

NON-CARDIAC CHEST PAIN AND SHORTNESS OF BREATH

Epidemiology / Introduction

Chest pain (CP), or acute chest discomfort, and shortness of breath (SOB), or dyspnea, are two of the most common causes of emergency room and doctor visits in the United States. The potential etiologies are varied and range from immediately life-threatening to benign causes. Cardiac ischemia is one of the most common life-threatening causes of both symptoms and should be evaluated with an electrocardiograph (ECG). Other “cardiac” related causes include aortic dissection and pericardial tamponade, which may be seen on a Chest CT with IV contrast and an echocardiogram respectively. Patients may present with urgent symptoms and go to the ER or present with chronic symptoms ranging from days to years. A thorough history can guide the provider with the differential. If a cardiac cause has been ruled out, gastroesophageal reflux disease (GERD) becomes the next most likely cause of chest discomfort, but more life-threatening causes, such as pneumothorax or pulmonary embolism must be ruled out. Other esophageal-related concerns, such as a perforation, can also be life-threatening if not recognized and treated early. Reflux, esophageal spasm, pneumonia, and empyema can all cause symptoms but are less likely to cause early hemodynamic compromise and death. This section will focus on non-cardiac/aortic causes of chest pain and dyspnea.

Overview

When a patient presents with either chest pain or shortness of breath, the acuity must first be determined. Did symptoms start suddenly? Have they been gradually getting worse over days or longer? What has been the patient’s recent history and associated symptoms? Have they had fevers? What is their past medical and surgical history? Outlined below are “classic” symptoms associated with a variety of disorders related to chest pathology and causes of CP and SOB.

PULMONARY EMBOLUS (PE)

Pathophysiology

PE’s are often underdiagnosed due to other common causes of CP and SOB. They are caused by the development of blood clots in the venous system, usually due to stasis and/or hypercoagulable states. When they develop in the larger veins of the lower extremity (femoral veins, iliac veins) or pelvis, they may dislodge and move with venous blood through the inferior vena cava (IVC), through the heart (right atrium to right ventricle) and into the pulmonary arterial system. The clots can become lodged in the pulmonary arteries and cause a central obstruction. This can result in a ventilation-perfusion (VQ) mismatch, where the lung is ventilated in the corresponding bronchiole but, there is no blood flow to the alveoli to allow for gas exchange. Patients may notice shortness of breath as they are not getting adequate oxygen to their blood. Long-term obstruction of the pulmonary arteries can result in right heart strain and potential right heart failure. When a patient presents with a PE, the providing

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caregiver must be concerned about more clots passing into the lungs which can be immediately fatal if there is a large enough VQ mismatch.

Signs and Symptoms

Risk factors include hypercoagulable states due to cancer, recent surgery, or other chronic diseases such as obesity. Recent histories of travel with long periods of sitting or stasis may be noted. Prior history of deep venous thrombus (DVT) or PE may also be present. Symptoms include chest pain, shortness of breath, or palpitations. Some or all of these symptoms may be present with varying degrees of severity. Calf swelling and tenderness may also be present. Signs include tachycardia and dyspnea which may be subtle. They may be tachypneic and have unexplained reduction in oxygen saturation and require supplemental oxygen. Homan's sign (pain in calf or posterior knee on passive dorsiflexion) may be positive.

Relevant Diagnostic Studies

A chest x-ray and ECG are the first steps to rule out other causes of SOB and CP (pneumonia, pneumothorax, etc.). A chest x-ray will usually not have obvious specific findings, though many will have lower lobe atelectasis or have a "Hampton's Hump" (a peripheral opacity) or "Palla's sign" (a large right descending pulmonary artery). These findings on a chest x-ray are not sensitive though for a diagnosis of a PE, but again, will rule out other problems. Historically, a VQ scan would be performed to show ventilation-perfusion (VQ) mismatch. Currently, a CT chest with IV contrast can usually be obtained much more quickly in most emergency settings and will show emboli within the pulmonary artery system. (See Figure 1.) A CT scan will provide you with information about other potential issues such as pneumonia or hemothorax/empyema. A CT scan with oral contrast can also evaluate for esophageal perforation if contrast is seen leaking out of the esophagus or pneumomediastinum is present. A venous duplex scan should be performed to rule out clots in the legs as a potential source of more emboli. If a VQ scan or CT chest cannot be obtained quickly, and a venous duplex shows blood clots in the legs, anticoagulation should be started immediately in a high-risk patient until further work up is possible. The risk of PE's from upper extremities DVT is low overall but may remain a potential source. Upper extremity venous scans should be performed if there is a history of risk factors for upper extremity clots, including central lines, upper arm thrombophlebitis, and thoracic outlet syndrome.



Figure 1: CT chest showing bilateral pulmonary emboli (blue arrows)

Non-operative Management

Acute PE's are not managed with traditional surgery, although endovascular procedures may be done in the operating room. The mainstay of treatment is systemic anticoagulation with IV heparin and transition to oral anticoagulation, or subcutaneous injections, on an outpatient basis. The anticoagulants will prevent new clots and aid in the thrombolysis of present clots, though this process takes weeks to months. There is growing experience with localized thrombolytic therapy by interventional radiologists/cardiothoracic surgeons, but this is usually reserved for patients demonstrating heart strain/failure or who are unable to maintain oxygenation despite mechanical ventilation. Depending on the risk of more clots, the current PE burden, and the ability to tolerate systemic anticoagulation, an IVC filter may be placed. These filters do not prevent clots from forming, but rather prevent large clots from travelling through the IVC to the heart. Filters may be temporary (placed for a few weeks and removable by a secondary endovascular procedure) or be long term/permanent. Filters and systemic anticoagulation may be used in high-risk patients or patients needing surgery in the near future.

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Operative Management

Pulmonary embolectomy is an option for patients with chronic PEs and who demonstrate heart strain. This is only performed by select cardiac surgeons and higher volume referral centers. This is not an “urgent” surgery and requires a multidisciplinary approach to work up and decision making. In the acute setting, if a patient presents with heart failure and cardiac arrest, an emergent non-sterile left anterolateral thoracotomy can be performed to allow access to the heart for manual cardiac compressions. This is rarely successful and can be fraught with risk to the medical providers from accidental cuts with scalpels.

Basic Postoperative Care

Anticoagulation guidelines for PE’s and DVT’s mandate systemic anticoagulation for 6-12 months. The use of an IVC filter does not change this length of time. Emergent thoracotomies should be managed with chest tubes and pain control if patients survive the process.

PLEURAL DISEASES

(PNEUMOTHORAX / TENSION PNEUMOTHORAX / HEMOTHORAX / EMPYEMA)

Pathophysiology

Pleural diseases can be categorized by what is causing lung compression in the pleural space; air-pneumothorax, blood-hemothorax, and fluid/pus-effusion/empyema. Air can be introduced into the pleural space from an external source through trauma or from the lung via blunt or penetrating mechanisms. The chest is usually a negative pressure cavity which allows for air to be drawn into the lungs for normal breathing. This also draws air in from a chest wall injury/defect or from within the lung which can compress the lung and lead to symptoms of SOB and CP. There is no “exit” path for air in the pleural space and, therefore, pressure can build up and cause a tension pneumothorax. A tension pneumothorax causes a shift of mediastinal contents to the contralateral side causing external compression of the superior vena cava (SVC), the IVC, and the heart. This loss of inflow into the heart results in hypotension, tachycardia, decreased cardiac output, and impending death. A simple pneumothorax can become a tension pneumothorax and is the reason why all pneumothoraces must be observed and/or treated with drainage.

