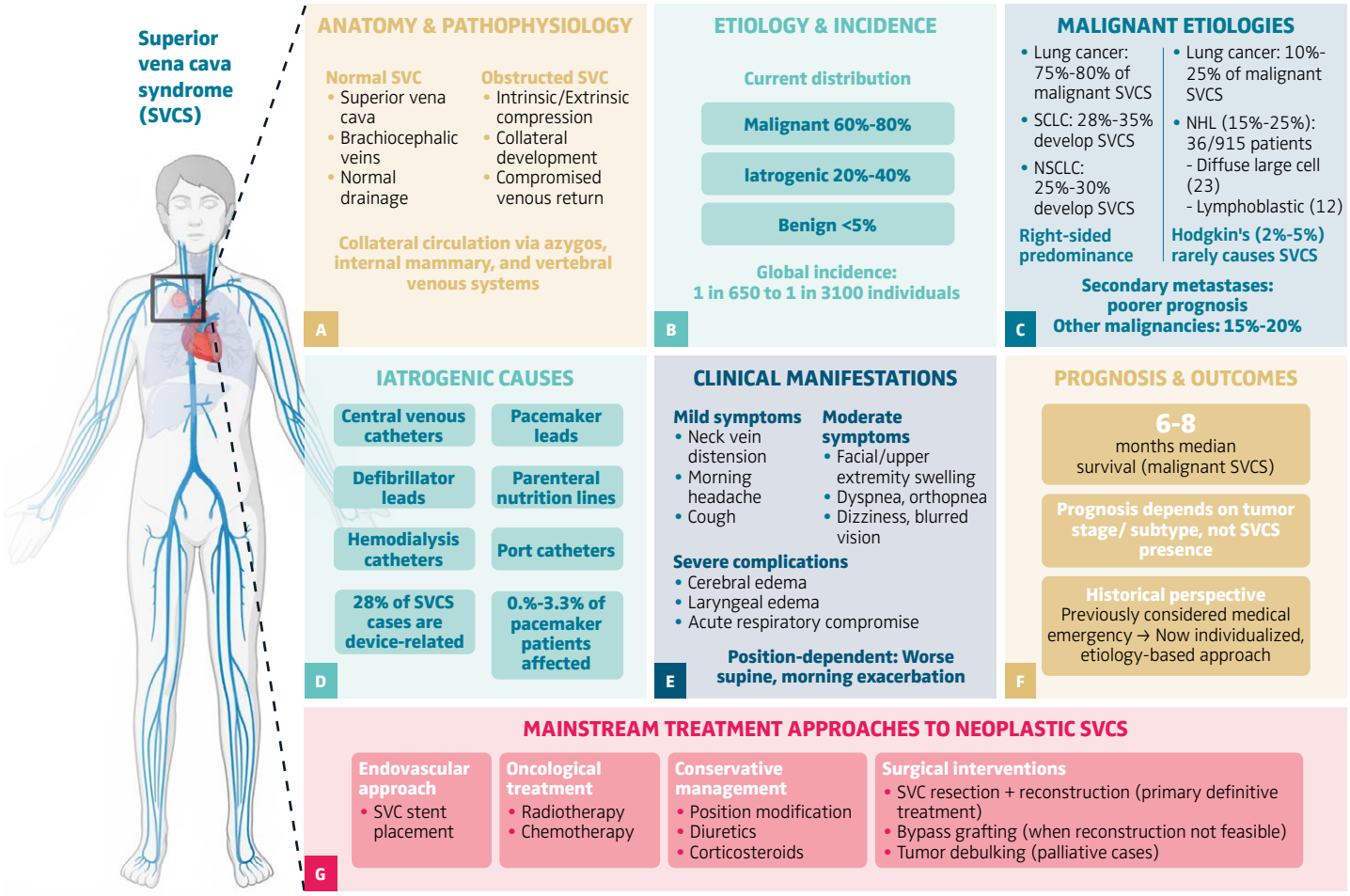

Figure 1. Malignant superior vena cava syndrome: a comprehensive overview.

The figure illustrates the comprehensive clinical spectrum of superior vena cava syndrome (SVCS). (A) Anatomy and pathophysiology: Normal SVC anatomy demonstrates venous drainage through brachiocephalic veins with collateral circulation via azygos, internal mammary, and vertebral venous systems, and obstructed SVC shows intrinsic/extrinsic compression mechanisms and compromised venous return. (B) Etiology and incidence: Global epidemiological patterns reveal malignant causes account for 60%-80% of cases, iatrogenic causes 20%-40%, and benign etiologies <5%, with global incidence ranging from 1 in 650 to 1 in 3100 individuals. (C) Malignant etiologies: Lung cancer represents 75%-80% of malignant SVCS cases, with SCLC accounting for 28%-35% of malignant SVCS (develops SVCS in ~10% of SCLC patients) and NSCLC representing 25%-30% of malignant SVCS (develops SVCS in ~2% of NSCLC patients). Lymphomas account for 10%-25% of malignant SVCS, including non-Hodgkin lymphoma (15%-25% of malignant SVCS cases) with diffuse large B-cell lymphoma being most common, while Hodgkin lymphoma rarely causes SVCS (2%-5% of malignant SVCS cases). Other malignancies represent 15%-20% of malignant SVCS, including secondary metastases from breast, testicular, and thyroid cancers, with metastatic disease conferring poor prognosis. (D) Iatrogenic causes: Iatrogenic causes encompass various indwelling devices (central venous catheters, defibrillator leads, hemodialysis catheters, pacemaker leads, parenteral nutrition lines, port catheters), representing 20%-40% of all SVCS cases, with pacemaker-associated complications occurring in 0.1%-3.3% of patients with these devices. (E) Clinical manifestations: Clinical manifestations range from mild symptoms (neck vein distension, morning headache, cough) to moderate (facial/upper extremity swelling, dyspnea, orthopnea, dizziness, blurred vision) to severe complications (cerebral edema, laryngeal edema, acute respiratory compromise), characteristically worse in supine position with morning exacerbation. (F) Prognosis and outcomes: Prognosis shows median survival of 6-8 months for malignant SVCS with outcomes dependent on tumor stage/subtype rather than SVCS presence itself. Historical perspective shows transition from infectious etiology to malignancy-predominant pattern, with modern individualized, etiology-based treatment approaches replacing previous emergency management protocols. (G) Mainstream treatment approaches: Treatment strategies include endovascular approaches (SVC stent placement), oncological treatment (radiotherapy, chemotherapy), conservative management (position modification, diuretics, corticosteroids), and surgical interventions (SVC resection + reconstruction for primary malignant involvement, bypass reconstruction when feasible, tumor debulking for palliative cases).

