STANDARDS OF PRACTICE

Society of Interventional Radiology Reporting Standards for Thoracic Central Vein Obstruction

Endorsed by the American Society of Diagnostic and Interventional Nephrology (ASDIN), British Society of Interventional Radiology (BSIR), Canadian Interventional Radiology Association (CIRA), Heart Rhythm Society (HRS), Indian Society of Vascular and Interventional Radiology (ISVIR), Vascular Access Society of the Americas (VASA), and Vascular Access Society of Britain and Ireland (VASBI)

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ABBREVIATIONS

AV = arteriovenous, BCV = brachiocephalic vein, CDE = common data element, CVWG = Central Vein Work Group, IJV = internal jugular vein, IVC = inferior vena cava, SCV = subclavian vein, SVC = superior vena cava, TCVO = thoracic central vein obstruction

PREAMBLE

This effort to produce a thoracic central vein obstruction (TCVO) reporting document arose from informal discussions at meetings of several professional societies. These societies share a common interest in promoting responsible and sustainable vascular access through education, research, and clinical practice initiatives. TCVO is not only a major clinical problem that impacts vascular access but is also encountered in patients with end-stage renal disease, cancer, infection, parenteral nutrition requirements, cardiac dysrhythmias, and many other conditions. It was recognized that a multidisciplinary coalition would be required to develop a useful comprehensive reporting system acceptable to all major stakeholders in this domain.

Participants and Methods

This multidisciplinary coalition, called the Central Vein Work Group (CVWG), consists of clinicians and basic scientists from academic and private practices. The clinical specialties of interventional radiology, nephrology, vascular surgery, transplant surgery, pediatrics, hematology/oncology, cardiology, and clinical anatomy were represented. This document was drafted during a series of Web-based meetings and conference calls. After an organizational outline was established, individual topics were discussed until resolved. Whenever possible, statements contained in this document are based on peer-reviewed literature. All included references are relevant to current practice and free from relationships with industry or other sources of bias.

Scope of Document

The scope of these reporting standards is intentionally narrow. It is not the purpose of this document to catalog the spectrum of central venous obstruction in exhaustive fashion. Nor is this document intended to serve as a tool for determining the presence of TCVO. Rather, it identifies the

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Tables E1 and E2 and Appendices A–C are available online at *www.jvir.org*.

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manifestations of TCVO and seeks to reduce these diverse manifestations to 4 anatomic patterns of obstruction and to define 5 key reporting dimensions that allow clinicians to describe TCVO in a straightforward and reproducible manner.

Development of These Reporting Standards

All CVWG members are listed in **Table E1** (available online at *www.jvir.org*). No CVWG members received compensation or commercial support for their time and effort. Some members chose to participate in the process of writing, whereas others did not. Those who contributed to writing of this document are listed as authors. The Society of Interventional Radiology voluntarily contributed time and personnel to review, edit, and approve this manuscript without payment or commercial support.

INTRODUCTION

TCVO often interferes with vascular access and may cause disability of varying degree and, on occasion, death. One of the first reports of TCVO was made by Hunter (1), who described a patient with superior vena cava (SVC) obstruction caused by a syphilitic thoracic aortic aneurysm. Although clinically evident TCVO has long been recognized, its incidence, prevalence, and the disability that it causes remain unknown. Without consistent use of reporting standards, a unified perspective of TCVO will never be possible.

Although thoracic aneurysms rarely cause TCVO today, there are a multitude of other conditions that do. Thoracic malignancy, particularly lung cancer and lymphoma, and other neoplastic, infectious, and inflammatory mediastinal processes may obstruct the thoracic central veins (2–5). Paget–Schroetter syndrome and subclavian venous thrombosis may cause TCVO (6–12). TCVO is frequently associated with the use of indwelling venous devices such as infusion ports, peripherally inserted central catheters, and transvenous cardiac rhythm device leads (13–20). Chronic central venous catheters in children and adults have been associated with TCVO (21–26). Patients receiving hemodialysis who have had previous venous catheter access or cardiac rhythm device leads are known to have a high prevalence of symptomatic TCVO (27–38). Some cases of TCVO cannot be attributed to any particular cause (39).

The CVWG recognized that it is challenging, and often impossible, to compare TCVO reports because of inconsistency. For example, there are different ways to describe outcomes after TCVO treatment, including venographic patency (40–44), clinical findings (45–49), or both (50–52). In addition, it is often difficult to tell which method was used because most work is retrospective, without consistent definitions or endpoints. Additionally, anatomy and terminology of TCVO vary. The axillary vein is sometimes considered a thoracic central vein (45), and the brachiocephalic vein (BCV) has been incorrectly termed the innominate vein (45,53). Therefore, the foremost purpose of these reporting standards is to provide a simple, consistent, and useful way for clinicians and researchers to describe TCVO across all disciplines and for every patient. Use of these TCVO reporting standards will facilitate future analysis of incidence, prevalence, and outcomes for patients with TCVO, whether treated or not.