A primary spontaneous pneumothorax is defined as a new pneumothorax seen in a person with no obvious inciting factor, i.e., no known lung pathology on chest-x-ray or CT chest. These patients are usually younger, male, tall, and thin. They almost always have small apical lung blebs which may need to be addressed. A secondary spontaneous pneumothorax is seen in patients with previously identified pulmonary diseases, such as COPD/emphysema, cystic fibrosis, or pulmonary fibrosis. Patients with these diseases are at risk for pneumothoraces from chronic lung injuries that can evolve into holes in the lung allowing air to escape into the pleural space.

All people have a small amount of fluid around their lungs which allows for lubrication and smooth movement of the lungs against the chest wall. Increased fluid can be due to increased

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fluid production or decreased fluid absorption. Increased fluid production may be a result of volume overload. This fluid can be termed a transudative effusion. Alternatively, in the setting of inflammation or infection, the capillaries within the chest become “leaky” and allow proteins to slip into the pleural space and draw fluid into the chest by oncotic pressure. This is called an exudative effusion. Transudative effusions are seen with systemic problems causing fluid overload, such as heart failure, nephrotic syndrome, etc. Exudative effusions are seen in the setting of cancers, infections, or lymphatic system problems. Up to 30% of patients with pneumonia will develop a pleural effusion, but these rarely need to be treated and will resolve with treatment of the pneumonia. The fluid is usually sterile initially but can become infected. The higher protein/fibrin concentration in the fluid can lead to “loculations” or pockets of fluid with thin rinds of proteinaceous material limiting the fluid from moving about the chest. When infected fluid leads to pus development, more loculations can occur and a thick rind can develop around the lung of fibrin and protein. This can compress the lung and cause SOB and CP.

Once an infection develops, it can progress rapidly as there is no mechanism to drain the pleural space without some external intervention. Malignant pleural effusions (secondary to cancer) usually are more insidious in development but can lead to acute symptoms. They are usually related to increased fluid production from tumors seeding the chest wall and/or lymphatic obstruction of the lymphatic chain. Cancer-related effusions are often bloody due to chronic oozing from raw surface of pleural disease. Chylothorax occurs when the thoracic duct becomes injured (surgery) or occluded (cancer) and lymph (chyle) builds up in the pleural space. Hemothorax will be covered in the trauma module but can cause acute SOB or chest pain when related to bleeding from cancers that have spread to the chest or arise within the chest. Blunt or sharp accidents or iatrogenic injuries from procedures such as thoracentesis or central line insertion can injure an intercostal vessel and lead to bleeding in the chest. The blood will not be reabsorbed and will cause pulmonary compression.

Signs and Symptoms

Patients presenting with CP/SOB and pleural injuries need a thorough history and physical exam. For primary spontaneous pneumothoraces, patients are often young without any known lung concerns. They may be tall and thin in appearance. Symptoms are usually of sudden onset. For other causes of pneumothorax, a recent history may elucidate trauma or procedural interventions. A medical history may give a history of prior pulmonary disease or malignancy which can raise the possibility of a secondary spontaneous pneumothorax or malignant effusion. A history of fevers could indicate pneumonia and an associated effusion or empyema. On physical exam, vital signs could reveal a fever and tachycardia for a pneumonia, and empyema or tachycardia for a tension pneumothorax. One should note the breath sounds for quality and distribution. Patients with a pneumothorax may have hyper-resonant percussion sounds or absent breath sounds upon auscultation on the affected side. Patients with an effusion/hemothorax/empyema may have diminished breath sounds only at the base of one side, although large fluid collections can cause complete compression of the lung and lead to absent breath sounds. Signs of a tension pneumothorax include jugular venous distension, tachycardia, and tracheal deviation away from the affected side. Hypotension is also seen when a patient's cardiac function is compromised.

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Relevant Diagnostic Studies

The first step should be a chest x-ray for a suspected pleural problem. These should be done promptly, as a tension pneumothorax can progress to a fatality within minutes. (See Figure 2 and Figure 3.) A pneumothorax can be identifiable easily on a routine chest x-ray, although more subtle findings may require a Chest CT scan to show 3-dimensional structural changes within the lung. For a young patient without a prior history, a chest x-ray alone should be adequate. If other lung pathology is suspected, a CT Chest with IV contrast is needed. (See Figure 4.) If an effusion is seen on the chest x-ray, it may be the result of fluid, blood, or pus, and a thoracentesis is needed to further aid in the diagnostic work up. The fluid could appear serous, or clear and yellow, to bloody, to frank blood, or frank pus. Chyle from a chylothorax can appear similar in gross appearance to pus. Testing includes sending some fluid for cytology to evaluate for malignant cells. Fluid should be sent for Protein and Lactate Dehydrogenase (LDH) levels to evaluate for a transudative versus exudative effusion. Light's criteria can then be used to aid in this differential, with values >0.5 of serum values (for protein and LDH) indicating an exudative effusion. Fluid should be sent for culture if an infectious process is suspected. Triglyceride levels will help identify chyle, if present. Systemic blood work can show leukocytosis and identify patients with ongoing infections. A low hemoglobin value may be seen in patients with active bleeding into their chest space.

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Figure 2: Left-sided tension pneumothorax. Mediastinal contents (trachea, heart) can be seen pushing to the patient's right side.

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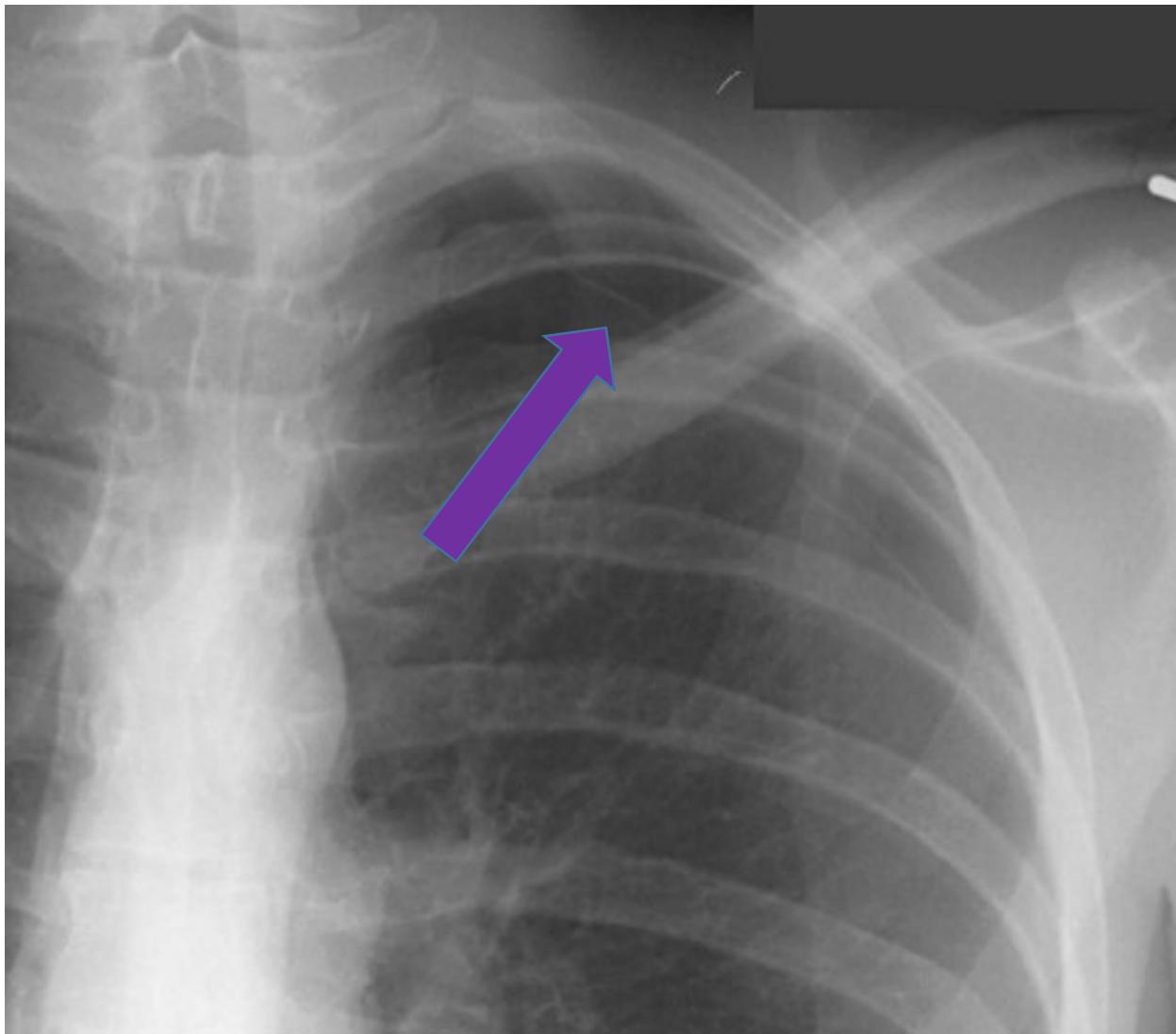


