Oncologic Emergencies, Part I: Spinal Cord Compression, Superior Vena Cava Syndrome, And Pericardial Effusion

A 65-year-old woman with a history of non-small-cell cancer is brought in by her family to the emergency department (ED) with a chief complaint of progressive lethargy for the past 2 days. According to the family, she had 2 weeks of cough and shortness of breath, and she was given antibiotics for pneumonia a week ago in urgent care, with no improvement. She became confused yesterday and is less interactive today. The family thinks that her neck and arms are more swollen than usual, which you confirm by examining her driver’s license picture, taken last year. She is afebrile and has a blood pressure (BP) of 160/95 mm Hg, heart rate of 70 beats per minute, and respiratory rate of 24 breaths per minute with an oxygen saturation of 98% on room air. She has intact cough and gag, but you can hear mild stridor. An indwelling catheter in her left chest was used recently for chemotherapy. Although you consider superior vena cava syndrome, you are not sure if you can put everything together with that diagnosis. As you try to look up more on superior vena cava syndrome, a nurse grabs you, stating that your patient’s respirations have increased to 36 breaths per minute.

Later in your busy shift, a 45-year-old man with altered mental status is brought in by ambulance. At his bedside, a prehospital care provider tells you that the patient is unable to provide a history and that the family is on the way to the ED. The patient appears pale and lethargic and has labored breathing. The ambulance crew reports that they administered 500 mL of Ringer’s lactate in response to an initial blood pressure of 86/60 mm Hg.
Oncologic emergencies are not everyday occurrences in the ED, even at tertiary care centers. Yet, because of the growing number of patients in cancer treatment, those with problems related to their disease and its treatment will inevitably present to EDs. Since patients with cancer are at higher risk for significant morbidity and mortality at baseline, the potentially catastrophic complications of malignancy can quickly turn lethal. Therefore, it is essential for the emergency clinician to recognize these infrequent presentations and be prepared to treat them. This issue of Emergency Medicine Practice provides a review of 3 important cancer-related complications: malignant epidural spinal cord compression, superior vena cava syndrome, and malignant pericardial effusion and tamponade. The March 2010 issue of Emergency Medicine Practice, Oncologic Emergencies Part II, will address the topics of neutropenic fever, tumor lysis syndrome, and hypercalcemia of malignancy.

Critical Review Of The Literature

An Ovid MEDLINE® (www.ovid.com) and PubMed (www.pubmed.gov) search was performed using the terms malignant epidural spinal cord compression, superior vena cava syndrome, and malignant pericardial tamponade in articles published between 1966 and 2008. The search yielded numerous studies ranging from case series to randomized, double-blind, placebo-controlled trials; in addition, standard textbooks were reviewed. Of the 3 topics, the literature on malignant epidural spinal cord compression offered the best evidence to support recommendations for its management. Evidence regarding management recommendations for the other 2 conditions was largely based on case reports and observational cohort studies.

Prehospital Care

Prehospital care is directed toward the presenting complaint, with a focus on stabilizing the patient and identifying the transport destination that is best prepared to manage the patient’s illness. Ideally, each cancer patient should be taken to the hospital with which his or her oncologist is affiliated; unfortunately, the available literature does not help define the impact on outcome when the patient is transported to a different hospital, and clearly more research is needed on this topic.

Paramedics charged with caring for patients with oncologic emergencies should be familiar with the region-specific transport protocols and be aware that the length of time that elapses before evaluation and treatment in the ED can significantly alter outcomes. Gathering as much medical history as possible from caregivers and resources at the scene, including code status and advance directives, is extremely valuable to medical personnel who receive the patient in the ED.

Malignant Epidural Spinal Cord Compression

Malignant epidural spinal cord compression (ESCC) is a serious complication of cancer, with an incidence of 5% in the population with cancer and a median survival of only 6 months after diagnosis. Although prompt recognition and timely treatment will ensure good functional outcomes in the majority of patients, diagnosis of this complication is often delayed because the presentations are frequently varied and atypical and clinicians’ index of suspicion is low. In a prospective, observational study of 310 patients, a delay in diagnosis was the primary cause for postponing treatment for a median of 2 months from the onset of back pain and for a median of 2 weeks from the onset of actual signs consistent with spinal cord compression. Since the vast majority of cases of malignant spinal cord compression result from epidural (extradural) compression, it is the focus of this review. Other less common causes include intramedullary metastases (intradural involvement) and leptomeningeal metastases (dural involvement).

Etiology Of ESCC

Spinal cord compression is generally defined as a condition in which thecal sac impingement is evident on radiography but may or may not manifest clinical findings that correspond to the level of impingement. Tumors invade the epidural space in essentially 2 ways: (1) directly from vertebral bone metastases, or (2) from extension through the neural foramina from nearby structures, such as lymph nodes or nerve bundles. Compression of the epidural sac and encasement of the spinal cord ensue, causing an elevation in pressures within the venous plexus that supplies the cord. Vasogenic edema occurs and leads to spinal cord ischemia.
Approximately 90% of cases of ESCC are due to extension of metastases in the vertebral bodies. Metastatic deposits in the vertebral bodies come primarily from hematogenous (arterial and venous) sources. The amount of arterial blood flow that a vertebra receives depends on the volume of bone present. Therefore, sections of vertebrae with more bone volume (specifically the lumbosacral and thoracic spine) have a greater likelihood of being seeded by vascularly spread metastases. In approximately 70% of patients with malignant cord compression, the metastasis is located within the thoracic spine, 20% are located in the lumbosacral spine, and 10% in the cervical spine. The disproportionately greater percentage of cases of ESCC in the thoracic region is thought to result from the relatively smaller available free space within the thoracic canal, which makes metastatic disease at this level more frequently symptomatic. Though most metastases are of arterial origin, many lumbosacral vertebral metastases arise from venous sources. In general, this is most likely due to the absence of valves in the venous plexus that connects the lumbosacral spine to organs such as the prostate. In the remaining 10% of cases of ESCC, metastases gain access to the spinal cord by penetrating the neural foramina. Although less common, this mechanism of spinal cord compression is important because of the complete absence of abnormalities associated with it on plain radiography, as will be discussed later.

Since the majority of cases of ESCC are caused by vertebral metastases, the likelihood of epidural compression is related to both a tumor’s incidence and its affinity for vertebral bone metastasis. Bone, breast, lung, and prostate cancers account for 60% of cases of ESCC. Approximately 25% of cases are associated with non-Hodgkin’s lymphoma, renal cell cancer, and multiple myeloma.

### History For ESCC

Targeted questions during the patient interview help stratify risk in individuals who present with symptoms that suggest malignant ESCC. In patients with a history of cancer, any suggestive symptoms should be considered red flags for the presence of ESCC, since back pain due to nerve damage, tumor expansion, or vertebral collapse is the first symptom in 95% of patients with ESCC, usually preceding all other symptoms by up to 2 months. Although back pain is a sensitive indicator of ESCC and is usually what prompts a patient to seek medical attention, it is rarely specific for the site of the lesion. The classic teaching that pain worsens with recumbence was not validated in a prospective series of 170 patients.

Certain findings elicited on history-taking are associated with a higher risk for epidural compression; these include pain at night and a low Karnofsky score (a global assessment of independent function that is often used to evaluate patients with cancer). Other key features in the history can also provide insight into the cause of a patient’s pain. For example, about 75% of patients with ESCC present with focal weakness, 50% with bowel or bladder dysfunction, 40% to 90% with sensory abnormalities, and many others with radicular pain or ataxia (due to impingement of the spinocerebellar tract along the posterior aspect of the spinal cord). Motor abnormalities typically follow a sequential pattern of progressive weakness, ataxia, and finally, paralysis.

