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NARRATIVE REVIEW

Surgical approach to superior vena cava syndrome associated with malignant neoplasia: A narrative review.

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Abstract

Introduction: Superior vena cava syndrome (SVCS) is a rare pathology, associated in most cases with neoplasms of malignant origin. In advanced stages represents a medical-oncological emergency that compromises the patient's life.

Purpose of the review: The objective of the study is to outline the role of the different surgical and percutaneous alternatives for the treatment of SVCS. We look for reports that include the best survival results for the other current therapies.

Recent findings: The medical literature describes treatments such as radiotherapy, chemotherapy, bypass, endovascular therapy, and vascular reconstruction to manage SVCS; however, not all respond with the same efficacy during a vital emergency. By SVCS, this product of the intrinsic and extrinsic factors of the patient. The social condition stands out among the outside elements, which becomes a challenge when carrying out integral patient management in border cities, were data that the doctor knows about the patient is limited, or the patient does not have a previous diagnosis.

Conclusions: The starting point of treating the patient with SVCS is differentiating the emergency and the stability using the severity classification table. In emergent cases there, are two alternatives: endovascular therapy and radiotherapy. The surgical approach with Bypass is contraindicated. The definitive treatment with vascular reconstruction with a prosthesis has advantages and disadvantages that must be defined individually considering the etiology of associated cancer.

Keywords:

MESH: vena cava; vena cava, superior; superior vena cava syndrome; etiology; neoplasms; therapy.

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Introduction

Superior vena cava syndrome (SVCS) corresponds to a group of clinical manifestations, such as the appearance of collateral circulation, facial and cervical edema, and slave edema. It is caused by the limitation of the venous drainage of the superior vena cava, which can be due to both extrinsic compression and intraluminal blockage of the superior vena cava. [1, 2]. This compression is associated with both neoplastic and non-neoplastic causes. Most cases respond to causes of malignant neoplastic origin [3].

Although it is a rare pathology, superior vena cava syndrome can lead to an emergency in its most advanced stages. Therefore, establishing an early clinical and radiological diagnosis is essential for adequate therapeutic management [4].

The treatment of SVCS seeks to solve the compression of the superior vena cava with endovascular therapy, bypass, and vascular reconstruction; however, in some cases associated with neoplasms, radiotherapy and chemotherapy can be used either as alternatives or concomitant with surgical management [5, 6].

The objective of the present narrative review was to make the state the question of the subject on the most relevant and updated literature.

Superior vena cava syndrome

Superior vena cava syndrome (SVCS) is a medical and oncological emergency. It is due to the obstruction of blood flow through the superior vena cava due to an external or internal process that reduces the vessel lumen. The signs and symptoms are located in blood drainage from the superior vena cava, such as the head, neck, torso, and upper limbs; the nervous system, larynx, pharynx, face, chest, and upper limbs are affected [5- 11].

Epidemiology and etiology

5-10% of patients with a malignant intrathoracic lesion develop SVCS. Due to the location of the superior vena cava on the right side, any mass in the right hemithorax could affect the cava. It is observed that between 30 and 40 years, the etiology is more frequently due to a benign cause; between 41 and 60 years, it is due to a malignant etiology; in children, it is usually secondary to congenital stenosis. SVCS affects men more frequently due to their higher rate of lung cancer, and the distribution in terms of the race depends on the susceptibility of each of them to lung cancer and lymphomas [9]. In the United States, it affects approximately 15,000 people. Before the era of antibiotics, the two most common causes of SVCS were syphilitic aortitis and bulky mediastinal adenopathy due to tuberculosis [12]. In Colombia, there is no epidemiological follow-up on SVCS (table 1).