Figure 3: Small left-sided pneumothorax with arrow showing edge of lung.

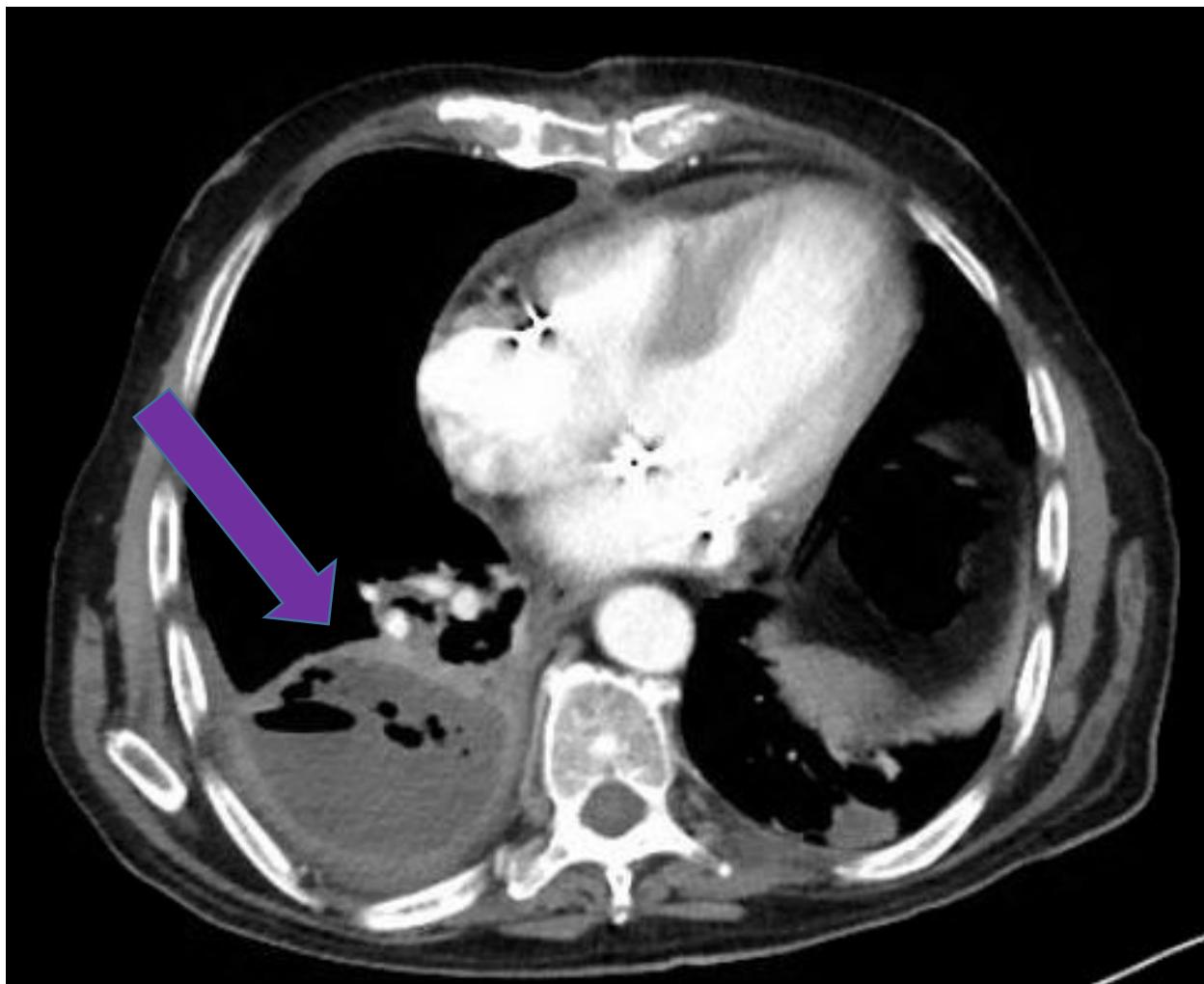


Figure 4: CT Chest showing a small right posterior loculated effusion with gas (arrow), likely signifying an empyema.

Non-operative Management

Management of pleural disease depends on the severity of symptoms. Usually, the easiest and quickest procedure can be a pigtail drain or a larger chest tube placement which allows for drainage of air/fluid/blood/pus and can relieve the pressure on the lung and in the chest. The drainage from the chest tube can also be sent for analysis as outlined above. Small pneumothoraces in patients with mild symptoms may be observed with serial chest x-rays for 24 hours or drained by percutaneous drainage of the air alone. The only downside of this approach would be that the lung doesn't fully expand allowing for the potential small blebs causing the air leak to seal against the chest wall. Historically, 70% of "treated" primary spontaneous pneumothoraces will not recur, and operative management is reserved for the 30% of patients with a recurrence. For patients with fluid, the next step of treatment depends on the degree of expansion of the lung after thoracentesis and/or chest tube, and if symptoms of

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SOB or CP have resolved. If a simple effusion/hemothorax/chylothorax is drained with resolution of symptoms and minimal further chest tube drainage after 24-48 hours, the drainage is all that may be needed. For patients with pneumonia and a concurrent pleural effusion, broad spectrum antibiotics may be needed. For patients with incomplete drainage, but who are too frail to go to the operating room, placement of fibrinolytic therapy through the drain may be required. Instilling tPA (tissue plasminogen activator) into the pleural space, allowing it to dwell for 30 min - 2 hours and then draining the fluid allows for the chemical break up of loculations and improved drainage of fluid. For early empyemas, before a rind has developed around the lung, tPA may be all that is needed.