Modified with permission from original figure created in BioRender. Habib, SM. (2025) https://app.biorender.com/profile/template/details/t-68af2458e9155023e110f842-superior-vena-cava-syndrome-a-comprehensive-clinical-overvie. Figure link: https://app.biorender.com/profile/template/details/t-68af2458e9155023e110f842-superior-vena-cava-syndrome-a-comprehensive-clinical-overvie

NHL, non-Hodgkin lymphoma; NSCLC, non-small cell lung cancer; SCLC, small cell lung cancer; SVC, superior vena cava; SVCS, superior vena cava syndrome.

are undersized, and they may interfere with the collateral circulation.

Outcomes, patency, recurrence, and complication

Endovascular stenting for SVCS demonstrates high efficiency, with reported technical success and fast symptomatic relief for most patients.[30-33] The primary patency rate is usually 85% to 90% in the first year, although it may fall over time, whereas the secondary patency after subsequent interventions remains high at 72% to 89%.[30] Serious complications are rare, and overall complication rates remain low at 6% to 9%, including stent migration or cardiac complications.[30,32,34] In cases of malignant SVCS, the survival mainly reflects tumor rather than the stent; however, stents offer rapid, lasting relief of symptoms and are more effective than medical or RT alone when immediate palliation is required.[31,32,35]

Comparative analysis of endovascular and other approaches

For the patient with malignant SVCS, percutaneous stent placement has emerged as the primary treatment, providing rapid relief and demonstrating excellent efficacy and safety. According to a meta-analysis of 2249 patients, the technical and clinical success was 96.8% and 92.8%, respectively. The patency rate was 90% at 1 year, and the reintervention was 9.1%, with a low complication rate of 5.8%.[33] Both retrospective and prospective studies confirm these outcomes. Over a 17-year period (2006-2023), a single-center cohort study of 42 patients achieved 97.6% technical success and 91% to 93% 12-month patency, with low procedure-related mortality.[36] In an additional series of 32 patients (2015-2019), the technical success was 100% and symptoms fully improved within 7 days, with only minimal complications.[37] Between 1993 and 2008, a multicenter cohort (208 stents) showed prompt clinical improvement in more than 80% of patients, with excellent long-term outcomes, supporting stenting as first-line palliative intervention in malignant SVCS.[38] Chemotherapy and radiation therapy provide delayed symptom relief (3 to 4 weeks), and have 46% to 80% effectiveness, with a 10% to 50% recurrence rate.[7] Surgical bypass is reserved for last due to its invasiveness and morbidity.

Clinical outcomes following the endovascular intervention: pooled results of meta-analyses

Technical and clinical success rates

Endovascular stenting has demonstrated consistently high success rates across multiple large-scale systematic reviews and meta-analyses. The most comprehensive analysis encompassing 54 studies with 2249 patients over 27 years (1993-2020) reported pooled technical success rates of 96.8% (95% CI, 96.0%-97.5%) and clinical success rates of 92.8% (95% CI, 91.7%-93.8%) for all-cause SVCS.[1] When analyzing malignant SVCS specifically, these rates remained similarly impressive, with 2015 patients demonstrating the reliability of endovascular intervention as a first-line therapeutic approach.[1]

Systematic reviews focusing on bronchogenic carcinoma-induced SVCS reported that stent insertion provided symptomatic relief in 95% of cases, significantly higher than conventional treatments such as chemotherapy and RT alone.[20,26] This finding was particularly relevant for malignant etiologies, where rapid symptom resolution is often clinically imperative.[20,26] Analysis specifically examining Wallstent deployment in 701 individuals with 930 stents demonstrated complete symptomatic resolution within 48 hours of intervention, with a 30-day morbidity rate of 8% and mortality rate of 3%.[39] Notably, female sex was associated with higher 30-day morbidity (P<0.03), suggesting potential sex-specific considerations in patient selection and perioperative management.[39]

Patency outcomes and long-term durability

Patency rates represent a critical metric for evaluating the durability of endovascular interventions in SVCS management. A comprehensive meta-analysis of 39 studies involving 1539 patients revealed primary patency rates of 81.5% (95% CI, 74.5%-86.9%) up to 1-year post procedure.[39] However, primary patency demonstrated temporal decline, falling to 63.2% (95% CI, 51.9%-73.1%) at 12 to 24 months post intervention.[40]

The distinction between primary, primary-assisted, and secondary patency becomes clinically relevant when considering long-term outcomes. At ≥24 months, primary-

assisted patency reached 72.7% (95% CI, 49.1%-88.0%), whereas secondary patency achieved 76.6% (95% CI, 51.1%-91.1%).[40] These findings suggest that whereas primary patency may decline over time, interventional salvage procedures can successfully maintain vessel patency in the majority of cases.[40] Pooled patency remained above 90% during the first year, corroborating the findings regarding early durability.[1] The Wallstent-specific analysis demonstrated long-term patency rates of 92% following successful recanalization procedures, emphasizing the salvageability of failed stents.[39]