Table 1. Etiology of superior vena cava syndrome

Etiology of superior vena cava syndrome	
Malignant (60-85%)	Benign (15-40%)
Lung cancer • Large cells (most common) • small cells	Thrombosis Mediastinal Fibrosis
lymphomas • Non-Hodgkin lymphoma ➢ L. Primary B-cell media ➢ L diffuse	vascular disease infections
Other malignant and benign tumors found in the thoracic cavity	cardiac causes Post-radiation fibrosis

Pathophysiology

Superior vena cava (SVC) drainage is a low-pressure, thin-walled system that can be affected by its anatomy, anomalous venous flow, and damage to the integrity of the vessel wall (Figure 1) [6]. This syndrome's most representative physiopathological characteristic corresponds to collateral networks formed in several weeks, mainly if the obstruction occurs above the insertion of the azygos vein. Patients may not have symptoms as long as there is a collateral venous drainage system or if there is a partial obstruction [9, 13].

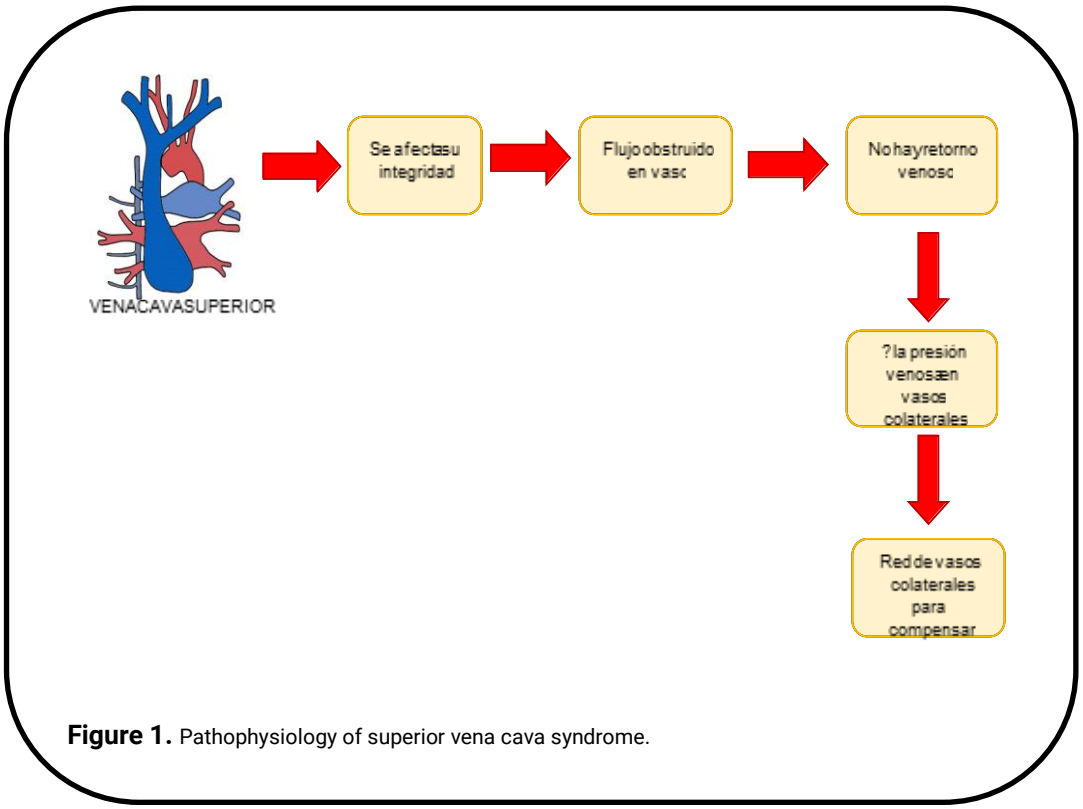

Figure 1. Pathophysiology of superior vena cava syndrome.

Signs and symptoms

A gradual onset characterizes the symptomatology; as the obstruction progresses, dyspnea appears to be one of the main symptoms. Other signs and symptoms include swelling of the face, neck, trunk, and upper extremities; hoarseness, pain, stridor, cough, dilated thoracic veins, weight loss, jugular venous distention, phrenic nerve paresis, plethora, dysphagia, cyanosis, stupor, and even coma [1, 2]. Complications of SVCS may include laryngeal edema, cerebral edema, decreased cardiac output and secondary hypotension, pulmonary embolism in the presence of a thrombus, local irritation, or thrombosis of veins in the upper extremities [9, 13]. The most frequent systems can classify the signs and symptoms as neurological, laryngopharyngeal, and facial [5].