Operative Management

Operative management may be needed for symptom control and/or source control of the underlying cause of the pleural disease, if a drainage procedure is not adequate, or if continued problems are expected due to continued air leak or more fluid/blood/pus. Patients can receive an exploratory thoracoscopy (VATS procedure). The lung is not ventilated, and the remaining fluid is evacuated. In the setting of a pneumothorax, an apical bleb should be removed by a wedge resection and mechanical pleurodesis performed (roughing up the chest wall pleural surface) to create adhesions between the lung and chest wall to prevent future pneumothoraces. If there is known lung pathology, the site of the lesion should be removed, if possible, and if not, at least identified for drain placement near the damage. Sources of bleeding should be identified and controlled. In the setting of a loculated effusion or empyema, all the fluid should be removed and the rind around the lung peeled off (decortication). This may require converting the surgery to an open thoracotomy as the rinds are often tenacious. A similar fibrinous rind can be seen after a longstanding hemothorax and is treated the same way. A thoracic duct injury, if suspected, should be treated with ligation of thoracic duct by clips or suture. If a thoracic duct injury is suspected, interventional radiology has a growing experience at an endovascular approach with embolization of the duct by coils or glue.

Basic Postoperative Care

Chest tubes should be left in the chest until the fluid output is low (< 100 or 150 ml per day) and any air leaks have resolved. For a post-pleurodesis patient, many would leave the chest tubes in for 72 hours prior to removal, to allow for the lung to adhere to the chest wall. For patients with lung injuries and air leaks that were not amenable to resection, and that continue to have air leaks after 3-5 days, conversion from a water seal drainage system to a small one-way valve attached to the tube allows for patients to be discharged home with a return for drain removal in clinic when the leak has resolved. For infected pleural spaces (empyemas), conversion of one of the tubes to an "empyema tube" means disconnecting the tube from the one-way valve drainage box and leaving the tube "open to air". The lung usually stays inflated as there are adhesions to the chest wall. In this setting, the empyema tube serves as a wick for the infected space and is slowly removed (1-3 cm every 1-2 weeks) over a period of weeks to months. This allows the infection to clear with slow removal of the tube. Antibiotics are often used concurrently to treat an associated pneumonia or to treat the pleural space alone.

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ESOPHAGEAL PERFORATION / RUPTURE

Pathophysiology

Esophageal rupture usually occurs after a trauma or a sudden increase in intrathoracic pressure resulting in disruption of the esophageal wall. The classic scenario of “Boerhaave’s syndrome” was identified due to excessive forceful vomiting which led to a full thickness esophageal rupture. This is still a frequent cause of esophageal injury and can be seen in younger people who vomit after excess drinking or other causes. Other causes may be external trauma, such as a car accident leading to increased intrathoracic pressure, or penetrating trauma into the neck. Any foreign body placed into the esophagus can result in an injury and tear. Endoscopy probes placed for esophagogastroduodenoscopy (EGD), endoscopic ultrasound (EUS), or transesophageal echocardiogram (TEE) can all cause injuries, as can associated procedures such as balloon or bougie dilation. Food can become stuck in the esophagus causing tears. All of these etiologies cause a mechanical injury that penetrates the esophageal wall, resulting in leakage of air and fluid into the mediastinum or the pleural space. This air and/or fluid can lead to a reactive effusion and the development of an empyema as bacteria translocate from the esophagus into the enclosed chest spaces. Patients with esophageal diseases such as scleroderma or esophagitis may be more prone to esophageal injury.

Signs and Symptoms

Patients will almost always present with a significant amount of chest pain with some associated SOB. The pain is usually very acute in development and can worsen quickly. A history to identify trauma or prior vomiting is essential. Fevers are common but may not be seen early in the presentation. Tachycardia is common and due to pain, anxiety, and also pericardial irritation from the leaking esophageal contents. There may not be other signs or symptoms, so a thorough history and a low threshold for concerns should lead to further studies.

Relevant Diagnostic Studies

A chest x-ray may demonstrate pneumomediastinum (air in the mediastinum) or pleural effusions, but it cannot identify a leak/injury. A barium or gastrografin esophagogram is the most sensitive test to identify a leak and the exact location of a leak. (See Figure 5.) A CT chest with IV and oral contrast may also identify a leak but is not as accurate at showing the site of the leak compared to esophagogram. Conversely, the CT scan can show related changes in the chest such as pneumomediastinum, pleural or pericardial effusions, mediastinal fluid collections, etc. Bloodwork will usually reveal leukocytosis. A diagnostic endoscopy is usually not warranted, as it can cause further injury, and should be reserved during the operative management phase of treatment.

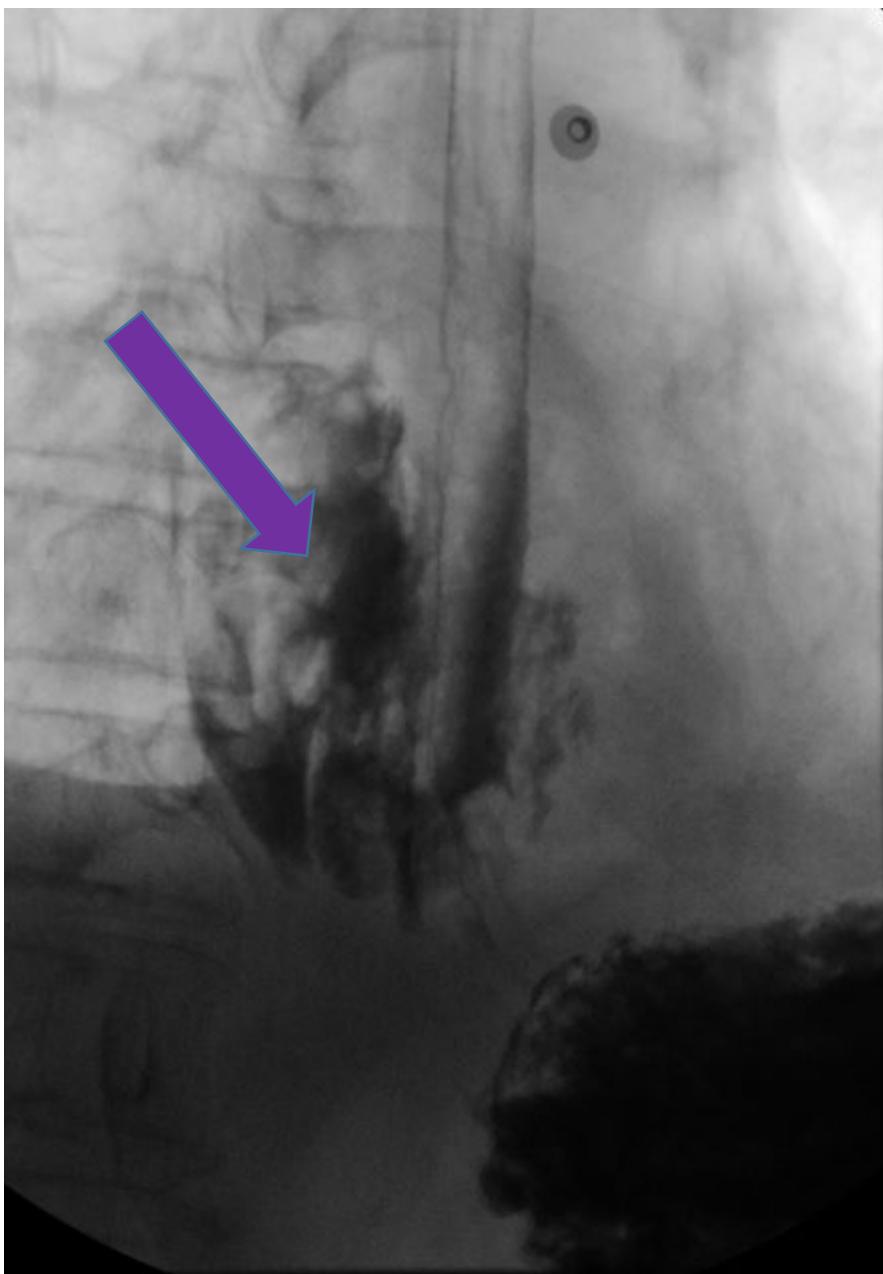


Figure 5: Barium Esophagogram showing a perforation with a leak of contrast into the mediastinum (arrow).