The emergency clinician should ask patients about the following 6 subjects: (1) the type of cancer they have and whether there are known metastases; (2) whether they have undergone or are undergoing radiation therapy or chemotherapy; (3) the results of previous imaging studies; (4) recent trauma; (5) risk factors that might point to other causes for the symptoms; and (6) information about advance directives and their wishes regarding workup and treatment for possible oncologic complications.

### Diagnostic Studies For ESCC

The history and physical examination alone are insufficient to diagnose or rule out ESCC. Radiologic evidence of cord compression provides the basis for the diagnosis of ESCC.

### Laboratory Testing

Although there is no evidence to suggest that routine laboratory data aid in the diagnosis of malignant cord compression, such information may be
Clinical Pathway For Evaluation Of Suspected Malignant Epidural Spinal Cord Compression

Patient with back pain and history of malignancy

- Normal neurological exam
  - Low suspicion of ESCC
    - Excellent follow-up
      - Outpatient MRI or CT myelogram within 24 hours (Class III)
  - High suspicion of ESCC
    - Admit; consult oncology, neurosurgery, and radiation oncology as appropriate
  - Abnormal neurological exam
  - Emergent MRI or CT myelogram (Class I)
  - Unreliable follow-up
    - Administer empiric steroids (see page 6 for dose) (Class II)

No ESCC
  - Consider alternate diagnosis

ESCC
  - Admit; consult oncology, neurosurgery, and radiation oncology as appropriate

Class Of Evidence Definitions

Each action in the clinical pathways section of Emergency Medicine Practice receives a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness
Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful
Level of Evidence:
- Generally higher levels of evidence
- Non-randomized or retrospective studies: historic, cohort, or case control studies
- Less robust RCTs
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments
Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research
Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling


This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient’s individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

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helpful in the management of comorbidities and in preparing for further invasive interventions (eg, platelet count prior to lumbar puncture, or serum creatinine levels prior to contrast-enhanced imaging studies). In rare cases, cerebrospinal fluid (CSF) analysis to detect leptomeningeal metastasis may be performed, and an alternative diagnosis may be found to explain a patient’s presentation.

**Plain Radiography**

Clinicians should not rely on plain radiography as the only tool for diagnosing cord compression. Vertebral compression cannot be visualized unless there has been bone loss of up to 50%. Ten percent of cases of ESCC are caused by neuroforamen invasion without significant bone destruction. In fact, even when vertebral collapse is evident on an x-ray film, the overall sensitivity of plain radiography for ESCC is approximately 75%, which drops to 66% in patients with lymphoma. In the absence of other available radiographic modalities, x-ray evidence of compression coupled with clinical findings that are consistent with that level of sensory abnormality can aid in the diagnosis and management of clinically relevant ESCC.

**Magnetic Resonance Imaging**

If ESCC seems likely, based on history and physical examination, the diagnostic evaluation should proceed directly to magnetic resonance imaging (MRI). Despite slight variations in readings, and depending on the experience of the radiologist, the overall accuracy of MRI for ESCC is high (93% sensitivity, > 97% specificity, > 95% positive predictive value). Additional benefits of MRI, including its noninvasive nature, cost-effectiveness, and ability to image the entire spine to look for compression, intramedullary lesions, and bone metastases, make this modality the study of choice for suspected ESCC. Approximately one-third of all patients diagnosed with ESCC have multiple sites of epidural metastases, so being able to image the entire spine is extremely valuable. If MRI is unavailable or if the patient cannot tolerate the lengthy imaging time, focused imaging of those areas that arouse clinical suspicion is a reasonable temporary alternative.

In a study of 280 patients with confirmed ESCC, if clinical findings were suspicious for thoracic or lumbar cord injury, less than 1% of the patients had cervical lesions. Therefore, it is probably safe to forgo MRI of the cervical spine if plain films initially verify thoracic or lumbar cord compression which is then corroborated on the clinical examination. Nevertheless, careful imaging of the thoracic and lumbar spine should be performed in all ESCC patients to rule out multiple epidural metastases. Overall, it is important to note that the sensory level affected does not necessarily correspond to the area of suspected cord compression, as evidenced by 27% of the 280 patients with ESCC mentioned earlier whose sensory levels were either 4 vertebral segments below or 2 vertebral segments above the MRI-confirmed area of compression. Contrast-enhanced images with gadolinium provide better visualization of the less-common leptomeningeal metastases and intramedullary tumors, but the evaluation of epidural spinal cord compression does not require intravenous (IV) contrast.

**Computed Tomography With Myelography**

Myelography is an inherently invasive imaging modality in which a lumbar puncture is performed and contrast dye is injected into the spinal canal during fluoroscopy to highlight any existing flow obstructions that may indicate spinal cord compression. For many years, it was the standard imaging modality used in the assessment of patients suspected of having ESCC. Although myelography was useful for detecting blockages in the spinal canal, it was not particularly helpful in assessing the degree of cord injury or other anatomic abnormalities proximal to the obstruction.

With the advent of MRI and computed tomography (CT), imaging quality has been greatly enhanced, allowing for much better anatomic detail of the spine. Since spinal CT alone (with or without contrast) does not adequately image the spinal cord.
itself or the epidural space, it is not recommended in the evaluation of ESCC. However, in the absence of available MRI or CT myelography, noncontrast spinal CT could be used to evaluate the vertebrae for possible compression fractures or cortical bone discontinuity that could suggest cord compression.21

CT imaging is very useful when used in conjunction with myelography. In fact, when the flow of contrast in the CSF is not halted by a completely occluded spinal canal, CT myelography allows good-quality visualization of paraspinal and spinal masses, with sensitivity and specificity that is comparable to those of MRI (both over 95%) in the evaluation of ESCC.33,34 The invasive nature of CT myelography often prohibits its use in many cancer patients, who are often severely thrombocytopenic. Moreover, patients with complete subarachnoid blockage may neurologically decompensate when lumbar puncture reduces CSF pressure below the block; no studies have been carried out to determine what risk factors might predict this rare occurrence. Despite its risks, CT myelography is a worthwhile and proven alternative to MRI when the latter is unavailable or contraindicated.

Bone Scintigraphy
More efficient, accurate, and comprehensive radiographic modalities have largely supplanted the bone scan in the detection of ESCC. However, when both MRI and CT myelography are contraindicated or not available, bone scintigraphy can still be of value. Plain radiographs in conjunction with bone scintigraphy have been reported to have a sensitivity as high as 98% for ESCC.35 In these difficult cases, consultation with the radiation oncologist, neurosurgeon, or primary oncologist is recommended.

Treatment For ESCC
The neurologic status of patients before treatment for ESCC is the most important prognostic factor for neurologic status after treatment.21 The timing of a patient’s presentation may also have prognostic value, with 1 prospective study failing to demonstrate any improvement in neurologic status if a patient’s deficits developed rapidly within the 48-hour period prior to presentation.22 The emergency clinician must understand the significant prognostic implications of the patient’s presenting neurologic status, and treatment goals for each individual patient should be well-defined before therapy is initiated.

It is imperative to involve the appropriate consultants in the early and definitive management of patients with ESCC. In most circumstances, the goals are to improve or maintain a patient’s pretreatment neurologic function, to control local tumor growth, to stabilize the spine, and to control pain.35 Standard treatment typically involves a combination of steroids, surgery, and/or radiation.

Corticosteroids
Vasogenic edema of the spinal cord associated with ESCC can be mitigated by the use of corticosteroids, a mainstay of treatment that should be started in the ED. The benefits of steroids are generally well-accepted. In the most-quoted randomized controlled trial of corticosteroids for patients with myelography-confirmed ESCC treated with radiation therapy, high-dose dexamethasone (IV bolus of 96 mg, followed by 24 mg orally 4 times daily for 3 days, and then tapered over the next 10 days) was compared with no steroid therapy.36 The 3-month post-radiation evaluation demonstrated a significantly higher rate of ambulation in the group treated with steroids (81% of patients) vs the group without steroid therapy (63%).

