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Postoperative Pulmonary Complications: Clinical and Imaging Insights

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Postoperative pulmonary complications (PPCs) continue to be a major cause of illness and death among surgical patients, affecting recovery and increasing healthcare expenses. This in-depth review examines the wide range of PPCs, covering infectious, inflammatory, mechanical, airway, vascular, lymphatic, and transplant-related issues. It highlights the vital function of imaging-especially computed tomography-in promptly identifying, correctly diagnosing, and managing these problems. Frequent complications like pneumonia, empyema, acute respiratory distress syndrome, atelectasis, and pulmonary embolism are covered, as well as rarer but serious conditions such as lung torsion, bronchopleural fistulas, and pseudoaneurysms. The review describes clinic signs and matches them with imaging characteristics to support diagnosis and treatment planning. It also underscores the value of a team-based approach involving surgeons, radiologists, and critical care experts to enable timely care and better patient results.

INTRODUCTION

Postoperative pulmonary complications (PPCs) are a major source of morbidity and mortality following surgery, hindering recovery and raising healthcare expenditures. These complications include a wide range of respiratory conditions, making prompt detection and precise diagnosis essential for appropriate management.[1-3] Imaging is central to this process; chest radiographs are generally the initial tool, while computed tomography (CT) is used for more comprehensive evaluation when needed.[1,3] This review outlines PPCs, focusing on clinical features, important imaging findings, guideline-directed treatment, and prognostic considerations to assist radiologists, pulmonologists, surgeons, and intensivists in daily practice.

POSTOPERATIVE PULMONARY COMPLICATIONS

Infectious complications

Pneumonia

Pneumonia is one of the most frequent PPCs, with higher rates in older patients, those with chronic lung disease, or those who have required extended mechanical ventilation.[1,2] Clinically, patients often present with fever, productive cough, shortness of breath, and elevated white blood cell counts.[2] While chest X-rays commonly show new lung infiltrates, they can fail to detect pneumonia in up to one-third of patients; CT scans have higher sensitivity and can reveal smaller or more widespread areas of lung consolidation.[1,3] As per the American College of Radiology guidelines, chest radiography should be the first imaging test for suspected pneumonia, but CT is advised in severe, atypical, or treatment-resistant cases because it can uncover hidden pneumonia or other possible diagnoses[4] (Figure 1). A definitive diagnosis of postoperative pneumonia is made by combining imaging results with microbiological evidence from sputum or bronchoalveolar lavage cultures. Current recommendations suggest starting empiric antibiotic treatment based on local antibiograms and patient-specific risk factors to improve outcomes and limit resistance.[4-6] With prompt treatment, most patients recover; however, delayed therapy may lead to severe infection or sepsis.[6]

Empyema

Empyema is characterized by the buildup of pus within the pleural cavity, commonly developing as a complication of pneumonia, chest surgery, or thoracic trauma. Patients usually exhibit fever, sharp chest pain worsened by breathing, and difficulty breathing.[5,6] Chest ultrasound is useful for detecting pleural fluid and often shows internal septations or loculations, which are indicative of an exudative effusion.[7] CT imaging plays an important role in evaluating empyema because it clearly defines the pleural anatomy and helps differentiate empyema from simple pleural effusions or lung abscesses. On contrast-enhanced CT, empyemas typically present

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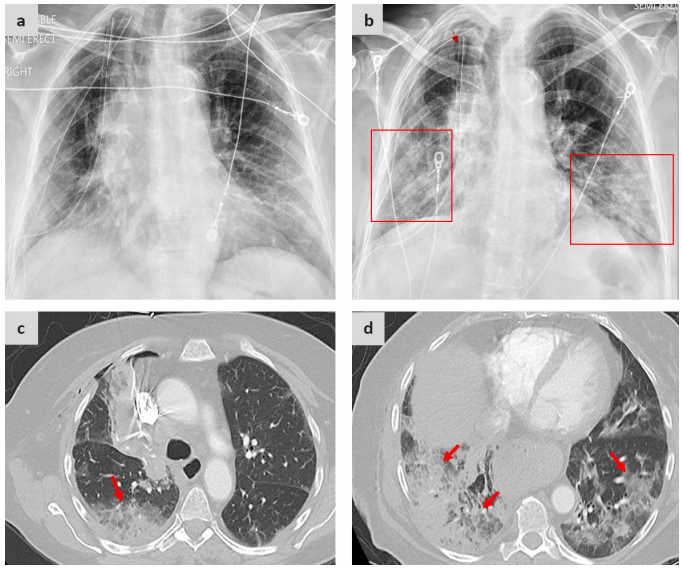

FIG. 1. Pneumonia in a patient with right upper lobectomy due to lung adenocarcinoma. (a) Frontal chest radiograph on postoperative day (POD) 0 demonstrates two apical chest tubes, reduced right lung volume, and a moderate hiatal hernia. (b) Follow-up frontal chest radiograph on POD 4 shows new bibasilar, patchy airspace opacities (rectangles). (c, d) Axial chest CT images reveal postoperative changes of right upper lobectomy, including mild anterior pneumothorax and right hilar distortion. New consolidative and ground-glass opacities predominate in the right lower lobe (greater than left), findings compatible with pneumonia (arrows). A large hiatal hernia is also evident.
CT, computed tomography.

as lens-shaped fluid collections that enlarge the pleural space and compress the surrounding lung and mediastinum. A well-known feature is the "split pleura sign," where both the visceral and parietal pleura appear thickened and enhanced by contrast, separated by the fluid; while this sign strongly points to empyema, its absence does not exclude the condition[8] (Figure 2). CT may also show the presence of pleural air or an air-fluid level, especially if there is a connection with a bronchopleural fistula (BPF). When empyema is confirmed, image-guided thoracentesis is carried out to verify the purulent nature of the fluid, which typically demonstrates low pH, low glucose, and elevated lactate dehydrogenase levels. Treatment focuses on timely drainage and suitable antibiotic administration.[9] Early management, guided by imaging findings, is essential to avoid chronic organizing empyema (fibrothorax) and to facilitate lung re-expansion.[7-9]

Sepsis

Sepsis is a severe, life-threatening systemic inflammatory response to infection that can lead to multiorgan failure.[6,10] Among postoperative patients, it often arises from issues such as pneumonia, intra-abdominal abscesses, or urinary tract infections. Early clinical signs include fever, rapid heart rate, low blood pressure, and changes in mental status, accompanied by lab findings that indicate inflammation and organ damage.[1,6] Imaging methods like chest X-rays and CT scans help detect lung consolidations, empyemas, or abscesses that could be the source of infection (Figure 3).

