REVIEW ARTICLE

Advances in Interventional Radiology for Superior Vena Cava Obstruction: Pathophysiology, Techniques and Challenges

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ABSTRACT

Superior Vena Cava Obstruction (SVCO) is a serious disorder that frequently arises from benign sources like thrombosis from pacemakers and indwelling catheters or malignant processes like lung cancer or lymphoma. Compared to conventional therapy, endovascular stenting provides quick symptom relief, making it a popular therapeutic option. This review will examine the pathophysiology, interventional methods, and perioperative treatment to determine the best practices for enhancing clinical results and reducing problems.

An extensive literature review was performed using Google Scholar, Web of Science, PubMed, and Scopus. Keywords like "superior vena cava obstruction," "SVCO stenting," "malignant SVCO," and "clinical outcomes" were employed to find pertinent studies. Pathophysiology, diagnostic methods, interventional strategies, complications, and post-stenting care were the main topics of the review.

With an overall symptom resolution rate of 90-95%, endovascular stenting exhibits good technical success and prompt symptom relief. Long-term patency is enhanced with covered stents, but migration issues still exist. Although they are rare (8-10%), complications can be fatal and include pulmonary embolism, cardiac tamponade, and stent thrombosis. Standardized guidelines are necessary since the best perioperative care, which includes beta-blockers, anticoagulation, and hemodynamic instability monitoring, is still up for discussion.

In summary, endovascular stenting is a crucial development in the treatment of SVCO, offering quick symptom alleviation and positive short-term results. However, more research is needed on post-procedural care techniques and long-term consequences. Optimizing outcomes and lowering mortality in high-risk patients require a multidisciplinary approach and updated guidelines.

Keywords: *Superior vena cava obstruction, superior vena cava syndrome, malignant SVCO, endovascular stenting.*

INTRODUCTION

Superior vena cava obstruction (SVCO), as its name suggests, is the partial or complete obstruction of the superior vena cava (SVC) resultant from a range of aetiologies, which can cause a collection of signs and symptoms known as superior vena cava syndrome (SVCS). The low blood pressure and thin walls of the SVC make it especially susceptible to mechanical obstruction, and given its proximity to the lung and regional lymph nodes, favours those with intrathoracic diseases [1]. SVCS was first described by William Hunter in 1757 from a case of a syphilitic aortic aneurysm [2]. In fact, before the use of antibiotics, SVCS was a common infectious etiology; aortic aneurysms from tertiary syphilis and tuberculosis-related mediastinal adenopathy were common causes of this, with a cause prevalence of 40% based on a review of 274 well-documented SVCS cases in 1954 [3, 4].

Malignancy is currently the most common etiology of SVCS, accounting for around 70% of cases, with the remainder being of benign causes [5]. SVCO caused by malignant etiology usually results from external compression of the SVC, intraluminal tumor growth, or thrombosis secondary to the hypercoagulable state of cancer [3, 6]. Non-small cell lung cancer accounts for around 50% of malignant SVCS, with small cell lung cancer accounting for around 25% of cases, and other cancers such as lymphomas, thymomas and metastases accounting for the remainder **(Fig. 1)** [7]. Benign causes of SVCO have however seen a recent increase in prevalence, mainly from the increase in implanted transvenous cardiac devices and long-term central venous catheters resulting in SVC thrombosis [3, 8-10]. Previously the most common cause of benign SVCO was mediastinal fibrosis [9, 11].

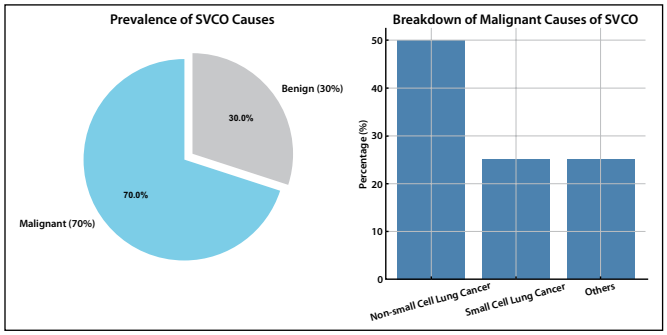

Fig. (1): The prevalence of benign and malignant SVCO and the percentage of malignant causes.

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SVCO arises as a consequence due to impaired blood flow through the SVC leading to elevated central venous pressures and venous congestion in the upper body [5, 7]. Pathologically, this can be classified per etiology as a malignant or non-malignant cause. Malignant causes include lung cancers, and lymphomas whereas non-malignant causes arise from thrombotic events from indwelling catheters or certain genetic predispositions [12, 13]. The obstruction has a cascade of effects such as venous congestion, collateral vessel formation, and impaired preload. This systemically manifests as hypotension, hypoperfusion, and life-threatening complications that would require urgent intervention [6, 7, 14].

The signs and symptoms of SVCS can be correlated to the pathophysiology of the syndrome of which the most common is a direct result from the venous obstruction and associated upstream pooling of blood; facial/neck plethora, upper extremity edema, and distended neck/chest veins [5, 7]. Symptom severity increases with a greater degree of SVCO and reduces with increased venous collateral formation [15]. Symptoms may worsen when supine, due to further impairment of venous return [16].

The first-line management of malignant SVCO has transitioned from radiotherapy, chemotherapy, and surgical bypass to endovascular therapy with stenting since its first introduction in 1986 by Charnsangavej [17]. Over the past two decades, stenting has become commonplace secondary to its high efficacy with symptom relief achieved in up to 95% of patients, high technical success, and low complication rates [3, 7, 12, 18, 19]. Despite this, however, there seems to be a lack of standardized protocols regarding pre- and post-procedural management in the literature to maintain SVC patency and for favorable long-term outcomes.

For instance, certain studies recommend pre-dilatation of the SVC before stent placement whilst others recommend post-dilatation after stent placement, and much variation exists surrounding anticoagulation therapy to prevent stent thrombosis [20-22].

Despite low complication rates and a procedure mortality rate of around 2%, complications can be very severe and can include pulmonary embolism, cardiac tamponade, SVC rupture, and stent migration [7, 23]. The aim of this review therefore is to analyse and identify optimal perioperative management in malignant SVCO stenting, yielding favourable long-term treatment outcomes and minimising complications.

