Nonvascular, Nontraumatic Mediastinal Emergencies in Adults: A Comprehensive Review of Imaging Findings

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Given their high frequency, mediastinal emergencies are often perceived as being a result of external trauma or vascular conditions. However, there is a group of nonvascular, nontraumatic mediastinal emergencies that are less common in clinical practice, are less recognized, and that represent an important source of morbidity and mortality in patients. Nonvascular, nontraumatic mediastinal emergencies have several causes and result from different pathophysiologic mechanisms including infection, internal trauma, malignancy, and postoperative complications, and some may be idiopathic. Some conditions that lead to nonvascular, nontraumatic mediastinal emergencies include acute mediastinitis; esophageal emergencies such as intramural hematoma of the esophagus, Boerhaave syndrome, and acquired esophagorespiratory fistulas; spontaneous mediastinal hematoma; tension pneumomediastinum; and tension pneumopericardium. Although clinical findings of nonvascular, nontraumatic mediastinal emergencies may be nonspecific, imaging findings are often definitive. Awareness of various nonvascular, nontraumatic mediastinal emergencies and their clinical manifestations and imaging findings is crucial for making an accurate and timely diagnosis to facilitate appropriate patient management.

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Abbreviations: AEF = aortoesophageal fistula, DNM = descending necrotizing mediastinitis, IHE = intramural hematoma of the esophagus

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**Introduction**

Among adults who visit emergency departments, as many as 5% present with acute chest pain (1). As many as 30% of adults who present with acute chest pain receive a diagnosis of acute myocardial infarction or unstable angina (2). Other mediastinal causes of acute chest pain include blunt or penetrating trauma and vessel-related conditions such as pulmonary thromboembolism and acute aortic syndromes. Although they are less common, some nonvascular, nontraumatic mediastinal conditions also may cause acute chest pain, such as acute mediastinitis; esophageal emergencies such as intramural hematoma of the esophagus (IHE), Boerhaave syndrome, and acquired esophagorespiratory fistula; spontaneous mediastinal hematoma; tension pneumomediastinum; and tension pneumopericardium (Table). These conditions demonstrate characteristic pathophysiologic and imaging findings at projectional and cross-sectional imaging, a feature that helps in making a timely diagnosis and providing optimal patient management.

In this article, we discuss the types of nonvascular, nontraumatic mediastinal emergencies that may occur in adults, as well as the pathophysiologic, projectional, and cross-sectional imaging findings of these conditions.

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**Imaging Approach**

Chest radiography is the first imaging examination that is performed to evaluate patients with suspected nonvascular, nontraumatic mediastinal emergencies. Both anteroposterior and lateral views of the chest are obtained. Findings seen at chest radiography are considered diagnostic in patients with pneumomediastinum or pneumopericardium, although it may be difficult to identify a tension component of these conditions at radiography (2). In addition, conditions such as acute mediastinitis, esophageal emergencies, and mediastinal hematomas may be indicated on the basis of chest radiographic findings. Contrast esophagography is indicated in patients with suspected IHE, esophageal perforation, or esophagorespiratory fistula. The use of hydrosoluble contrast material is preferred over barium in these patients, and computed tomography (CT) is the imaging modality of choice. It is readily available, rapid, noninvasive, easy to perform, and useful in detecting various mediastinal emergencies. CT is indicated for patients with chest radiographic findings indicative of mediastinal conditions and those with normal chest radiographic findings but other strong indications of mediastinal conditions. In a typical CT protocol for evaluating mediastinal conditions, 1.5-mm section thickness and 3-mm reconstruction intervals are used. Intravenous contrast material is routinely used because many patients present with chest pain, and vascular abnormalities are often suspected. The use of oral contrast material is recommended in patients suspected of having esophageal conditions (3). With the advent of multidetector CT, coronal and sagittal reconstructions are readily available and are most useful for evaluating mediastinal emergencies. In addition, CT provides guidance for draining mediastinal fluid collections and aspirating mediastinal and pericardial air. Magnetic resonance (MR) imaging is rarely required for evaluation of nonvascular, nontraumatic mediastinal emergencies.

