Review Article | Symptom Management in Palliative Medicine and Palliative Care

Management of malignant superior vena cava syndrome

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Abstract: Superior vena cava (SVC) syndrome occurs due to obstructed blood flow through the SVC. It can present clinically on a spectrum, between asymptomatic and life-threatening emergency. Patients commonly report a feeling of fullness in the head, facial, neck and upper extremity edema, and dyspnea. On imaging, patients commonly have superior mediastinal widening and pleural effusion. The majority of cases are due to malignant causes, with non-small cell lung cancer, small cell lung cancer, and lymphoma the most commonly associated malignancies. When evaluating patients, a complete staging workup is recommended, as it will determine whether treatment should be definitive/curative or palliative in intent. If the patient requires urgent treatment of venous obstruction, such as in the cases of acute central airway obstruction, severe laryngeal edema and/or coma from cerebral edema, direct opening of the occlusion by endovascular stenting and angioplasty with thrombolysis should be considered. Such an approach can provide immediate relief of symptoms before cancer-specific therapies are initiated. The intent of treatment is to manage the underlying disease while palliating symptoms. Treatment approaches most commonly employ chemotherapy and/or radiation therapy depending on the primary histology. Mildly hypofractionated radiation regimens are most commonly employed and achieve high rates of symptomatic responses generally within 2 weeks of initiating therapy.

Keywords: Superior vena cava syndrome (SVC syndrome); palliative care; lung cancer; non-small cell lung cancer (NSCLC); radiation therapy

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Introduction

The clinical manifestations of superior vena cava (SVC) syndrome relate to its underlying pathology of obstructed flow through the SVC. Clinical presentations can vary on a spectrum between asymptomatic and life-threatening emergency, and such symptoms can develop acutely or insidiously. The global incidence of SVC syndrome that has been reported in the literature ranges from as many as 1 in 650 to up to 1 in 3,100 patients (1).

Historically, the majority of SVC syndrome cases were due to infectious diseases, including syphilitic aortic aneurysm and tuberculosis (2,3). However, with the evolution of medicine over the past century, most cases are now due to chronic, rather than infectious causes (4). In fact, the majority of cases (over 60%) are due to malignant causes (5,6). Of these, non-small cell lung cancer (NSCLC) (50%), small cell lung cancer (SCLC) (22%) and lymphoma (12%) are the most common malignant etiologies (7). Among the non-malignant causes, the most common etiologies are related to intravascular devices, such as catheters and pacemakers, causing thrombosis, and the

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incidence of SVC syndrome due to intravascular devices has steadily increased over the past decades (8).

Anatomy and pathophysiology

The SVC is a great vessel of the heart that drains venous blood from systemic circulation into the right atrium of the heart. It is formed by the merger of the right and left brachiocephalic veins at the level of the sternal angle. The left and right brachiocephalic veins, in turn, collect venous blood from smaller veins and venules, originating from capillary beds in the head, neck, upper extremities, and upper thorax. The azygos vein drains blood from the posterior wall of the thorax and abdomen and also drains into the SVC at the level of the sternal angle.

The SVC is located in the right superior mediastinum. Its neighboring anatomical landmarks include the sternum anteriorly, the aorta medially, the right lung laterally, and the right main bronchus and pulmonary vessels posteriorly. The SVC is surrounded by lymph nodes that drain the right thoracic cavity. As the SVC has a relatively thin, compliant wall, it is easily compressible and susceptible to any enlarged pathologies and extrinsic compression from structures within the superior mediastinum.

In the event of any structural enlargements or presence of space-occupying pathologies, the SVC may be partially or fully occluded which may result in compartment syndrome. If insidious, collaterals systems may form, which typically lead to less-severe symptomatic presentations than acute presentations due to the recruitment of azygos, lateral thoracic, paraspinal, internal mammary, and/or esophageal venous network and may result in externally visible venous collaterals at the skin surface (9).

Etiology

SVC syndrome is more commonly caused by malignant etiologies (5,6), and less commonly by non-malignant etiologies.

Malignant causes

Over 60% of patients with SVC syndrome are known to have a malignancy (7,10,11). Patients often present with a bulky central mass involving the mediastinum, with a histology of squamous cell carcinoma of the lung and SCLC (12). Lymphoma also accounts for SVC syndrome (7,10,11). Diffuse large-cell lymphoma and lymphoblastic lymphoma account for the majority of lymphoma-associated SVC syndrome cases. More rarely, SVC syndrome can manifest in patients with pleural mesothelioma, thymoma or thymic carcinoma, primary mediastinal germ cell tumors, and intrathoracic sarcomas.