The safety of high-dose vs low-dose steroids is still a matter of controversy. One case-control study compared high-dose dexamethasone (same regimen as noted above) with a low-dose regimen (10 mg IV bolus, then 4 mg IV daily, tapered over 2 weeks). Although the 2 regimens had similar efficacy rates, the high-dose regimen was associated with many side effects.37 Despite inconclusive evidence regarding the proper steroid dose for ESCC, expert recommendations are to administer high-dose steroids (dexamethasone, 100 mg IV bolus, with a rapid taper, halving the total daily dose every 3 days if the patient is stable) for patients with myelopathy or abnormal neurologic findings on examination, and low-dose steroids (dexamethasone, 10 mg IV bolus with 4 mg IV doses daily, tapered over 2 weeks) for all other patients, such as those in whom only radiographic evidence of ESCC exists in the absence of corresponding clinical findings.9,40 Large-scale studies are needed to determine the most appropriate dose of steroids for ESCC.

Surgery
The generally accepted indications for surgical decompression include spinal instability (defined by gross spinal deformity or bony retropulsion into the canal), previous radiation therapy in the compressed area, a need for tissue diagnosis of a primary malignancy, disease progression despite radiation and steroids, and the presence of radioresistant tumors (eg, melanoma, sarcoma, or renal cell carcinoma).20,41

The debate over the appropriate criteria for selecting surgery and/or radiation therapy is ongoing. Various retrospective studies report similar neurologic outcomes in patients who undergo radiation plus laminectomy and those who receive radiation only,6,42 with some studies demonstrating slightly improved outcomes with the latter.33 As previously discussed, the majority of cases of malignant ESCC originate from the vertebral bodies. However, in earlier studies (mostly in the 1980s), the surgical arm involved posterior laminectomy, which conceptually would relieve pressure within the epidural space.
itself without addressing the site of metastasis. A more recent large, prospective, randomized trial assigned patients to either radiation therapy alone or to radical surgical decompression (involving only the anterior vertebral body in 60% of cases) followed by postoperative radiation. Ambulation time was significantly longer for the group treated with surgery plus radiation than for those treated with radiation alone (median time = 126 vs 35 days). The conclusions reached in this study and in similar studies have made surgical management a much more attractive initial treatment option for patients who present with ESCC, although formal guidelines from specialty societies have not yet been issued.45

**Radiation Therapy**

Radiation therapy is of benefit in patients with ESCC who have already undergone radical spinal decompression,44,45 who have no spinal compression or instability, who have subclinical cord compression (radiographically evident but without clinical signs or symptoms consistent with compression),21 and who are not eligible for surgery. Although the value of radiation therapy in these circumstances has been well-proven, the ideal dosages and schedule regimens for the treatment of ESCC have still not been determined. The poor survival rates of patients treated with radiation (median = 6 months) is thought to be associated with the severity of the underlying malignancy rather than with the radiation therapy. Intuitively, it is assumed that radiation would be more successful in the recovery of neurologic function in patients with radiosensitive tumors (eg, lymphoma, myeloma, breast, prostate, and small-cell lung cancers) than in patients with radioresistant ones (eg, melanoma, sarcoma, and renal cell carcinoma). Nearly half of all patients who receive radiation therapy and who are still alive 1 year after the procedure maintain their ability to ambulate. Further clarification is needed to identify which subgroups benefit from particular combinations of radiation therapy and surgery.

**Special Circumstances: Malignant ESCC In Children**

The prevalence and management of particular malignancies in children differ from those in adults. Pediatric malignant ESCC most frequently arises from paravertebral tumors that invade the epidural space through the neuroforamen, leaving spinal bone intact. Ewing’s sarcoma and neuroblastoma are the 2 most common causes of ESCC in children. Ewing’s and other sarcomas are best treated by surgical decompression alone or in conjunction with postoperative medical therapy. However, neuroblastomas, Hodgkin’s disease, and germ cell tumors are best treated with chemotherapy, with or without radiation. Two-thirds of children whose pretreatment neurologic function is normal will remain ambulatory regardless of the type of treatment.46

**Summary Of ESCC**

A delay in the diagnosis of malignant ESCC often results in significant morbidity. Therefore, early suspicion, diagnosis, and treatment are paramount in obtaining the best neurologic outcome. Back pain is usually the earliest symptom, and MRI is the diagnostic test of choice, although CT myelography is a reasonable alternative. Imaging of the entire spine is recommended, since patients can have multiple sites of metastasis. Initiation of steroids in the ED is indicated, after which most patients with ESCC are treated with either radiation alone or surgical decompression along with radiation therapy. Pretreatment ambulatory status is the most important determinant of posttreatment ambulation and should therefore be well-documented at the time of diagnosis.

**Disposition Of Patients With ESCC**

Patients who are diagnosed in the ED with ESCC should be admitted to the hospital for pain control, frequent neurologic assessments, and definitive treatment. The involvement of all the appropriate specialties (eg, neurosurgery, radiation oncology, and oncology) is critical to ensure the most effective level of care.

**Superior Vena Cava Syndrome**

Superior vena cava syndrome (SVCS) is a rare but potentially serious complication of an underlying malignancy. Throughout the second half of the 20th century, the condition was almost exclusively limited to malignancy-related obstruction of the superior vena cava. However, with the growing number of patients dependent on dialysis, the incidence of catheter-related thrombosis as a cause of SVCS has increased significantly. SVCS is generally considered an “urgency” rather than emergency related to cancer.47

**Epidemiology Of SVCS**

SVCS, which affects approximately 15,000 people per year in the United States, was first described in 1757 by Hunter in a case report of a patient with syphilis in whom a thoracic aortic aneurysm was compressing the superior vena cava. Until the 1950s, SVCS was most commonly associated with syphilis and tuberculosis.48,49 In the late 20th century, however, 85% to 97% of SVCS cases are caused by malignancy.46 Approximately 80% of these cases result from lung cancer (most commonly bronchogenic), 10% from lymphoma (mostly non-Hodgkin’s), and 10% from metastatic disease (eg, breast cancer). Interestingly, only a small fraction of patients with lung cancer (3% to 5%) will have SVCS during the course of their illness.48 Nonmalignant causes of
SVCS include infection (eg, histoplasmosis, tuberculosis, syphilis, actinomycosis, mediastinitis), vascular problems (eg, aortic aneurysm), and trauma and local thromboses related to central venous catheters and pacemaker wires. In their retrospective case series, Rice et al found that of 78 patients with SVCS, 40% of cases were due to causes other than cancer, with the majority stemming from catheters inserted for chemotherapy access and dialysis.

Pathophysiology Of SVCS
SVCS results from the occlusion of the superior vena cava (SVC) due to either external compression or internal obstruction. A short review of the relevant anatomy will help to explain its pathophysiology. The SVC is formed by the junction of the brachio-cephalic veins. Before entering the right atrium, it merges posteriorly with the azygos venous system. It is a low-pressure vessel with thin walls surrounded by right paratracheal, perihilar, and subcarinal lymph nodes; the ascending aorta; and other mediastinal structures such as the trachea, right bronchus, pulmonary artery, and thymus. Significant enlargement of any of these structures can compress the SVC. The surrounding lymph nodes drain mostly from the right side of the lung, which may explain why right-sided lung cancer is 4 times more common than left in patients with SVCS due to lung cancer. If the obstruction is above the level of the azygos vein, this vein can partially accommodate an increase in venous flow from the head, neck, and upper extremities. If the obstruction is below the azygos attachment, the venous flow has to go through the abdominal venous system. Owing to the abdomen’s more limited capacity to shunt venous flow, the symptoms of obstruction can be more severe.