Non-operative Management

Esophageal perforations need treatment on two fronts: 1) to address the esophageal injury itself and 2) to treat related sequelae of the injury like effusions or empyemas. There are no real non-operative options for patients with a true esophageal rupture. Some patients will present with symptoms of pain and be found to have a pneumomediastinum, but no leak of contrast on

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barium study or CT scan. In this scenario, there was likely a small leak of air that has sealed. Observation, NPO status until resolution of pain, and IV antibiotics is the most conservative approach. Theoretically, if air has escaped into the mediastinum, then bacteria may have also, and can develop into abscesses in the chest. Keeping patients NPO can limit food passing by a healing injury. As long as a patient is improving clinically, they may be observed in this setting. Related pleural effusions can be treated with chest tube drainage and can be done concurrently with more definitive treatment of the injury. In patients who are critically ill and septic and would not tolerate an operation, endoscopy with stent placement and bedside drainage of fluid collections can allow for source control of the infection and management of the fluid collections. The use of esophageal stents has increased over the last several years. Placement of a drain near the esophageal injury allows for a de facto “esophagocutaneous” fistula which can temporarily control the infection.

Operative Management

There are a number of operative strategies, but the key ones include primary repair of the injury, if possible, control of the leak/infectious source, and management of the sequela of the leak as stated earlier. Generally, leaks that occur in normal esophageal tissue can be primarily repaired within 24-48 hours. This may require a right thoracotomy, left thoracotomy or laparotomy (abdominal) approach, or some combination of them. The identification of the location of the leak within the esophagus and its drainage pattern by contrast study before going to the operating room is critical. On-table esophagoscopy should be performed to evaluate the esophagus and potentially find the leak, but it is notoriously unreliable to always find the injury and can delay the more definitive treatment. Along with repair of the leak, addressing any infectious sequela is needed. If there is pneumomediastinum, the mediastinal space can be opened to drain into the pleural space and a drain placed thoracoscopically. Loculated pleural effusions may require thoracoscopy and may not be drained by chest tube placement alone. If pericardial effusions are present, making a window in the pericardium can be performed thoracoscopically. For small leaks/injuries, there is a growing body of evidence supporting the use of esophageal stents with concomitant drainage of effusions. These strategies may avoid a thoracotomy that can be associated with greater risks of long-term pain. For larger injuries, or in the setting of a chronic esophageal disease that would prevent a primary repair from healing, like a distal esophageal stricture or achalasia, resection of the esophagus with primary anastomosis should be performed at an experienced center. If a patient is likely to be NPO after the operation, a feeding jejunostomy tube may be placed to allow for early enteral feeding. A gastrostomy tube is generally avoided in case the esophageal injury does not heal and the stomach is needed for replacement of the esophagus.

Basic Postoperative Care

Postoperative care allows for the esophageal repair to heal before instituting oral intake. Patients are left with nasogastric tubes (NGTs) at the end of surgery. Most centers would wait at least 4-7 days before removing the NGTs and obtaining a contrast study (barium

esophagogram) to show no leak prior to starting clear liquids. The diet can then be advanced slowly. Chest tubes can be managed as described in previous sections for effusions or empyemas.

GASTROESOPHAGEAL REFLUX DISEASE (GERD)

Pathophysiology

Gastroesophageal reflux disease (GERD) is one of the most common causes of non-cardiac chest pain. It is caused by excess acid exposure in the distal esophagus due to high gastric acid levels, and incompetent lower esophageal sphincter (LES), a hiatal hernia, or any combination of stated factors. In normal physiologic conditions, the LES works with the muscles of the diaphragm hiatus, the crura, to allow food boluses to pass from the esophagus to the stomach and to block acid from going in reverse from the stomach to the esophagus. Due to the negative pressure in the chest, the LES is always under some “pressure” to slide into the chest. When people have weakened tissues and/or increased abdominal pressure (due to obesity), the stomach and LES can be pushed up into the chest leading to discordant functioning of the muscles and increased acid exposure to the esophagus. The loss of the angle of His, between the LES and the cardia of the stomach can also lead to the loss of the valve-like function of the LES. Over time, this acid exposure can lead to the formation of Barrett's esophagitis, or the development of intestinal metaplasia in the esophagus. This abnormal tissue is a risk factor for developing esophageal cancer in the long term. Acid exposure alone can cause symptoms of chest pain and if the acid moves proximally to the upper esophagus, it can be aspirated into the lungs leading to pneumonitis and asthma-like symptoms. Long-term acid exposure and the chronic inflammation of the lower esophagus can also cause a stricture of the distal esophagus leading to dysphagia and other symptoms. This was more common in the era before oral H₂ blocking medications. Increased acid exposure can be the result of increased gastrin production, as seen with gastrinomas and Zollinger-Ellison syndrome.

People can have GERD in the setting of normal stomach and diaphragm anatomy. However, many have a concurrent hiatal or paraesophageal hiatal hernia though, with the LES and proximal stomach “sliding” into and out of the chest (Type I Hiatal Hernia). A Type II hernia is defined when the LES is at the level of the crura but the fundus has herniated through into the chest, while a Type III is a formal “paraesophageal” hiatal hernia with the fundus AND LES pulled up in to the chest, and lastly, a Type IV is defined by the presence of other organs herniating next to the stomach, like the colon or pancreas. When 1/3 or more of the stomach is in the chest, it can torsion and even strangulate, resulting in severe chest pain and possible stomach necrosis requiring urgent surgery. Achalasia is a separate esophageal motility problem that can often be mistaken for GERD, as it leads to dysphagia and regurgitation. Achalasia is usually defined by a lack of peristalsis in the esophagus (aperistalsis) and a normo- or hypertensive LES with no relaxation, which results in food staying in the esophagus and esophageal dilation.

Signs and Symptoms

The symptoms of reflux disease can sometimes be indistinguishable from cardiac chest pain. When it presents with classic symptoms, patients usually have a relationship between symptoms and food, with pain following 30 min. after food intake. Patients may have dysphagia if a stricture is present. In the acute setting, patients can be tachycardic, tachypneic, and diaphoretic. Other typical causes of chest pain and SOB must be ruled out first. A thorough history can elucidate a known history of reflux. A history of a hiatal hernia can also lead to a concern about GERD. A history of ulcer disease in the stomach or duodenum is usually indicative of excess acid production or exposure distal to the LES and may not be related to GERD. A physical exam may be underwhelming as there are no specific physical signs for reflux. Patients with hiatal hernias may present with chest pain due to food dilating the proximal stomach or torsion of the stomach, shortness of breath, or anemia from chronic gastritis secondary to the hernia. Patients with achalasia usually present with the triad of dysphagia, regurgitation/emesis, and weight loss.