Malignant versus benign etiology outcomes

The underlying etiology of SVCS significantly influences clinical outcomes and patency rates. Primary patency was significantly higher in patients with malignant stenosis compared with benign stenosis at both 1 to 3 months and 12 to 24 months post intervention.[40] This finding likely reflects the different pathophysiological mechanisms underlying malignant versus benign obstruction, with malignant compression potentially responding more favorably to stent deployment.[40]

Differential patency outcomes based on etiology showed cumulative median patency for nonmalignant etiology reaching 550 days (interquartile range [IQR]: 14-1080) compared with 120 days (IQR: 0-925) for malignant cases ($P<0.03$).[27] This apparent contradiction with other findings may reflect different patient populations, stent types, or measurement methodologies, highlighting the complexity of comparing outcomes across heterogeneous study populations.[39] Specific analysis of benign SVCS outcomes reported identical technical and clinical success rates of 88.8% (95% CI, 83.0%-93.1%) for benign cases, which, although lower than malignant cases, still represent acceptable therapeutic outcomes.[1]

Complications and reintervention rates

Complication profiles vary across studies but generally demonstrate the safety of endovascular intervention. Average complication rates of 5.78% (standard deviation [SD]=9.32) across all studies were reported, with reintervention rates of 9.11% (SD=11.19).[25] The relatively low standard deviations suggest consistent safety profiles across different patient populations and institutional experiences.[1]

Morbidity following stent insertion was greatest when thrombolytics were administered, suggesting careful consideration of adjunctive therapies.[20,26] SVCS recurrence rates of 11% following stent insertion were reported, though recanalization was possible in the majority of cases, resulting in long-term patency rates of 92%.[20,26] The pediatric population demonstrated higher morbidity (30%) and mortality (18%) rates, with acute complications occurring in 55% of cases.[41] However, multimodal anticoagulation therapy improved outcomes by >50% ($P=0.004$), particularly in cases with concomitant thrombosis (present in 36% of pediatric cases).[42]

Prognostic factors and patient selection

Several studies identified specific factors associated with improved outcomes. Infant age ($P=0.04$), lack of collateral circulation ($P=0.007$), and presence of acute complications ($P=0.005$) possessed prognostic utility for outcome prediction in pediatric populations.[42] The presence of collateral circulation appeared particularly important, suggesting that patients with well-developed collateral networks may have different risk-benefit profiles for endovascular intervention.[42] The analysis of hepatocellular carcinoma risk in Budd-Chiari syndrome, though not directly related to SVCS stenting outcomes, provides insight into long-term complications in venous obstruction syndromes.[43] Their systematic review demonstrated significant geographic variation in complication rates, emphasizing the importance of population-specific outcome data.[43]

Comparative effectiveness with alternative therapies

When compared with conventional therapies, endovascular stenting demonstrates superior efficacy profiles. In SCLC, chemotherapy and/or RT relieved SVCS in 77% of cases, with 17% experiencing recurrence.[20,26] In NSCLC, conventional therapy achieved relief in 60% of cases, with 19% recurrence rates.[20,26] These figures compare unfavorably with the 95% relief rate achieved with stent insertion and the superior long-term patency rates.[20,26] The rapid symptom resolution achieved with endovascular intervention, often within 48 hours, represents a significant clinical advantage over conventional therapies, particularly in patients with severe symptoms requiring urgent decompression.[39]

Quality of evidence and study limitations

The grade analysis determined that the certainty of evidence for all outcomes was very low, reflecting the predominance of retrospective observational studies in this field.[40] This limitation underscores the need for higher-quality prospective studies and randomized controlled trials to establish definitive evidence-based guidelines for SVCS management.[40]

The heterogeneity observed across studies, particularly in patency definitions and measurement time points, complicates direct comparison of outcomes. Future research should focus on standardizing outcome definitions and establishing consensus guidelines for surveillance and reintervention protocols.

Table I provides an overview of major retrospective cohort studies evaluating endovascular treatment of superior vena cava syndrome.[44-53] It provides a comprehensive overview of the causes of SVCS, technical and clinical success rates of stenting, preoperative assessment methods, stent types, procedural details, follow-up protocols, and management of the underlying malignancy. It highlights the diverse spectra of patient populations, procedural strategies, and follow-up approaches, reflecting the evolving role of endovascular stenting in SVCS management.

Table I. Summary of clinical studies on endovascular stenting for superior vena cava syndrome (SVCS).