**Acute Mediastinitis**

Acute mediastinitis is a life-threatening condition with high mortality and morbidity. It results from acute inflammation of the connective tissues and fat surrounding the mediastinal structures. Acute mediastinitis may be classified, on the basis of its cause, as being postoperative, secondary to esophageal perforation, a result of the spread of osteomyelitis from adjacent bone, a direct extension of head and neck infection, or a result of hematogenous spread of infection (4). It is important to know the cause of acute mediastinitis because the prognosis may vary depending on the cause. Most cases of acute mediastinitis are secondary to...
Figure 1. Sternal dehiscence after median sternotomy. (a) Anteroposterior chest radiograph shows lateral displacement of sternal wires (arrows) and a radiolucent stripe (arrowheads) in the sternal body, findings indicative of sternal dehiscence. (b) Contrast-enhanced CT image shows an 11-mm gap (arrowhead) between the sternal halves, a finding consistent with sternal dehiscence.

Postoperative complications and esophageal perforation (5). Although there are some common findings among the different types of acute mediastinitis, the imaging appearances of acute mediastinitis may vary depending on the cause.

Clinically, patients present with acute chest pain, high fever, chills, shortness of breath, and leukocytosis. Patients with a cervical infection may have neck swelling, dysphagia, and sore throat. Chest radiographic findings of acute mediastinitis include widening and loss of normal contours of the mediastinum, diffuse or focal gas bubbles within the mediastinum, and focal mediastinal fluid collections (4,6). The diagnosis of acute mediastinitis may be confirmed and the extent of disease determined at CT, the imaging modality of choice for evaluating this condition.

Common CT findings of acute mediastinitis include increased attenuation of mediastinal fat, free gas bubbles in the mediastinum, localized fluid collections, enlarged lymph nodes, pleural effusions, and empyema (7). Additional findings that depend on the cause of the condition are discussed in the following sections. Acute mediastinitis is treated with both surgical and medical methods, depending on the cause of the infection and geared toward its primary cause and the patient’s clinical presentation (5).

Postoperative Complications
Before the development of modern cardiovascular surgery, esophageal perforation and spread of infection from the head and neck were the most common causes of acute mediastinitis. Currently, most cases of acute mediastinitis result from complications of cardiovascular or other thoracic surgical procedures (8). Acute mediastinitis occurs in 0.5%-5% of patients who underwent median sternotomy and has a reported mortality rate of 7%-80% (4,9). Staphylococcus aureus is the most common causative microorganism of postoperative acute mediastinitis. Obesity, insulin-dependent diabetes, and internal mammary artery grafts (especially bilateral grafts) are important risk factors for the development of a postoperative infection. Clinically, patients may present with postoperative fever and signs of local inflammation at the surgical site. Sternal dehiscence is frequently associated with the development of acute mediastinitis. Radiographic findings of sternal dehiscence include displacement, rotation, and fracture of the sternal wires on serial frontal chest radiographs. Rarely, a midsternal lucent stripe may also be seen. This finding is indicative of dehiscence only if the stripe is thicker than 3 mm; the presence of a midsternal lucent stripe on axial CT images that is thicker than 3 mm is indicative of dehiscence (Fig 1) (10). In patients with dehiscence, CT findings of acute mediastinitis include air or fluid collections and free air in the mediastinum (Fig 2). CT also may show pleural and pericardial effusions, enlarged lymph nodes, and sternal and lung parenchymal abnormalities (4). The presence of mediastinal gas bubbles and fluid collections on CT images after the 14th postoperative day has sensitivity and specificity approaching 100% for the diagnosis.
of acute mediastinitis (9). In addition, CT may be used to help guide diagnostic and therapeutic aspiration of fluid collections.

**Esophageal Perforation**

Esophageal rupture of various causes also may lead to acute mediastinitis (4,6). Esophageal perforation may be traumatic (eg, iatrogenic—resulting from surgical, endoscopic, or radiologic procedures—or a result of ingestion of foreign bodies), neoplastic (eg, esophageal or lung cancers), or spontaneous (eg, Boerhaave syndrome) (4). Clinical features of esophageal perforation include chest pain, vomiting, and subcutaneous emphysema. Contrast-enhanced esophagography may demonstrate extravasation of contrast material into the mediastinum, although false-negative findings are present in as many as 10% of patients (11). CT findings of esophageal rupture include focal esophageal wall thickening, periesophageal fluid collections, free mediastinal air, and contrast extravasation into the mediastinum and pleural space (Fig 3) (4,6). The survival rate for esophageal perforation is as high as 80% if primary closure is performed within a day after the perforation occurs (4). Delayed diagnosis and treatment are associated with a poor prognosis and high mortality rate (12). As was reported in a recent study by Shaker et al (13), early diagnosis and treatment (within 24 hours) are crucial for a successful outcome in patients with rupture of the esophagus. In this study, the mortality rate
among patients with a delayed diagnosis was 40%, compared with 6.2% among patients who received a diagnosis and treatment within 24 hours. The mortality rate of acute mediastinitis secondary to esophageal rupture may be as high as 90% if repair of the rupture is delayed for longer than 48 hours (14).