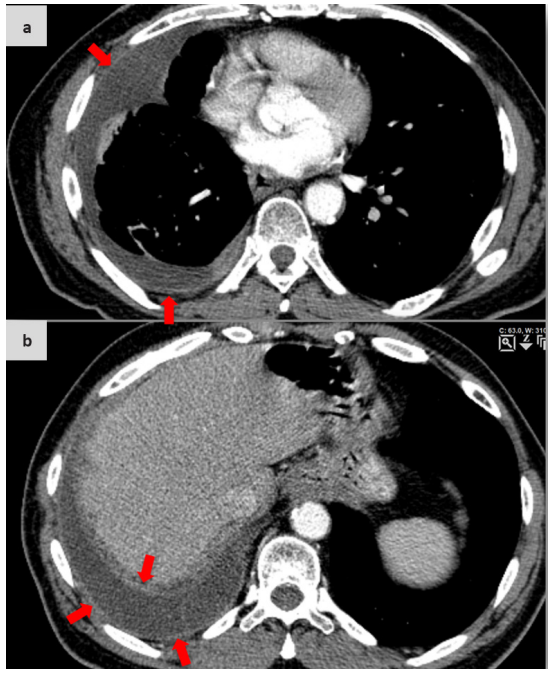

FIG. 2. Empyema in a patient following wedge resection for pulmonary metastasis. (a, b) Axial CT images at the level of the lower hemithorax demonstrate a small loculated pleural effusion with thickened and enhancing visceral and parietal pleura (the split-pleura sign; arrows), findings consistent with empyema.
CT, computed tomography.

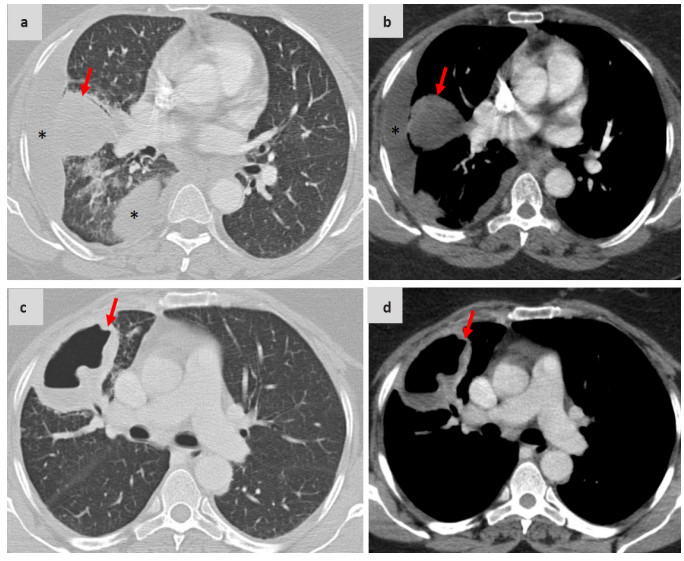

FIG. 3. Empyema and lung abscess in a patient following right upper lobectomy for primary lung adenocarcinoma. (a, b) Early postoperative axial chest CT images show a loculated right pleural effusion (*) and focal consolidation in the right lung (arrows). (c, d) Follow-up axial chest CT images obtained three weeks later demonstrate resolution of the pleural effusion and the development of a cavitary lung lesion consistent with an abscess (arrows). The lung abscess was confirmed histopathologically.
CT, computed tomography.

Recent studies show that CT is highly sensitive for identifying septic sources in emergency settings and that the timing of the scan does not significantly influence morbidity or mortality outcomes.[11] Once the infection source is confirmed, management focuses on rapid source control, administration of broad-spectrum antibiotics, and supportive measures for organ dysfunction.[10,11] Despite improved treatment approaches, sepsis still carries substantial mortality risk, highlighting the importance of careful clinical observation and timely imaging.

INFLAMMATORY COMPLICATIONS

Acute respiratory distress syndrome (ARDS)

Acute respiratory distress syndrome (ARDS) occurs when severe inflammation increases alveolar-capillary permeability, resulting in widespread pulmonary edema, impaired gas exchange, and profound hypoxemia.[12] After surgery, ARDS can develop due to sepsis, trauma, massive blood transfusions, or aspiration, usually appearing within a few days postoperatively with sudden shortness of breath, rapid breathing, and hypoxemia that does not respond well to oxygen therapy. Diagnosis depends on imaging: chest X-rays show bilateral diffuse infiltrates, while CT scans reveal widespread ground-glass opacities and consolidations, indicating edema and lung collapse (Figure 4).[1,12,13] The absence of significant pleural effusions helps differentiate ARDS from heart-related pulmonary edema.[1,3] Management is mainly supportive and includes lung-protective mechanical ventilation with low tidal volumes (4-6 mL/kg ideal body weight), appropriate positive end-expiratory pressure (PEEP), prone positioning in severe cases, conservative fluid strategies, and prompt treatment of underlying causes. Despite therapeutic improvements, ARDS remains associated with high mortality, and survivors often face extended recovery periods and possible long-term lung fibrosis.[12,13]

Ischemia-reperfusion injury

Ischemia-reperfusion injury (IRI) happens when blood supply returns after a period of ischemia, causing oxidative damage and inflammation.[14] It is most commonly seen in lung transplantation-where it is known as primary graft dysfunction (PGD)-but may also follow procedures like pulmonary thromboendarterectomy. IRI usually leads to sudden hypoxemia and decreased lung compliance within hours to days.[15] Imaging features are similar to those of ARDS: chest X-rays demonstrate bilateral diffuse opacities consistent with pulmonary edema, while CT shows ground-glass areas, patchy consolidations, and septal thickening. Since imaging cannot reliably distinguish IRI from other causes of lung infiltrates, clinical context is essential for diagnosis (Figure 5).[16] Management is supportive and includes lung-protective ventilation with adequate PEEP, careful