In addition to the above primary tumors of the thorax and mediastinum, SVC syndrome can also present secondary to metastases. Metastatic breast cancer is estimated to comprise one in 10 cases of malignant SVC syndrome (2,11). When diagnosed with SVC syndrome secondary to their metastases, patients generally have poorer prognosis (13).

Non-malignant causes

Nearly two in five patients with SVC syndrome have non-malignant etiologies (7,10,11). Etiologies include thrombosis from central vein catheters delivering chemotherapy agents or hyperalimentation, pacemakers and other cardiac devices, and cardiac interventions including radiofrequency ablation (14). Of note, the prevalence of non-malignant etiologies is markedly higher than several decades ago and will likely continue to increase due to an increased use of catheters and pacemakers (8).

Non-malignant causes of SVC syndrome can also be vasculitic conditions that increase the risk of thrombosis (i.e., Behçet disease), vascular anomalies/aneurysms that exert pressure on the SVC, and inflammatory mediastinal processes (i.e., fibrosing mediastinitis, Castleman's disease). Among children and adolescents, SVC syndrome is uncommon and most typically iatrogenic (i.e., due to sequelae of cardiovascular repair of congenital heart disease, orthotopic heart transplantation, ventriculoatrial shunt placement for the treatment of hydrocephalus, or SVC catheterization placement for the receipt of parenteral nutrition), or potentially from an infectious etiology (i.e., mediastinal fibrosis caused by histoplasmosis) (15,16).

Given the disparate treatment approaches for malignant and non-malignant causes of SVC syndrome and malignant causes being the dominant etiology of SVC syndrome cases, the remainder of this review will focus on SVC syndrome associated with malignant causes.

Clinical presentation

Patients with SVC syndrome commonly report a feeling of fullness in the head, facial edema, and dyspnea (17). On physical exam, patients may present with hypervolemia, namely with venous distention of the neck and chest wall,

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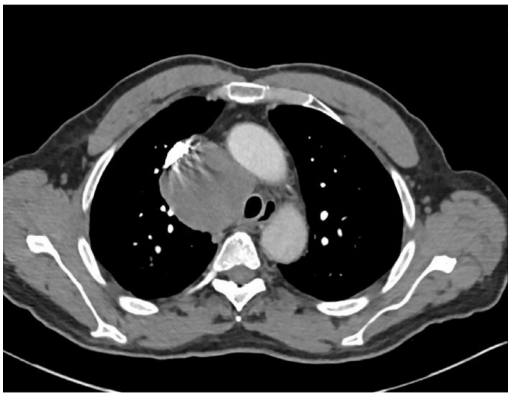

Figure 1 A 77-year-old patient with newly diagnosed locally advanced lung adenocarcinoma with radiographic evidence of superior vena cava compression presenting with cough and weight loss but no overt symptoms related to SVC syndrome. SVC, superior vena cava.

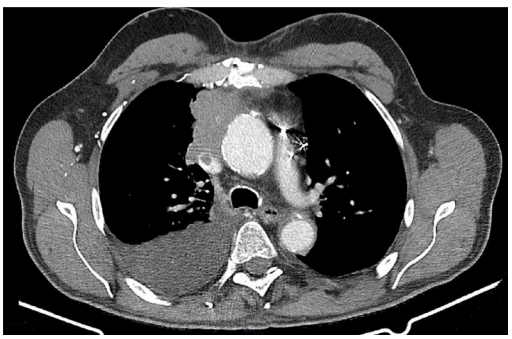

Figure 2 A 66-year-old patient with thymoma presenting with rapidly progressing dyspnea on exertion and facial and neck fullness found on imaging to have a 6 cm anterior mediastinal mass with superior vena cava encasement and invasion.

facial edema, and upper extremity edema; respiratory function may also be compromised, manifesting as central and peripheral cyanosis (6). Symptoms may worsen when the patient is placed supine or leaning forward/tilted anteriorly around their pelvis. Patients may also present with a pleural effusion, which is commonly exudative and chylous.