Extension of Osteomyelitis from Adjacent Bone
Acute sternal osteomyelitis and septic arthritis of the sternoclavicular joint may spread into the adjacent mediastinum, resulting in acute mediastinitis (4,10,15). This type of acute mediastinitis is extremely rare, and no exact prevalence has been reported in the literature (4). Risk factors for primary sternal osteomyelitis include intravenous drug use, acquired immunodeficiency syndrome (AIDS), and other immunodeficiency conditions (10). *Staphylococcus aureus* infection is the most common cause of this type of acute mediastinitis, although other bacterial and fungal infections also may be responsible.

Septic arthritis of the sternoclavicular joint is commonly seen in patients with diabetes or rheumatoid arthritis and in those who use intravenous drugs (10). *Staphylococcus aureus* and *Pseudomonas aeruginosa* are the most common causative organisms. Clinically, patients present with fever, swelling, and redness over the anterior chest wall. CT may depict cortical destruction of the sternum and the adjacent sternoclavicular joint, widening of the joint space, irregular sternum contours, peristernal soft-tissue swelling, and gas or fluid collections in the mediastinum (Fig 4) (4,10,15).
Direct Extension of a Head and Neck Infection (Descending Necrotizing Mediastinitis)

Descending necrotizing mediastinitis (DNM) is an acute, polymicrobial infection of the mediastinum that spreads from oropharyngeal, cervical, and odontogenic infections. It is a serious condition, with reported mortality rates ranging from 30% to 50% (16). DNM is also a rare condition and was first reported by Pearse (17) in 1938. Brunelli et al (18) and Corsten et al (19) reported 69 cases of DNM from 1960 to 1995. Continuity of the fascial planes between the neck and mediastinum may allow infection to spread from the oral cavity and neck into the mediastinum, which leads to acute mediastinitis (4). There are three potential pathways for the spread of infection from the neck to the mediastinum: the pretracheal route to the anterior mediastinum, the lateral pharyngeal route to the middle mediastinum, and the retropharyngeal-retrovisceral route to the posterior mediastinum. Of these, the most common route is by way of the retropharyngeal-retrovisceral space (16). The “danger space” is located posterior to the retropharyngeal space and is separated by the alar fascia (Fig 5) (16). It extends from the base of the skull to the level of the diaphragm, providing a pathway into the posterior mediastinum and pleural spaces. Neck radiography may depict subcutaneous emphysema, prevertebral soft-tissue swelling, mediastinal air, and widening of the superior mediastinum (4,16). In addition to mediastinal air and fluid collections, neck CT findings of DNM include thickening of the subcutaneous tissues in the neck, thickening or enhancement of cervical fascia and muscles, fluid collections, and enlarged lymph nodes (Fig 6) (16,20). Establishing pathways for the spread of infection from soft tissues in the neck to the mediastinum is key in diagnosing DNM (21). On the basis of CT findings, Endo et al (22) proposed a classification system to define the extent of disease and aid patient management. In this system, type 1 disease is localized above the carina, type 2A disease extends to the lower anterior mediastinum, and type 2B disease extends to the anterior and posterior mediastinum (22). Although patients with type 1 disease may not always require aggressive mediastinal drainage, this is not the case in patients with type 2 disease: Those with type 2B disease must undergo complete mediastinal drainage and débridement via thoracotomy, and those with type 2A disease may benefit from subxiphoid mediastinal drainage without sternotomy (22).
Hematogenous Spread of Infection

Rarely, acute mediastinitis may result from hematogenous spread of an extrathoracic infection such as septic arthritis (5). This type of acute mediastinitis occurs in those who abuse intravenous drugs, are immunocompromised, and who have chronic debilitating disease (5,8). At CT, mediastinal air and multiple fluid collections commonly are seen in the anterior and posterior mediastinum. Fluid collections may extend into the soft tissues of the chest wall (Fig 7). There are no definitive imaging findings that pinpoint extrathoracic infection as the cause of mediastinitis; however, histologic analysis of microbial organisms obtained from mediastinal and extramedias-
tinal sites may help establish a diagnosis.