Relevant Diagnostic Studies

In the acute setting, testing should be done to rule out other causes of chest pain as described earlier. A chest x-ray, barium contrast study, and Chest CT may be done quickly to ensure no other problems. A barium esophagogram can rule out a perforation, but also identify a hiatal hernia and even show the presence of reflux if the barium moves in reverse after entering the stomach. (See Figure 6.) In achalasia, the classic finding is a “bird’s beak”, reflecting a dilated esophagus that narrows to a “beak” at the LES which does not relax/open. (See Figure 7.) An upper endoscopy may be performed, but that is not the best test for determining excess acid, and even if Barrett’s changes are identified, GERD may not be the cause of symptoms.

Treatment with a cocktail of medicines that include topical esophageal medicine (Sucralfate) and acid blockers can be used, and if symptoms improve, can be diagnostic. Testing for H. Pylori infections, if positive, allows for antibiotic treatment. Other elective studies include pH testing and manometry which identify the presence of excess acid and symptoms, and the function of the esophageal muscle to push food down respectively. Manometry will help define achalasia or indicate the presence of other esophageal dysmotility disorders such as esophageal spasm, which is not treated with surgery. These tests, along with an EGD, aid in decision making for the long-term medical or surgical management of GERD or other esophageal diseases.

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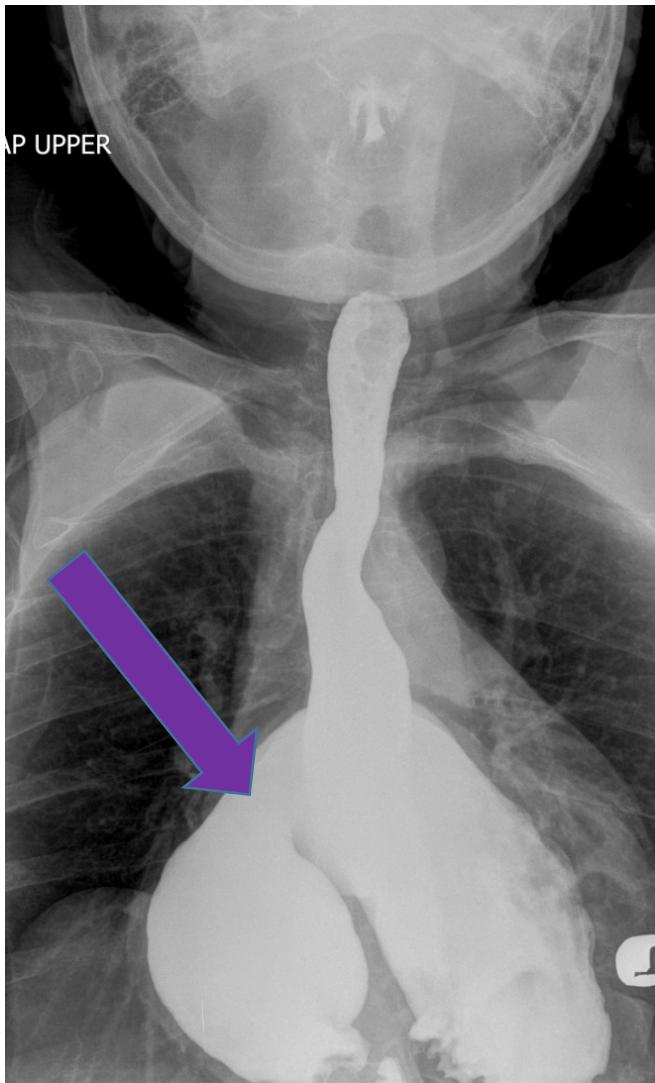


Figure 6: Barium Esophagogram showing a Type III paraesophageal hiatal hernia with the stomach inverted in the chest (arrow).

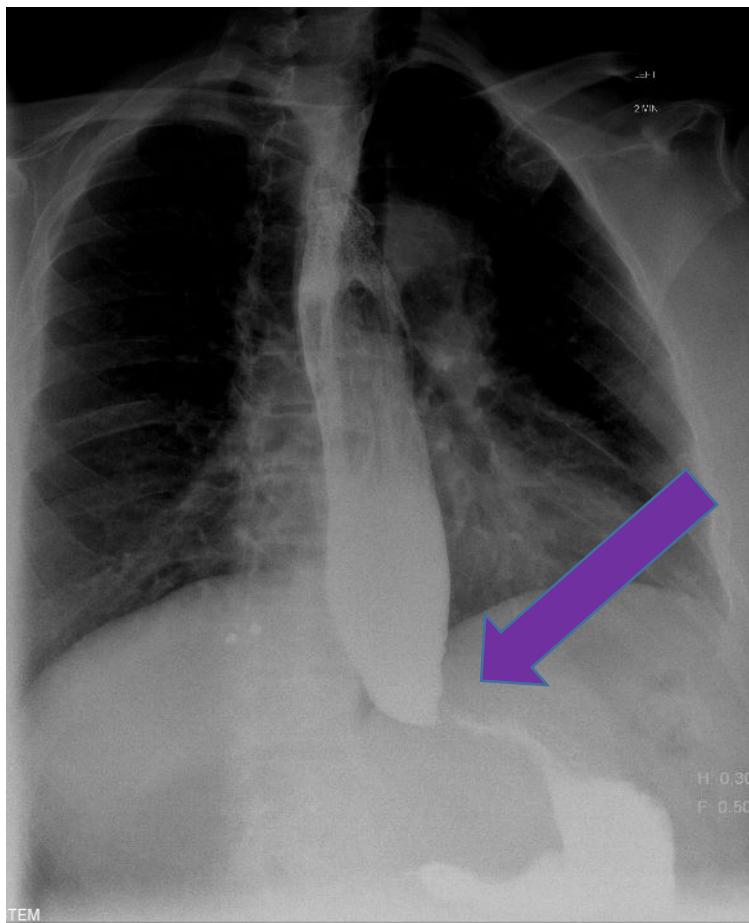


Figure 7: Barium Esophagogram showing a dilated esophagus with a narrow tapering at the distal esophagus reflecting a non-relaxing LES (arrow).

Non-operative Management

In the acute setting, medical management is key. Even in the long-term setting, management with H₂ blockers or proton-pump inhibitors and other medicines that suppress acid production, have become the mainstay of treatment. Ideally, lifestyle changes including diet modification (elimination of acid-producing foods like coffee, citrus, etc.) and weight loss can eliminate reflux and the need for medication. In recent years, there has been growing concerns about complications from the long-term use of acid suppressing medications, from the change in the gut microbiome to an association with dementia. This has led to more interest in the surgical treatment of reflux. Acid suppression may be able to limit the progression of Barrett's esophagitis. Once the presence of Barrett's esophagitis is known, surveillance EGD's are recommended with biopsies of the length and circumference of Barrett's mucosa to rule out dysplasia and early esophageal cancers. If high grade dysplasia is found on a biopsy, there is up to a 40% risk of concurrent esophageal adenocarcinoma present and the dysplasia should be treated. The historical treatment was esophagectomy, but now, endoluminal therapies (by EGD) including ablation or endomucosal resection are possible. Resection is often favored as it

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allows for pathologic examination of the tissue for possible occult cancers, but it is technically more challenging to perform than ablation. Hiatal hernias cannot be fixed without surgery, but reflux symptom management can minimize chronic symptoms. In the acute setting, nasogastric tube decompression is mandatory to reduce stomach distension and possible strangulation. For achalasia, endoscopic dilation with or without Botox injections to the LES can help manage symptoms but require repeat endoscopies.