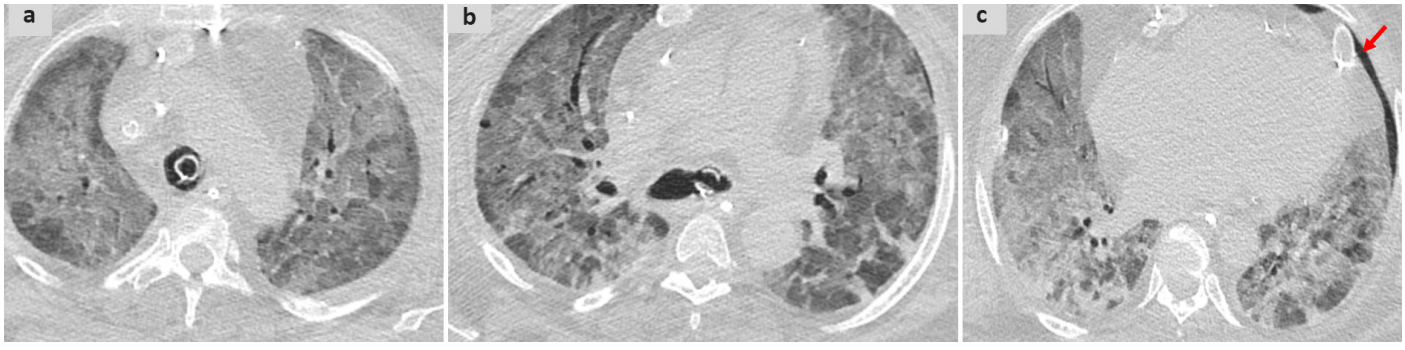

FIG. 4. Acute respiratory distress syndrome (ARDS) in a patient following coronary artery bypass surgery. (a-c) Axial chest CT images demonstrate extensive ground-glass opacities and patchy basilar consolidations, along with a trace left pneumothorax (arrow). The absence of significant pleural effusions favors ARDS over cardiogenic pulmonary edema. Note the presence of an endotracheal tube.
CT, computed tomography.

FIG. 5. Ischemia-reperfusion injury in a patient following mitral valve surgery. (a) Coronal and (b, c) axial chest CT images demonstrate extensive ground-glass opacities, patchy consolidations, and interlobular septal thickening. Echocardiography showed normal biventricular function, supporting a non-cardiogenic etiology.
CT, computed tomography.

fluid balance, and, in severe cases, veno-venous extracorporeal membrane oxygenation (ECMO). Outcomes vary widely, from full recovery in mild cases to death or persistent lung dysfunction in severe ones.[15,16]

Chronic bronchial inflammation

Chronic bronchial inflammation develops from ongoing irritation due to intubation, mechanical injury, or persistent infections, leading to structural changes in the airways and lasting airflow restriction. Patients usually report a persistent productive cough, wheezing, and shortness of breath that may continue for weeks after surgery. Diagnosis is mainly clinical, supported by pulmonary function tests-which often show an obstructive pattern-and imaging.[17] Chest CT findings tend to be subtle or non-specific, commonly showing bronchial wall thickening, narrowed lumens, and mosaic attenuation that suggests air trapping. Expiratory CT scans are especially helpful for confirming air trapping in small airway disease. Although these imaging findings are not specific enough for diagnosis alone, they help assess the extent of airway involvement and rule out other conditions like pneumonia or bronchiolitis obliterans.[3,18] Treatment includes bronchodilators, corticosteroids to reduce inflammation, chest physiotherapy to clear mucus, and management of any ongoing infection. Long-term measures such as smoking cessation and avoiding other irritants are also important for recovery.[17]

MECHANICAL COMPLICATIONS

Atelectasis

Atelectasis is a frequent postoperative issue following abdominal or thoracic procedures and is mainly caused by shallow breathing due to pain, mucus obstruction, or extended immobility.[1] Patients often present with shortness of breath, low oxygen levels, and reduced breath sounds. Chest X-rays indicate volume loss through findings like increased opacity, an elevated diaphragm, rib crowding, and a shift of the mediastinum, while CT offers a clearer view of the collapsed lung and any underlying causes such as mucus plugs or masses. CT can also help distinguish atelectasis-where enhancement occurs without an air bronchogram-from pneumonia, which typically shows an air bronchogram (Figure 6).[3,17] Management aims to re-expand the lung using pulmonary toilet techniques, incentive spirometry, good pain control, early mobilization, and chest physiotherapy; bronchoscopy is performed if mucus plugging is present.[3] With timely treatment, postoperative atelectasis usually resolves fully and has an excellent outcome.

Pulmonary edema

Postoperative pulmonary edema can occur due to cardiac dysfunction that raises hydrostatic pressure or from inflammatory processes like ARDS, which cause increased capillary permeability and fluid buildup outside the blood vessels. These conditions require different treatment approaches, so distinguishing cardiogenic from non-cardiogenic edema through clinical assessment and imaging is essential.[1,3] On chest X-rays, cardiogenic edema typically appears as bilateral perihilar "bat wing" opacities, Kerley B lines, pleural

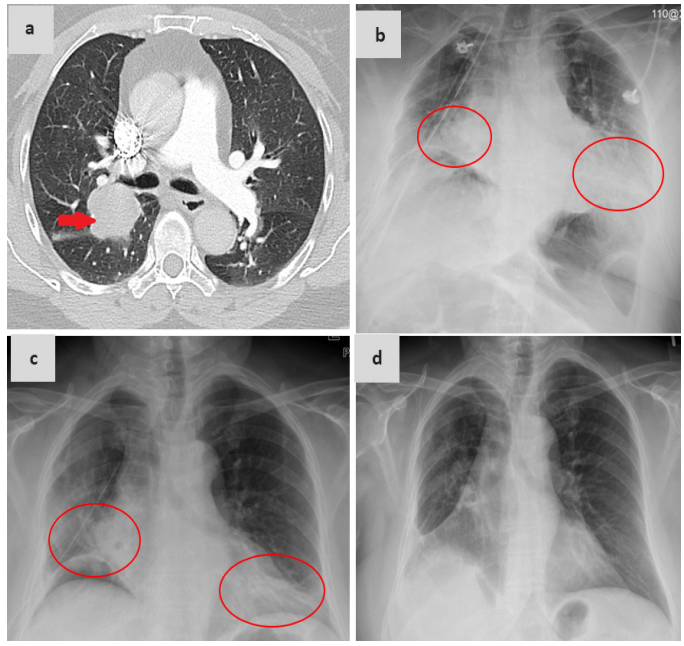

FIG. 6. Bibasilar atelectasis after right upper lobectomy. (a) Pre-operative axial chest CT demonstrates a right upper lobe mass (arrow). (b) Postoperative day 2 chest radiograph shows bibasilar patchy opacities (circles), consistent with atelectasis. (c) On postoperative day 4, there is a slight decrease in the bibasilar opacities (circles). (d) By postoperative day 7, the bibasilar opacities have significantly decreased.
CT, computed tomography.