Diagnostic evaluation

Given that the differential diagnosis for SVC syndrome can vary significantly and consequently have very different treatment, diagnosis is a critical step in guiding management. Complete staging workup is ideal, as it can indicate whether treatment should be definitive/curative or palliative in intent.

Patients with SVC syndrome have a characteristic pathology on imaging of superior mediastinal widening and pleural effusion (*Figures 1*,*2*). With more clinically significant cases readily seen on routine computed tomography (CT) scans of the chest without and especially with contrast, SVC obstruction can be optimally visualized by superior vena cavogram, which is limited to only surveying the SVC and can identify the site of SVC obstruction, extent of stenosis, any associated thrombus, and any hemodynamic significant findings (18). CT with intravenous contrast can allow detailed investigations of the SVC, its tributaries, and other critical mediastinal structures, including the trachea, bronchi, esophagus and spinal cord (19). In patients with chronic kidney disease and/or contraindications for iodinated CT contrast agents, magnetic resonance venography may be used instead (18). Patients with a greater number of involved veins, presence of pleural effusion, older age, more advanced cancer stage and more severe SVC syndrome have been reported to have poorer 30-day mortality from SVC syndrome (20).

Patients without a known history of malignancy and a new clinical diagnosis of SVC syndrome should undergo investigation for the underlying cause of cancer. Modern minimally invasive diagnostic procedures should be used. Depending on disease presentation, cytologic sampling can be conducted to confirm malignancy. Sampling can be conducted by bronchoscopy and endobronchial fine-needle aspiration, mediastinoscopy and biopsy, CT-guided or ultrasound-guided needle biopsy, or supraclavicular lymph node biopsy (7). Endobronchial fine-needle aspirations are accurate and provides sufficient malignant cells for cytologic evaluation in the majority of cases of lung cancer (21). A percutaneous transthoracic CT-guided needle biopsy is an effective and safe alternative. For patients who cannot be pathologically staged via less invasive techniques, a mediastinoscopy frequently can provide a diagnosis, and it has a low complication rate (22). If all else fails, a thoracoscopic biopsy or open thoracotomy may be necessary. Additionally, a ^{18}F-fluorodeoxyglucose (FDG)-positron emission tomography (PET) scan can be used to assist with staging and design of a radiotherapy field (23,24).

Treatment

Treatment intent for malignant SVC syndrome is both to manage the underlying disease while palliating symptoms

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from the SVC syndrome. If the patient requires urgent treatment of venous obstruction, such as in the cases of acute central airway obstruction, severe laryngeal edema and/or coma from cerebral edema, direct opening of the occlusion by endovascular stenting and angioplasty with thrombolysis should be considered. Such an approach can provide immediate relief of symptoms before cancer-specific therapies are initiated and may be appropriate to use as standard treatment for immediate symptomatic management (25,26). Otherwise, a full diagnostic workup and staging of cancer is needed, and cancer-specific therapies should be initiated based on the stage of the respective malignancy. During evaluations, the patient may benefit from supportive oxygen administration. Some clinicians may favor use of diuretics and/or corticosteroids for patients who are symptomatic and uncomfortable; this should be administered after pathologic confirmation of the etiology of SVC syndrome, as steroid initiation can reduce the diagnostic sensitivity of a biopsy, especially for cases induced by hematologic malignancies. The rate of clinical improvement is similar among those receiving diuretics and/or corticosteroids (11).

Radiation therapy is a commonly used and effective treatment option for patients with malignant SVC syndrome to shrink the underlying compressive/invasive malignant mass(es) and thereby provide rapid relief of the obstruction. The radiation portal should include all known gross disease. Additional margins should be added to account for microscopic disease extension (clinic target volume; CTV) and set-up errors and uncertainties (planning target volume; PTV). The etiology and specific histology of malignancy causing the SVC compression should be taken into account when choosing the dose-fractionation regimen of radiotherapy. Chemotherapy is also indicated in many scenarios. Surgery may be explored in select cases. However, given the extent of the procedure often needed and the invasiveness of tumors typically causing SVC syndrome, gross total resections are often difficult to achieve, and the use of surgery is typically reserved for when other treatment options of stenting, chemotherapy and radiation have been exhausted.