Finally, the CVWG recognizes the need to develop a comprehensive strategy for thoracic central venous access. Progressive loss of central vein patency may be related to 1 or more central venous access events. Only with standard terminology and a systematic approach for reporting TCVO can practice patterns be developed to assure preservation of thoracic central veins.

THORACIC CENTRAL VEIN ANATOMY

The Normal Thoracic Central Veins

Thoracic veins can be categorized as central (systemic veins), somatic (azygos/hemiazygos, superficial, body wall veins), or visceral (pulmonary veins, coronary sinus). This document will discuss obstruction of the thoracic central veins, which can be broadly considered a continuation of the deep veins of the head, neck, and upper extremities. However, before addressing the thoracic central veins, it is worth noting that somatic veins (including the azygos/hemiazygos system and the superficial, paraspinal, epidural, and body wall veins) often provide collateral circulation as TCVO develops. These collateral pathways play a role in mitigating the clinical effects of TCVO. Although collateral venous pathways are important, it is beyond the scope of these reporting standards to discuss TCVO collateral pathways in detail.

The thoracic central veins (**Fig 1**) include intrathoracic segments of the internal jugular veins (IJVs), subclavian veins (SCVs), BCVs, SVC, and the suprahepatic portion of the inferior vena cava (IVC). These veins are located central to the superior thoracic aperture (C7–T1 intervertebral disc level), central to the lateral margin of the first rib margin, and superior to the diaphragmatic caval opening (54). Common data elements (CDEs) for the normal thoracic central veins are described in **Table E2** (available online at *www.jvir.org*; 55).

The SVC serves as the final pathway for thoracic central venous return to the right atrium. The azygos vein drains into the SVC between the confluence of the BCVs and the right atrium and serves as an important collateral pathway in the setting of many cases of TCVO. Obstruction of the central SVC (ie, between the azygos vein and heart) prevents antegrade azygous venous drainage to the right atrium and therefore defines the most central type of TCVO (see *TCVO Patterns of Obstruction*).

The suprahepatic IVC is a thoracic central vein because it lies above the diaphragm (and is therefore in the thorax). However, obstruction of this venous segment has a markedly different clinical presentation than obstruction of the thoracic central veins above the right atrium. It will not be further considered in these reporting standards.

Anomalies of the thoracic central veins have been described. The most common, found in 0.3% of people, is persistence of the left SVC, typically seen along with a right-sided SVC (56). The left SVC is almost always an incidental finding, and carries venous flow from the left BCV to the coronary sinus. Given its infrequent occurrence, the role of a left-sided SVC in TCVO remains unknown. Other thoracic central vein anomalies are much less common and therefore not mentioned in these reporting standards.

TCVO

Overview

Venous obstruction is defined as a pathophysiologic venous luminal narrowing that impedes blood flow. Obstruction may be partial (ie, stenosis) or complete (ie, occlusion). In this reporting document, obstructions are considered to be central (ie, closer to the right atrium; BCVs and SVC) or peripheral (ie, further from the right atrium, eg, IJV and SCV obstructions).

Mechanisms of Obstruction

Although many conditions cause TCVO, there are 3 predominant mechanisms of obstruction. Extrinsic compression is caused by arterial compression, musculoskeletal compression, postoperative scarring, fibrosis, or compression as a result of tumor. Venous wall thickening may be caused by de novo smooth muscle hyperplasia, organized mural thrombus, or fibrosis or secondary to stent, stent graft, catheter, or implanted cardiac rhythm device leads. Tumor infiltration, infection, inflammation, intramural dissection, or hematoma can cause wall thickening. Endoluminal obstruction is commonly caused by thrombus, but may be caused by endoluminal device implants such as stents or stent grafts, catheters, or cardiac rhythm device leads that occupy luminal space or by secondary formation of adherent tissue (ie, "fibrin") sheath or neointimal tissue. Rarely, it is the result of tumor (eg, angiosarcoma) or congenital or acquired webs or membranes.

TCVO Patterns of Obstruction

The CVWG considered different schema for categorizing TCVO patterns. The simplest and most useful, proposed in the subsequent section, has only 4 types of obstructing patterns determined by location of obstructed veins and the remaining patent segments (**Fig 2**). Within each of the 4 types of obstructions, there are diagrammatic examples. These examples represent some of the more obvious patterns that may be seen in any of the 4 types of obstruction and are not intended to be comprehensive.