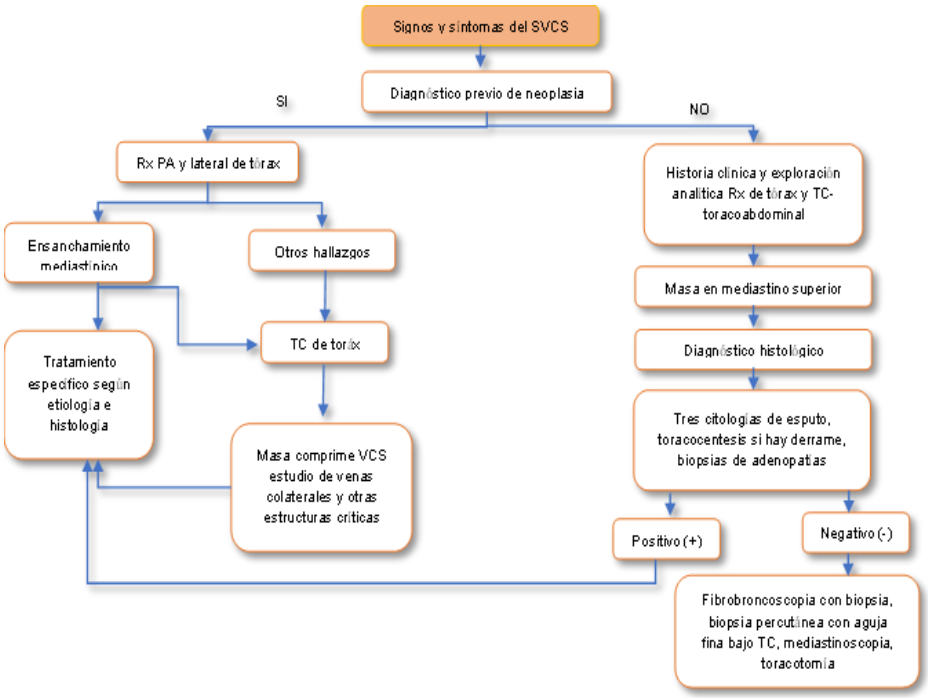

Figure 2. Diagnosis of superior vena cava syndrome Superior vena cava syndrome (AMF 2013) From a symptom [Internet]. Amf-semfyc.com. 2013.

Malignant pathologies associated with superior vena cava syndrome

Malignant neoplasms explain between 60-85% of SVCS, and SVC obstruction is the symptom that occurs in 60% of cases of a tumor not yet diagnosed. In the data collected in the study by Armstrong et al., the presence of SVCS in patients with giant cell lung cancer was 50% of cases with an age of onset above 50 years and with a history of smoking. In patients with small cell lung cancer, SVCS has a prevalence of 22% and has the same suggestive clinical features as large cell lung cancer. Lymphomas have an SVCS prevalence of 12%, patients have extrathoracic lymphadenopathy, and the age of onset is less than 65 years. The prevalence of SVCS in patients with metastatic breast cancer is 9%. In patients with germ cell cancer, it was 3%. It occurs more frequently in men under 40 years of age, who usually have high tumor markers, such as beta-human chorionic gonadotropin and alpha-fetoprotein. In patients with thymoma, the prevalence of SVCS is 2%, in which radiographic findings are evident at the base of the thymus and associated with parathymic syndrome. Patients with mesothelioma and other cancers have a prevalence of 1%; mesothelioma patients have a history of asbestos exposure [In patients with thymoma, the majority of SVCS is 2% radiographic findings are evident at the base of the thymus and associated with parathymic syndrome. Patients with mesothelioma and other cancers have a prevalence of 1%; mesothelioma patients have a history of asbestos exposure [In patients with thymoma, the majority of SVCS is 2% radiographic findings are evident at the base of the thymus and associated with parathymic syndrome. In patients with

mesothelioma and other cancers, it is 1%; mesothelioma patients have a history of asbestos exposure [14]. Both lung cancer and NHL are estimated to be responsible for approximately 95% of cases of SVCS syndrome that are caused by malignancy [15].