MATERIALS AND METHODS

For this study, a thorough literature review was conducted using various databases, including PubMed, Scopus, Web of Science and Google Scholar to collect relevant studies and data on SVCO to see what the ideal treatment before and after stent insertion should be. A wide range of terms was utilized, including “superior vena cava obstruction,” “SVCO,” “mediastinal tumors,” “oncology,” “stenting in SVCO,” and “clinical outcomes”. We narrowed down the specific studies that were relevant to the clinical management of SVCO, which encompassed various aspects such as clinical indications, pathophysiology, diagnostic approaches, procedural techniques, therapeutic interventions, complications, and long-term outcomes. All the findings were collated to provide a comprehensive understanding of current clinical practices and to identify gaps in the literature that would require further investigation.

DISCUSSION

The superior vena cava (SVC), formed by the left and right brachiocephalic veins, drains the head, neck, torso, and upper extremities. Located in the superomedial mediastinum and surrounded by the sternum, trachea, right mainstem bronchus, and lymph nodes, the SVC’s thin walls and low pressure make it vulnerable to compression by adjacent masses. This compression can compromise blood flow, leading to superior vena cava obstruction (SVCO) [7]. Collateral pathways, including the azygos-hemiazygos system and contributions from the lateral thoracic, vertebral, and internal mammary pathways, facilitate retrograde blood flow to the right atrium during occlusion. However, venous pressure often remains elevated, causing the characteristic signs and symptoms of SVCO [5].

The etiopathophysiological mechanisms of SVCS involve impaired blood flow through the SVC, leading to elevated central venous pressure and venous congestion in the upper body [5, 7, 24, 25]. Aetiologies are broadly categorized as malignant or non-malignant [26].

In MSVCO, thoracic malignancies, such as small cell and non-small cell lung cancers, and hematological malignancies like lymphomas, are the most common causes [26]. These malignancies invade the SVC due to their proximity in the superomedial mediastinum.

Conversely, non-malignant SVCO results from factors other than tumor compression, such as thrombotic events linked to central venous catheters or pacemakers, which promote a hypercoagulable state [12, 27]. Genetic predispositions, including Factor V Leiden and antiphospholipid syndrome, also contribute to venous thrombosis [13, 28].

The pathophysiological consequence of SVC compression, irrespective of etiology, results in impaired hemodynamic stability and reduced venous return from the upper body to the heart [5, 7, 15, 26]. Partial obstruction may cause mild dyspnoea, particularly during exertion, as increased venous pressure affects pulmonary circulation. Facial fullness upon waking can occur due to venous pressure and fluid accumulation. With persistent obstruction, compensatory vascular remodeling leads to collateral venous formation. Upregulation of pro-angiogenic factors like vascular endothelial growth factor (VEGF) and hypoxia-induced factor (HIF) alters venous return pathways, primarily involving the azygos venous system. This adaptation can produce symptoms such as edema, cyanosis, and, in severe cases, airway compression [14, 29].

The altered venous return reduces blood volume entering the right atrium, decreasing end-diastolic volume (EDV) and preload. Collateral pathways fail to fully restore normal venous function, leading to insufficient preload restoration. According to Frank-Starling's law, decreased preload reduces cardiac output, potentially causing organ hypoperfusion. This can result in systemic hypotension, manifesting as fainting, dizziness, or hypovolemic shock in severe cases [30].

Post-SVC stenting, blood flow restoration to the right atrium improves venous return. However, the immediate physiological changes can trigger a cascade of events, increasing post-procedural mortality risk [4, 5, 7, 14, 31]. Restoration of blood flow after prolonged ischemia elevates oxidative stress, marked by excessive reactive oxidative species (ROS) production. This oxidative stress induces vascular inflammation, tissue edema, and endothelial dysfunction, heightening the risk of thrombotic events such as pulmonary embolism [32, 33].

The Bainbridge reflex, triggered by increased venous return, results in heightened heart rate *via* atrial stretch receptors. This also induces the release of atrial natriuretic peptide (ANP) [34, 35]. A sudden heart rate increase post-stenting raises the risk of cardiac arrhythmias, particularly atrial fibrillation, complicating recovery. Additionally, ANP release promotes vasodilation and renin inhibition, potentially causing rapid vasodilation and diuresis, which, if unmanaged, can result in life-threatening hypotension [36].

The sudden increase in preload post-SVC stenting can lead to acute right ventricular (RV) overload, resulting in right-sided heart failure characterized by elevated central venous pressure, hepatic congestion, and reduced cardiac output **(Fig. 2)**. If untreated, this can cause systemic hypoperfusion and end-organ dysfunction. In patients with pre-existing conditions like pulmonary hypertension, such hemodynamic instability may result in systemic venous congestion and fatal hypotension [30, 32].

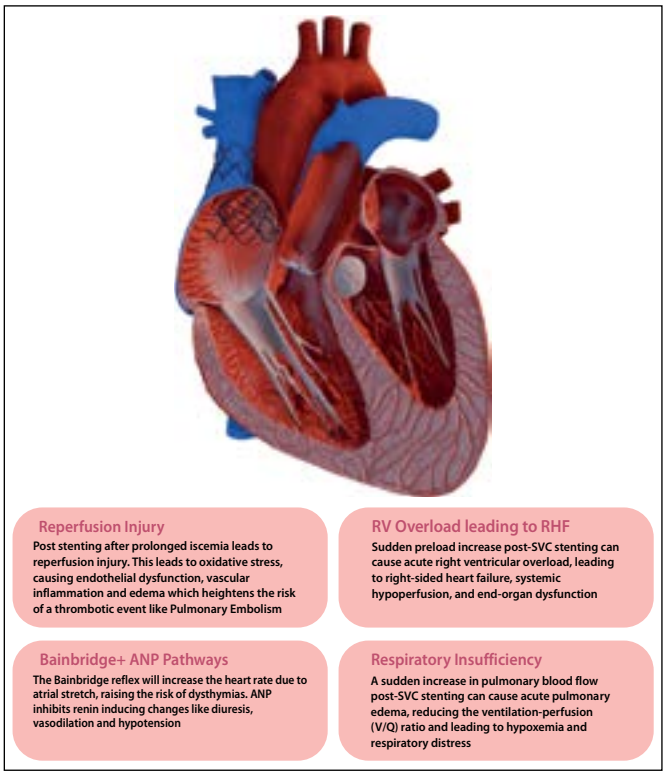

Fig. (2): The pathophysiological changes post SVC stent insertion.

Additionally, the sudden influx of blood into the pulmonary circulation can precipitate acute pulmonary edema, reducing the ventilation-perfusion (V/Q) ratio and leading to hypoxemia and potentially respiratory distress, which are life-threatening if not managed promptly [37, 38].