Study URL	Reference	Study design	No. of patients	Mean age (y)	Cause of SVCS	Condition	Vessels involved	Tech success rate (%)	Clinical success rate (%)	Preoperative assessment modality	Type of stent used	Procedure details	Follow-up protocol	Malignancy treatment
https://publishing.rcseng.ac.uk/doi/epdf/10.1308/rcsann.2020.7127	Irace et al,[44] 2021	Retrospective cohort	42	72±3	Nonresectable lung tumors with SVC invasion or compression (34), (81%) Non-Hodgkin lymphoma (5), (12%) Metastatic lymphadenopathy (3), (7%): 2 from breast cancer, 1 from gastric cancer	SCVS	Stanford and Doty classification: Type II (10), (24%) Type III (17), (40%) Type IV (15), (36%)	100%	NR	Preoperative work-up evaluation consisted of whole-body contrast-enhanced CT scan or MR angiography	Memotherm; Wallstent	The procedures were performed in an operating room using a mobile C-arm (GE OEC9800 Plus) under local anesthesia and conscious sedation. Patients received a 100 IU/kg heparin bolus before intervention. Access was most often via the ipsilateral jugular vein, with a 0.035-inch guidewire (Terumo) supported by a catheter to cross the stenosis; thrombolysis with urokinase (150 000 IU) was given if thrombus burden was extensive. After crossing, the wire was exchanged for an Amplatz Super Stiff Guidewire, and predilatation with a 10 mm balloon was performed when needed. Nitinol or steel stents were then deployed, with technical success defined as achieving luminal patency.	Patients were followed every 4 months clinically and annually with CT venography; suspected graft occlusion was confirmed by symptoms, Duplex ultrasound, and CT venography.	No treatment was done for malignancy.
https://dirjournal.org/pdf/beb8919b-f013-4ea1-b1c8-40332e840fe1/articles/dir.2020.19282/Diagn%20Interv%20Radiol-27-72-En.pdf	McDevitt et al,[45] 2021	Retrospective cohort	30	48.6 (16–89)	Prior central line placement (58), (42.3%) Extrinsic compression (29), (21.2%) Postsurgical anastomatic stenosis (27), (19.7%) Liver transplant (25), (18.2%) DVT (19), (13.9%) Tumor compression of IVC (18), (13.1%) Tumor compression of SVC (9), (6.6%) IVC atresia (4), (2.9%) Fibrosing mediastinitis (2), (1.5%) Isolated cardiac transplant or hepatectomy (1 each), (0.7%)	SCVS	Thoracic central veins were treated in (61), (44.5%): Type 1C (8), (5.8%) Type 2B (19), (13.9%) Type 4 (34), (24.8%) Stents were placed in: SVC (44), (21.2%) Brachiocephalic veins (27: 15 right, 12 left) Subclavian veins (13: 6 right, 7 left)	100%	NR	Each patient was assessed by an interventional radiologist.	Gianturco Z-Stent	Procedures were performed under conscious sedation with midazolam/fentanyl (55.5%) or general anesthesia (44.5%). Blunt recanalization was attempted with a vertebral-tip catheter and stiff glidewire; if unsuccessful, sharp recanalization was performed with an 18-gauge BRK needle and snare/vascular plug. Intraluminal position was confirmed by venography (100%) and intravascular ultrasound (30.7%). A 16F Z-stent sheath was advanced, and Gianturco Z-stents were deployed with 25%–50% overlap, followed by heparinization and balloon angioplasty. Completion venography and ultrasound were performed post stenting, and no stents were placed across the costoclavicular junction.	Mean follow-up was 43.6±52.7 months (range 1–207), with primary patency rates of 84.2% at 1 and 3 years, and 82.1% at 5 years.	No treatment was done for malignancy.
https://pmc.ncbi.nlm.nih.gov/articles/PMC6774659/	Karakhanian et al,[46] 2019	Retrospective cohort	28	52.5 (37–68)	Malignant disease (18), (64.3%) Prolonged use of catheters (10), (35.7%)	SVCS	Type III SVCS (stenosis between 90%-100% with retrograde azygos vein flow)	96.4	96.4	From 2002–2005, phlebography with cavography was used to assess lesions, with femoral/jugular access in swollen arms and 5000 IU heparin given. From 2006, thoracic CT replaced phlebography for procedural planning.	Wallstent; Sinus; Sioxx	The stenosis/occlusion was crossed using a 0.035 hydrophilic guidewire with MP 5F catheter support, sometimes exchanged for a stiffer Amplatz wire. Lesion length and vein diameter were measured with a pigtail catheter, and predilatation with 8×40/60 mm balloons was performed in subocclusions. Stents (10–24 mm, 40–80 mm), mainly Wallstents, were deployed—proximal first, with overlapping if needed—extending up to the right atrium. Alternative stents (Sinus, Sioxx) were used in select cases; 2 patients required multiple stents. Postdilatation (16–18 mm balloons) was performed, fibrinolysis (rtPA 20 mg) used in 1 case, and all patients received dual antiplatelet therapy (1 anticoagulated).	All patients were followed for 90 days to assess symptom relief, recurrence, and procedure-related complications.	No treatment was done for malignancy.
https://doi.org/10.1007/s00520-017-3997-9	Anton et al,[47] 2018	Retrospective cohort	31	67 ± 8	Malignant tumors: Bronchial carcinoma (26), (83.9%) Lymphoma (2), (6.5%) Metastases of extrathoracic carcinomas (3), (6.6%)	SVCS	Required port removal (13), (42%) Stenosis found (24), (77.4%) Total occlusion found (7), (22.6%) Most had bronchial carcinoma (26), (83.9%) with lymphoma (2), (6.5%) or extrathoracic metastases (3), (6.6%) Obstruction involved: Only the SVC (21), (67.7%) Left BCV (5), (16.1%) Right BCV (4), (13%) Both BCVs (1), (3.2%)	100	100	y contrast-enhanced CT (ceCT).	Sinus XL; OptiMed; Protégé; EverFlex; Covidien; Ireland	The intervention was performed under local anesthesia in an angiography suite, with venous access obtained via ultrasound-guided subclavian (5F) or femoral (6–10F) sheaths. After 3000 IU heparin, venography classified stenoses per the Stanford system, and lesions were crossed using hydrophilic or CTO guidewires with catheter support; a through-and-through technique was established when needed. Predilatation was routinely performed, vessel sizing assessed, and in patients with TIVAPs, the port was modified for guidewire access. One or two self-expanding nitinol stents (Sinus XL or Protege EverFlex) were implanted with ≥10 mm overlap, followed by postdilatation. Final venography confirmed revascularization, TIVAPs were repositioned if required, and hemostasis was achieved with manual compression and bandaging.	Clinical follow-up occurred at a mean of 37±58 days, while imaging follow-up (available in 18 patients) averaged 184±172 days.	Before stent/TIVAP implantation, 12 received no therapy, 11 chemotherapy, 7 chemo+RT, and 1 was not applicable. After stenting, 1 had no treatment, 1 RT, 16 chemotherapy, and 13 chemo+RT.