effusions, and an enlarged heart, while non-cardiogenic edema generally presents with bilateral infiltrates but without notable heart enlargement or significant effusions.[19] CT imaging offers additional information: cardiogenic edema often shows smooth thickening of the interlobular septa and widespread, centrally located ground-glass opacities with frequent pleural fluid, whereas edema due to ARDS tends to have more irregular, peripheral or dorsal ground-glass opacities, along with areas of consolidation and dependent atelectasis.[3,13,17] Management depends on the cause-cardiogenic edema is treated with diuretics, afterload reduction, and measures to support cardiac function, while non-cardiogenic edema or ARDS is handled with supportive ventilation and careful fluid management.[13]

Persistent air leak

A persistent air leak occurs when damage to the alveoli or small airway allows air to continuously escape into the pleural space, often observed as ongoing bubbling in the chest tube for more than 24-48 hours after surgery. This complication is common following lung resections or extensive pleural dissections, where bronchopleural or alveolopleural fistulas can develop, leading to subcutaneous emphysema and preventing full lung re-expansion, which may cause respiratory distress.[20] Chest X-rays may reveal a lasting pneumothorax and subcutaneous emphysema, while CT-especially when a BPF is suspected-can help identify the air leak directly or indirectly (Figure 7).[1,3,20] Initial management focuses on

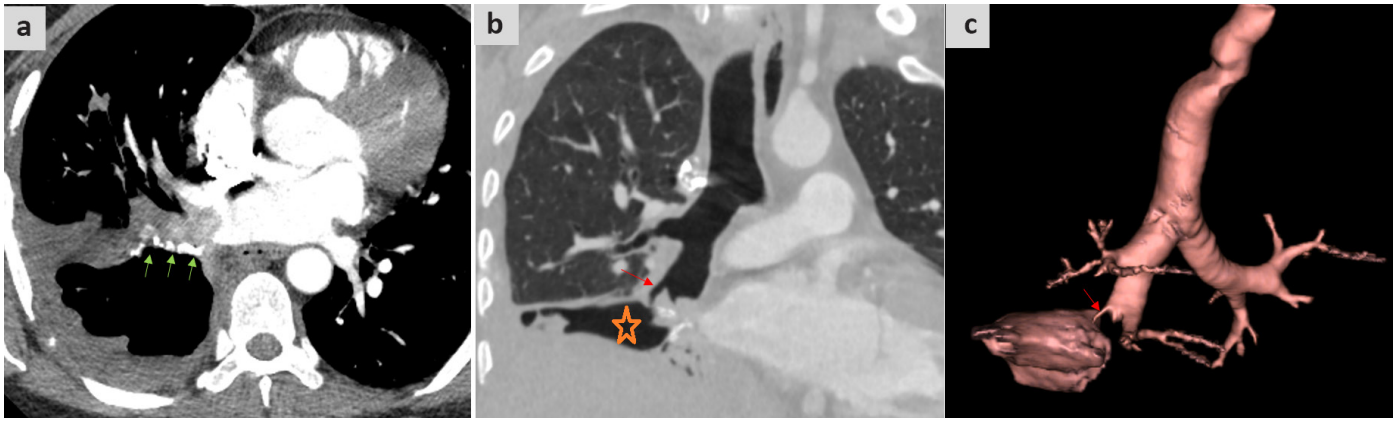

FIG. 7. (a) Axial chest CT shows surgical clips along the resection margin (arrows). (b) Coronal chest CT demonstrates a small bronchopleural fistula (arrow) and a loculated pneumothorax (star). (c) Coronal three-dimensional volume rendering CT image confirms the presence of the bronchopleural fistula (arrow).

CT, computed tomography.

ensuring that the chest tube is functioning well; many minor leaks resolve with intensive pulmonary exercises. However, leaks that persisted beyond 5-7 days or are large may need further treatment such as pleurodesis, bronchoscopic procedures, or surgical repair with resuturing and tissue reinforcement.[20]

Lung torsion

Lung torsion is an uncommon but life-threatening complication in which a lung lobe-or, rarely, an entire lung-rotates around its bronchovascular pedicle, resulting in blood flow obstruction and airway blockage.[3,21] This usually happens shortly after lung resections (especially upper lobectomies, which can leave the middle lobe at risk) or following chest trauma. Although it is rare (with an incidence of about 0.1%-0.4%), its non-specific symptoms-sudden breathing difficulty, chest pain, shortness of breath, rapid breathing, low oxygen levels, and occasionally fever or coughing up blood-require high clinical suspicion, as they can be mistaken for pneumonia or pulmonary embolism (PE).[22] Initial chest radiographs may show new lung opacities and abnormal hilar positions, but CT is more definitive, directly demonstrating the twisted bronchovascular pedicle, narrowed bronchus, and poor lobe enhancement due to compromised blood flow. CT angiography with 3D reconstruction can further show abrupt cut-offs in the pulmonary artery and vein (Figure 8).[3,21,22] Immediate surgical treatment, such as detorsion or lobectomy if the lobe is necrotic, is essential to prevent infarction, sepsis, or death.[22]

Postpneumonectomy syndrome

Postpneumonectomy syndrome is an uncommon delayed complication-most frequently seen after a right pneumonectomy-in which significant mediastinal shift compresses the bronchus and blood vessels of the remaining lung.[23] Over time, the mediastinum shifts toward the empty side of the chest, sometimes causing the tracheobronchial tree to kink-e.g., the left main bronchus can become compressed between the aorta and the spine. Clinically, patients experience worsening shortness of breath, wheezing that