Thus, while SVC stenting restores hemodynamic flow, it can paradoxically trigger complications that significantly impact prognosis. Recognizing these potential complications and their mechanisms is critical for improving outcomes. Adequate post-stenting monitoring and tailored interventions are essential to mitigate life-threatening consequences. Given the predominantly cardiac and pulmonary nature of these events, input from specialist cardiologists and pulmonary physicians is vital. Recommended measures include beta-blockers, prophylactic diuretics, and intensive cardiac monitoring in the intensive therapy unit (ITU) post-stent insertion [5, 7, 15, 24, 26].

SVCO is primarily a clinical diagnosis, with facial edema being the most commonly reported initial symptom, occurring in approximately 60% of patients

Table 1: Kishi scoring system.

Signs and Symptoms	Score
Neurological symptoms	
Stupor, coma, blackout	4
Blurry vision, headache, dizziness, amnesia	3
Changes in mentation	2
Uneasiness	1
Laryngopharyngeal or thoracic symptoms	
Orthopnoea or laryngeal oedema	3
Stridor, hoarseness, dysphagia, glossal oedema, or shortness of breath	2
Cough or pleural effusion	1
Nasal and facial signs or symptoms	
Lip oedema, nasal stiffness, epistaxis, rhinorrhoea	1
Facial swelling	1
Venous dilatation	
Neck vein or arm vein distention, upper extremity swelling, or upper body plethora	1

[39-42]. Other frequent presentations include arm edema and distended veins in the arms and chest [3, 40-43]. Patients may also exhibit a variety of additional signs and symptoms, categorized by Kishi *et al.* [44] into four groups: neurological (*e.g.,* headaches, dizziness, amnesia, blurry vision, coma, changes in mentation, uneasiness), laryngopharyngeal (*e.g.,* orthopnoea, laryngeal edema, stridor, hoarseness, dysphagia, glossal edema, shortness of breath, cough, pleural effusion), facial (*e.g.,* facial edema, proptosis, lip edema, nasal stiffness, epistaxis, rhinorrhoea), and chest (*e.g.,* neck vein or arm vein distention, upper extremity swelling, upper body plethora) [42, 44]. These symptoms are scored by severity as determined by Kishi *et al.* [44], with neurological symptoms like coma scoring highly, while neck and vein distension are scored significantly lower, as shown in Table **1**.

Another simplified scoring system for assessing the severity of SVC syndrome is the Yale SVC grading system, developed in 2008 by Yu *et al.* [47]. This system grades severity from "0," where patients are asymptomatic and SVCO is incidentally found on imaging, to "5," where SVCO has caused patient death, as detailed in Table **2**.

Both the Kishi Scoring System and the Yale grading system guide the assessment of symptom severity in SVCO [47]. The Yale system simplifies the evaluation of a patient's condition, while the Kishi system offers a quantitative measure of severity. A Kishi score of 4 or above indicates the need for immediate intervention, such as stenting [45, 48].

Table 2: Yale grading system for SVC syndrome.

Grade	Definition
0	Asymptomatic: radiographic superior vena cava obstruction in the absence of symptoms
1	Mild: oedema in the head or neck, cyanosis, plethora
2	Moderate: oedema in the head or neck with functional impairment (mild dysphagia/ mild or moderate impairment of the head, jaw, or eyelid movements, visual disturbance)
3	Severe: mild to moderate cerebral oedema, laryngeal oedema, diminished cardiac reserve
4	Life threatening: significant cerebral oedema, laryngeal oedema, haemodynamic compromise
5	Fatal: cause of death

Table 3: Stanford classification for degree of obstruction.

Stanford Type	Description	Management as Advised by Stanford *et al.* [50]
Type I	Partial obstruction (up to 90% stenosis) of the SVC with patency of the azygos -right atrial pathway	Irradiation and chemotherapy
Type II	Near complete-to-complete obstruction (90-100%) of the SVC with patency and antegrade flow in the azygos-right atrial pathway	Interventional treatment when there is airway compromise or cerebral venous hypertension
Type III	Near complete-to-complete obstruction (90-100%) of the SVC with reversal of azygos blood flow	SVC bypass
Type IV	Complete obstruction of the SVC and one or more of the major caval tributaries including the azygos systems	Interventional treatment when there is airway compromise or cerebral venous hypertension

The severity of SVCS correlates with the degree and rapidity of obstruction impeding venous return [3, 42, 43, 49]. Stanford *et al.* introduced a classification system for the magnitude of obstruction, using venography to associate obstruction severity with management options, as shown in Table **3** [50, 51].

In general, symptoms develop over weeks if not months [3] although life expectancy when MSVCO is involved is on average 6 months [3], with onset of symptoms classes into two groups, acute (onset of symptoms less than two weeks) and subacute or chronic (onset of symptoms greater than two weeks) [52]. With a slower development of the obstruction, collateral venous supply can be created and thus result in better prognostic outcomes [42, 43]. Collateral venous supply includes significantly the azygos hemiazygos and accessory hemiazygos veins.

Azizi *et al.* [7] proposed a modification to the Stanford classification to include the anatomical location of the SVC obstruction into the severity of the anatomical

Table 4: Azizi classification.

Type	Description	Symptoms
Type I	SVCO involves the brachiocephalic veins. This results in moderate to severe symptoms using the Yale Grading System.	Moderate to severe
Type II	SVCO is proximal to the azygos vein. Collateral blood supply is directed into the azygos vein *via* the right superior intercostal vein.	Severe
Type III	SVCO is at the level of the azygos vein. Blood cannot be directed into the azygos vein and is redirected to chest wall collateral veins, including the superior epigastric and internal mammary veins.	Severe
Type IV	SVCO is distal to the azygos vein. This results in retrograde flow into the azygos and hemiazygos veins.	Mild (compared to other grades)

location of the obstruction and which describes the collateral venous supply that arises from different anatomical locations of SVCO. The grades of SVCO that Azizi *et al.* [7] are described in Table **4**.

Considering the Yale Scoring system, Kishi Scoring System, Stanford classification, and Azizi classification, the severity of the symptoms is most acutely compromising and necessitate intervention for Grade 3 on the Yale Scoring system (severe to life-threatening symptoms), for a Kishi score of greater than 4 and/or Type II, III, IV on the Stanford Classification (near to or complete SVCO) or Type I-III using the Azizi classification system (SVCO is at the level or proximal to the azygos vein with or without brachiocephalic venous supply involvement [44, 47, 50, 51].