Clinical Presentation Of SVCS
The severity of symptoms of SVCS varies, depending on the site of obstruction relative to the azygos vein. Incomplete occlusion may be asymptomatic, whereas complete and extensive occlusion involving the collateral circulation can cause significant morbidity. In addition to the location and extent of obstruction, its acuity plays an important role. When obstruction occurs rapidly, there is not enough time for collateral circulation to develop. In general, sudden presentations are the exception. Delayed presentations tend to be the rule, making diagnosis more difficult. More than 75% of patients have signs and symptoms of SVCS for longer than 1 week before seeking medical attention. It is not uncommon for patients to complain of SVCS-related symptoms for weeks, or even months.

The symptoms and findings on examination reflect the underlying venous congestion. Symptoms tend to be worse upon awakening and improve as the day progresses. Patients typically have facial, neck, and upper-extremity swelling, especially when they are lying down or bending over. Dyspnea, cough, facial flushing, and hoarseness often occur, but frank airway obstruction from laryngeal edema is rare. Patients may also experience headache, confusion, or lethargy as venous drainage from the intracranial space becomes compromised. In the case series by Rice et al, the most common signs and symptoms were face or neck swelling (82%), upper-extremity swelling (62%), dyspnea at rest (53%), cough (50%), and dilated chest veins (38%). Interestingly, certain symptoms were significantly more common in SVCS due to cancer than in SVCS due to other causes; these included dyspnea at rest (64% vs 35%), cough (70% vs 19%), and chest or shoulder pain (28% vs 2%). These findings suggest that the symptoms attributed to SVCS may actually be caused by the underlying cancer.

Diagnosis Of SVCS
Clinical suspicion for SVCS depends on a thorough history and physical examination. Many patients present to the ED more than once before the true etiology is uncovered. Imaging studies are needed to confirm the clinician’s suspicion and can elucidate the cause, exact location, and extent of the obstruction.

Plain Radiography
A chest radiograph can provide clues to the diagnosis. In a retrospective case series involving 125 patients, Armstrong et al found that 78% of patients with SVCS had an abnormal chest radiograph (59% with a superior mediastinal mass and 19% with a right hilar mass).

Chest CT
This modality is preferred in the emergency setting for several reasons: it is widely available, provides anatomic details of both the extent of obstruction and collateral flow, and can potentially identify causes of the syndrome (eg, compression vs indwelling catheter-related disease). The presence of any collateral veins in the abdomen and thorax on CT is an accurate predictor of SVCS (sensitivity 96%, specificity 92%). In a retrospective review of 19 patients with pathologically confirmed SVCS, 94.7% had at least 1 enlarged collateral vein at or below the level of the diaphragm that was apparent on abdominal CT scans. No reports in the literature specifically address the sensitivity or specificity of CT for SVCS nor are there any contraindications to the use of this modality other than those inherent in the use of contrast-enhanced media.

MRI
Although MRI is considered the diagnostic modality of “choice” because of the excellent anatomic detail it provides, it may not be routinely available in EDs, and its use may be limited by the presence of incom-
patible metals within a patient, the inability to lie flat for an extended amount of time, or an allergy to or contraindication to the use of gadolinium. Hansen et al found that MRI detected thoracic venous obstruction with 94% sensitivity and 100% specificity.

Contrast Venography
CT, MRI, and nuclear imaging have largely replaced this invasive procedure; however, contrast venography is still considered a useful adjunctive modality for patients being evaluated for surgical or radiologic interventions.

Radionuclide Venography
This test requires nuclear tracers and a significant amount of time, thus limiting its usefulness in the ED setting. It is less invasive than contrast venography but also less specific in defining patency and flow.37 If a patient has a contraindication to both CT and MRI, this study is a viable option to diagnose SVCS. Discussion with a radiologist is encouraged when choosing the optimal imaging modality for the individual patient.

Treatment Of SVCS
The cause of SVCS should be determined before initiating treatment for the following reasons:
1. The syndrome is usually insidious, so one typically has time to make a diagnosis.
2. There is no role for empiric radiation or chemotherapy in nonmalignant causes of SVCS.
3. Different cancers have variable sensitivities to radiation and chemotherapy.
4. In SVCS caused by cancer, patients do not usually die from SVCS itself, but rather from progression of the underlying malignancy, and the prognosis depends on the type of tumor.

Since the long-term prognosis for patients with malignancy-related SVCS is generally poor, treatment should focus on improving quality of life. Tissue diagnosis and definitive treatment typically begin after the patient has been admitted to the hospital. Coordination of subspecialties such as oncology, radiation oncology, interventional radiology, and pulmonology is often necessary to develop an appropriate therapeutic regimen. Once a malignant cause has been established, decisions regarding potential chemotherapy, radiation treatment, or even endovascular stent placement can be addressed. Multiple factors will affect this process, including a patient’s clinical stability, comorbidities, functional status, personal preference, and possible hospice status. In patients with SVCS associated with small-cell lung carcinoma, nonrandomized trials have demonstrated that chemotherapy and/or radiation therapy relieves SVC occlusion in 77% of patients, with a recurrence rate of only 17%.57 For patients with SVCS associated with non-small-cell carcinoma (NSCC), 60% had relief of the SVC occlusion following chemotherapy and/or radiation therapy, and the rate of recurrence for occlusion was 19%.57 In general, surgical bypass is reserved for patients with nonmalignant causes of SVCS, whose better long-term prognosis justifies an invasive approach.47,53 On the other hand, endovascular stent is a less invasive and yet very effective alternative for cancer-related SVCS. This procedure is especially useful among patients with acute and severe symptoms for whom immediate treatment is needed, and for whom radiation or chemotherapy has failed.53 In the study of NSCC patients, placement of an endovascular SVC stent relieved SVC occlusion in 95% of patients, with a recurrence rate of 11%.57

The emergency clinician must bear in mind various key elements when caring for patients who present with malignancy-induced SVCS. Addressing the “ABCs” is always the priority, and particular attention should be paid to the patency of airway. In extreme cases, patients can lose their airway secondary to cerebral edema or stridor from SVCS. Elevating the patient’s head and providing supplemental oxygen are 2 simple measures that are generally recommended despite the lack of quality evidence to support their benefit.50 Diuretics often result in dehydration in this setting and have not been proven to be efficacious; therefore, they are not recommended for patients with SVCS. In addition, no evidence supports the use of empiric corticosteroids, and they should not be routinely administered in the ED (although they may be considered if a tissue diagnosis has been established and the tumor is known to be responsive to steroids). Consultation with the primary oncologist is recommended to assist with this decision.

If the SVCS is caused by the large acute or subacute thrombosis, the emergency clinician should consult with an interventional radiologist, who can give thrombolytic therapy directly into the thrombus by infusion catheters that are centered in the thrombus. The literature has not addressed the issue of the ideal location for IV access in patients with SVCS, but the authors recommend obtaining femoral access if a central line is needed. Internal jugular and subclavian central lines may further complicate SVCS with local thrombosis; however, since many of these patients only have partial SVC obstruction, and since those with complete obstruction usually have some degree of collateral circulation, upper-extremity peripheral venous access is generally sufficient.

SVCS Summary
The clinical signs and symptoms of SVCS are often subtle and subacute, and can easily divert the ED clinician from the true diagnosis. Consequently, it is not unusual for a patient with SVCS to present many
times before the correct diagnosis is established. The clinician must be astute and focus on the relative chronicity of symptoms in order to entertain the infrequent diagnosis of SVCS. Once the diagnosis is suspected, it can be confirmed by CT or MRI. Treatment in the ED should focus on an assessment of the patient’s airway and coordination of care with appropriate subspecialties to ensure the lowest morbidity and mortality after hospital admission.