Operative Management

The operative management of GERD is tied to the presence of hiatal hernias and the response of patients to acid suppression. If diagnostic testing has confirmed excess acid exposure in the lower esophagus and normal, or near normal esophageal motility, a laparoscopic Nissen fundoplication is the operation of choice. During the operation, the crura are dissected free and repaired to narrow the esophageal hiatus. A fundoplication is performed to reinforce the LES by wrapping the mobilized fundus of the stomach in a 360 degree fashion around the LES. With the crural closure and wrap, most people will have a significant amount of symptom relief. If there is abnormal esophageal motility, and there is a concern that a full wrap will block food from entering the stomach, a partial wrap can be performed (Toupet fundoplication). Concurrent hiatal hernias, if present, are repaired surgically in the same manner (fundoplication, crural repair) as one would for GERD alone, although for moderate to large hernias, approaches may be either through the left chest (thoracotomy) or the abdomen (laparoscopic or open). Laparoscopic Nissen fundoplications have been shown to stop the progression of Barrett's esophagitis. The reduction in GERD symptoms is highest in those patients that have had good symptom relief by medications prior to surgery. In the absence of a hiatal hernia, there are endoluminal therapies designed to recreate the angle of His by internally plicating the LES and stomach. The success of these therapies has yet to be proven in long-term studies. Achalasia has been treated by performing a laparoscopic (Heller) myotomy of the lower esophagus, through the LES, and onto the stomach with a concurrent partial fundoplication. Endoscopic myotomies are becoming more mainstream.

Basic Postoperative Care

Postoperative care for fundoplications and myotomies are fairly straightforward. Some surgeons will place an NGT after surgery and remove it 1-2 days postoperatively with a barium contrast study to demonstrate no evidence of a leak. Patients will be discharged on clear liquids or a soft diet. As most operations are being performed by a laparoscopic approach, patients are ambulating quickly with minimal pain concerns. Up to 30-40% of patients with GERD will have dysphagia after a fundoplication. This almost always resolves in 2-3 months and does not require dilations. Patients with achalasia will often still have some symptoms of dysphagia but are much improved.

PNEUMONIA

Pathophysiology

Pneumonia can arise in a variety of different settings. Our lungs are normally colonized with a variety of bacteria that can become pathologic due to immune suppression or some imbalance in the bacterial flora leading to overgrowth of one dominant species. The most common bacterial causes of pneumonia are *Streptococcus pneumoniae* and *Staphylococcus aureus* (gram positive bacteria), although gram negative bacteria such as *Haemophilus influenza* and *Klebsiella pneumoniae* from the gastrointestinal tract can also cause pneumonia. The overgrowth of bacteria leads to the recruitment of macrophages and other white blood cells to the area of the lung, leading to local inflammation and congestion of the tissue. These changes in the lung parenchyma can lead to shortness of breath, if a significant amount of lung is affected, and cough due to airway irritation. The congestion of the lung can lead to a parapneumonic effusion which can be sterile or infected. Most of these effusions will resolve if the pneumonia is treated, but a small percentage can become larger as the bacteria in the pleural fluid multiply, resulting in more fluid in the chest and the development of pus as white blood cells are released to fight the infection in the pleural fluid.

Signs and Symptoms

Pneumonia can present with only chest pain or only SOB, but often has other symptoms such as cough and fevers. Patients may have had a prior upper respiratory infection in the recent few weeks and may have secondary symptoms of malaise and fatigue. A medical history of immune suppression can raise the likelihood of pneumonia. Patients with a history of other lung diseases, such as emphysema, are more prone to pneumonias also. Physical exam findings may include a fever, tachypnea, and tachycardia. A lung exam may reveal diminished breath sounds, especially if an effusion is present, or even wheezing and crackles. Oxygenation levels may be diminished also.

Relevant Diagnostic Studies

The first steps will be blood work to look for leukocytosis and a chest x-ray will show some evidence of congestion/consolidation, and an effusion if present. (See Figure 8.) Sometimes, early or subtle pneumonias may be missed on chest x-ray but can be seen on a Chest CT scan performed during the early work-up phase. Once pneumonia is suspected or confirmed, an induced sputum sample may be sent for culture to identify the responsible organisms. In patients with immune suppression, a diagnostic bronchoscopy may be warranted to get better sputum samples from within the lung.

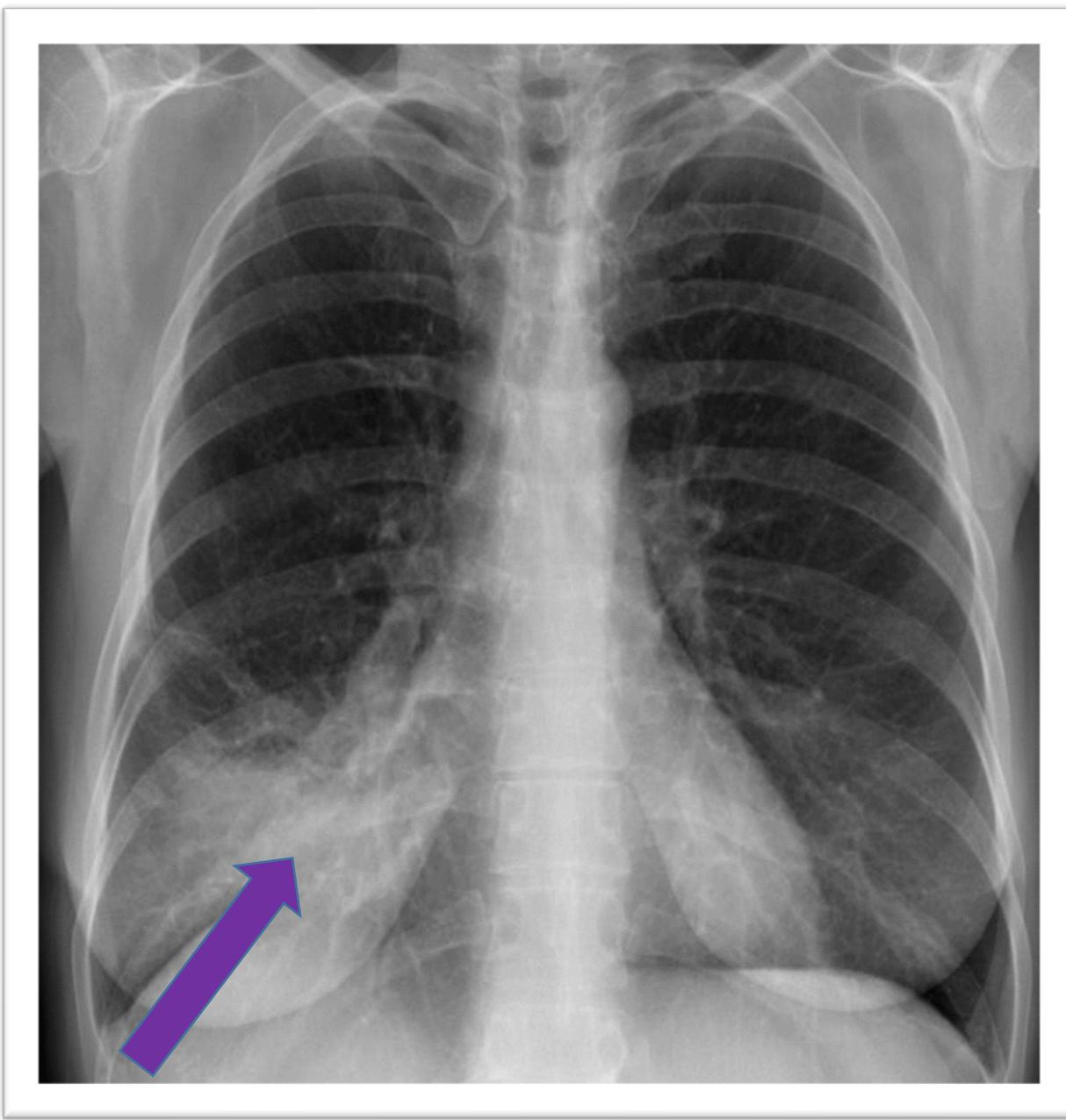


Figure 8: Right Middle Lobe pneumonia. Consolidation seen at the right base (arrow).