The National Early Warning Score (NEWS score) is an early warning scoring system that uses physiological observations to determine the likelihood of clinical deterioration in patients. This uses the parameters seen in Table **5** to determine the severity of the disease [53-55]. Established in 2012 in the United Kingdom, it has quickly become integrated into all parts of the National Health Service (NHS) from acute medicine to community services or general practice [53-55]. Acutely, it is beneficial in being able to predict sepsis when scoring more than 5 than other scoring systems such as quick sequential organ failure assessment (qSOFA) or the systemic inflammatory response criteria (SIRS) [56, 57]. Also, it can reliably predict patient mortality within 24 hours [56, 58]. After 24 hours, it is unreliable to predict mortality as other factors, not included in the score, affect long-term mortality such as functional ability, comorbidities, and age [56, 58, 59]. Other arguments against the NEWS scoring system include it is reductionist as it removes other parameters such as urine output, so acute conditions such as acute kidney injury can be missed [56].

SVCO symptoms revolve primarily around venous congestion, subsequently causing overall hemodynamic instability. Consequently, this can lead to increased pulmonary venous pressure as well as edema, especially in the head and neck area causing dyspnoea [7, 60, 61]. With this being said, these parameters are monitored in the NEWS scoring system, especially blood pressure, respiratory rate, and oxygen saturation.

Suspicion of SVCO is primarily based on the characteristic clinical presentations. However, a definitive diagnosis requires imaging. Chest X-rays may indirectly suggest potential causes of SVCO through signs of mediastinal widening or pleural effusion, but lack the specificity required to determine the underlying etiology [3]. Duplex ultrasound may detect the presence of a thrombus within the jugular, subclavian, and axillary veins, but is often limited by rib and lung shadows that prevent direct visualization of the SVC [7]. Digital subtraction venography remains the gold standard for SVCO diagnosis and is typically carried out before stenting. It comprehensively defines the extent of thrombus burden, and venous collateralization, and offers superior vessel visualization when compared

Table 5: The NEWS scoring system. Consciousness is determined as being alert or CVPU: new confusion, voice, pain and unresponsiveness. SpO2 Scale 2% is typically used in the presence of type 2 respiratory failure.

Physiological Parameters	3	2	1	0	1	2	3
Respiration rate (per minute)	≤8	-	9-11	12-20	-	21-24	≥25
SpO2 Scale 1 (%)	≤91	92-93	94-95	≥96	-	-	-
SpO2 Scale 2 (%)	≤83	84-84	86-87	88-92 ≥93 on air	93-94 on oxygen	95-96 on oxygen	≥97 on oxygen
Air or Oxygen?	-	Oxygen	-	Air	-	-	-
Systolic Blood Pressure (mmHg)	≤90	91-100	101-110	111-219	-	-	≥220
Pulse (per minute)	≤40	-	41-50	51-90	91-110	111-130	≥131
Consciousness	-	-	-	Alert	-	-	CVPU
Temperature (*C)	≤35.0	-	35.1-36.0	36.1-38.0	38.1-39.0	≥39.1	-

to CT [62]. However, it is limited by its invasive nature and the inability to identify extrinsic causes of SVCO. Contrast-enhanced CT (CECT) is used to diagnose SVCO given its high sensitivity (92%) and specificity (92%) [63]. CECT provides detailed imaging of the SVC, allowing for accurate delineation of the site and extent of obstruction. Importantly, it can distinguish intrinsic from extrinsic obstruction, thereby revealing important pathological processes contributing to SVCO. This includes findings such as mediastinal tumors, lymphadenopathy, or intraluminal filling defects (in the case of thrombosis). In patients with contrast allergies, magnetic resonance imaging (MRI) offers a non-invasive alternative approach to diagnose SVCO. Irrespective of the imaging modality used, further work-up is essential when determining the cause of SVCO, given that it is often the initial presentation of an occult malignancy. Therefore, additional investigations including needle biopsies, fluid or sputum cytology, and even bronchoscopies, may be warranted to determine the underlying cause [3, 7].

The management of MSVCO has been improving over the past few decades, transitioning from pharmaceutical and surgical management to now endovascular stenting being at the forefront. The benefits of this management cannot be denied, with rapid symptom resolution within hours in comparison to other therapies [5, 6]. However, from this review, its limitations have also been evaluated. Limitations include premature restenosis mainly due to stent migration, a reduction of primary stent patency, tumor invasion or thrombosis, and rarely SVC revascularization complications.

Additionally, the management of SVCO is also based on the severity of symptoms and aetiologies. It requires a multi-disciplinary approach where different treatment modalities are evaluated in terms of their benefit to patient care and prognosis. Initial management consists of elevation of the head of the bed as it helps decrease hydrostatic pressure in the head and neck Further management focuses on relieving immediate symptoms whether it be endovascular stenting, medication, or therapies [7, 64].

Pre-SVCO syndrome management primarily involves interventions aimed at addressing the underlying malignancy and reducing the risk of SVCO development. This is significantly impacted by the stage of the disease, histology type previous treatment, and overall prognosis [65].

Systemic therapies such as chemotherapy, targeted, or immunotherapy often chosen based on tumor histology

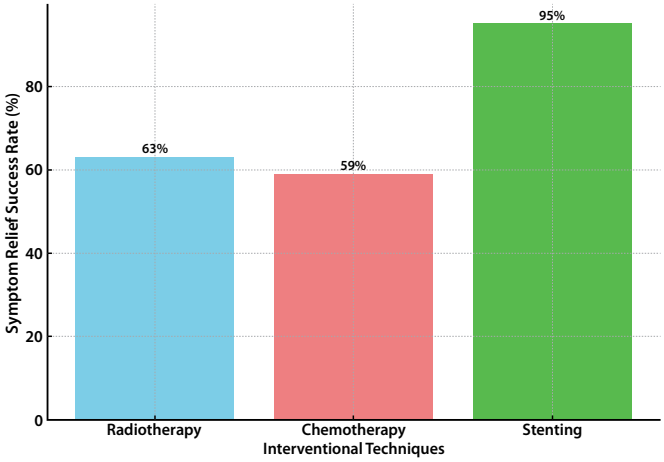

Fig. (3): The estimated success rates of interventional techniques for SVCO.

and biochemical markers, can help target malignancies. This is effective in reducing tumor size, thereby relieving or preventing compression on the SVC. In NSCLC, targeted therapies such as EGFR Inhibitors or immunotherapies like checkpoint inhibitors are crucial in the indirect prevention of SVCO-related symptoms [15]. Chemotherapies were found to relieve symptoms in 59% of patients and radiotherapy in 63% of patients **(Fig. 3)** [7]. Similarly, protocols like R-CHOP for diffuse large B-cell lymphomas have helped manipulate malignancies and improve survival rates. Chemotherapy has been shown to provide fast relief in germ-cell tumors as they are chemosensitive. Whilst these treatments have brought many benefits, they also come with the risk of treatment-induced toxicities [15].