Study URL	Reference	Study design	No. of patients	Mean age (y)	Cause of SVCS	Condition	Vessels involved	Tech success rate (%)	Clinical success rate (%)	Preoperative assessment modality	Type of stent used	Procedure details	Follow-up protocol	Malignancy treatment
https://doi.org/10.1080/21548331.2017.1342507	Juscafresa et al,[48] 2017	Retrospective cohort	33	57.6 (34–71)	sNSCLC (24), (73%) SCLC (9), (27%)	SVCS	NR	100	85	Clinical symptoms and CTa for confirmation.	Wallstent; Protégé	All procedures were performed under local anesthesia with continuous monitoring, and CT angiography was used preoperatively to assess SVC anatomy. Access was obtained via right femoral vein (4–5F sheath) with hydrophilic guidewire and catheterization to the left brachiocephalic trunk; upper extremity access was added if needed. Venography was performed, and the wire was exchanged for a stiff guidewire before stent placement. A 9–10F sheath was inserted, lesion measurements confirmed, and predilatation performed when necessary, followed by deployment of a self-expandable stent (14–20 mm; Wallstent or Protege) and postdilatation. Final venography confirmed patency, femoral compression and bandaging achieved hemostasis, and chest radiography verified stent position.	At study end, 21 patients (63.6%) had died, mostly from lung cancer progression; one death was due to sepsis after stent thrombosis. Limited follow-up reflected advanced disease stages. Kaplan-Meier analysis showed primary stent patency of 94% and secondary patency of 97% at 1–12 months. Twelve patients survived without restenosis, with survival rates of 77%, 62%, 58%, and 36% at 1, 3, 6, and 12 months, respectively. Median survival after stenting was 13 months.	Patients completed their therapy with the oncology department after finishing the endovascular intervention.
https://link.springer.com/article/10.1007/s11547-017-0767-1	Niu et al,[49] 2017	Retrospective cohort	47	NR	Lung cancer (42) Esophageal cancer (1) Mediastinal tumor (0) Metastases lymph nodule (4)	SVCS	Type of obstruction: SVC only (20) SVC and unilateral BCV (8) SVC and bilateral BCV (19)	100	100	SVC syndrome was diagnosed based on patients' history, clinical presentation, and thoracic contrast enhanced computed tomography (CT) findings.	Sinus XL; Zilver; Luminexx; Smart	All SVC stents were bare self-expanding metallic types (Sinus-XL, Zilver, Luminexx, Smart), sized 2 mm larger than the SVC and extending ≥10 mm beyond the obstruction. Procedures, performed by 3 interventional radiologists under fluoroscopic guidance via right femoral vein access, used a VER catheter and guidewire to cross the lesion, with venography confirmation, predilatation (10–14 mm balloon), and deployment of 7–10 Fr delivery systems. Venography and pressure gradients were assessed before and after stenting. Stent placement strategies varied depending on involvement of unilateral/bilateral brachiocephalic veins or isolated SVC lesions. Post procedure, patients received LMWH for 3 days followed by warfarin (INR 2–3).	Follow-up included thoracic CT (at 1 month, then every 2–3 months) and telephone checks every 2 months until death or study end (Dec 2016). The primary end point was survival, with secondary end points of procedure-related complications and stent dysfunction.	Patients completed their therapy with the oncology department after finishing the endovascular intervention.
https://www.sciencedirect.com/science/article/abs/pii/S1051044314011105	Mokry et al,[50] 2015	Retrospective cohort	23	62.5 y 8.5	NSCLC with acute SVC obstruction: Stage IIIA (1) Stage IIIB (4) Stage IV (18)	Acute SVCS	Stanford classification (7): Type I (5) Type II (9) Type III (4) Type IV (5)	100	NR	All patients underwent multiphase contrast-enhanced multidetector CT before the intervention to plan the target stent length and stent diameter.	Sinus XL	The Sinus-XL is a self-expanding nitinol stent (16–34 mm × 30–100 mm) with closed-cell design, antijump mechanism, and radiopaque markers for precise placement, though not FDA approved. Procedures used combined right jugular and femoral access under local anesthesia (left-sided access if thrombosed), with venography to classify SVC obstruction (Stanford/Doty types I–IV). Recanalization was achieved with a hydrophilic guidewire and catheter, then exchanged for a super-stiff wire, followed by placement of a 10-F sheath and deployment of the Sinus-XL stent sized 2 mm larger than the native SVC. Technical success (SIR definition) required complete coverage and <30% residual stenosis, with angioplasty (12–24 mm balloons) performed if needed. Patients received 2000 IU intraprocedural heparin and 1 week of anticoagulation post procedure.	Mean follow-up was 66±83 days (range 1–305). One patient was lost to follow-up, and another developed stent occlusion at 44 days requiring re-stenting, but died from pulmonary embolism after reintervention. Primary and secondary patency rates were 95.7% and 100%, respectively.	Before stenting, 2 had no treatment, 1 RT, 3 chemotherapy, 15 chemo+RT, and 8 surgery. After stenting, 8 had no treatment, 1 RT, 11 chemotherapy, and 3 chemo+RT.