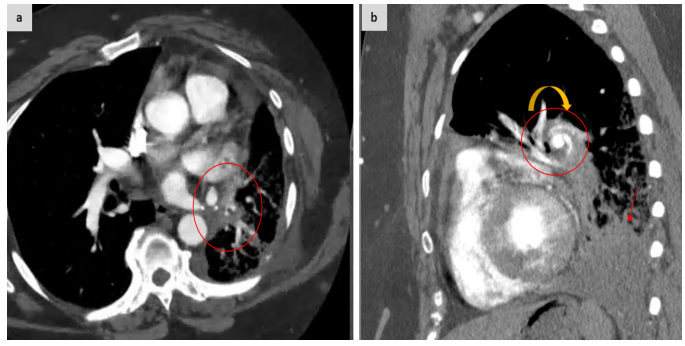

FIG. 8. (a) Axial and (b) sagittal CT angiography images demonstrate a twisted bronchovascular pedicle (circles), bronchial narrowing, and decreased enhancement of the left upper lobe (arrow), consistent with vascular compromise. In A, note the abrupt cut-off of the pulmonary artery and vein (circle), indicative of vascular torsion.

CT, computed tomography.

changes with position, coughing, repeated pneumonias, and a sense of air hunger. Chest X-rays show pronounced mediastinal shift, an elevated diaphragm on the affected side, and overexpansion of the remaining lung, while CT provides detailed views of the rotated mediastinum, narrowed bronchus, and compressed vessels (Figure 9).[21-23] Treatment is surgical and usually involves repositioning the mediastinum using a saline-filled tissue expander or a similar spacer.[23]

Lung hernia

Postoperative lung hernia is a rare complication that can develop after thoracic surgery when lung tissue pushes through a defect in the chest wall.[1,24] Clinically, patients may notice a visible or palpable bulge near the surgical site, which can enlarge with activities that raise intrathoracic pressure and may be accompanied by discomfort or pain. Imaging-particularly CT-is crucial for confirming the diagnosis,

showing the degree of lung tissue protrusion and detailing the chest wall defect, while chest X-rays may reveal a soft-tissue shadow that changes in size (Figure 10).[24] Management depends on symptom severity: conservative treatment is suitable for asymptomatic patients, while surgical repair is indicated for those with significant pain, functional problems, or risk of complications.[22,24]

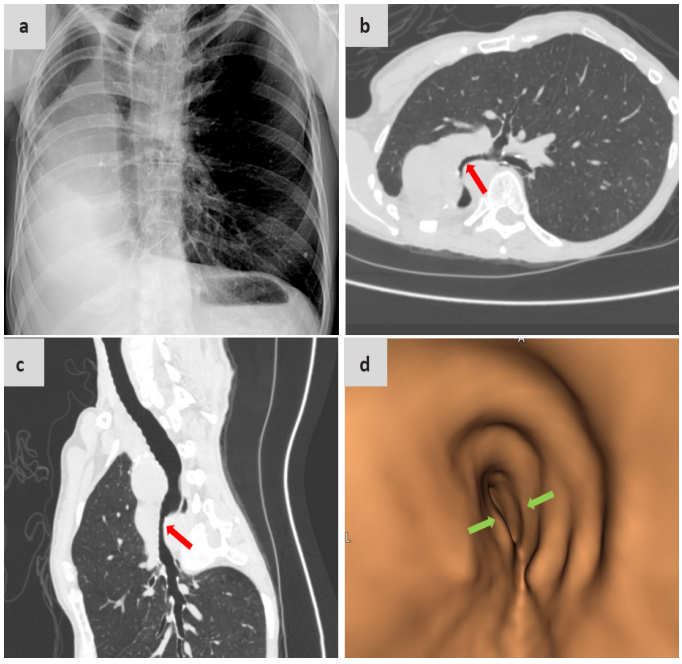

FIG. 9. Post-pneumonectomy syndrome in a patient following right pneumonectomy. (a) Frontal chest radiograph shows post-right pneumonectomy changes with marked mediastinal shift. (b) Axial oblique multiplanar reconstruction (MPR) and (c) curved MPR CT images highlight the pronounced mediastinal shift as well as elongation and severe (> 70%) narrowing of the left mainstem bronchus (red arrows). (d) Endoluminal volume-rendered movie and static 3D volume-rendered image confirm the severe mainstem bronchial stenosis (green arrows).
CT, computed tomography.

PLEURAL COMPLICATIONS

Pleural effusion

Pleural effusions commonly occur after surgery, particularly following cardiothoracic or upper abdominal operations. Most are small, do not cause symptoms, and result from fluid shifts, blood transfusions, or surgical inflammation, while larger effusions may lead to shortness of breath, pleuritic chest pain, and cough, with physical exam findings of reduced breath sounds and dullness to percussion.[3,17,22] A chest X-ray is an appropriate first test-moderate to large effusions obscure the costophrenic angle and show a meniscus sign, but small amounts of fluid can be difficult to see on supine films.[19] Ultrasound is very effective for detecting even small fluid collections, distinguishing free-flowing from loculated fluid, and guiding thoracentesis.[7] CT is mainly used for atypical, large, or persistent effusions, as it helps differentiate pleural fluid from lung consolidation and defines features of complicated effusions.[1,21,22] Most simple effusions resolve with conservative treatment, but symptomatic or large collections usually require thoracentesis for symptom relief and diagnostic evaluation.[3,22]

Pneumothorax

Pneumothorax is a known postoperative issue that may occur after lung biopsies, placement of central lines, positive-pressure ventilation, or surgery.[21,22] Patients often present with sudden pleuritic chest pain and shortness of breath, with exam findings of decreased breath sounds and hyperresonance on the affected side.[3,17] A chest X-ray typically shows a visible pleural line without lung markings beyond it, though supine films may show subtle signs like the deep sulcus sign (Figure 11).[19] CT is more sensitive, especially for detecting loculated air, bullae, or subtle lung damage. Treatment depends on the size and severity: small, asymptomatic pneumothoraces (< 2 cm) can be managed with observation and oxygen therapy, while larger or symptomatic pneumothoraces require chest tube placement, and tension pneumothorax requires immediate needle decompression followed by chest tube insertion.[1,21,22]