Radiation therapy (RT), which is usually seen in more urgent cases of symptomatic obstruction or where chemotherapy is contraindicated, has been effective in radiosensitive tumors such as SCLC and lymphomas [15]. This results in prolonged survival rates with symptomatic relief in about 80% of patients however it can take weeks to observe these improvements. 5-20% of patients who've experienced RT, only benefit temporarily due to recurrence of the syndrome whereas endovascular stenting has been found to provide more immediate relief of symptoms [40]. Combinations of radiotherapy & immunotherapy have not resulted in greater relief of SVCO **(Table 6)** [21].

There is a noticeable debate regarding the optimal management post-stenting regarding anticoagulation and antiplatelet treatment. The location of the stent in the venous vasculature may suggest that anticoagulants should be favored, whilst the thrombotic risk posed by the foreign material suggests antiplatelets should be favored [66]. As stated by Azizi *et al.* [7], common practice is to start heparin during the procedure

Table 6: A comparison between different interventions and their impact on survival rates, symptoms and complications.

Intervention	Survival	Symptomatic Improvement	Complications
Chemotherapy	Prolonged (tumour control)	Indirect (prevents SVCO)	Systemic toxicities
Radiation therapy	Moderate (radiosensitive tumors)	Symptom relief in weeks	Radiation-related tissue damage
Anticoagulation	Limited to thrombotic causes	Effective in thrombotic SVCO	Bleeding risks
Steroids	Minimal	Temporary (inflammatory oedema)	Long-term steroid related side effects
Prophylactic stenting	Limited	High (if obstruction develops)	Risk of thrombosis and stent dysfunction

itself and then after endovascular treatment to reach therapeutic activated clotting times. This is before the use of subsequent anticoagulation/antithrombotic therapy and has been described by many studies [67-70]. After this stage, however, guidance regarding the use of antiplatelet or anticoagulation therapy, or none at all, is of much debate.

A review of 164 subjects with malignant SVCS treated with endovascular stenting found no statistical significance in the recurrence rates or complications experienced between those treated with heparin, aspirin, or coumarin derivatives, which included patients with thrombosis. They concluded that the type of antithrombotic given long-term does not influence the recurrence of SVCS [20]. Lanciego *et al.* [21] found that there was no significant difference in survival between those who had antiplatelet therapy and those who had anticoagulant therapy with coumarin derivatives after stent placement in a cohort of 208 subjects.

Other studies have found that patients who receive anticoagulation post-stent placement have no significantly reduced risk of thrombosis and long-term stent patency compared to those who do not receive any treatment whatsoever. Shah *et al.* [71] stated that long-term stent patency rates did not differ significantly between a study conducted by Rosch *et al.* [72] who used long-term anticoagulation and Irving *et al.* [73] who did not post stent insertion. Ratzon *et al.* [74] found that thrombosis rates were similar in patients who received anticoagulation in the form of heparin or heparin and warfarin for a median of 68 days and those who did not receive anticoagulation, however, acknowledged that patients who required anticoagulant therapy were more likely to have higher grade occlusions to begin with, which could have confounded these results.

Many studies offer different insights into the use of anticoagulants or antiplatelets post stent insertion resulting in a lack of definitive guidelines or practice. Aspirin however seems to be the preferred option post-initial heparin treatment, given the reduced risk of bleeding [66]. Thony *et al.* [68] found no incidence of thrombosis in long-term follow-up in twenty-four patients treated with aspirin post-initial heparin therapy, hence also suggested this to be adequate. They stated that coumarin was only used in preference to aspirin in a patient with a concomitant DVT. In keeping with this, Marcy *et al.* [67] stated that they generally use aspirin for uncomplicated procedures, and reserve warfarin treatment for those with residual venous stenosis post-procedure or those who need thrombolytic therapy. It therefore seems in keeping with much of the literature that after initial heparin treatment during and immediately post-endovascular treatment, aspirin is preferred in those with uncomplicated procedures given the reduced bleeding risk [66]. Further study needs to be conducted in this area nevertheless, given the heterogeneity of results present in the literature.

Steroids such as dexamethasone or prednisolone are administered in high-risk cases to reduce peritumoral inflammation and edema. Anecdotal evidence supports steroids for short-term relief in rapidly progressing SVCO syndromes [15] The impact on survival is minimal however there is short-term relief from airway compression. Its minimal benefit on survival outcomes is weighed against the high risk of immunosuppression and long-term side effects that come with using steroids. Its efficacy in improving outcomes overall in SVCO remains uncertain due to the lack of data.

Endovascular stenting in recent times has been utilized mainly as the first-line management for treating MSVCO. Most studies report that stenting should be utilized as its sole first-line management with Wei *et al.* slightly differing [7, 21, 46, 75, 76]. They instead suggest a combination of stenting and anti-vascular targeted medication [76]. The indications according to the Cardiovascular and Interventional Radiological Society of Europe (CIRSE) guidelines for SVC stenting include symptomatic malignant or benign SVCO - either pre or post-failed radiotherapy/ chemotherapy [77].

There is limited research regarding the optimal timing of stent insertion in the management of MSVCO. One small cohort study conducted by Guerrero-Macías *et al.* demonstrates a relationship between acute

onset of symptoms and mortality. It was concluded that the earlier successful endovascular stenting occurred, the better the clinical result [78]. Due to the absence of sufficient data or research studies, we recommend further studies for the investigation of the exact time of agent insertion in terms of optimization of mortality outcomes.

Most studies, instead explored the relapse of stents, with few exploring the post-stenting management and evaluating clinical outcomes. Regarding stent type, covered stents have been shown in multiple studies to have increased long-term patency in comparison to uncovered stents in both benign and MSVCO [18, 21, 79-81]. However, Liu *et al.* expressed concern over covered stenting having higher rates of migration, increased cost, and potential to block collateral vessels [31]. Interestingly, there are high rates of covered stent migration in gastrointestinal and ureteric stenting, especially oesophageal stricture stenting. Thomas *et al.* displayed a total of 23% fully covered self-expandable stent migration in malignant oesophageal strictures [82]. Notably, there is a lack of adequate data or research studies that investigate the rate of covered stent migration in MSVCO management.

Other stent types refer to balloon expandable and self-expandable stents [7]. Stents typically used for MSVCO that have been explored in the literature include the Wallstent Endoprosthesis (a self-expandable stent), Palmaz Stent, and the Gianturco Z stent (balloon expanding stent) [7]. Out of the three, the Wallstent is most likely to migrate if not placed precisely on the stenosis [83]. Hammer *et al.* suggest to reduce overall stent migration, in venous stenting, an oversized stent - by 2-4mm should be utilized to ensure stent-to-wall contact [84].