Disposition Of SVCS Patients
Patients with respiratory compromise are best managed in the intensive care unit (ICU) setting. Coordination with other specialties (eg, radiation oncology) is essential before considering intervention to relieve the obstruction. Most patients will be clinically stable and can be given a regular floor bed on the appropriate medical service. In select cases, discharge may be appropriate if the primary doctor (eg, oncologist) is aware of the diagnosis of SVCS but judges that admission is not indicated (eg, for a hospice patient).

Malignant Pericardial Effusion And Cardiac Tamponade
Cardiac tamponade is one of the true emergencies in emergency medicine. The list of causes of cardiac tamponade is long and ranges from trauma to malignancy. With increased awareness of this complication and relatively easy access to bedside echocardiography, it is likely that emergency clinicians will be diagnosing more cases of malignant pericardial effusion and tamponade in the future. Although it is not necessary to diagnosis every case of pericardial effusion in the ED, it is crucial to identify cardiac tamponade promptly, since early intervention can be lifesaving.

Epidemiology Of Malignant Pericardial Effusion And Tamponade
Pericardial effusion is the most common complication associated with metastases to the heart and pericardium. One might believe that metastasis to the pericardium is a relatively uncommon phenomenon, but an autopsy series of patients with cancer reported by DeLoach and Haynes showed that 15% to 20% of patients had metastases to the heart and pericardium. The majority of these patients did not have a history of metastatic burden; in most cases, this complication remains clinically silent during a patient’s lifetime. Of the 137 patients with cardiac metastases, 22 had pericardial effusions but only 2 had symptoms attributable to the effusions. In contrast, another case series reported by Thurber et al demonstrated that 55 of 189 (29%) patients with pericardial metastases had symptoms believed to be related to the effusion. Once a malignant pericardial effusion has been diagnosed, median survival is estimated to be 2 months; only about 25% of patients live for an additional 2 years. Metastatic pericardial involvement is approximately 16 times more common than are primary tumors of the pericardium.

Theoretically, any cancer can metastasize to the pericardium. Cancers originating above the diaphragm are the most common sources of metastatic pericardial disease, with lung and breast cancer accounting for about half of all cases. Lymphoma, leukemia, and melanoma account for an additional 25% of metastatic disease. It is generally believed that the majority of patients with pericardial metastases have a known underlying cancer with metastatic disease in other organ systems. However, in a small case series of 23 patients with cardiac tamponade, Haskell et al found that 8 patients had cardiac tamponade as the first clinical manifestation of previously undiagnosed cancer. Seven patients were seen by a physician prior to the ultimate diagnosis of tamponade, and 6 patients were incorrectly diagnosed as having bronchitis, congestive heart failure, or pneumonia despite abnormal findings on chest radiograph that should have raised suspicion for malignancy. These findings reinforce the need for clinicians to consider pericardial effusion and tamponade in patients who present with signs and symptoms of concern.

Patients with an underlying malignancy can have a pericardial effusion that is not caused by the cancer. In addition to malignancies of idiopathic origin, other etiologies include radiation, medications, hypothyroidism, infection, end-stage renal disease, and autoimmune disorders. In roughly 20% to 40% of malignant pericardial effusions, the results of cytologic analysis are negative; therefore, experts recommend that a pericardial fluid sample be tested for protein, lactic dehydrogenase, hematocrit, glucose, cell count, and culture to aid in the diagnosis.

Pathophysiology Of Malignant Pericardial Effusion And Tamponade
The pericardium consists of 2 membranes: parietal and visceral. The visceral membrane is composed of a single layer of mesothelial cells that adhere to the myocardium, and the parietal membrane is a fibrous layer surrounding the heart. The fluid between these 2 membranes (up to 50 mL) functions as a physiologic lubricant. In order for a significant pericardial effusion to arise from a malignant lesion, 3 conditions must be present: (1) neoplastic invasion of the mediastinal or hilar lymph nodes; (2) local obstruction of lymphatic flow; and (3) implantation of the tumor on the serosal surface of the pericardial membranes, with subsequent exudation of fluid into the pericardial space.

Kline et al demonstrated that the sequence of events begins with mediastinal lymph node metastases from primary tumor sites. Retrograde migration
of cancer cells through the lymphatic system into the pericardium follows, with subsequent fluid production and accumulation within the pericardial sac. The shared lymphatic drainage of the chest structures into the hilar and mediastinal lymph nodes may explain why lung and breast cancers are the most common causes of pericardial metastases. Direct invasion and hematogenous metastases are also potential sources of cardiac and pericardial metastases and are associated with a significantly increased risk of dysrhythmia or tamponade.66

The development of clinical symptoms depends on the time course of fluid accumulation. Patients can develop symptoms with as little as 200 mL of pericardial fluid if accumulation is rapid — a situation typically seen in the setting of trauma. Symptomatic malignant effusions occur more insidiously, allowing the parietal pericardium to accommodate a greater volume of fluid. In a minority of patients, several liters of fluid may accumulate without subsequent tamponade, although the literature is not clear regarding the percentage of patients in whom such large effusions develop.64

Cardiac tamponade occurs when the pericardial fluid begins to impede cardiac diastolic filling, resulting in inadequate cardiac output. Unfortunately, no single sign or symptom has sufficient sensitivity or specificity to confirm or exclude this diagnosis. Patients may present in extremis or even in cardiac arrest due to tamponade.

Patients with cardiac tamponade may deteriorate rapidly; the emergency clinician must have a high index of suspicion for this diagnosis to facilitate its early recognition. The literature describes a “last-drop phenomenon,” in which the pressure exerted on the cardiac chambers by the accumulation of pericardial fluid reaches a critical level. The “last drop” is the tipping point at which just a small amount of additional fluid in the pericardial space causes sudden clinical deterioration because the cardiac chambers can no longer adequately fill to sustain the necessary cardiac output.67

History For Malignant Pericardial Effusion And Tamponade

The most common symptom in malignant pericardial effusion, by far, is dyspnea (91%). Thurber et al reported other common symptoms: cough (65%), thoracic pain (47%), and orthopnea (38%).69 Bishiniotis et al reviewed 19 patients with confirmed malignant pericardial effusion causing cardiac tamponade. Common symptoms were dyspnea (95%), orthopnea (58%), cough (47%), chest pain (42%), and peripheral edema (36%).69

If the patient is clinically stable, a thorough search should be undertaken to determine the underlying cause. Questions should specifically target a possible history of cancer or cancer-related symptoms (eg, cachexia, weight loss), thyroid disease, end-stage renal disease, autoimmune disease (eg, systemic lupus erythematosus, rheumatoid arthritis), infectious disease (eg, HIV infection, tuberculosis), a history of chest irradiation, and current medications.

Physical Examination For Malignant Pericardial Effusion And Tamponade

The majority of patients with pericardial effusion will present with relatively preserved blood pressure and variable symptoms and signs, including dyspnea, tachypnea, and tachycardia. Guberman et al reviewed the charts of 56 patients seen between 1963 and 1980 for cardiac tamponade of nontraumatic etiology (eg, malignancy, myocardial infarction).69 Almost two-thirds of the patients had an average systolic arterial blood pressure greater than 100 mm Hg, and almost all patients were stable and provided a thorough history. Contrary to common medical school teaching, these same authors reported that most patients with a gradual onset of tamponade did not present with Beck’s triad (hypotension, distant heart sounds, and jugular venous distention).69 This triad is found in less than 20% of cases of tamponade unrelated to trauma.