Esophageal Emergencies

Intramural Hematoma of the Esophagus

IHE is a rare condition characterized by hemorrhage within the esophageal wall and formation of a localized hematoma. No precise prevalence of IHE is reported in the literature. It is part of the spectrum of esophageal injuries, which include mucosal (eg, Mallory-Weiss tear) and transmural esophageal (eg, Boerhaave syndrome) injuries (3). A hemorrhagic episode within the submucosal layer of the esophagus is the first event in the development of IHE. Continued bleeding leads to formation of a hematoma, which may superiorly and inferiorly dissect the submucosa (3,23). The distal esophagus is the most common site of IHE because of its lack of striated muscle and support from adjacent structures such as the trachea and
Figures 8, 9. (8) Concentric type of IHE. Drawings of the axial (left) and lateral (right) views of the neck show a concentric IHE (red areas), which causes enlargement of the esophagus and luminal narrowing (arrow). (9) Eccentric type of IHE. Drawings of the axial (left) and lateral (right) views of the neck show an eccentric hematoma (red areas) within the esophageal wall, which leads to lateral displacement of the esophageal lumen (arrow).

IHE may be concentric or eccentric depending on the extent of the hematoma within the esophageal wall (Figs 8, 9) (3). IHE may be classified into the following five subtypes on the basis of the nature of the hemorrhage: traumatic (eg, postendoscopic and food-induced trauma), emetogenic (eg, an intermediate stage between a Mallory-Weiss tear and Boerhaave syndrome), abnormal hemostasis–related (eg, hemophilia, anticoagulation, and renal failure), aorta-related (eg, aortoesophageal fistula), and spontaneous (ie, with no identifiable cause) (3). IHE commonly occurs in middle-aged patients, and it has a slight female predominance. Acute chest pain, dysphagia, and odynophagia are the most common presenting symptoms (3,25). Hematemesis is usually a late symptom of IHE and is indicative of rupture of the mucosal surface.

Typically, no abnormalities are seen at chest radiography; however, widening of the mediastinum and a hyperlucent, elongated mass may be seen in the retrocardiac region in an expected anatomic location in the esophagus (Fig 10a) (3). At water-soluble contrast-enhanced esophagography, a well-defined filling defect is seen in the presence of a focal hematoma, a “double-barreled esophagus” secondary to communication between the hematoma and esophageal lumen, or narrowing of the esophageal lumen due to a large submucosal mass (3,23,26). Intramural dissection of the esophagus may create false and true esophageal lumina, which are separated by a radiolucent mucosal stripe and give rise to the so-called double-barreled esophagus. Intramural dissection of the esophagus may progress to frank esophageal rupture with extravasation of contrast material (Fig 10b) (27). CT is the modality of choice for evaluation of IHE and typically depicts symmetric or asymmetric esophageal wall thickening and a well-defined, nonenhancing, high-attenuation intramural esophageal mass that extends along the esophageal wall (Fig 10c, 10d) (3,28,29). Attenuation values of the hematoma may vary depending on its age. CT also may depict air within the hematoma, a finding indicative of a mucosal tear or infection with gas-forming bacteria (Fig 11) (3). At upper gastrointestinal
Figure 10. IHE in three different patients. (a) Anteroposterior chest radiograph in a young man who presented with chest pain after forceful vomiting shows widening of the superior mediastinum (arrow). (b) Water-soluble contrast-enhanced esophagogram obtained in a 37-year-old man shows a double-barreled esophagus, in which contrast enhancement of the false lumen (white arrow) and a radiolucent mucosal stripe (black arrow) are seen. Extraluminal contrast enhancement (arrowhead) also is seen, a finding consistent with esophageal perforation. (c) Contrast-enhanced CT image obtained in the same patient as a shows diffuse hyperattenuating (60-HU) esophageal thickening (arrow) and luminal narrowing, findings indicative of the concentric type of IHE. (d) Contrast-enhanced CT image, obtained in a 68-year-old woman with nausea and retching while recovering from general anesthesia after undergoing ophthalmologic surgery, shows a hyperattenuating (55-HU) hematoma (arrow) in the distal esophagus with lateral displacement of the esophageal lumen (arrowhead), findings consistent with the eccentric type of IHE.
endoscopy, a bluish submucosal hematoma is seen, with bulging of overlying mucosa into the esophageal lumen (3,25,30). At endoscopic ultrasonography, IHE appears as a homogeneously hypoechoic lesion within a submucosal layer (3,23,31). In addition, endoscopic ultrasonography may help distinguish mediastinal vascular structures from an IHE.