Currently, there are no absolute contraindications for SVC endovascular stenting however the relative contraindications for stenting still stand [77]. Contraindications include manageable bleeding disorders, being unable to lie in position for treatment, or infections such as severe sepsis [77]. In addition, a theorized contraindication may include the breech of the obstructing tumor into the superior vena cava [9].

Endovascular stenting has demonstrated high success rates, effective symptomatic palliation, and low complication rates [23, 76, 77]. It offers rapid symptomatic improvement within hours for MSVCO, outperforming other therapies [23, 85]. Symptoms such as neck, facial, and laryngeal edema, as well as dyspnoea, are significantly alleviated [7, 60, 61]. Unlike radiotherapy, stenting does not adversely affect subsequent surgical bypass or chemo-radiotherapy outcomes, enhancing its palliative benefit [7, 9, 85, 86].

Stenting provides immediate relief from venous congestion in most cancer patients, whether partial or complete [23, 69, 78, 87-89]. A meta-analysis reported symptomatic improvement in 91.7% of patients across 32 studies, with a range of 53%-100% [90]. Nicholson *et al.* demonstrated significantly improved symptom relief with stenting compared to radiotherapy (96% *vs.* 56%, $p < 0.001$) [22]. Literature reviews confirm that symptomatic improvement persists before and after cancer-targeting therapies [23].

The most recent systematic review by Chawla *et al.* reported high technical success and primary patency rates of 81.5% at 12 months and 63.2% at 24 months [60]. The malignant subgroup experienced lower rates of primary re-stenosis. However, limitations include combining benign and malignant SVCO cases over a 24-month period, which may not accurately reflect outcomes in malignant cases. Lanciego *et al.* reported primary stent patency rates of 85% at 6 months and 75% at 24 months in 149 cancer patients [21]. Notably, follow-up data is limited due to cancer-related deaths, but for surviving patients, the median survival was 10.6 months (range, 15 days-36 months) [21]. All patients maintained a good response to stenting until death.

The efficacy of pre- and post-dilatation of the SVC around stent insertion is well documented in the literature. Pre-dilatation is performed mainly to facilitate the entry of the stent into the SVC, such as if the occlusion blocks the pathway of the stent delivery system. Post-dilation meanwhile is performed to allow for the full expansion of stents, or if there seems to be residual stenosis of greater than 30% [83, 91]. Dondelinger *et al.* [92] found pre-dilatation of the vessel very useful to better visualize and analyze the stricture before stent placement. Conversely, Dyet *et al.* [93] found post-dilatation beneficial concerning allowing the stent to expand to a greater extent and more quickly, but with the increased risk of stent migration, further highlighted by Taylor *et al.* [94]. The indications of both methods are well documented, but there is yet to be a direct comparison regarding the efficacy of these procedures. Given the inherent risk of stent migration resulting from dilatation, Lingegowda *et al.* [48] state that pre- and post-dilatation should not be routinely performed.

Previous meta-analysis measured the weighted re-stenosis rate to be 10.5% (95% CI 8.4-12.6) over a 24-month period (**Fig. 4**) [90]. However, rates were

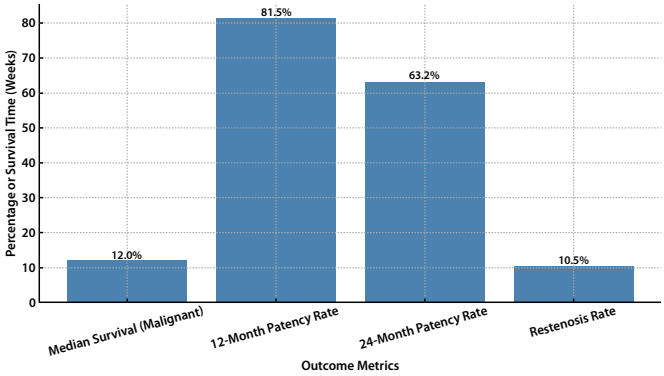

Fig. (4): The survival outcomes and stent performance post insertion of the stent.

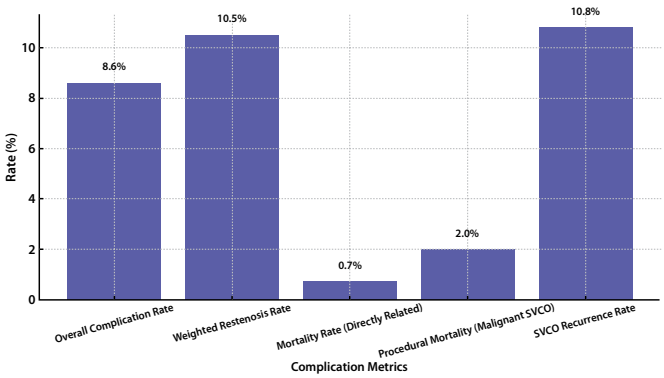

Fig. (5): The possible post-stenting complications in patients with SVCO.

quite variable ranging from 2.6-34%. Causative factors for premature re-stenosis have been majority due to stent migration particularly in cases of stent shortening or undersized stents or incorrect placement [7, 20, 21, 69, 95].

Moreover, recurrence of SVC syndrome in stented patients was found to be 10.8%, occurring at an overall mean of 12.9 months (range 2-42 months) [90]. In 41% of SVCO recurrence, it has been due to stent invasion, intraluminal thrombosis or hyperplasia, or external compression **(Fig. 5)** [79]. Specifically, in MSVCO stent occlusion most often occurs due to external compression from inward tumor growth, or due to thrombosis which was reported in 24% of MSCVO cases [7, 74].

Complications of SVC revascularization and SVCO recurrence must be carefully considered. While overall complication rates are low, with a meta-analysis reporting 8.6% (95% CI 7.3-9.9%) [90], they range from minor issues like site bleeding, hematoma, and local infection (1.1%) to severe outcomes such as thrombosis, stent migration, arrhythmia, pulmonary embolism, cardiac tamponade, and respiratory distress [7, 20, 69, 90]. Rarely, SVC rupture may occur [42]. Some complications arise from rapid hemodynamic flow restoration, leading to increased preload and right heart failure (RHF) or pulmonary congestion [96]. Pulmonary edema and respiratory insufficiency may result, in requiring diuretics, PEEP ventilatory support, and ITU monitoring, as recommended by CIRSE 2003 guidelines [94, 96]. Pre-intervention echocardiography is advised for high-risk patients with poor cardiac reserve or valve diseases [94]. Diuretics effectively reduce preload in overloaded patients but are contraindicated in cases of intravascular depletion [12, 97-100].