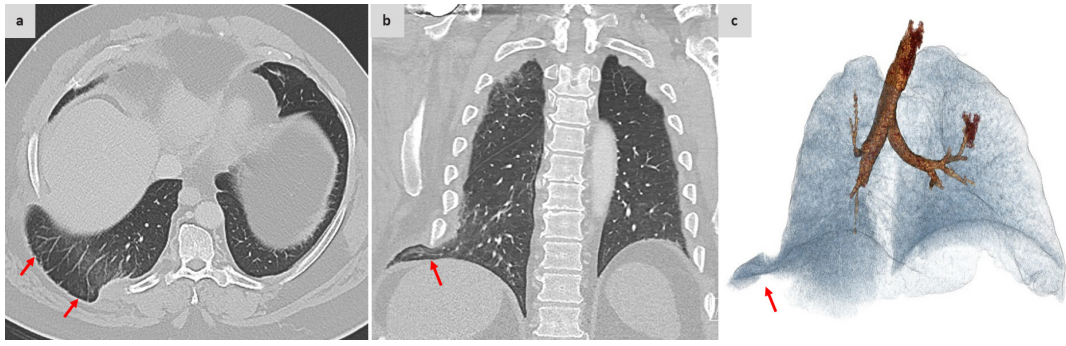

FIG. 10. Intercostal lung herniation in a patient with a history of right lower lobe wedge resection. (a) Axial, (b) coronal, and (c) three-dimensional volume-rendered CT images demonstrate intercostal lung herniation through the chest wall defect (arrows).
CT, computed tomography.

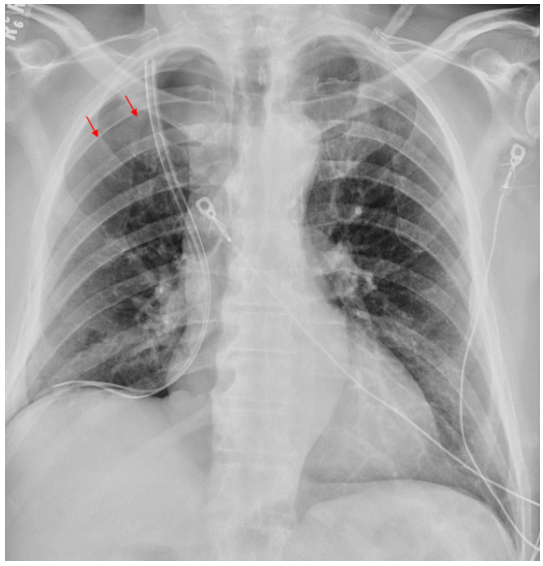

FIG. 11. Pneumothorax in a patient following right upper lobectomy. Frontal chest radiograph obtained on postoperative day 1 shows a right apical pneumothorax (arrows) and an apically positioned chest tube.

Hemothorax

Hemothorax refers to the accumulation of blood within the pleural cavity, most commonly developing after surgery due to operative bleeding, coagulopathy, or trauma.[25] Patients may report chest pain, difficulty breathing, and signs of low blood volume, with physical exam findings of reduced breath sounds and dullness to percussion. Chest X-rays usually show pleural opacification, while CT-especially with contrast-helps distinguish fresh clotted blood from simple fluid, detect active bleeding through contrast extravasation, and differentiate hemothorax from other conditions like empyema (Figure 12).[21] CT can also pinpoint the source of bleeding, such as an intercostal artery. Management generally involves urgent chest tube drainage, with surgical intervention or interventional radiology embolization needed for severe or ongoing blood loss (> 200 mL/hour).[26] When treated promptly, hemothorax typically resolves without lasting issues, though delays can complicate recovery or require decortication.

AIRWAY COMPLICATIONS

Anastomotic dehiscence

Anastomotic dehiscence in the airway-where a surgical connection in the tracheobronchial tree partially or completely separates, such as after tracheal resection or lung transplant-is rare but serious and can result in life-threatening air leaks or mediastinitis.[1,21] Patients often present within days of surgery with subcutaneous emphysema, pneumomediastinum, breathing difficulty, and, if infection occurs, sepsis. Imaging is vital: chest X-rays may show pneumomediastinum, an enlarging pneumothorax, or a pleural effusion, while CT can directly identify airway disruption through extraluminal air, wall defects, and surrounding inflammation (Figure 13).[1,27,28] Emergent bronchoscopy confirms the diagnosis by

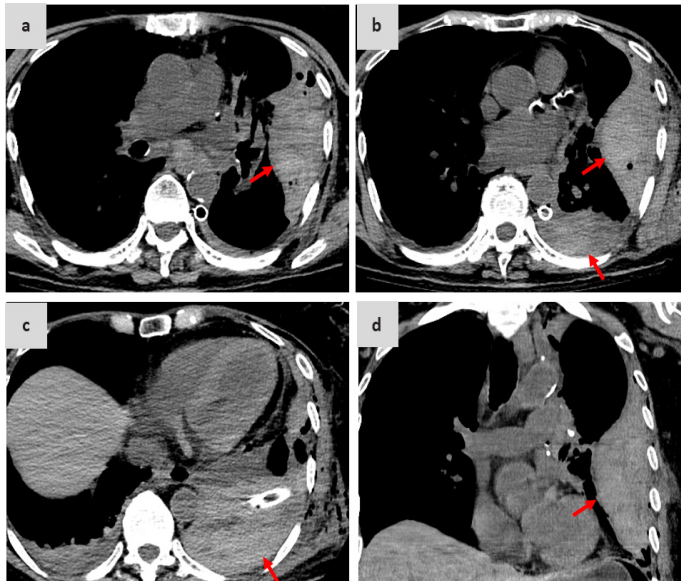

FIG. 12. Large hemothorax following left upper lobectomy. The patient developed a hematocrit drop on postoperative day 2. (a-c) Axial and (d) coronal non-contrast chest CT images demonstrate a complex, heterogeneous, hyperattenuating pleural collection with dominant posterior and lateral basilar components and small gas locules (arrows), consistent with a large hemothorax containing acute blood products. A partially imaged left basilar chest tube is in place.
CT, computed tomography.