Study URL	Reference	Study design	No. of patients	Mean age (y)	Cause of SVCS	Condition	Vessels involved	Tech success rate (%)	Clinical success rate (%)	Preoperative assessment modality	Type of stent used	Procedure details	Follow-up protocol	Malignancy treatment
https://www.kjronline.org/DOIx.php?id=10.3348/kjr.2014.15.1.87	Cho et al,[51] 2014	Retrospective cohort	40	61.4 (35-81)	Tumor histology: Squamous cell carcinoma (14) Adenocarcinoma (12) Small cell carcinoma (10) Others (4)	SVCS	SVC and both brachiocephalic veins (20) Only SVC (15) SVC and unilateral brachiocephalic vein (5)	100	92	Contrast venography	ComVi	Procedures were performed by 2 senior interventional radiologists under moderate sedation (pethidine + lidocaine) with cardiopulmonary monitoring, without prophylactic antibiotics. Venous access was primarily via the right femoral vein (jugular if required), using a 9–10 Fr sheath, hydrophilic guidewire, and catheter for venography and pressure gradient measurement, followed by exchange to a stiff Amplatz wire and predilatation (6–12 mm balloons). A triple-layered ePTFE-covered ComVi stent (10–14 mm, 6–10 cm) with bare extensions was deployed, oversized by 10%–15% relative to SVC diameter, and positioned to extend ≥1 cm beyond the lesion. Stent placement strategies varied by involvement of the brachiocephalic veins or venous confluence, with unilateral stenting preferred if 1 BCV remained patent. Poststenting venography and pressure gradients confirmed efficacy, with further balloon dilatation if residual gradients >10 mm Hg; anticoagulation was prescribed in most patients.	Follow-up venography was not routinely performed unless the patient required additional intervention.	Patients were treated with the oncology department.
https://pubs.rsna.org/doi/10.1148/radiol.12120517	Gwon et al,[52] 2013	Retrospective cohort	73	60.3 (35–81)	Tumor histology: Squamous cell carcinoma (27) Adenocarcinoma (25) Small cell carcinoma (15) Thymic carcinoma (3) Invasive thymoma (2) Breast cancer (1)	SVCS	SVC and both brachiocephalic veins (44) Only SVC (26) Unilateral brachiocephalic vein (3)	100	93.2	Pre-stent contrast-enhanced CT was used for anatomical assessment and planning, followed by venography and stent insertion by experienced interventional radiologists.	Covered ePTFE vs uncovered	Procedures were performed under moderate sedation (pethidine + lidocaine) with venography to evaluate SVC stenosis/occlusion. Access was via femoral, jugular, or subclavian veins, using 6F sheaths for uncovered and 9–10F for covered stents; lesions were crossed with a 5F catheter and hydrophilic guidewire, or alternative routes if needed. Pressure gradients were measured before stenting, and predilatation (6–12 mm balloons) guided stent sizing and placement. Stent deployment strategies varied depending on involvement of SVC and brachiocephalic veins, with overlapping stents used for longer lesions; postdilatation was performed if gradients >10 mm Hg. Patients received heparin infusion for 2–5 days followed by long-term anticoagulation (warfarin, INR 2.0) or aspirin for ≥3 months.	Covered stent patients were prospectively followed, whereas uncovered stent data were retrospectively collected, though follow-up protocols were similar. Clinical and radiologic data, technical details, and complications were recorded. Covered stent patients were seen at 1, 3, 6, 9, and 12 months, then followed monthly by phone, with CT at 1 and 6 months and during/after oncologic therapy. Uncovered stent patients had clinical and CT follow-up at 1 month, with additional survival data obtained by phone.	
https://link.springer.com/article/10.1007/s00270-011-0310-z	Fagedet et al,[53] 2013	Retrospective cohort	164	59.9	Lung cancer (132), (80.5%) NSCLS (82), (50%) SCLC (50), (30.5%) Other: metastasis, lymphoma (32), (19.5%)	SVCS	SVC and both brachiocephalic veins (82) Only SVC (67) Unilateral brachiocephalic vein (15)	84.5	NR	Angiography	Wallstent; Memotherm; SMART; Strecker; Protégé	SVCS was treated by endovascular therapy immediately after diagnosis, under local anesthesia with cardiopulmonary monitoring in an angiography suite. Femoral access was preferred, with jugular or brachial access added if dual entry was needed. Lesions were crossed with a multipurpose catheter and hydrophilic stiff wire, predilated by angioplasty, and treated with stents as required, followed by repeat angioplasty and control venography; 3000 IU heparin was administered intra-procedurally. Stent selection depended on lesion extent: SVC only (isolated stent), SVC + BCV (unilateral/bi-BCV stenting), with Wallstent for compressed lesions, nitinol stents for large SVC, and Smart stents for BCV. Thrombus management included thromboaspiration, fibrinolysis (urokinase 100,000–300,000 IU bolus or prolonged infusion), fragmentation, or balloon crushing.	Follow-up was measured from the procedure to last contact or death, with data collection ending February 28, 2009. Antitumor therapy followed international guidelines. Patients had CT every 6 months in the first year, then annually, and recurrent cases were offered repeat endovascular therapy when feasible.	Treatment was given as per the guidelines for each malignancy.

BCV, brachiocephalic vein; CT, computed tomography; CTO, chronic total occlusion; DVT, deep vein thrombosis; ePTFE, expanded polytetrafluoroethylene; FDA, Food and Drug Administration; INR, international normalized ratio; IVC, inferior vena cava; MR, magnetic resonance; NR: not reported; NSCLC, non-small cell lung cancer; RT, radiotherapy; SCLC, small cell lung carcinoma; SIR, Society of Interventional Radiologists; sNSCLC, squamous non-small cell lung cancer; SVC, superior vena cava; SVCS, superior vena cava syndrome; TIVAP, totally implantable venous access port.

Clinical implications and future directions

The pooled evidence strongly supports endovascular stenting as an effective first-line intervention for both malignant and benign SVCS, with high technical success rates, acceptable complication profiles, and durable medium-term patency. The ability to achieve rapid symptom resolution makes this approach particularly valuable in the management of acute or severe presentations. However, the decline in primary patency beyond the first year highlights the importance of structured surveillance programs and the need for patient counseling regarding potential reintervention requirements.[40]

The differential outcomes observed between malignant and benign etiologies suggest that etiology-specific treatment algorithms may optimize patient outcomes.[40]

Future research priorities should include prospective comparative effectiveness studies, standardization of outcome definitions, investigation of optimal anticoagulation strategies, and development of evidence-based surveillance protocols to guide long-term patient management.

Conclusions

Neoplastic SVCS, usually secondary to malignancy, demonstrates a wide spectrum of severity, ranging from mild symptoms to life-threatening complications. Endovascular stenting represents the first-line approach, delivering immediate, safe, and durable patency outcomes. When the patient is stable, histologic confirmation should be obtained before initiating oncologic therapy, where severe cases require urgent stenting. Future investigations should include harmonizing outcome measures, anticoagulation optimization, and long-term monitoring protocols.