Diagnosis

The following classification scales are used for SVCS diagnosis: the CTCAE (Common Terminology Criteria for Adverse Effects) of the NCI National Cancer Institute and the classification proposed by Wilson and Detterbeck. (Table 2). Chest radiography may be helpful, as more than half of patients with SVCS have abnormal images; in the study by Parish et al., 84% of 86 patients with SVCS had chest radiograph abnormalities: the most common abnormalities were widening of the mediastinum and pleural effusion in 64% and 26%, respectively [9]. Regarding laboratories, complete blood counts and coagulation tests (PT, aPTT) can be ordered, and the evaluation of the increase in tumor markers to follow the evolution of neoplasms can be beneficial for those patients who have already been diagnosed with any previous neoplasia (HCG, AFP) [1, 4]. The use of diagnostic images depends on the severity of the symptoms. For patients with severe or life-threatening symptoms, catheter venography is best used. As appropriate, it provides timely diagnosis and immediate treatment to relieve SVC obstruction with intravenous recanalization and stenting [7, 9, 14]. For patients presenting with mild or moderate symptoms, the initial imaging study may be a venous duplex study or imaging such as CT or MRI (nuclear magnetic resonance imaging) [7, 9, 14]. CT is the most appropriate initial study for patients with mild to moderate clinical features of SVCS. It can define the level and extent of venous blockage, identify and map collateral venous drainage pathways, and often identify the underlying cause of venous obstruction. The presence of collateral vessels on CT is a strong indicator of CVS syndrome, with a specificity of 96% and a sensitivity of 92%, according to Bechtold et al.; Kim, Ki,m and Chung; and Eren, Karaman, and Okur. [7, 9, 14].

A histological study is a prerequisite to choosing the appropriate treatment for patients with SVC syndrome associated with malignancy. Minimally invasive examinations can be performed, such as sputum cytology and pleural fluid cytology. CT-guided biopsy of the enlarged peripheral lymph nodes (e.g., supraclavicular) may be diagnostic in up to two-thirds of cases. On the other hand, there are more invasive tests, such as bronchoscopy, mediastinoscopy, video-assisted thoracoscopy, and thoracotomy, when a definitive diagnosis cannot be established otherwise. [7, 9, 14].

CT-guided percutaneous transthoracic or endoscopic ultrasound-guided biopsies are alternatives to mediastinoscopy or thoracotomy to establish a definitive diagnosis in high-risk patients (Figure 2) [1, 4].

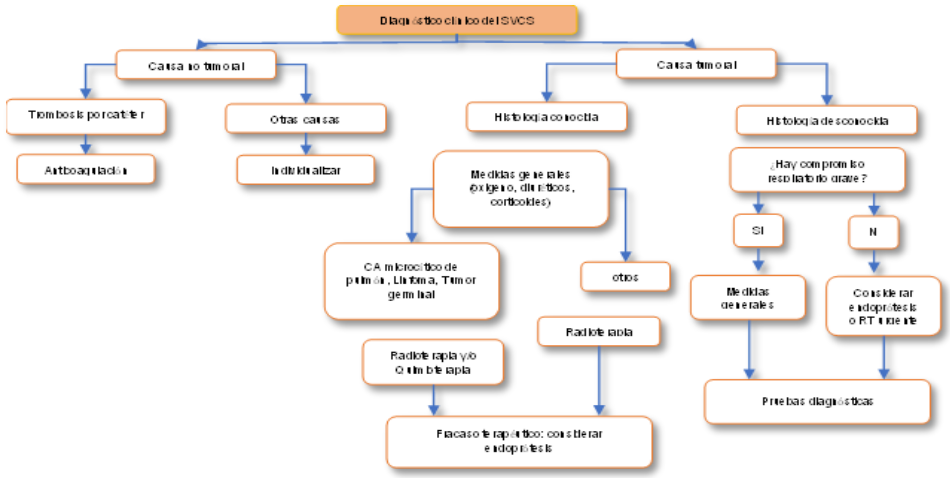

Figure 3. Superior vena cava syndrome diagnosis and treatment algorithm. Navarro Expósito F, López González J, Molina Villaverde R, Lamarca Lete A. Diagnostic and therapeutic protocol for superior vena cava syndrome. Medicine - Accredited Continuing Medical Education Program. 2013;11(24):1500-1503.