Aortoesophageal fistula (AEF) is a subtype of IHE that deserves special mention. AEF is a rare, life-threatening cause of upper gastrointestinal bleeding, and it may develop secondary to aortic or esophageal conditions. AEF accounts for about 3.5% of all deaths that result from upper gastrointestinal bleeding, and aortic conditions are responsible for 75% of all cases of AEF (3,32). Aortic causes of AEF include ruptured aortic aneurysm, ruptured atheromatous plaque, penetrating ulcer, complications of prosthetic aortic grafting, and aortic reconstructive surgery. Esophageal causes of AEF include foreign body ingestion, esophageal malignancy, and corrosive esophagitis (33). CT depicts AEF as an enhancing submucosal esophageal mass with a fistulous tract between the aorta and the esophagus (Fig 12) (3,34). Because a definite fistula may not always be seen, the presence of a hyperattenuating enhancing esophageal mass with an associated aneurysm of the adjacent aorta may be indicative of AEF (3).

The differential diagnosis of IHE includes all causes of symmetric (eg, esophagitis and diffuse esophageal spasm) and asymmetric (eg, leiomyoma, leiomyosarcoma, and carcinoma) esophageal wall thickening. The morphologic characteristics, distribution, attenuation values, and enhancement pattern may help differentiate IHE from other types of esophageal emergencies (3). Treatment of IHE is mainly medical, rarely surgical, and depends on the cause of the hemorrhage. Although many cases of IHE spontaneously resolve with conservative management, there is a 7%–9% mortality rate, even with aggressive treatment (3).

**Boerhaave Syndrome**

Boerhaave syndrome is defined as complete transmural laceration of the esophagus that results from violent straining or vomiting. It has high mortality and morbidity rates and is uncommon, affecting one in 6000 people (6). However, it is more common in middle-aged men than in other patient groups, and 50% of patients have a history of alcoholism or heavy drinking. Clinically, patients present with excruciating subster-
nal or epigastric pain after an episode of severe retching and vomiting. Patients also typically present with the Mackler triad of symptoms, which includes vomiting, sudden severe chest pain, and subcutaneous emphysema (35). Odynophagia, tachypnea, cyanosis, fever, and shock may develop later in the course of the disease.

In patients with Boerhaave syndrome, esophageal rupture results from a sudden increase in intraesophageal pressure caused by straining or vomiting. Esophageal tears are most commonly located in the left posterior wall of the lower one-third of the esophagus, above the squamocolumnar junction and where the “clasp” and oblique (“sling”) fibers of the esophageal musculature merge (36). On average, tears are about 2 cm long and are located 3–6 cm above the diaphragm (37). Sudden straining or vomiting—a result of neuromuscular incoordination—may cause the cricopharyngeus muscle to fail to relax, which may lead to a sudden rise in intraesophageal pressure and subsequent rupture.

Chest radiography is usually performed first and may depict widening of the mediastinum, pneumomediastinum, pleural effusions (more commonly on the left side), subcutaneous emphysema, hydropneumothorax, and patchy pulmonary infiltrates (35). Frontal chest radiography may depict a V-shaped lucency filled with air in the lower left area of the mediastinum.

Figure 12. AEF. (a) Drawings of the axial (left) and lateral (right) planes of the neck show aneurysmal dilatation of the thoracic aorta, which ruptures into the esophageal wall and leads to development of an intramural hematoma (red areas). The intramural hematoma displaces the esophageal lumen laterally (arrow). (b) Unenhanced CT image obtained in a 65-year-old man with excruciating retrosternal chest pain shows a hyperattenuating (66-HU) esophageal hematoma (black arrow) displacing the esophageal lumen laterally (arrowhead) and a thoracic aortic aneurysm (white arrow). (c) Contrast-enhanced CT image shows a fistulous tract from a penetrating aortic ulcer (arrow), a finding consistent with AEF.
and pneumomediastinum, a finding known as the V sign of Naclerio (Fig 13a). Although the V sign of Naclerio is not specific for Boerhaave syndrome, it may be an early indication of esophageal perforation in appropriate clinical scenarios (38). Contrast-enhanced esophagography is the modality of choice for evaluating esophageal rupture and may depict extravasation of contrast material, submucosal collection of contrast material, and esophagopleural fistula (most commonly on the left side) (Fig 13b) (6,35). The use of hydrosoluble contrast material is preferred over barium in patients with suspected esophageal rupture because of the risk for mediastinitis, a result of irritation caused by barium. However, false-negative results may occur in as many as 10% of patients with Boerhaave syndrome at hydrosoluble contrast material–enhanced esophagography (6). For this reason, follow-up esophagography with barium contrast material is indicated in patients with Boerhaave syndrome because it provides

Figure 13. Boerhaave syndrome in two different patients. (a) Frontal chest radiograph obtained in a young man with severe chest pain shows a pneumomediastinum, subcutaneous emphysema, and the V sign of Naclerio (arrows). (b) Contrast-enhanced esophagogram obtained in a 40-year-old man with retrosternal pain after vomiting shows contrast extravasation (arrow) from the distal one-third of the esophagus. (c) Axial oral and intravenous contrast-enhanced CT image obtained in the same patient as b shows periesophageal air collections (arrow) and contrast extravasation (arrowhead) surrounding the distal esophagus, findings consistent with Boerhaave syndrome.