CORRESPONDING AUTHOR

Almamoon I. Justaniah, MD, FSIR

P.O. Box 40047, Jeddah 21499,
Saudi Arabia

EMAIL: *ajustaniah@kfshrc.edu.sa*

Dr Almamoon I. Justaniah received an honorarium from Servier for the writing of this article.

References

1. Azizi AH, Shafi I, Shah N, et al. Superior vena cava syndrome. *JACC Cardiovasc Interv*. 2020 28;13(24):2896-2910.
2. Friedman T, Quencer KB, Kishore SA, Winokur RS, Madoff DC. Malignant venous obstruction: superior vena cava syndrome and beyond. *Semin Intervent Radiol*. 2017;34(4):398-408.
3. Cheng S. Superior vena cava syndrome: a contemporary review of a historic disease. *Cardiol Rev*. 2009-;17(1):16-23.
4. Rice TW, Rodriguez RM, Light RW. The superior vena cava syndrome: clinical characteristics and evolving etiology. *Medicine (Baltimore)*. 2006;85(1):37-42.
5. Deshwal H, Ghosh S, Magruder K, Bartholomew JR, Montgomery J, Mehta AC. A review of endovascular stenting for superior vena cava syndrome in fibrosing mediastinitis. *Vasc Med*. 2020;25(2):174-183.
6. Schraufnagel DE, Hill R, Leech JA, Pare JA. Superior vena caval obstruction. Is it a medical emergency? *Am J Med*. 1981;70(6):1169-1174.
7. Wilson LD, Detterbeck FC, Yahalom J. Clinical practice. Superior vena cava syndrome with malignant causes. *N Engl J Med*. 2007;356(18):1862-1869. Erratum in: *N Engl J Med*. 2008;358(10):1083.
8. Seligson MT, Surowiec SM. Superior vena cava syndrome. 2022 Sep 26. In: *StatPearls [Internet]*. StatPearls Publishing; 2025 Jan–.
9. Schechter MM. The superior vena cava syndrome. *Am J Med Sci*. 1954;227(1):46-56.
10. Klein-Weigel PF, Elitok S, Ruttloff A, et al. Superior vena cava syndrome. *Vasa*. 2020;49(6):437-448.
11. Chee CE, Bjarnason H, Prasad A. Superior vena cava syndrome: an increasingly frequent complication of cardiac procedures. *Nat Clin Pract Cardiovasc Med*. 2007;4(4):226-230.
12. Breault S, Doenz F, Jouannic AM, Qanadli SD. Percutaneous endovascular management of chronic superior vena cava syndrome of benign causes: long-term follow-up. *Eur Radiol*. 2017;27(1):97-104.
13. Herscovici R, Szyper-Kravitz M, Altman A, et al. Superior vena cava syndrome - changing etiology in the third millennium. *Lupus*. 2012;21(1):93-96.
14. Ostler PJ, Clarke DP, Watkinson AF, Gaze MN. Superior vena cava obstruction: a modern management strategy. *Clin Oncol (R Coll Radiol)*. 1997;9(2):83-89.
15. Yellin A, Rosen A, Reichert N, Lieberman Y. Superior vena cava syndrome. The myth--the facts. *Am Rev Respir Dis*. 1990;141(5 Pt 1):1114-1118.
16. Lochridge SK, Knibbe WP, Doty DB. Obstruction of the superior vena cava. *Surgery*. 1979;85(1):14-24.
17. Nogeire C, Mincer F, Botstein C. Long survival in patients with bronchogenic carcinoma complicated by superior vena caval obstruction. *Chest*. 1979;75(3):325-329.
18. Ahmann FR. A reassessment of the clinical implications of the superior vena caval syndrome. *J Clin Oncol*. 1984;2(8):961-969.
19. Sculier JP, Evans WK, Feld R, et al. Superior vena caval obstruction syndrome in small cell lung cancer. *Cancer*. 1986;57(4):847-851.
20. Rowell NP, Gleeson FV. Steroids, radiotherapy, chemotherapy and stents for superior vena caval obstruction in carcinoma of the bronchus: a systematic review. *Clin Oncol (R Coll Radiol)*. 2002;14(5):338-351.
21. Perez-Soler R, McLaughlin P, Velasquez WS, et al. Clinical features and results of management of superior vena cava syndrome secondary to lymphoma. *J Clin Oncol*. 1984;2(4):260-266.
22. Gucalp R, Dutcher J, Fauci AS, et al. Oncologic emergencies. In: *Harrison's Principles of Internal Medicine*. 17th ed. New York McGraw-Hills. 2008:1730.
23. Picquet J, Blin V, Dussaussoy C, Jousset Y, Papon X, Enon B. Surgical reconstruction of the superior vena cava system: indications and results. *Surgery*. 2009;145(1):93-99.
24. Quencer KB. Superior vena cava syndrome: etiologies, manifestations, and treatments. *Semin Intervent Radiol*. 2022;39(3):292-303.
25. Grant JD, Lee JS, Lee EW, Kee ST. Superior vena cava syndrome. *Endovascular Today*. 2009. Accessed August 29, 2025. https://evtoday.com/articles/2009-july/EVT0709_09-php/pdf.
26. Sasikumar A, Kothiwale VA. Superior vena cava syndrome: a comprehensive review. *Int J Res Med Sci*. 2025;13(3):1331-1334.
27. Ponti A, Saltiel S, Rotzinger DC, Qanadli SD. Insights into endovascular management of superior vena cava obstructions. *Front Cardiovasc Med*. 2021;8:765798.
28. Anyagwa OE, Dairo OA, Tak RS, et al. Endovascular stenting for superior vena cava syndrome: a systematic review. *Emerg Med J*. 2024;9(2):154-167.
29. Wright K, Digby GC, Gyawali B, et al. Malignant superior vena cava syndrome: a scoping review. *J Thorac Oncol*. 2023;18(10):1268-1276.
30. Chawla S, Zhang Q, Gwozdz AM, et al. Editor's Choice - A systematic review and meta-analysis of 24 month patency after endovenous stenting of superior vena cava, subclavian, and brachiocephalic vein stenosis. *Eur J Vasc Endovasc Surg*. 2025;69(1):139-155.
31. Léon D, Rao S, Huang S, et al. Literature review of percutaneous stenting for palliative treatment of malignant superior vena cava syndrome (SVCS). *Acad Radiol*. 2022;29 Suppl 4:S110-S120.
32. Azizi AH, Shafi I, Zhao M, et al. Endovascular therapy for superior vena cava syndrome: a systematic review and meta-analysis. *EClinicalMedicine*. 2021;37:100970.
33. Aung EY, Khan M, Williams N, Raja U, Hamady M. Endovascular stenting in superior vena cava syndrome: a systematic review and meta-analysis. *Cardiovasc Intervent Radiol*. 2022;45(9):1236-1254.
34. Cao S, Zou Y, Lyu T, et al. Safety and efficacy of stent graft implantation for malignant superior vena cava syndrome. *Heart Surg Forum*. 2021;24(6):E952-E957.
35. Guerrero-Macías S, Beltrán J, Buitrago R, Beltrán R, Carreño J, Carvajal-Fierro C. Outcomes in patients managed with endovascular stent for malignant superior vena cava syndrome. *Surg Open Sci*. 2023;16:16-21.
36. Rioja Artal S, González Martínez V, Royo Serrando J, Delgado Daza R, Moga Donadeu L. Results of palliative stenting in malignant superior vena cava syndrome analyzing self-expanding stainless steel and nitinol venous bare metal stents. *J Endovasc Ther*. 2024:15266028241242926.
37. Liu H, Li Y, Wang Y, Yan L, Zhou P, Han X. Percutaneous transluminal stenting for superior vena cava syndrome caused by malignant tumors: a single-center retrospective study. *J Cardiothorac Surg*. 2021;16(1):39.
38. Lanciego C, Pangua C, Chacón JI, et al. Endovascular stenting as the first step in the overall management of malignant superior vena cava syndrome. *AJR Am J Roentgenol*. 2009;193(2):549-558.
39. Rowell NP, Gleeson FV. Steroids, radiotherapy, chemotherapy and stents for superior vena caval obstruction in carcinoma of the bronchus. *Cochrane Database Syst Rev*. 2001;(4):CD001316. Update in: *Cochrane Database Syst Rev*. 2015;(3):CD001316.
40. Kordzadeh A, Askari A, Hanif MA, Gadhvi V. Superior vena cava syndrome and Wallstent: a systematic review. *Ann Vasc Dis*. 2022;15(2):87-93.
41. Sheth S, Ebert MD, Fishman EK. Superior vena cava obstruction evaluation with MDCT. *AJR Am J Roentgenol*. 2010;194(4):W336-W346.
42. Nossair F, Schoettler P, Starr J, et al. Pediatric superior vena cava syndrome: an evidence-based systematic review of the literature. *Pediatr Blood Cancer*. 2018;65(9):e27225.
43. Ren W, Qi X, Yang Z, Han G, Fan D. Prevalence and risk factors of hepatocellular carcinoma in Budd-Chiari syndrome: a systematic review. *Eur J Gastroenterol Hepatol*. 2013;25(7):830-841.
44. Irace L, Martinelli O, Gattuso R, et al. The role of self-expanding vascular stent in superior vena cava syndrome for advanced tumours. *Ann R Coll Surg Engl*. 2021;103(4):296-301.