Pulsus paradoxus is an exaggeration of the normal variations in systolic blood pressure during the inspiratory component of respiration. It remains a key bedside diagnostic test for pericardial tamponade. (See Table 1.) In the Guberman study cited earlier, 53 of 56 patients with tamponade exhibited a documented decrease in systolic blood pressure of more than 10 mm Hg during inspiration.69 The mean drop in systolic blood pressure in the study group as a whole was 49 mm Hg; however, clinicians should be aware that pulsus paradoxus occurs in conditions other than tamponade, such as asthma, pulmonary embolus, obesity, and mitral stenosis. In a meta-analysis, Roy et al found the pooled sensitivity of pulsus paradoxus to be 82% for cardiac tamponade.70 In a retrospective review of 19 patients, Bishiniotis et al found that the most common signs of pericardial tamponade in women with breast cancer were pulsus paradoxus (79%), tachycardia (74%), jugular venous distention

<table>
<thead>
<tr>
<th>Table 1. Detecting Pulsus Paradoxus When Using A Manual Sphygmomanometer</th>
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<tr>
<td>Step 1: Note SBP when Korotkoff sounds are audible during expiration only.</td>
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<tr>
<td>Step 2: Note SBP when Korotkoff sounds are audible during both expiration and inspiration.</td>
</tr>
<tr>
<td>Step 3: Pulsus paradoxus present if: (SBP on expiration only) - (SBP on inspiration and expiration) &gt; 10 mm Hg</td>
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Abbreviation: SBP, systolic blood pressure.
(68%), hepatomegaly (63%), distant heart sounds (58%), and hypotension (53%). Kussmaul’s sign (26%) and friction rub (16%) were rare. In another study, the most common findings were evidence of pleural effusion (53%), hepatomegaly (51%), cyanosis (35%), and leg edema (31%). It is interesting to note that a friction rub and distant heart sounds were each found in only 1 patient (1.8%). To help explain this phenomenon, researchers have suggested that heart sounds may be preserved by neoplastic bridges between the visceral and the parietal pericardium. 

**Diagnostic Studies For Malignant Pericardial Effusion And Tamponade**

**Plain Radiography**
Chest radiographs are often abnormal in patients with cardiac effusions and may be the first sign to suggest this diagnosis. An enlarged cardiome-diastinal silhouette on posteroanterior and lateral radiographs can correlate with as little as 250 mL of pericardial fluid. Two studies involving a total of 139 patients reported this sign in 71% to 95% of patients with pericardial effusions. Other findings may include a globular (ie, “water-bottle”) heart shadow, irregular and nodular contours of the cardiac chambers, a pericardial fat stripe, mediastinal widening, hilar adenopathy, or a hilar mass. (See Figure 2.) In a study of malignant pericardial effusion and tamponade, Bishiniotis et al reported that 95% of patients had a cardiothoracic ratio above 0.5. As shown in Figure 3, this is the ratio of the transverse diameter of the heart (distance from A to B) to the greatest internal diameter of the thorax (distance from C to D) on a posteroanterior chest film. They also identified pleural effusions and malignant parenchymal disease in 68% and 42% of patients, respectively.

**Electrocardiography**
Patients who present with pericardial effusions and tamponade frequently have abnormal electrocardiographic findings. In 1962, a retrospective autopsy review of 189 patients showed that 27 with obtainable ECGs had “significant” pericardial metastatic lesions; for those patients with metastatic lesions in the pericardium, the findings included T-wave abnormalities in 25 (93%), low QRS voltage in 22 (88%), and sinus tachycardia in 15 (56%). Direct tumor invasion of the atria can lead to atrial fibrillation, atrial flutter, and premature atrial contractions. Note that these dysrhythmias may be resistant to typical medical therapy. Electrical alternans — ie, beat-to-beat variations in the direction, amplitude, or duration of any component of the ECG — has classically been associated with pericardial effusion and tamponade, but may also be found in other conditions, such as hypertrophic cardiomyopathy. (See Figure 4.) Total electrical alternans, in which...


![Figure 3. The Cardiothoracic Ratio](Reprinted from Journal of Cardiology, Vol. 54(1), Kunihisa Miwa and Masatoshi Fujita, Cardiac function fluctuates during exacerbation and remission in young adults with chronic fatigue syndrome and “small heart.” Pages 29-35, Copyright 2009, with permission from Elsevier.)
the P wave, QRS complex, and T wave all exhibit beat-to-beat variability, is rare (5% to 10%) and is seen most commonly with large pericardial effusions. 61

**Echocardiography**

The standard diagnostic tool for confirming cardiac tamponade is echocardiography (a Class I recommendation by the American College of Cardiology). 62 (See Figure 5.) A growing number of emergency clinicians are now facile using ultrasound to detect pericardial effusion; however, the presence of a pericardial effusion does not always equal a diagnosis of cardiac tamponade. Echocardiographic findings suggestive of cardiac tamponade include early diastolic collapse of the right ventricle and late diastolic collapse of the right atrium. 73 Experienced echocardiographers may visualize abnormal diastolic movement of the anterior mitral valve leaflet. One great benefit of using sonography in the ED setting is that if the clinician is confident that the pericardium has been adequately visualized, absence of an effusion may be enough to rule out this diagnosis.

**Treatment For Malignant Pericardial Effusion And Tamponade**

The initial management of a patient suspected of having cardiac tamponade begins immediately, with the insertion of 2 large-bore IV lines for aggressive volume resuscitation using normal saline or Ringer’s lactate. Patients with tamponade become preload-dependent, so agents that reduce preload (eg, nitroglycerin) should be strictly avoided. If volume resuscitation is not immediately successful in stabilizing the patient, pericardiocentesis should be performed.

**Pericardiocentesis**

For patients in imminent danger of cardiac arrest, pericardiocentesis may be done blindly. The ster-

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**Figure 4. ECG Showing Electrical Alternans In A Patient With Pericardial Effusion**

ECG of patient with pericardial effusion showing tachycardia, low voltage, and electrical alternans (adapted by permission from www.askdrwiki.com)

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**Figure 5. Echocardiogram Of Large Pericardial Effusion With Tamponade**

Abbreviations: RV, right ventricle; LV, left ventricle.

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the track of the needle, it is prudent to choose this method whenever possible (a Class IIa recommendation by the American College of Cardiology). Echocardiography-guided pericardiocentesis appears to be the safest approach, with no fatalities reported during more than 600 attempts, according to a study by Callahan et al. In another study of 32 such procedures, the success rate was 97%, with a complication rate of 12.5% (generally due to cases of pneumothorax or ventricular lacerations). An added benefit of ultrasound is that the Seldinger technique can be used to insert a catheter to allow continuous drainage once the location of the needle is confirmed to be within the pericardial sac. Although no studies have formally assessed the usefulness of this technique, a triple-lumen central venous catheter can be placed to facilitate drainage if a pericardial drain kit is unavailable. If a patient with cardiac tamponade or pericardial effusion suffers a cardiac arrest, emergent pericardiocentesis may revive the patient and restore circulation.

**Examination Of The Pericardial Fluid**

Pericardiocentesis should be considered both a diagnostic and a therapeutic procedure in all patients with nontraumatic tamponade. As much fluid as possible should be aspirated to improve the patient’s hemodynamic status and to maximize yield for cytologic examination to detect malignant cells. Cytology is believed to be positive in 60% to 80% of patients with a malignant pericardial effusion. Cytology will assist in the determination of a primary cancer and will therefore indicate the patient’s prognosis. For example, breast cancer patients diagnosed with a pericardial effusion may survive 10 to 13 months, whereas patients with an effusion associated with lung cancer may survive less than 6 months.

In addition to cytologic analysis, routine studies of the pericardial fluid should include a cell count and differential, hematocrit, bacterial culture, and levels of protein, glucose, and lactic dehydrogenase. The color of the pericardial fluid sample is not an accurate predictor of the cause of the effusion. Malignant effusions may be serous, serosanguineous, or grossly bloody. If bloody, the fluid should not easily form a clot, and the fluid hematocrit should be lower than the serum hematocrit. If the fluid clots easily and the hematocrit is similar to the serum hematocrit, consider the possibility that an intracardiac sample was inadvertently obtained. If the patient is stable enough to allow echocardiogram-guided pericardiocentesis, a catheter should be inserted and left in the pericardial space to allow continued drainage. After the catheter is sutured to the skin, it may be affixed to a 3-way valve or Hemovac®. Sterile technique should always be used to avoid infectious complications.