Teaching Point
increased sensitivity for contrast-enhanced examinations (39). CT is the modality of choice in patients with equivocal esophagography results. Common CT findings of esophageal perforation include esophageal wall thickening, periesophageal air collections, pneumomediastinum, mediastinal fluid collections, pleural effusions, extravasation of contrast material, and esophagopleural fistula (Figs 13c, 14) (35). Rarely, CT may depict a tear in the esophageal wall. The presence of a supradiaphragmatic periesophageal air collection in a typical location (the left posterior wall in the lower one-third of the esophagus) is characteristic of Boerhaave syndrome.

Treatment options for patients with Boerhaave syndrome depend on the time of presentation and the patient’s clinical condition (37). Thus, treatment may be conservative, endoscopic, or surgical (37).

**Acquired Esophagorespiratory Fistula**

Acquired esophagorespiratory fistula in adults is a rare but serious clinical entity. Depending on the cause, fistulous communication may develop between the esophagus and the trachea, bronchi, or lung parenchyma; tracheoesophageal fistulas are most common. Acquired esophagorespiratory fistula may result from benign or malignant causes. Benign causes include corrosive esophageal injuries, prolonged intubation, surgical or endoscopic procedures, esophageal infection, simultaneous rupture of mediastinal lymph nodes into the esophagus and airways, and ingestion of foreign bodies (6,40,41). Malignant causes of esophagorespiratory fistula include esophageal carcinoma, bronchogenic carcinoma, and lymphoma (6,42). Fistulas may develop in 5%–10% of patients with advanced esophageal cancer, and the risk for esophagorespiratory fistulas increases after radiation therapy (6,43). Clinical features of esophagorespiratory fistulas include attacks of coughing when swallowing liquids, dryness of mouth, neck and chest pain, spontaneous coughing, and sputum that contains particles of food. The classic manifestation of esophagorespiratory fistulas is the swallow-cough sequence referred to as the Ono sign. Although the Ono sign may not be seen in all cases, its presence is indicative of esophagorespiratory fistula (41).

Chest radiographic findings of esophagorespiratory fistula are nonspecific and may include recurrent episodes of pulmonary consolidation secondary to aspiration, lung abscesses, and pleural effusions. In addition, findings indicative of fistula caused by primary disease—such as radiopaque foreign bodies, mediastinal lymph nodes, and esophageal malignancy—may be seen at

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**Figure 14.** Boerhaave syndrome in a 45-year-old man who presented with chest pain and vomiting after heavy drinking. (a) Axial CT image shows periesophageal air, pneumomediastinum (arrows), and a left pleural effusion (arrowhead). (b) Axial oral and intravenous contrast-enhanced CT image (obtained at a different level than a) shows a periesophageal air collection and contrast extravasation (arrowheads).
Spontaneous mediastinal hematoma

Spontaneous mediastinal hemorrhage with associated hematoma is a rare condition, with about 50 cases reported in the literature. The condition was first described by Capps (46) in 1934. Spontaneous mediastinal hemorrhage may occur in the following clinical settings: (a) in patients with bleeding disorders or who are undergoing chest radiography. If esophagorespiratory fistula is suspected, contrast-enhanced esophagography should be performed with the use of hydrosoluble contrast material rather than barium because of the increased risk for aspiration. A fistulous tract may be demonstrated by the passage of ingested contrast material into the trachea, bronchi, and lung parenchyma (Fig 15a). Endoscopy (tracheobronchoscopy and esophagoscopy) may be used to help identify an esophagorespiratory fistula. CT also plays an important role in determining the site, number, and extent of fistulous tracts and the condition of lung parenchyma (Figs 15–17) (44). However, multidetector CT with sagittal and coronal reconstructions and virtual CT endoscopy are more useful for depicting esophagorespiratory fistulas (45). Treatment of patients with esophagorespiratory fistulas resulting from benign conditions is primarily surgical and consists of closing the fistulous communication. Malignant conditions are treated according to the stage of disease.
anticoagulation therapy; (b) in the presence of a sudden increase in intrathoracic pressure due to coughing, vomiting, or sneezing; (c) in the presence of a sudden, sustained rise in systemic blood pressure due to malignant hypertension; and (d) secondary to hemorrhage into a mediastinal organ or mass with no underlying bleeding disorder (47–49). Dyspnea is the most common manifestation of spontaneous mediastinal hemorrhage in patients with known risk factors for mediastinal bleeding. Other symptoms of spontaneous mediastinal hemorrhage include neck and chest wall ecchymosis, dysphagia, dysphonia, chest pain, and tachycardia. Middle-aged and elderly patients are most commonly affected, and there is a slight male predominance.
Spontaneous mediastinal hematoma in a 50-year-old man who underwent anticoagulation therapy for a pulmonary embolism in the postoperative stage of intrathoracic goiter resection. Contrast-enhanced CT images (a obtained at a different level than b) show ill-defined soft-tissue thickening in the mediastinum (arrows in a) and a well-defined soft-tissue mass in the paraesophageal space (arrowheads in b).

