DIFFERENT SURGICAL MODALITIES IN TREATMENT OF SUPERIOR VENA CAVA SYNDROME

An Essay

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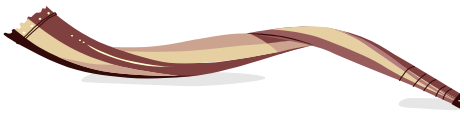

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Dedication

Words can not express my thanks, gratefulness, respect and love to my great **parents, my Brothers, Sisters, my friends.** Without their help, support, patience and encouragement, I would have never achieved any success.

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List of Abbreviations

Abbreviation	Meaning
CPB	Cardiopulmonary Bypass
CT	Computed Tomography
DVT	Deep Venous Thrombosis
ECG	Electrocardiography
ECMO	Extra-Corporeal Membrane Oxygenation
EVR	Endovascular Repair
INR	International Normalized Ratio
IVC	Inferior Vena Cava
MPR	Multi-planar Reconstruction
MRA	Magnetic Resonance Angiography
MRI	Magnetic Resonance Imaging
NHL	Hodgkin lymphoma
OSR	Open Surgical Reconstruction
PET	Positron Emission Tomography
PTFE	Polytetrafluoroethylene
SFV	Superficial Femoral Vein
SV	Saphenous Vein
SVC	Superior Vena Cava
SVCS	Superior Vena Cava Syndrome
TEE	Transesophageal Echocardiography

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1. INTRODUCTION

The superior vena cava (SVC) syndrome, which was first described by William Hunter in 1757.

SVC syndrome encompasses a constellation of symptoms and signs resulting from obstruction of the superior vena cava, which is a thin-walled vessel carrying blood at low pressure (2 – 6 mmHg). The increased venous pressure in the upper body results in edema of the head, neck, and arms, often with cyanosis, plethora, and distended subcutaneous vessels. Edema may cause functional compromise of the larynx or pharynx, manifested as cough, hoarseness, dyspnea, stridor, and dysphagia. Cerebral edema may lead to headache, confusion, and coma. [15]

The seriousness of the SVC syndrome depends on its onset rapidity and on the duration of the SVC obstruction, as well as on the related possibility of the development of collateral circulation. Moreover, it depends on the location of obstruction. If there is a stenosis or obstruction above the azygos vein ostium, the collateral flow is ensured via this vein contrary to the obstruction below the azygos vein ostium. [1]

SVC syndrome may be caused either by an external SVC compression, or possibly by a stenosis related thrombosis or a direct infiltration of a tumour into the vessel. [17]

The causes of SVC obstruction have changed during the years. Nowadays more than 90% of SVC syndromes are triggered by an advanced malignant disease, most frequently by bronchogenic carcinoma, accounts for more than 80% of cases, and particularly its small-cell type, by non-Hodgkin lymphoma (NHL), accounts for 5% to 15% of cases, and by metastatic mediastinal tumours. Thyroid carcinomas and thymomas are rarer causes[15,4]

Currently, benign aetiology is a cause of SVC syndrome, occurring in approximately 15-20 % of all cases.[1] Fibrosing mediastinitis is one of the important infiltrative benign etiologies causing SVC obstruction, accounts for 60% to 70% of benign causes. Other important non-infiltrative causes of SVC obstruction are substernal goitre, Riedel's thyroiditis, aortic aneurysm and thymoma.

Of the other non-malignant causes of SVC syndrome, thrombosis from central venous instrumentation (catheter, pacemaker, guidewire) is an increasingly common event, especially as these procedures become more common. [15,2]

The age range of the SVC syndrome is 18-76 years, with a mean age of 54 years.[7] SVC syndrome is rare in children and appears at presentation in 12% of paediatric patients with malignant mediastinal tumours. [6]

If the syndrome is fully expressed, the diagnosis is already evident from the medical history and physical examination. Location, degree, and the cause of the SVC obstruction should be characterized in every case. [2]