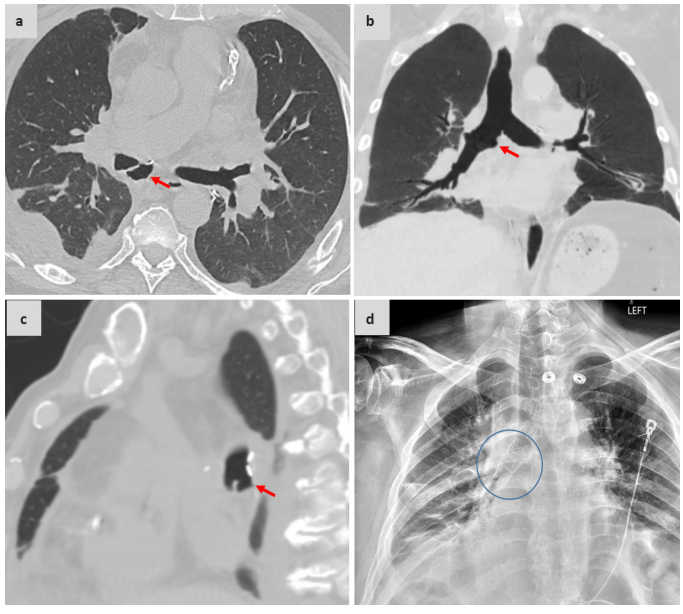

FIG. 13. Bronchial anastomotic dehiscence in a patient following bilateral lung transplantation. (a) Axial, (b) coronal, and (c) sagittal chest CT images reveal airway disruption characterized by extraluminal air collection and suspected bronchial wall defects (arrows), suggestive of anastomotic dehiscence. Bronchoscopy confirmed the dehiscence, and a bronchial stent was placed. (d) Frontal chest radiograph demonstrates the right mainstem bronchial stent (circle).
CT, computed tomography.

visualizing the suture line. Management depends on the extent of the defect: small separations may be managed conservatively with antibiotics and close monitoring, while larger dehiscences or those complicated by sepsis require urgent surgical repair to prevent severe mediastinitis and high mortality.[28]

Airway malacia (bronchomalacia or tracheomalacia)

Airway malacia refers to the weakness of the tracheal or bronchial cartilage, resulting in excessive collapse of the airway during expiration.[29] In postoperative patients, this can be caused by extended intubation or surgical handling. Patients typically show signs of central airway obstruction, including trouble clearing secretions, expiratory wheezing, a persistent cough, and shortness of breath that worsens when lying down or with exertion. Although dynamic bronchoscopy remains the gold standard for diagnosis, dynamic expiratory CT offers a non-invasive alternative by showing more than a 50% reduction in airway diameter during expiration and helps distinguish whether the malacia is diffuse or localized (Figure 14).[29] Management depends on severity and ranges from respiratory therapy, positive airway pressure, and anti-inflammatory treatments for mild cases to airway stenting or surgical correction, such as tracheobronchoplasty, for severe cases.[29,30]

Tracheoesophageal or bronchopleural fistula

A tracheoesophageal fistula (TEF) is an abnormal passage between the trachea and esophagus, most often acquired due to prolonged intubation, cancer, or surgical injury. In postoperative patients-particularly after an esophagectomy or mediastinal surgery-a TEF should be considered if there is excessive coughing while swallowing, repeated aspiration pneumonia, or abdominal bloating from air passage.[21,31] Similarly, a BPF-an abnormal connection between the bronchial tree and pleural space-commonly occurs after lung resections and usually presents with continuous air leaks, subcutaneous emphysema, or sometimes tension pneumothorax.[21,22] Diagnosis relies on imaging: a water-soluble contrast esophagogram may show contrast material entering the airways in TEF, while chest CT can confirm and define small or hidden fistulas by showing a direct air-filled tract or indirect signs such as mediastinal air and localized wall thickening (Figure 15). CT with oral contrast can further help detect the defect and plan treatment.[3,21] Bronchoscopy and upper endoscopy allow direct visualization and may enable stent placement. Because spontaneous closure is rare and ongoing aspiration or air leaks pose serious risks, both TEFs and BPFs usually need active treatment-generally surgical repair to close the defect in TEF cases, and prompt bronchoscopic or surgical intervention with pleural drainage for BPF, with endoscopic stenting used as a temporary or palliative measure.[31]

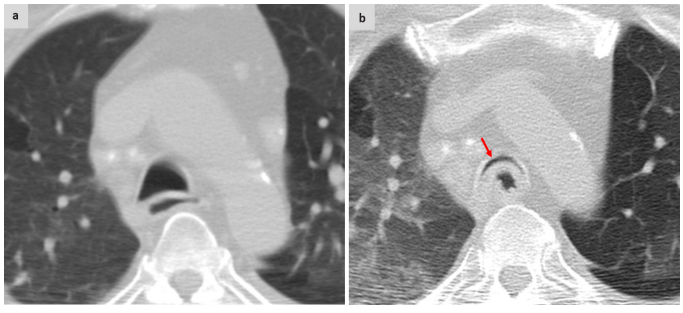

FIG. 14. Tracheomalacia in a patient following prolonged ICU stay after coronary artery bypass surgery. The patient presented with expiratory wheezing, cough, and dyspnea. (a) Inspiratory and (b) expiratory axial chest CT images demonstrate dynamic airway collapse, with more than 50% reduction in the tracheal lumen on expiration (arrow), consistent with tracheomalacia.

CT, computed tomography; ICU, intensive care unit.