If drainage of the pericardial fluid does not alleviate symptoms or improve the patient’s hemodynamic status, the clinician should consider the possibility of an alternative diagnosis or procedural failure (eg, improper placement of the catheter in the pericardial space). When mechanical ventilation is being considered or has already been instituted for possible cardiac tamponade, the emergency clinician should be aware that positive-pressure ventilation may impede venous return and further reduce preload.

Once the patient has been stabilized, consultation with other hospital services — oncology (in cases of known malignancy), cardiology, and surgical services — is advised to thoroughly plan any intervention. Whenever pericardiocentesis is the sole treatment modality, malignant effusions are reported to recur at a rate of 56%, often within 24 to 48 hours. Consequently, these patients should be offered other interventions that may reduce the recurrence rate. The patient’s oncologist and the consulting surgeon may base their decision for intervention on the patient’s functional status, the specific characteristics of the tumor, and overall prognosis. Procedures that are usually considered include thoracotomy with pericardectomy; thoracotomy with a pericardial, subxiphoid, or transcutaneous-balloon pericardial window; local infiltration of sclerosing agents or chemotherapeutic agents; systemic chemotherapy; and radiation therapy. Minimally invasive approaches should be considered for patients with poor functional status.

The success of any of these procedures is measured by the 30-day survival rate and the absence of pericardial effusion during those 30 days. Given the relatively poor prognosis associated with malignant pericardial effusion, a thorough discussion with the family and the patient should be undertaken by the consulting services to ensure that invasive or risky operative interventions are warranted.

**Summary For Malignant Pericardial Effusion And Tamponade**

Although the presentation in the ED of a patient with acute cardiac tamponade can be a frightening event, most cases of nontraumatic cardiac tamponade tend to be more insidious. Medical histories are often vague, and the “typical” complaints of dyspnea, cough, and orthopnea generate a broad differential diagnosis. Findings on physical examination that are suggestive of, but not definitive for, cardiac tamponade include pulsus paradoxus, tachycardia, jugular venous distention, hepatomegaly, distant heart sounds, and hypotension. The serious consideration of tamponade within the differential diagnosis is the first step in diagnosing and managing this oncologic emergency. Routine laboratory tests are of little help in this process, but the ECG and chest film are of value. Low-voltage or electrical alternans on the ECG, coupled with a markedly enlarged cardiac silhouette on a chest film, should prompt the
emergency clinician to perform or request urgent bedside echocardiography. Although many emergency clinicians are capable of detecting a large pericardial effusion and signs of tamponade, a cardiologist’s interpretation is often necessary to confirm a subtle diagnosis of tamponade by means of formal echocardiography. If a patient with a pericardial effusion becomes markedly unstable or if tamponade is grossly visible on the echocardiogram, the clinician needs to be familiar with the equipment, supplies, and steps required to perform pericardiocentesis. It is important to reiterate that for all stable patients, consultation with other hospital services (cardiology, oncology [if appropriate], and surgery) will allow the team to thoroughly coordinate any interventions and inpatient care.

Disposition Of Patients with Malignant Pericardial Effusion And Tamponade

All patients who are diagnosed with cardiac tamponade in the ED should be admitted to the ICU for close monitoring until definitive treatment can be delivered. Prior to admission to the ICU, some patients may be sent directly to surgery or to the angiography department for interventional treatment. Any patient who undergoes pericardiocentesis in the ED will require intensive care to monitor for signs of decompensation. Patients who are believed to have a stable pericardial effusion without cardiac tamponade can be considered for admission to the medical floor if no coexisting conditions dictate a higher level of care.

Case Conclusions

During the emergent intubation of your patient with SVCS, laryngeal edema was evident. Her post-intubation chest x-ray shows opacity, likely representing her underlying tumors. Her head CT shows diffuse cerebral edema without herniation or abnormality, and her chest CT with intravenous contrast reveals a tumor compressing the SVC and an extensive clot burden within the vessel. The patient is admitted to the ICU, and specialists in radiation oncology and interventional radiology are consulted. On the same day, the patient is taken to the interventional radiology suite where a local thrombolytic agent is infused at the site of thrombosis and a stent is placed in the SVC. Within 24 hours of the intervention, her facial and arm swelling improves significantly. A few days later, the patient’s mental status and laryngeal edema has improved to the extent that she can be extubated. Her quality of life was improved with this treatment of SVCS and her palliative chemotherapy was continued, to maximize valuable time with her loved ones.

The family of your 45-year-old male arrives and states that he has a history of lung cancer. The ECG demonstrates low-voltage amplitudes in all leads. This finding, in conjunction with this patient’s clinical presentation and history, makes you suspect cardiac tamponade. You wheel over the bedside ultrasound equipment, and the echocardiogram reveals an extremely large pericardial effusion with what appears to be very restricted ventricular activity. The patient’s hypotensive state improves within 10 minutes after bedside pericardiocentesis yields 400 mL of bloody fluid. The cardiothoracic surgeon arrives at the bedside and consults with the family regarding the possible creation of a pericardial window to more definitively treat his condition. Your high level of suspicion for this catastrophic complication of malignancy and quick actions averted what could have been a lethal situation.

Summary

The presentation and management of oncologic emergencies are infrequently encountered by most emergency clinicians; however, what were once thought to be relatively rare presentations in the ED are now becoming more common as patients with cancer are living longer and receiving a broad array of treatments. The presentation of SVCS can be subtle and easily missed if the physician does not suspect this diagnosis. Patients with epidural metastases and cardiac tamponade may present in a more dramatic fashion and will require prompt intervention to achieve a favorable outcome. It is important to remember that such patients often have other serious comorbidities that contribute to their poor clinical status. In conclusion, this review highlights the importance of obtaining a thorough history, maintaining a high index of suspicion for these 3 complications, and acting quickly on these concerns for cancer patients who present to the ED.

Acknowledgments

The authors thank Linda Kesselring for her assistance in the preparation of this article, and Anis Frayha, MD for helping to acquire the images.

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To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study, will be included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, will be noted by an asterisk (*) next to the number of the reference.


**Risk Management Pitfalls For Oncologic Emergencies (continued on page 17)**

1. “Even though the patient whom I just diagnosed with malignant spinal cord compression wasn’t able to walk into the ED, I’m going to tell the family that he should be all right once treatment is initiated.”

   The neurologic status of patients before treatment for ESCC is the most important prognostic factor for neurologic status after treatment. Understanding the significant prognostic implications of the patient’s presenting neurologic status should help guide your discussions with patients and their families.

2. “This lady has ESCC. She needs to get radiation now!”

   Radiation therapy is not indicated in all patients. Its greatest benefits are seen in the following situations: recurrences following surgical decompression, no spinal compression or instability, subclinical cord compression, radiosensitive tumors, or in patients who are not candidates for surgery.

3. “He said his back has been hurting for 2 months, making it difficult for him to walk, but he appeared fine to me in the ED and had a normal x-ray.”

   A new diagnosis of an underlying malignancy is made in 20% of patients who present with ESCC, so clinical suspicion must always remain high for any patient with relevant symptoms. Back pain is the first symptom in 95% of patients with ESCC, and it usually predates all other symptoms by up to 2 months.

4. “I know he had lung cancer, but his back pain doesn’t seem too bad. I’ll just get an x-ray and send him home with some acetaminophen if it’s normal.”

   Not seriously considering ESCC in the differential diagnosis in any cancer patient with back pain is a recipe for disaster. Also, relying on a plain radiograph alone for the diagnosis of ESCC is inadequate. Since plain radiography is only 75% sensitive for ESCC even when vertebral collapse is evident, MRI is the study of choice.

5. “I couldn’t tell the patient’s face was swollen. I’ve never seen him before and I thought he always looked like that.”

