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Caval Thrombus Management: The Data, Where We Are, and How It Is Done

Brittany Harrison, MD, Frank Hao, MD, Nii Koney, MD, MBA, Justin McWilliams, MD, FSIR, and John M. Moriarty, MD, FSIR

Thromboses of the superior and inferior vena cava, either isolated or associated with distal deep venous thrombosis, are uncommon, but confer potentially serious morbidity and mortality. Incidence is increasing, especially with the prominence of intravascular devices. The range of treatment options is also expanding to include medical management, surgery, and endovascular techniques which are now frequently considered first line therapy due to lower reintervention rates and decreased periprocedural morbidity. Currently, there are no official guidelines for screening or treatment. This article reviews the etiology, diagnosis, and management of caval thromboses, including equipment, procedural steps, outcomes, and complications, particularly with regard to endovascular techniques, such as catheter-directed thrombolysis, pharmacomechanical catheter-directed thrombolysis, angioplasty, and stenting.
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Introduction

Thromboses of the superior vena cava (SVC) and inferior vena cava (IVC) are within the spectrum of proximal deep venous thrombosis (DVT). Isolated caval thrombosis is reportedly rare and may be clinically silent, but carries a risk of serious morbidity and mortality associated with pain, swelling, venous hypertension, infection, pulmonary embolism (PE), loss of vascular access, and postthrombotic syndrome (PTS). Using data from the *United States National Hospital Discharge Survey*, Stein et al[1] reported that the incidence of vena cava thrombosis (VCT) (either SVC or IVC) was 1.7 per 100,000 from 2001-2005 which increased with patient age and was frequently associated with malignancy. PE occurred in 12% of those with isolated VCT.

The pathophysiology of thrombus formation is multifactorial. Events may be classified as primary or secondary and the latter is often discussed in the setting of foreign bodies or malignancies. Distinguishing the etiology may affect expected outcomes and management.[2] Of note, vena cava filters are a major cause of acquired thrombosis, but will be discussed in detail elsewhere in this edition.

Currently, there are no official guidelines for screening or management of SVC or IVC thrombosis, and data are often extrapolated from more general venous thromboembolism (VTE) literature. It is often diagnosed incidentally on imaging such as when patients with suspicion of lower extremity DVT undergo imaging of the IVC to plan for filter placement. Management strategies are evolving, with the current range of options including anticoagulation, graduated elastic compression stockings, endovascular techniques including catheter-directed thrombolysis (CDT) and mechanical thrombectomy, and surgery. This review article will summarize the relevant anatomy, etiology, diagnosis, and treatment of caval thromboses as related to modern practice.

Anatomy

Congenital anomalies are frequent and are usually due to irregularity of normal embryologic processes. Thus, there is usually time for collateralization, and patients may, consequently, be asymptomatic. Nonetheless, identifying the anomaly influences various aspects of patient management including venous interventional planning.

Department of Interventional Radiology, University of California, Los Angeles, CA.

Address reprint requests to Brittany Harrison, MD, Department of Interventional Radiology, University of California, 757 Westwood Plaza, Los Angeles, CA 90095. E-mail: blharrison@mednet.ucla.edu

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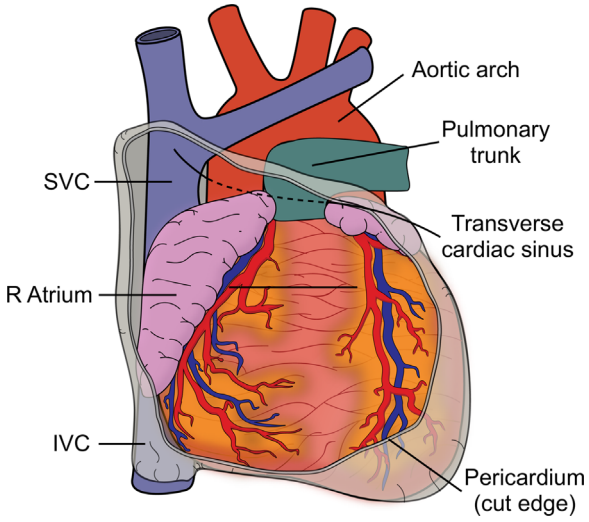

Figure 1 Relationship of the SVC to the Pericardium. *Schematic diagram by Anthony N. Hage, BS.* (Color version of figure is available online.)

SVC: Normal Anatomy and Clinical Correlations

The SVC delivers deoxygenated blood from the upper third of the body to the right atrium and forms at the junction of the bilateral brachiocephalic veins, then courses along the right mediastinum to the right of the trachea and ascending aorta and drains into the right atrium. The distal SVC is surrounded by fibrous pericardium, of great importance, if there is rupture distal to this point (Fig. 1). Although various thoracic veins empty into the SVC, the azygous vein is the main tributary, draining posteriorly into the distal SVC. If obstruction is distal to azygous insertion, then compensation occurs through retrograde azygous flow. If obstruction is proximal to the azygous, then flow must bypass the SVC and return via the smaller caliber internal mammary, superficial thoracoabdominal, or vertebral venous systems to the IVC, resulting in high venous pressures.[3]

SVC: Congenital Variations

Anomalies of the SVC are more common in patients with congenital heart disease, although they are overall less common than IVC variants.[4]

Persistent Left SVC

The most common aberration of the central thoracic veins occurs in up to 10% in those with congenital heart disease (0.5%-2% prevalence in the general population).[5] The SVC arises from the left subclavian and jugular veins, to the left of the aortic arch, then traverses the left heart with the Ligament of Marshall, connecting to right atrium via a dilated coronary sinus. Often, it is an incidental finding due to either an abnormal course of a central venous catheter (CVC) or a dilated coronary sinus seen on imaging. These patients may still have a normal or small right SVC. About 10% of cases drain directly into the left atrium and may cause a right-to-left shunt, increasing the risk of paradoxical emboli.[6,7]

Right Upper Lobe Partial Anomalous Pulmonary Venous Return (Papvr)

This anomaly occurs when the right upper lobe pulmonary vein does not drain into the left atrium, but rather into the SVC and right atrium. Prevalence has been reported at 0.5%-0.7% of the general population. It is associated with sinus venosus atrial septal defects which provide an interatrial communication due a deficiency of the common wall between the SVC and right-sided pulmonary veins.[8]

IVC: Normal Anatomy and Clinical Correlations

Conventional IVC anatomy consists of a single, right-sided venous structure formed by the confluence of the common iliac veins, coursing cranially to drain into the right atrium. There are 4 anatomical segments in the mature IVC: hepatic, suprarenal, renal, and infrarenal. The superior margin is marked by the Eustachian valve (fetal remnant forming a ridge at the inferior cavoatrial junction), which may regress to a variable degree and mimic a thrombus.

IVC: Congenital Variations

IVC anomalies are more common in males and usually present in the 3rd or 4th decade of life. The anatomical variants may be organized by the pertaining IVC segment and are pictorially summarized in Figure 2.

Hepatic IVC

Membranes or Webs

These obstructions are due to either a congenital vascular malformation or sequelae of an organized thrombus of the hepatic IVC. They often occur at the level of the diaphragm, consisting of a complete or fenestrated fibrous occlusion (also known as 'membranous obstruction of the IVC' (MOVC)). They may cause hepatic outflow obstruction and are a common cause of Budd-Chiari syndrome in Asian and African populations.[9,10]

Suprarenal IVC Congeital Variants

Absence of the Suprarenal IVC

The IVC drains either into the azygous or hemiazygous. There is no intrahepatic IVC, so the hepatic veins drain