Table 2. Classification of the severity of superior vena cava syndrome (SVCS) according to the NCI CTCAE

Degree	Findings	Estimated incidence (%)
0	Asymptomatic - radiological finding of superior vena cava obstruction in the absence of symptoms	10
1	mild- head or neck edema (vascular distension), cyanosis, facial plethora	25
2	moderate - edema of the head or neck with symptoms of functional impairment (mild dysphagia, cough, mild or moderate impairment of movements of the head, jaw, eyelids, and loss of visual acuity due to ocular edema)	50
3	severe - mild or moderate cerebral edema, mild or moderate laryngeal edema, decreased cardiac reserve (syncope after squatting)	10
4	life-threatening - significant cerebral edema (obtundation, confusion), laryngeal edema with stridor, significant hemodynamic compromise (syncope without precipitating factors, hypotension, renal failure)	5
5	Death- fatal outcome	>1

Treatment

In clinical symptoms such as hypotension, cerebral edema, or laryngeal edema in a cancer patient with SVCS, radiotherapy can be used as an emergent treatment [5, 16]. Initial measures include head elevation, Fowler's position, fluid restriction, diuretics, steroid therapy, and supplemental oxygen [17, 18].

Chemotherapy and radiotherapy

Chemotherapy can be used in sensitive tumors such as SCLC, non-Hodgkin's lymphoma, and germ cell tumors [5, 19, 20]. Chemotherapy should be the first line of treatment in patients who do not have life-threatening symptoms. The efficacy of chemotherapy is greater than 65% in cases associated with chemosensitive tumors. According to the extension, polychemotherapy is used as a specific treatment and is evaluated while associated with radiotherapy. Remission of symptoms usually occurs between 5 and 10 days from the start of therapy [20, 21].

Radiotherapy

Radiotherapy is the treatment of choice for chemoresistant tumors. It reduces the tumor mass and significantly reduces symptoms in the first 72 hours, with a complete disappearance of the same after two weeks with an efficacy of 80%. A dose of 50-60 Gy is administered with different types of fractionation. Management and endovascular therapy are recommended due to the most fibrosis stenosis that can occur in the long term. Radiotherapy is a fundamental part of definitive or palliative treatment. By reducing the mass's size, the obstruction's length also decreases, and therefore, there is a relief of symptoms; however, it does not entirely resolve the block [5, 17, 21]. Although malignant neoplasms sensitive to chemotherapy and radiotherapy have high rates of resolution of symptoms, high rates of complications are also recorded; for this reason, joint management with steroids acts as a moderator in the pathophysiological processes that favor the development of these complications [5, 7]. Steroids are administered intravenously in tumors that respond to treatments such as thymoma and lymphoma [20].

Endovascular therapy

Endovascular therapy offers an alternative for symptomatic relief in a shorter time interval. However, its approach as the first line of treatment in SVCS associated with malignant neoplasms remains controversial [5, 16, 21, 22, 23].

The indication for endovascular treatment is for three groups of patients:

1) patients with severe acute symptoms, such as respiratory distress due to laryngeal edema or airway obstruction and altered mental status due to elevated intracranial pressure.
2) patients with moderate symptoms, persistent despite chemotherapy.
3) Patients in whom chemotherapy and radiation are contraindicated [5].