At chest radiography, spontaneous mediastinal hematoma commonly manifests as mediastinal widening associated with sudden onset of chest pain and dyspnea in high-risk patients. Other frontal chest radiographic findings of mediastinal bleeding include abnormal aortic contours, widening of the right paratracheal stripe (5 mm or more), and deviation of the nasogastric tube to the right of the T4 spinous process (50). At unenhanced CT, a mediastinal hematoma appears as a soft-tissue-attenuation mass with areas of high attenuation (>60 HU), a finding indicative of hemorrhage (51). Other CT appearances include a focal heterogeneous mass and an area of ill-defined soft-tissue thickening in the mediastinum that may extend into the cervical region (Fig 18). CT also may depict compressive effects on the trachea, main bronchi, and great vessels of the mediastinum. CT angiography with coronal and sagittal reconstructions may help exclude a vascular source of mediastinal bleeding. At MR imaging, the signal intensity within the hematoma may vary depending on the age of the hematoma; however, MR imaging is rarely required for diagnosis of spontaneous mediastinal hematoma. Catheter angiography of the aortic arch and brachiocephalic vessels does not usually reveal arterial lesions that may cause hemorrhage. Although some patients require surgical or thoracoscopic removal of the hematoma, those who are clinically stable may be evaluated with serial follow-up imaging to assess for resolution of the hematoma (49,52).

Tension Pneumomediastinum

Pneumomediastinum is characterized by the presence of free air around the mediastinal structures. Common causes of pneumomediastinum include blunt or penetrating trauma, esophageal perforation, recent interventions in the esophageal or tracheobronchial tree, pulmonary infections, gas-forming infections in the mediastinum, cocaine inhalation, and extension of air from a pneumothorax (53,54). In addition, the presence of mediastinal air may be caused by a spontaneous pneumomediastinum, an uncommon, self-limited, benign condition that usually affects young men. Spontaneous pneumomediastinum is triggered by vomiting, asthma flare-ups, severe coughing, and exercise (53,55). Tension pneumomediastinum is a rare, potentially fatal complication of pneumomediastinum that can lead to substantial hemodynamic compromise. Tension pneumomediastinum is caused by a substantial increase in intramediastinal pressure, which results from accumulation of free air. Increased intramediastinal pressure may compress the heart, resulting in decreased venous return and compression of the tracheobronchial tree. All of these changes may lead to sudden, profound
cardiovascular and respiratory collapse (56). In patients undergoing ventilation, pneumomediastinum may result from barotrauma and progress to tension pneumomediastinum. Clinically, patients with pneumomediastinum present with chest pain, breathlessness, and subcutaneous emphysema. In these patients, sudden development of severe hypoxia, hypotension, tachycardia, metabolic acidosis, and high ventilation pressure should be suspicious for tension in the mediastinum, which may lead to cardiovascular and respiratory compromise.

In approximately 50% of patients with tension pneumomediastinum, mediastinal air appears as a thin line outlining the borders of the heart at frontal chest radiography (56). In almost all patients, pneumomediastinum may be seen on lateral chest radiographs with associated subcutaneous emphysema. However, it is difficult to identify tension pneumomediastinum at radiography. CT is the imaging modality of choice for patients who are strongly suspected of having tension pneumomediastinum. CT findings include substantial mediastinal free air, flattening of the anterior cardiac contour, compression of the right atrium, uplifting of the heart off of the diaphragm, distention of the inferior vena cava, compression of the mediastinal vessels, and flattening of the main bronchi (Fig 19) (57–59). In an appropriate clinical setting, the presence of these findings in a patient with pneumomediastinum should raise suspicion for a tension pneumomediastinum. Tension pneumomediastinum is an emergency and requires prompt and definite treatment (56). Percutaneous needle aspiration of mediastinal air, with or without CT-guided catheter placement, may be used to efficiently treat tension pneumomediastinum and reverse cardiovascular and respiratory compromise (56,59).