A quantity of non-invasive and invasive examination methods can be used in diagnosis of SVC syndrome. An X-ray of the heart and lungs may reveal a widened mediastinum, pleural exudate and mediastinal or hilar tumour, particularly on the right side. Sometimes dilatation of the azygos vein can be apparent. [6]

Contrast phlebography remains the gold standard in diagnostics. It makes it possible to display the level and extent of the obstruction and of the collateral circulation, as well as potential presence of thrombi. [1]

Computed tomography (CT) and nuclear magnetic resonance imaging (MRI) can display in detail anatomical structures, the cause and extent of obstruction and of the collateral circulation. However, MRI is contraindicated in patients with an implanted pacemaker. [12]

Positron emission tomography (PET) is sometimes useful.[2] Digital contrast angiography, venous phase, and two-dimensional echocardiography are also helpful in assessing collateral circulation and in evaluating clot formation on

central venous catheters and other foreign devices, respectively. [2]

Other diagnostic methods, including cytology, isotope venography and lymph node biopsy are indicated for individual cases. Bronchoscopy has a diagnostic yield of 50 to 70% and transthoracic needle-aspiration biopsy has a yield of approximately 75%, whereas mediastinoscopy or mediastinotomy has a diagnostic yield of more than 90%. [20,13]

Management of SVC syndrome depends on the cause of the obstruction, the gravity of symptoms and the patient's prognosis. It includes both operative and non-operative strategies. [12]

Non-operative strategies include pharmacotherapy (usage of diuretics, corticosteroids and anticoagulant therapy), radiotherapy, chemotherapy, transluminal balloon angioplasty, stent implantation and local thrombolysis. [2]

If the aetiology of the SVC syndrome is malignancy, radiotherapy, chemotherapy or a combination of the two - depending on the histological type of the tumour - form the basis of treatment. [14]

Percutaneous placement of an intravascular stent and transluminal balloon angioplasty to bypass the obstruction of the superior vena cava are other possible interventions,

especially for benign causes of SVC syndrome. But, angioplasty is generally performed only in preparation for stent placement because of a lack of durable benefit from angioplasty alone.

Drawbacks of stent placement have been reported including infection, pulmonary embolus, stent migration, hematoma at the insertion site, bleeding, and, very rarely, perforation and concomitant use of thrombolytic therapy.[15, 16,17]

Operative interventions in SVC syndrome include bypass operations and direct operations on SVC. Until recently a bypass operation, using venous conduits, was the only alternative treatment in the event of failure of conservative therapy with a very good and long patency (88 % of patient bypasses), and it still remains an alternative if there is failure of endovascular treatment or if a radical resection of a tumour can be effected. [12]

Autologous venous conduits include spiral saphenous vein, femoral vein, straight saphenous vein and composite autologous vein grafts. [2]

In spiral saphenous vein graft, the saphenous vein is mobilized, opened longitudinally, wrapped in spiral fashion around a temporary tubular stent such as a chest tube, and then running a monofilament suture to construct the tube graft. The

technique is widely versatile and has been used to replace a number of venous passages of varying sizes including the internal jugular vein.[18]

Spiral vein grafting for benign SVC obstruction demonstrates excellent long-term results with nearly 90% of patients experiencing total resolution of symptoms relating to the SVC syndrome. In addition to benign disease, spiral vein grafting may well be useful for malignant causes of SVC obstruction and in other situations where there is the need for a large calibre vein graft.[19]

Unmodified intact saphenous vein grafts are too small to relieve venous obstruction unless two or more grafts are constructed from veins above the caval obstruction to the right atrium. Also, the real problem with femoral bypass graft is their fixed diameter, which may be too small to relieve obstruction. [2]

In unusual situations, alternative venous conduits such as azygos vein-inferior vena cava (IVC) or jugular vein-femoral vein grafts can be used. In the absence of availability of autologous venous tissues, aortic allograft, venous allograft and pericardial tube construction can serve as conduit. Prosthetic graft materials are generally inferior to autologous tissue grafts. Aortic and venous allografts have long-term