Specific treatment options by malignancy for lung cancer and for lymphoma—two of the most common causes of malignant SVC syndrome—are reported below. In brief, patients with NSCLC can be treated with chemotherapy and/or radiotherapy. Patients with SCLC can be managed with systemic therapy alone, including platinum-based chemotherapy, radiotherapy, or combination therapy. Patients with lymphoma may also be managed with chemotherapy, with radiotherapy often used for local consolidation.

Lung cancer

Among patients with NSCLC, chemotherapy was reported to relieve SVC syndrome in 59% of patients, and radiotherapy in 63% of patients (25). Improvement after radiotherapy was more successful in patients who had previously received therapy; as such, radiotherapy may be an effective salvage therapy, in patients with recurrent SVC syndrome (25).

Currently, no consensus exists on the ideal fractionation scheme. Patients are commonly treated with modest hypofractionation regimens of 3–4 Gy per fraction in an attempt to achieve a more rapid treatment response. This is typically delivered to 5 to 10 fractions for patients with metastatic disease. For patients with non-metastatic disease, a limited number of these hypofractionated treatments can be delivered before proceeding with conventional 2 Gy fractions for the remainder of the course in order to reach a total dose with curative intent per current guidelines (27,28). Notably, however, no randomized trials comparing different dose fractionation regimens for SVC syndrome relief with radiation therapy have been conducted to date, and as a result no consensus currently exists on the ideal fractionation scheme (7,29). Immunotherapy, either alone or in combination with chemotherapy, may also be part of a first-line or subsequent management option for SVC syndrome, but current data on the use of immunotherapy specifically for SVC syndrome are sparse (27).

Given that the response rates for chemotherapy are generally higher and achieved faster in SCLC than for NSCLC, patients with extensive stage SCLC are commonly initiated on systemic therapy alone at the time of SVC syndrome diagnosis, frequently with thoracic radiotherapy considered as consolidative therapy (28,30,31). One study did note, however, that upfront thoracic radiation therapy in combination with chemotherapy may be associated with improved survival versus chemotherapy alone or chemotherapy followed by consolidative radiotherapy in patients with extensive stage SCLC and SVC syndrome (32), and that relief from SVC syndrome typically occurs within 2 weeks after therapy. Akin to radiation therapy for NSCLC, patients are commonly treated with modest hypofractionation regimens of 3–4 Gy per fraction in an attempt to have a more rapid treatment response.

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Immunotherapy, with anti-programmed death ligand 1 (PD-L1) or anti-programmed death 1 (PD-1) inhibition, is effective in patients with extensive stage SCLC (33,34). However, while chemotherapy (carboplatin or cisplatin with etoposide) in combination with immunotherapy (atezolizumab or durvalumab) is now the standard first line approach for newly diagnosed extensive stage SCLC, no literature to date reports on the effectiveness of immunotherapy alone or in combination with chemotherapy on relieving symptoms from SVC syndrome among patients with SCLC.

Lymphoma

Patients with lymphoma who are symptomatic with dysphagia, hoarseness or stridor were reported to have poorer survival (35). While systemic therapy remains the mainstem for treatment of diffuse large B cell lymphoma and of lymphoma-associated SVC syndrome, consolidation radiation therapy provides an opportunity for durable local control and also SVC syndrome-associated symptomatic relief. Patients treated with chemotherapy and/or radiation typically experience relief within 2 weeks of treatment (35). However, relapse is common, especially with chemotherapy alone in high grade lymphomas, which consolidative radiation therapy may help improve.

For patients with lymphoma, 1.8–2 Gy doses per fraction are recommended and administered to an increasing number of fractions until symptomatic relief is achieved (7). More hypofractionated regimens of 6–8 Gy per fraction to 2–3 fractions have been previously investigated and shown to be effective (36,37). Such approaches can achieve an overall response rate of 87%, partial response rate of 70% and complete resolution of symptoms in 28%. Further escalation of the total dose (i.e., increasing from 2 to 3 fractions), can allow for a partial response to be achieved in 96% of patients and complete resolution in 56% of patients. These fractionation regimens are generally well tolerated, with only mild and temporary side effects noted in the reports using these approaches.

Conclusions

SVC syndrome can be an asymptomatic or a life-threatening presentation. Complete workup should be pursued to determine a malignant or non-malignant pathology, and malignancies should subsequently be treated with definitive/curative or palliative intent. Treatment commonly involves chemotherapy and/or radiation therapy, with mildly hypofractionated radiation regimens most commonly employed and high rates of symptomatic response achieved.

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