Non-operative Management

The mainstay of treatment for pneumonia is broad spectrum antibiotics. If cultures are sent and a specific organism is identified, the antibiotic choice can be narrowed down. Intravenous antibiotics may be started for patients who appear sick, i.e. have malaise, leukocytosis, and SOB. Patients with milder pneumonias may be started on oral antibiotics and discharged home with short-term follow up with their primary care physician. Concordant effusions or empyemas should be managed as described earlier in this section.

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Operative Management

Historically, lung resection was an option for lung abscesses. In the current era, there is no role for surgery for pneumonia itself and even lung abscesses are better treated with percutaneous drainage and long-term antibiotics. Percutaneous drains of abscesses can lead to bronchopleural fistulae though, and the plan to place one should be discussed in a multidisciplinary manner with infectious diseases, surgery, pulmonary medicine, etc. prior to placement. Surgery can be used to help manage the sequelae of the pneumonia like an empyema, but infected lung tissue is fragile and even the grasping of consolidated lung tissue can lead to multiple inadvertent injuries.

Basic Postoperative Care

Postoperative care should be applied for treatment of the sequelae of pneumonias as described above.

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Questions

1. A 57-year-old man presents to the ER with sudden onset chest pain and shortness of breath for 2 hours. A CT chest reveals pulmonary emboli. The next best step in treatment is:
 - A. Systemic anticoagulation
 - B. Morphine, aspirin, and oxygen
 - C. Pulmonary embolectomy
 - D. Directed Thrombolytic therapy

2. An 80-year-old woman presents with a 10-day history of fevers, shortness of breath, and chest pain. A chest x-ray shows a left lower lobe pneumonia with an effusion. A CT chest is obtained that shows a small effusion and 5 cm left lower lobe lung abscess. The next best step in treatment is:
 - A. Surgical resection of the abscess
 - B. Pigtail placement into the abscess
 - C. Broad spectrum antibiotics
 - D. Chest tube to drain the effusion

3. A 42-year-old woman presents with a 4-hour episode of excruciating chest pain after a large meal. Coronary and pulmonary problems have been ruled out. A barium esophagogram shows a Type III Paraesophageal hiatal hernia with contrast in the proximal stomach above the diaphragm. The next best step in treatment is:
 - A. Surgery to reduce the hernia
 - B. Upper Endoscopy
 - C. Proton Pump Inhibitors
 - D. Nasogastric tube decompression

4. A 21-year-old man presents with a 6-hour history of chest pain and now worsening shortness of breath. He is alert and oriented x 3. A chest x-ray shows a large left-sided pneumothorax with mild tracheal deviation to the right. The next best step in management is:
 - A. Chest CT scan
 - B. Bronchoscopy
 - C. Intubation
 - D. Left sided chest tube placement

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Answers

1. A 57-year-old man presents to the ER with sudden onset chest pain and shortness of breath for 2 hours. A CT chest reveals pulmonary emboli. The next best step in treatment is:

- A. **Systemic anticoagulation**
- B. Morphine, aspirin, and oxygen
- C. Pulmonary embolectomy
- D. Directed thrombolytic therapy

Morphine, aspirin, and oxygen comprise the acute treatment for a heart attack. Pulmonary embolectomy and thrombolytic therapy are technically options, but only after documenting heart strain and having a multidisciplinary review of treatment options. The first step treating a PE is systemic anticoagulation.

2. An 80-year-old woman presents with a 10-day history of fevers, shortness of breath, and chest pain. A chest x-ray shows a left lower lobe pneumonia with an effusion. A CT chest is obtained that shows a small effusion and 5 cm left lower lobe lung abscess. The next best step in treatment is:

- A. Surgical resection of the abscess
- B. Pigtail placement into the abscess
- C. **Broad spectrum antibiotics**
- D. Chest tube to drain the effusion

Surgical resection is no longer indicated. Pigtail placement is an option but should be considered carefully as it can cause complications (bronchopleural fistula). Chest tube drainage for small effusions is unnecessary. Administering antibiotics is the core treatment for this condition.

3. A 42-year-old woman presents with a 4-hour episode of excruciating chest pain after a large meal. Coronary and pulmonary problems have been ruled out. A barium esophagogram shows a Type III Paraesophageal hiatal hernia with contrast in the proximal stomach above the diaphragm. The next best step in treatment is:

- A. Surgery to reduce the hernia
- B. Upper endoscopy
- C. Proton pump inhibitors
- D. **Nasogastric tube decompression**

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The patient is presenting with an acute obstruction and possible torsion and strangulation of her stomach. Surgery and endoscopy may be indicated, but urgent decompression by first placing a nasogastric tube is essential to reduce the size of the stomach. This alone can resolve the pain and urgency of the situation. Proton pump inhibitors can treat the reflux symptoms long term but have no role in the acute setting.

4. A 21-year-old man presents with a 6-hour history of chest pain and now worsening shortness of breath. He is alert and oriented x 3. A chest x-ray shows a large left-sided pneumothorax with mild tracheal deviation to the right. The next best step in management is:
 - A. Chest CT scan
 - B. Bronchoscopy
 - C. Intubation
 - D. Left-sided chest tube placement**

As he is alert and oriented, intubation is not required. A CT Chest and/or bronchoscopy may be done to identify the cause of the pneumothorax but only after the acute situation is addressed. A chest tube and/or needle decompression of the left side are immediate options to relieve the tension pneumothorax.

Problems

For each of the following problems, answer the following questions:

- What further data should be obtained from the patient's history?
 - What findings would you look for on physical exam?
 - What is your differential diagnosis?
 - What work-up would you recommend (include laboratory tests and diagnostic interventions)?
 - What therapy or treatment would you recommend?
1. A 75-year-old man presents with acute chest pain and shortness of breath after a long, trans-Pacific flight from Japan.
 2. A 45-year-old woman presents with post-prandial chest pain. She is short of breath and feels a fullness in her chest. Similar symptoms occurred 2 months ago but symptoms resolved after she vomited.
 3. A 19-year-old man was lifting weights and developed left sided chest pain and mild shortness of breath. He has a cousin with cystic fibrosis.

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Non-Cardiac Chest Pain and Shortness of Breath

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Authors/Contributors

Rishindra M. Reddy, MD, FACS (Content Author)
University of Michigan Department of Surgery, Ann Arbor, MI

Stephen C. Yang, MD, FACS (Section Editor and Goals and Objectives Author)
Johns Hopkins Medical Institutions, Baltimore, MD

Darshan Vummidi, MD (Images)
University of Michigan Department of Radiology, Ann Arbor, MI

Leslie Quint, MD (Images)
University of Michigan Department of Radiology, Ann Arbor, MI