Tension Pneumopericardium

Tension pneumopericardium is caused by a substantial amount of air within the pericardial sac, which leads to cardiac tamponade with resultant hemodynamic effects. Patients with a small amount of pericardial air are usually asymptomatic. Normal intrapericardial pressure is 50–100 mm of water. When intrapericardial pressure rises above 265 mm of water, the heart may undergo substantial compression, leading to cardiac tamponade and hemodynamic compromise (60). Trauma is the most common cause of pneumopericardium and may be blunt or penetrating, iatrogenic (eg, occurring during or after cardiac surgery), or related to barotrauma secondary to positive pressure ventilation. Nontraumatic causes of pneumopericardium include pericarditis with...
Figure 20. Tension pneumopericardium in a patient who recently underwent orthotopic heart transplantation. (a) Frontal chest radiograph shows air in the pericardial cavity outlining the borders of the heart (arrows), a finding consistent with pneumopericardium. (b) Unenhanced CT image shows a substantial amount of pericardial air (arrowheads) compressing and displacing the heart with associated collapse of the heart chambers.

gas-forming organisms, direct extension of an inflammatory process from adjacent structures, fistulous communication with air-containing structures (eg, the stomach, esophagus, airways, and lungs), and extension of a pneumomediastinum into the pericardial cavity (61,62).

Symptoms of pneumopericardium include chest pain, dyspnea, cyanosis, hypotension, and tachycardia. Auscultatory findings include a characteristic “mill wheel” murmur, metallic sounds of high frequency, and muffled heart sounds. Elevated ST segments and low-voltage waveforms may be seen at electrocardiography. At frontal chest radiography, pneumopericardium may appear as a continuous rim of air outlining the left and right borders of the heart, a finding referred to as the “halo” sign (Fig 20a) (61). In patients with pneumopericardium who experience sudden deterioration of hemodynamic status with an increase in central venous pressure, cardiac tamponade should be suspected. Mirvis et al (60) described the “small heart” sign, which is seen at frontal radiography in patients with tension pneumopericardium and denotes a sudden, substantial decrease in the size of the cardiac silhouette and is accompanied by pathophysiologic effects of cardiac tamponade. In patients with tension pneumopericardium, CT may depict substantial amounts of air in the pericardial cavity with compression and displacement of the heart and associated collapse of the heart chambers, flattening of the anterior border of the heart, and distention of the inferior vena cava (63) (Fig 20b). Tension pneumopericardium is an emergent condition and requires immediate pericardial decompression by needle pericardiocentesis and possibly placement of a pericardial drain (62,63). Some patients may require formal thoracotomy and pericardiotomy to correct the primary cause of the condition.

Summary
Nonvascular, nontraumatic mediastinal conditions such as acute mediastinitis, esophageal emergencies (eg, IHE, Boerhaave syndrome, and acquired esophagorespiratory fistulas), spontaneous mediastinal hematoma, tension pneumomediastinum, and tension pneumopericardium are uncommon but important causes of acute chest pain in adults. Recognizing the specific projectional and cross-sectional imaging findings of these conditions is important and may lead to timely diagnosis and treatment for patients with these conditions.
References

Nonvascular, Nontraumatic Mediastinal Emergencies in Adults: A Comprehensive Review of Imaging Findings

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Page 1143
Common CT findings of acute mediastinitis include increased attenuation of mediastinal fat, free gas bubbles in the mediastinum, localized fluid collections, enlarged lymph nodes, pleural effusions, and empyema (7).

Page 1148 (Figures on page 1149)
CT is the modality of choice for evaluation of IHE and typically depicts symmetric or asymmetric esophageal wall thickening and a well-defined, nonenhancing, high-attenuation intramural esophageal mass that extends along the esophageal wall (Fig 10c, 10d) (3,28,29).

Page 1152 (Figure on page 1152)
Contrast-enhanced esophagography is the modality of choice for evaluating esophageal rupture and may depict extravasation of contrast material, submucosal collection of contrast material, and esophagopleural fistula (most commonly on the left side) (Fig 13b) (6,35). The use of hydrosoluble contrast material is preferred over barium in patients with suspected esophageal rupture because of the risk for mediastinitis, a result of irritation caused by barium.

Page 1157 (Figure on page 1157)
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