Validation of a Novel Superior Vena Cava Syndrome Identification, Classification, and Management Algorithm

David Hampton MD, Christina Gamboa BS, John Zatarain MD, Brian Digs PhD, Charles R. Thomas, Jr. MD
Depts. of Radiation Medicine (Knight Cancer Institute) & Surgery, Oregon Health & Science University, Portland, Oregon, USA

Introduction

Superior vena cava (SVC) syndrome results from compromised venous return due to extrinsic compression of the great vessels, obstruction from an intramural thrombus and/or secondary to an indwelling catheter. SVC can escalate into a life threatening condition. The Yale classification system and management algorithm is based upon patient (pt) presentation and tumor characteristics and was devised as a starting point towards evidence based multi-disciplinary treatment, and to allow comparison of results from different institutions.

Hypothesis

We hypothesized this classification system and management algorithm may exclude clinical parameters which could lead to earlier identification of SVC syndrome from a broader range of causes. Our aim was to validate the Yale classification algorithm and develop a comprehensive model for SVC identification.

Methods

Retrospective data from Oregon Health and Science University pts diagnosed with ICD-9 code 459.2, compression of vein, between 2008-11 were collected. Pt demographics, vital signs, physical examination findings, radiographic studies, tumor characteristics, hospitalization records, and outcomes were collected and analyzed. Student-t and chi-squared tests were utilized to assess significant differences in clinical presentation between men and women (p ≤ 0.05).

Results

The study population consisted of 207 pts; 157 were removed due to compression of a vessel other than the SVC. Women (n = 23) were older than men (n = 27), 48 24 yrs vs 55 18 yrs (p = 0.02), and ventilated for a longer period of time (10 11 days vs 4 3 days (p < 0.01)). Otherwise there was no difference between the two groups with respect to length of hospitalization, duration of ICU stay, or admission vital signs. SVC syndrome was caused by: tumor (n=20), thrombus (n=11), fibrosis (n=5), and iatrogenic causes (n=3). The OHSU Grading system captured 47 patients, the Yale Grading system captured 30. Dyspnea, facial edema and cough were the most common physical examination findings. The OHSU’s severe grade captured the largest number of patients (n=22). Dyspnea was seen in 19 vs 3 (p≤0.01) and facial edema was seen in 18 vs 4 (p≤0.01). The Yale’s moderate grade captured 7 patients. Cough was seen in all 7 (p<0.01), and facial edema in 6 vs 1 (p=0.01). Mortality was not associated with the presence of a tumor. OHSU’s treatment courses mirrored the Yale classification system and management algorithm.

Discussion

SVC syndrome is often considered a surgical emergency. Most life threatening presentations involve tracheal deviation, mental status changes or cerebral edema. Death is usually related to non-vascular issues. Our population demonstrated that respiratory and esophageal compromise were common physical examination presentations. Awareness of other structures and the complications associated with the enlarging lesion may help target patients who otherwise may have been overlooked. Studies have demonstrated that most patients present with symptoms several years prior to developing SVC syndrome.

Small-cell lung cancer (SCLC) and lymphoma comprise 90% of the malignant causes of SVC syndrome. Chemotherapy is the usual management of SCLC with radiotherapy reserved for patients who relapse. Similar treatments are employed for lymphoma and other malignant causes. Those patients experiencing thrombus-related SVC syndrome benefit from thrombolytic therapy.

The Yale classification system and management algorithm was applicable to patients with a malignancy. It’s scope did not account for patients presenting with respiratory symptoms prior to the onset of those associated with vascular compromise (facial, neck, or extremity edema), nor were non-oncologic causes addressed. This approach excluded patients who presented with a thrombus or stricture secondary to an indwelling catheter or pacemaker leads, fibrosing mediastinitis, or who experience obstruction due to a previous intervention. Due to the development of collateral circulation, mortality in this population is rarely associated with vascular compromise.

Our algorithm and grading system, which is not limited to one etiology of SVC, places a greater emphasis on non-vascular symptoms and maintains the identical treatment plans seen in the Yale algorithm.

Conclusion

A new classification system which accounts for the escalation of symptoms due to mass effect on surrounding tissues and a new management algorithm incorporating their respective treatments may represent a more comprehensive approach to SVC syndrome treatment.

References


CT Images

Fig. 1 - Metastatic mediastinal lymph nodal mass, surrounding the vena cava. The patient's patency was secured secondary to a previously placed access port.
Fig. 2 - Near complete collapse of the SVC at the level of a right paratracheal mass
Fig. 3 - Venous thrombosis involving the central superior vena cava, brachiocephalic, both subclavian and auxiliary veins, and the left jugular vein
Fig. 4 - Venogram demonstrating thrombus in the right and left innominate veins and occlusion of the superior vena cava at the site of termination of left chest port catheter
Fig. 5 - Near occlusive stenosis at the carotid junction with proximal clot in the SVC extending cephalad to the junction of the right brachiocephalic vein. Focal stenosis is noted near the catheter tip. Likely triggered by a Port-A-Cath vs cavaotial junction stenosis.

There are no conflicts of interest to disclose.