Recent advancements in managing thrombotic SVCO include the use of thrombolytic agents such as Factor Xa inhibitors. These agents target the thrombotic etiology of SVCO with reduced bleeding risks compared to traditional anticoagulants. Additionally, thrombectomy devices have demonstrated efficacy in rapidly restoring SVC patency in cases where thrombolytics alone are insufficient. Although promising, these techniques require larger, controlled studies to establish their role in clinical practice [94].

Cardiac arrhythmias are a significant and potentially fatal risk of stenting, often triggered by stent migration into the right atrium [83, 100]. Patients with pre-existing arrhythmic conditions are at an even higher risk [7, 23, 83, 101]. Anand *et al.* reported a case of rapid ventricular response caused by stent migration in a patient with pre-existing atrial fibrillation [102]. Guidelines from the 2006 European Society of Cardiothoracic Surgery recommend perioperative beta-blocker use for all cardiac and thoracic surgeries unless contraindicated [103]. Studies have shown beta-blockers reduce postoperative atrial fibrillation rates from 37-50% to 12-16% in valvular and CABG surgeries [96, 104]. While their use in SVCO post-operative management is anecdotal and limited to complications [105], perioperative beta-blockers should be considered for patients with complex cardiac histories, as arrhythmias remain a major cause of death in MSVCO.

SVC rupture, caused by rapid blood flow restoration or iatrogenic injury, can result in extra-pericardial or intrapericardial extravasation, leading to catastrophic thoracic hemorrhage or cardiac tamponade, respectively [7, 42]. Despite intensive treatments like covered stents to seal rupture sites, the prognosis is often poor [106]. Although rare (0.1-0.8%), patients with recent radiation therapy face higher risks of major artery rupture and require extra surveillance [42, 107, 108]. Cardiac tamponade, which may occur independently of SVC rupture, necessitates prompt pericardiocentesis, especially in patients with necrotic tumor masses [7, 94, 109].

Table 7: A comparison of the current grading systems used to assess SVCO.

Criteria	Yale Scoring System	Kishi *et al.* [44]	Stanford Classification	Azizi *et al.* [7]
Basis	Grading based on overall severity of presentation	Quantitative assessment using multiple domains to gauge overall severity	Assessment upon the degree of obstruction through use of venography	Combines anatomical location with degree of stenosis
Use Case	Predominantly Symptom-based triage	Symptom-based Triage + threshold for endovascular intervention	Complicated cases involving airway or cerebral compromise	Intervention planning
Advantages	Rapid assessment of symptom severity	Comprehensive assessment of symptoms	Simple stratification based upon degree of occlusion	Comprehensive, objective assessment of the site and severity of occlusion. Can help guide intervention
Disadvantages	Lacks anatomical insights. Subjective interpretation of severity	Lacks anatomical insights	May not be applicable beyond emergency presentations	Does not take into consideration clinical presentation

Azizi *et al.* [7] recommend ensuring adequate facilities, equipment, and a surgical team ready for emergency interventions, including pericardiocentesis and surgery. Standardized techniques, such as maintaining wire position for potential tamponade balloon use, are crucial [7]. While CIRSE 2003 guidelines allow for balloon dilation catheters of 6-20 mm, studies caution against dilation exceeding 16 mm due to elevated risks of arrhythmias, pulmonary edema, and tamponade [12, 20, 94].

A meta-analysis of all aetiologies reported a mortality rate of 0.7% following SVCO stenting [90]. Literature reviews indicate that in MSVCO, mortality is primarily due to fatal hemorrhage (41%), arrhythmia (12%), cardiac tamponade (6%), myocardial infarction (6%), respiratory insufficiency (17%), and pulmonary embolism (6%) [23]. Procedural mortality from stent insertion in MSVCO patients is 2% [23]. However, the median survival duration for MSVCO remains 8-20 weeks, reflecting the natural progression of neoplasia [23, 69, 94, 95, 110].

Evidence for identifying and managing high-risk patients is limited. Comorbidities are likely significant contributors to stent-related deaths. Nguyen *et al.* highlighted that most MSVCO patients had underlying cardiac disease, with bronchogenic carcinoma linked to smoking and age [23]. Adverse outcomes are more common in patients with cardiopulmonary conditions such as arrhythmias, prior myocardial infarction, pulmonary edema, and respiratory insufficiency, emphasizing the need for enhanced cardiac monitoring. Sparse reporting on outcomes related to patient history and underlying disease underscores the need for better documentation, prospective studies, and post-mortems to establish guidelines for preempting complications in high-risk patients [23].

The Possibility of Adding a New Comprehensive Grading System

Table **7** displays a comparison of the current grading systems used to assess SVCO. Notably, there is no single system adopted throughout clinical practice. Those utilized within current practice are by no means perfect. Kishi *et al.* and Yu *et al.* focus on stratifying the severity of SVCO based on clinical presentation [44, 47]. They allow for a relatively rapid triage at the time of presentation and outline a threshold for intervention. However, the criteria devised by Yu *et al.* may be subjective and not entirely reproducible [47]. Moreover, both criteria do not take into regard the degree of occlusion or the presence of any collateral networks. Symptom severity is vital in determining the urgency of intervention but should not be the only consideration. The severity of SVCO is correlated with both the degree of narrowing and the rate of onset [7]. Given this, the Stanford classification and the criteria formed by Azizi *et al.* give significant weighting to the anatomical considerations to guide endovascular and/or surgical intervention. However, they fail to align this with clinical presentation [7, 50]. Moreover, the Stanford classification was established to identify patients at high risk of developing cerebral and airway compromise, thus the classification may be inappropriate for the broad spectrum of SVCO presentations [7]. Furthermore, all criteria in current use fail to account for the overall systemic burden of SVCO on the individual. This is a significant factor, given that reduced venous return in SVCO diminishes preload, causing a hemodynamic compromise by Starling's law [30]. Therefore, there is a need for a more holistic grading system for SVCO- one that integrates both the clinical presentation and anatomical findings. Only then can a more comprehensive evaluation of disease severity can be made.

The ideal grading system would be objective and make use of parameters that can assess both severity and

Table 8: The SVOSTA framework for severity assessment and treatment.