In the surgical context, the most commonly used stents are Palmaz (Cordis), Wallstent (Boston Scientific), and Gianturco-Z-Stent (Cook). There are no studies comparing stents. However, there is a tendency to use wall stents due to their characteristics of self-expansion and not compression by external forces [21]. Within 24 to 72 hours after stent placement in the patient, the resolution of symptoms such as facial edema and edema of the upper extremities and trunk can be observed [5]. Endovascular therapy leads to an increase in central venous return to the heart, becoming a predisposing factor in patients with heart disease to heart

failure and acute pulmonary edema, which is why peripheral hemodynamic monitoring is recommended as a prevention strategy for this complication [1, 6]. The recurrence of SVCS with stents is 20% and is caused by the progression of the disease or sometimes by the displacement of the stent; however, in 90% of the patients studied, an evident clinical improvement is observed in 24 to 48 hours [21].

Bypass

It is an invasive intervention with high morbidity and mortality rates, so it is not currently recommended [18, 24].

Vascular reconstruction

Vascular reconstruction with grafts is indicated in specific cases as an alternative for the treatment of SVCS of malignant origin resulting from pathologies such as mediastinal adenopathies or primary lung tumors, where the survival rate of these patients with this treatment at six months is reported from 40 to 54% and at 12 months from 19 to 34% [1, 7]. It has a 4.5-14% [3, 24, 25]. The most commonly used grafts in SVC reconstruction are Gore-Tex® synthetic polytetrafluoroethylene (PTFE) prostheses, custom-made heterologous pericardial prostheses (coils), and autologous pericardial grafts [2, 4]. Synthetic PTFE prostheses have a ring composition, prevent graft collapse with negative venous pressure, have a longer half-life than biological grafts, and are commercially available. Disadvantages are graft rejection, associated infections, early graft thrombosis [24, 25], and complications associated with anticoagulation required for six months after surgery.

Compared to PTFE, heterologous pericardial prostheses or coils have a lower risk of infection and thrombosis. However, this prosthesis does not exempt anticoagulants because the bovine pericardium is heterogeneous, and rejection requiring immunosuppressive therapy can still occur, which would increase the overall cost of the graft compared to an autologous pericardial graft [2, 4].

Shortly after implantation, the graft re-epithelializes with autologous epithelial cells in autologous pericardial grafts. It is associated with a low risk of infection, demand for reduced let deposition, and reduced thrombogenicity at the flow surface. The risk of antigenicity is wholly eliminated, and they do not require long-term anticoagulant therapy. However, its disadvantages include an unpopular method and the duration of the double surgical intervention to obtain the pericardium (Figure 3) [24, 26].

Important points

Patients with NSCLC and mediastinal lymph node involvement usually have a poor overall prognosis, and surgery should generally be avoided in these patients [2, 5].

Conclusions

The starting point of treating patients with SVCS is differentiating emergency conditions and stability using classification and severity tables. In emerging cases, there are two alternatives: endovascular therapy and radiotherapy: The surgical approach with bypass is contraindicated. Definitive treatment with vascular reconstruction with a prosthesis has advantages and disadvantages that must be defined individually, considering the etiology of associated cancer.

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Abbreviations

AFP: alpha-fetoprotein.
aPTT: activated partial thromboplastin time.
HCG: human chorionic gonadotropin.
NHL: non-Hodgkin's lymphoma.
NSCLC: non-small-cell lung cancer.
PT: prothrombin time.
SCLC: Small cell lung cancer.
SVC: superior vena cava.
SVCS: Superior vena cava syndrome.

Administrative information

Additional Files

The authors declare none.

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Author contributions

Marcel Quintero: conceptualization, methodology, resources, writing (review and editing), supervision, project management.

María Isabel García Gelvez: Conceptualization, methodology, research, resources, writing (original draft), writing (revision and edition), project administration.

Manuel Latorre Quintana: conceptualization, methodology, research, resources, writing (revision and edition), visualization, supervision, project administration.

All authors read and approved the final version of the manuscript.

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Ethics committee approval

Does not apply to narrative reviews.

Consent to publication

This does not apply to studies that do not publish explicit images such as CT scans, MRIs, and physical exam images.

Conflicts of interest

The authors declare that they have no conflict of interest or competence.

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