45. L McDevitt J, T Goldman D, J Bundy J, et al. Gianturco Z-stent placement for the treatment of chronic central venous occlusive disease: implantation of 208 stents in 137 symptomatic patients. *Diagn Interv Radiol.* 2021;27(1):72-78.

46. Karakhanian WK, Karakhanian WZ, Belczak SQ. Superior vena cava syndrome: endovascular management. *J Vasc Bras.* 2019;18:e20180062.

47. Anton S, Oechtering T, Stahlberg E, et al. Endovascular stent-based revascularization of malignant superior vena cava syndrome with concomitant implantation of a port device using a dual venous approach. *Support Care Cancer.* 2018;26(6):1881-1888.

48. Juscafresa LC, Bazo IG, Grochowicz L, et al. Endovascular treatment of malignant superior vena cava syndrome secondary to lung cancer. *Hosp Pract.* 2017;45(3):70-75. https://doi.org/10.1080/21548331.2017.1342507

49. Niu S, Xu YS, Cheng L, Cao C. Stent insertion for malignant superior vena cava syndrome: effectiveness and long-term outcome. *Radiol Med.* 2017;122(8):633-638.

50. Mokry T, Bellemann N, Sommer CM, et al. Retrospective study in 23 patients of the self-expanding sinus-XL stent for treatment of malignant superior vena cava obstruction caused by non-small cell lung cancer. *J Vasc Interv Radiol.* 2015;26(3):357-365.

51. Cho Y, Gwon DI, Ko GY, et al. Covered stent placement for the treatment of malignant superior vena cava syndrome: is unilateral covered stenting safe and effective? *Korean J Radiol.* 2014;15(1):87-94.

52. Gwon DI, Ko GY, Kim JH, Shin JH, Yoon HK, Sung KB. Malignant superior vena cava syndrome: a comparative cohort study of treatment with covered stents versus uncovered stents. *Radiology*. 2013;266(3):979-987.

53. Fagedet D, Thony F, Timsit JF, et al. Endovascular treatment of malignant superior vena cava syndrome: results and predictive factors of clinical efficacy. *Cardiovasc Intervent Radiol.* 2013;36(1):140-149.

Correspondent

Servier Affaires Médicales - 35, rue de Verdun - 92284 Suresnes Cedex - France

www.servier.com