Parameter	0	1	2	3	4
Radiological Findings	No Vascular compromise Normal SVC	Mild Narrowing: <50% narrowing, no collateral vessels	Moderate-Severe: Narrowing: 50-90%, formation of collateral vessels	Near Occlusion: >90%, retrograde blood flow through collateral vessels	Complete Occlusion: 100% extensive collateral network, SVC thrombus/land/ tumour
Symptom Severity	Asymptomatic	Asymptomatic or mild facial/limb edema, mild headache, cyanosed neck veins	Moderate: Dyspnea on exertion, neck/ mediastinal ache, dizziness, visual disturbance	Severe: Dyspnea at rest, orthopnea, laryngeal edema, severe upper limb swelling	Life-threatening: Cerebral edema, confusion, seizures, respiratory distress/ airway compromise
NEWS Score	0	0-4 (Normal/Slight Risk)	5-6 (Moderate Risk)	7-8 (High risk of deterioration)	9+ (Acute deterioration, requires urgent intervention)
Therapy	Nil	Nil	Decongestive therapy: Elevation of limbs, Oxygen, Diuretics Consider Beta Blockers	Decongestive therapy + Early intervention for underlying cause	Urgent intervention: Thrombolysis, SVC stenting consider bypass surgery

disease progression. Key scoring factors would thus include radiological imaging (to assess the degree and location of obstruction +/- the presence of collaterals), the clinical severity of venous congestion, and a physiological index of disease burden. Given that the NEWS score is often used in clinical practice to predict clinical deterioration, and that it integrates aspects of hemodynamic status (including heart rate and blood pressure), it can be used as an effective marker to capture disease burden. With all these factors in mind, we propose a new grading system that integrates the essential parameters to provide a holistic assessment of SVCO **(Table 8)**.

Our proposed system aims to address the limitations that exist within current clinical practice by integrating the degree of SVC narrowing with symptom severity. Both factors play a pivotal role in guiding the urgency of intervention within SVCO. Moreover, we have incorporated the NEWS score to account for the systemic burden of SVCO on the individual. When assessing patients, the highest severity score amongst the three parameters (Radiological findings, symptom severity, and NEWS score) is used to determine treatment escalation. This multi-faceted approach provides an objective and comprehensive assessment that better predicts disease severity and guides timely intervention in those with SVCO.

This algorithm should be useful in three ways: 1) identifying candidates for endovascular intervention, 2) guiding the timing of intervention, and 3) exploring treatment options for score-dependent escalation of treatment.

We propose this algorithm should be utilized in a two-step manner. Firstly, and most vitally, the score of radiologic findings should be used to identify the candidates requiring endovascular stenting. Secondly, the timing of intervention should be dictated following score-dependent assessment. Additionally, stenting should be carried out within the time frames advised in the algorithm. This will allow for more intervention to be done in an elective environment (Score 2-3) and reduce the need for emergency stenting (Score 4).

In all patients undergoing endovascular stenting, dexamethasone and beta-blockers should be considered. As previously mentioned, dexamethasone may provide short-term alleviation of airway compression and should be particularly considered in cases with respiratory deterioration [15]. Likewise, beta-blockers should be considered in the perioperative phase due to the arrhythmia risk imposed by the Bainbridge reflex, as well as possible stent migration. This is particularly the case for those with comorbid cardiac pathologies, especially in pre-existing arrhythmias/hemodynamic compromise. However, the lack of literature on steroid and beta-blocker use may preclude their clinical relevance in all SVC patients.

Diuretics and anti-coagulation should be used on a case-by-case basis. Patients with severe symptoms (score 2+) should be initiated on diuretics as they can effectively reduce preload and respiratory symptoms in overloaded patients [12, 94, 97-99]. However, caution should be used in hypotensive or intravascular-depleted patients [97, 100]. Anticoagulation therapy has demonstrated a good effect in studies of SVCO due to thrombotic causes and has shown to be a key aspect of pre- and post-operative management in these patients [74]. In particularly unstable or critical patients with thrombotic SVCO, emergency thrombolysis or thrombectomy

is warranted before revascularisation. This aids in reducing lesion size and prevents pulmonary embolism development [12].

It is important to acknowledge the limitations of our proposed model. The NEWS score is a marker of the overall disease burden on the individual but is not specific to SVCO. Patients with SVCO may have added comorbidities or infections that skew their NEWS score to a value that may not accurately reflect the severity of SVCO alone. For example, a patient with mild SVC narrowing (<50%) and stable observations (scoring a 1 according to the SVOSTA framework) may develop an infection. Consequently, tachycardia, hypotension, tachypnoea, and a new oxygen requirement may increase their NEWS score to 9. This aligns with a score of 4 in our proposed framework, despite the NEWS score being driven by infection and not the underlying SVCO. When used appropriately, however, the NEWS score can accurately capture the hemodynamic status of SVCO on the individual, forming an important physiological index to guide treatment escalation. Indeed, clinical judgment should always be used when determining the NEWS score within our proposed framework, especially in individuals with multiple comorbidities. This ensures that the NEWS score calculated reflects the impact of SVCO alone, minimizing the influence of any confounding factors.

CONCLUSION

The mainstay of treatment for Superior Vena Cava Obstruction (SVCO) is endovascular stenting, which provides quick and efficient symptom alleviation, especially in cases of cancer. In acute presentations, it outperforms traditional therapies like chemotherapy or radiation therapy because of its low procedural fatality rates, rapid venous flow restoration, and high technical success rate. Stent thrombosis, migration, restenosis, and the requirement for standardized post-procedural treatment protocols are among the issues that still exist despite their benefits.

A multidisciplinary strategy including intensivists, cardiologists, oncologists, and interventional radiologists is essential to maximizing results. Risk reduction requires targeted perioperative techniques, such as suitable anticoagulation, hemodynamic monitoring, and customized problem-solving. To direct action and forecast prognosis, a comprehensive framework that incorporates clinical presentation, anatomical findings, and systemic disease burden is required, as indicated by the absence of a standardized grading system.

Gaps in knowledge regarding the best stent selection, post-stent care, and the long-term durability of results should be filled by future studies. By tackling these issues, we can enhance endovascular treatments' effectiveness and safety even further, giving patients greater survival and quality of life results.

LIST OF ABBREVIATIONS

ANP: Atrial Natriuretic Peptide
CABG: Coronary Artery Bypass Graft
CECT: Contrast-Enhanced Computed Tomography
DVT: Deep Vein Thrombosis
EGFR: Epidermal Growth Factor Receptor
HIF: Hypoxia-Induced Factor
ITU: Intensive Therapy Unit
MSVCO: Malignant Superior Vena Cava Obstruction
NEWS: National Early Warning Score
ROS: Reactive Oxygen Species
SVC: Superior Vena Cava
SVCO: Superior Vena Cava Obstruction
SVCS: Superior Vena Cava Syndrome
VEGF: Vascular Endothelial Growth Factor

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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