   Although facial swelling is often obvious, it can be overlooked by one who is not familiar with the patient’s usual appearance. The emergency clinician must obtain a thorough history to elicit important clues, such as worsening of the swelling in the morning or a sense of facial fullness when bending down. Consider involving family or friends for their opinion or look at a patient’s driver’s license for comparison.

6. “I knew that the patient had SVCS, but it was subacute and I thought that she could follow up with her oncologist to take care of it. I
didn’t know that it could progress to stridor or cerebral edema.”

Their exact incidence is not known, but the 2 most feared complications of this syndrome are upper-airway obstruction and cerebral edema, both of which are attributable to diminished venous return to the head and neck. Even if the presentation is subacute, treatment should be provided promptly to avoid these complications. Given the coordination required among multiple disciplines, it is reasonable to admit most patients with SVCS.

7. “There is no facial edema and the chest film is normal — this patient doesn’t have SVCS.”

About 20% of patients with SVCS have no facial or neck edema, and about 20% have normal chest films. When abnormal radiographic findings are present, they usually include evidence of lung masses. Further imaging is warranted if SVCS continues to be suspected.

8. “A chest x-ray without an enlarged cardiac silhouette essentially rules out a pericardial effusion.”

In the case of malignant pericardial effusion, the effusion is usually large in volume, owing to the subacute nature of the fluid accumulation. The pericardium therefore has enough time to stretch and accommodate a large amount of fluid without affecting hemodynamic status. The classic “water-bottle” heart on chest x-ray certainly alerts physicians to the possibility of this diagnosis, but the lack of this finding is insufficient to rule out the diagnosis.

9. “After the pericardiocentesis, my patient was doing so well. He said he could follow up with his oncologist a week later, so I let him go home. I didn’t realize that fluid could reaccumulate that quickly!”

When pericardiocentesis is the sole treatment modality for a malignant pericardial effusion, it can recur at a rate of 56%, often within 24 to 48 hours. Patients should be admitted for close observation and a repeat echocardiogram. In cases of recurrent malignant pericardial effusions, more definitive treatment, such as a subxiphoid pericardial window, should be considered in consultation with the cardiothoracic surgery team.

10. “But the patient initially had a stable blood pressure. Nobody would have suspected cardiac tamponade.”

Initially, many patients with nontraumatic cardiac tamponade present with preserved blood pressure; however, they can deteriorate quickly because of the “last drop” phenomenon. Beck’s triad is rarely present in such patients. For any patient with a pulmonary malignancy (primary tumor or metastasis) who presents with heart failure-like symptoms, cardiac tamponade is often detected only by emergency clinicians who are alert for this possible diagnosis.


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1. **What is the most important prognostic factor for neurologic outcome in a patient with ESCC?**
   a. Back pain on recumbency
   b. Bladder dysfunction
   c. Night pain
   d. Pre-treatment neurologic function
   e. Type of cancer

2. **Which of the cancers listed below least frequently involves the bones?**
   a. Breast
   b. Lung
   c. Pancreas
   d. Prostate
   e. Multiple myeloma

3. **What is the diagnostic modality of choice for suspected malignant ESCC?**
   a. Bone scintigraphy
   b. CT scan
   c. MRI
   d. Physical examination
   e. Plain radiography

4. **What initial steroid type and dose is used for ESCC without evidence of myelopathy on examination?**
   a. Dexamethasone, 10 mg IV
   b. Dexamethasone, 50 mg IV
   c. Dexamethasone, 100 mg IV
   d. Prednisone, 40 mg PO
   e. Prednisone, 60 mg PO

5. **What is the most common tumor for malignancy-related SVCS?**
   a. Breast
   b. Esophageal
   c. Head and neck
   d. Lung
   e. Lymphoma
6. What is the diagnostic mode of choice for SVCS?
   a. Chest CT scan with IV contrast
   b. Chest CT scan without IV contrast
   c. Chest MRI
   d. Chest x-ray
   e. Physical examination

7. What is the next appropriate clinical step following a new diagnosis of SVCS due to a previously undiagnosed cancer?
   a. Consultation to assist with a tissue diagnosis prior to definitive treatment
   b. Consult oncologist for emergency chemotherapy
   c. Consult radiation oncologist for emergency radiation treatment
   d. Consult thoracic surgeon for bypass surgery

8. What is the most common symptom in patients with malignant pericardial effusions?
   a. Chest pain
   b. Cough
   c. Dyspnea
   d. Lightheadedness
   e. Palpitations

9. What percentage of patients with a malignant pericardial effusion have a positive result on pericardial fluid cytology?
   a. 20% to 40%
   b. 60% to 80%
   c. 100%

10. What is the recurrence rate of malignant pericardial effusions following pericardiocentesis?
    a. 0% to 10%
    b. 30% to 40%
    c. 40% to 50%
    d. 50% to 60%
    e. 80% to 90%
### Key Points

| Early suspicion of epidural spinal cord compression (ESCC) is fundamental in timely diagnosis and treatment, as clinical outcomes are dependent on the functional status of the patient at the time of diagnosis. | Neurologic status of a patient prior to treatment for ESCC is the most important prognostic factor for neurologic status after treatment.¹⁹ |
| MRI of the entire spine is appropriate in most circumstances for the diagnosis of ESCC. | The added benefits of a non-invasive modality, cost efficiency,²¹ and the ability to image the entire spine for ESCC, intramedullary lesions, and bony metastases make MRI the study of choice for patients with suspected ESCC. Imaging the entire spine is considered extremely valuable because approximately one-third of all patients diagnosed with ESCC have multiple sites of epidural metastases.²²,²³,⁶⁶,⁶⁸,⁷⁷ |
| Patients with superior vena cava syndrome (SVCS) often present with subacute complaints, and diagnosis can be further delayed without careful history and examination. | In general, sudden presentations are the exception, making the diagnosis more difficult. Delayed presentations are more of the rule, with more than 75% of patients having signs and symptoms of SVCS for greater than 1 week before seeking medical attention.⁴² Furthermore, it is not uncommon for patients to complain of SVCS-related symptoms for weeks, or even months.⁴³ |
| The preferred ED radiographic modality to diagnose SVCS is a chest CT with IV contrast. | This modality is preferred in the emergency setting for multiple reasons: it has wide availability, it provides both anatomic details of the extent of obstruction and collateral flow, and it can potentially identify causes of the syndrome (compression vs in-dwelling catheter-related disease). |
| Most malignant pericardial effusions are asymptomatic. | In DeLoach’s case series of 137 patients with cardiac metastases, 22 developed pericardial effusions, but only 2 of the patients had symptoms attributable to the effusions.⁴⁶ |
| Breast and lung cancers account for about half of pericardial metastases. | Theoretically, any cancer can metastasize to the pericardium. Cancers originating above the diaphragm are the most common sources of metastatic pericardial disease, with lung and breast cancer accounting for about half of all cases. Lymphoma, leukemia, and melanoma account for an additional 25% of metastatic disease.⁵² |
| Bedside echocardiography is a key instrument for both diagnostic and therapeutic use in cases of cardiac tamponade. | Echocardiographic findings suggestive of cardiac tamponade include right ventricular early diastolic collapse and right atrial late diastolic collapse.⁶² More experienced echocardiographers may visualize abnormal diastolic movement of the anterior mitral valve leaflet. A great benefit of the use of sonography in the ED setting is that if a physician is confident that adequate visualization of the pericardium has been obtained, then the absence of a pericardial effusion may “rule out” this diagnosis. |
REFERENCES

42. Young RF, Post EM, King GA. Treatment of spinal epidural metastases. Randomized prospective comparison of laminectomy and radiotherapy. J Neurosurg. 1980;53:741-748. (Prospective, randomized study; 29 patients)

CLINICAL RECOMMENDATIONS

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