LATE OR DELAYED-ONSET COMPLICATIONS

Chronic respiratory failure

Chronic respiratory failure, a long-term condition marked by inadequate gas exchange, can develop in surgical patients due to progressive lung disease or neuromuscular weakness that is worsened by surgery. It is most common in patients with chronic obstructive pulmonary disease , interstitial lung disease, or decreased lung reserve following thoracic operations like pneumonectomy.[32] Clinically, these patients have ongoing low oxygen levels and often need supplemental oxygen or ventilatory assistance for weeks to even months after surgery. Chest X-rays may show signs like hyperinflation or fibrotic reticular patterns, while CT scans provide clearer detail of emphysema and interstitial lung changes. Treatment focuses on the underlying cause, with lung transplantation being an option for carefully selected patients.[32]

Disease recurrence

Disease recurrence refers to the return of a condition that was previously treated surgically. In thoracic surgery, this usually involves lung cancer returning after resection, although other conditions, such as tuberculosis, may also recur.[33] Patients may have no symptoms or may present with cough, coughing up blood, or weight loss. Chest CT is vital for follow-up, as it can detect small nodules or

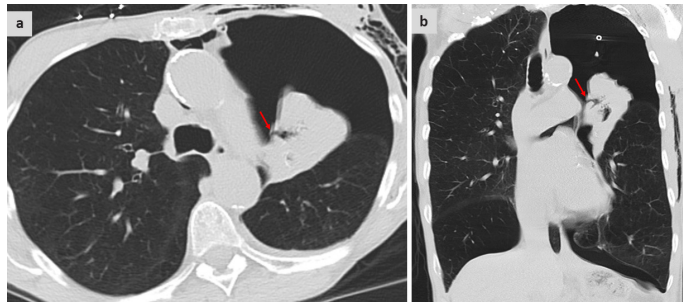

FIG. 15. Bronchopleural fistula following left upper lobe wedge resection. The patient underwent chest CT due to a persistent pneumothorax despite pleural catheter placement. (a) Axial and (b) coronal CT images demonstrate left upper lobe collapse and a small parenchymal defect consistent with a bronchopleural fistula (arrows). Note the chest catheter projecting over the left apex.

CT, computed tomography.

subtle mediastinal changes that might be missed on plain X-rays; positron emission tomography-CT can help differentiate active tumor from scar tissue, though it has limits with very small lesions. Early detection with imaging can sometimes allow for potentially curative treatment or long-term control, and the same monitoring approach is used for non-cancerous recurrences.[33]

TRANSPLANT-SPECIFIC COMPLICATIONS

Primary graft dysfunction

PGD is a type of IRI that usually develops within the first 72 hours after lung transplantation and is a significant cause of early post-transplant illness. It presents with low blood oxygen levels and widespread lung infiltrates on chest X-rays-ranging from mild interstitial markings to complete opacification-once other causes like fluid overload, acute rejection, or infection have been ruled out.[15,34] Although CT shows patterns similar to ARDS-such as bilateral ground-glass opacities, dependent consolidations, and septal thickening due to pulmonary edema-routine CT use is limited in critically ill patients (Figure 16).[12,13] Management focuses on supportive care with lung-protective ventilation, and severe cases may require ECMO, while studies continue to investigate treatments like inhaled nitric oxide. Mild to moderate PGD often resolves within a few days, but severe cases increase the risk of early death and may lead to chronic lung allograft dysfunction, highlighting the need for follow-up imaging to establish a new baseline.[34]

Chronic lung allograft dysfunction (CLAD)

Chronic lung allograft dysfunction (CLAD) is the long-term failure of a lung graft in transplant patients and includes bronchiolitis obliterans syndrome (BOS) and restrictive allograft syndrome (RAS).[16] BOS, the more frequent form, involves obliterative bronchiolitis and appears on CT as mosaic attenuation with patchy air-trapping, bronchiectasis, or thickened airway walls (Figure 17). RAS, in contrast, is marked by fibrosis in the upper lobes, pleural thickening, subpleural consolidations, scarring, volume loss, and sometimes honeycombing (Figure 18). CLAD generally occurs 1-5 years after transplantation, causing worsening shortness of breath, declining FEV_1, and sometimes a persistent cough despite maximum immunosuppressive therapy.[16,35] Detecting CLAD early is vital, as timely treatment-including stronger immunosuppression, photopheresis, or re-transplantation-is more effective. Routine

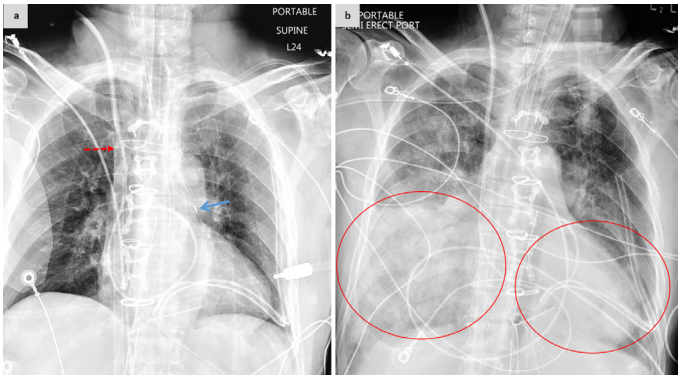

FIG. 16. Primary graft dysfunction (PGD) in a patient following cardiac transplantation. (a) Immediate postoperative chest radiograph shows clear lung fields and multiple supportive devices, including an extracorporeal membrane oxygenation cannula (dashed red arrow) and intra-aortic balloon pump marker (blue arrow). (b) Follow-up chest radiograph obtained 14 hours later demonstrates new bilateral basilar pulmonary infiltrates (circles). After excluding other causes such as fluid overload, acute rejection, and infection, the findings were attributed to PGD.

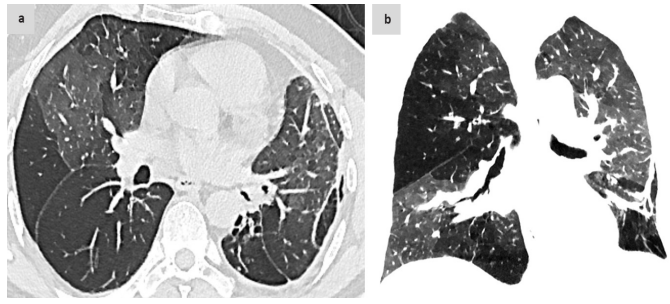

FIG. 17. Bronchiolitis obliterans syndrome (BOS) in a patient 4 years post-lung transplantation. (a) Axial and (b) coronal chest CT images demonstrate bilateral diffuse mosaic attenuation, consistent with air trapping, along with associated bronchial wall thickening-findings suggestive of BOS.

CT, computed tomography.

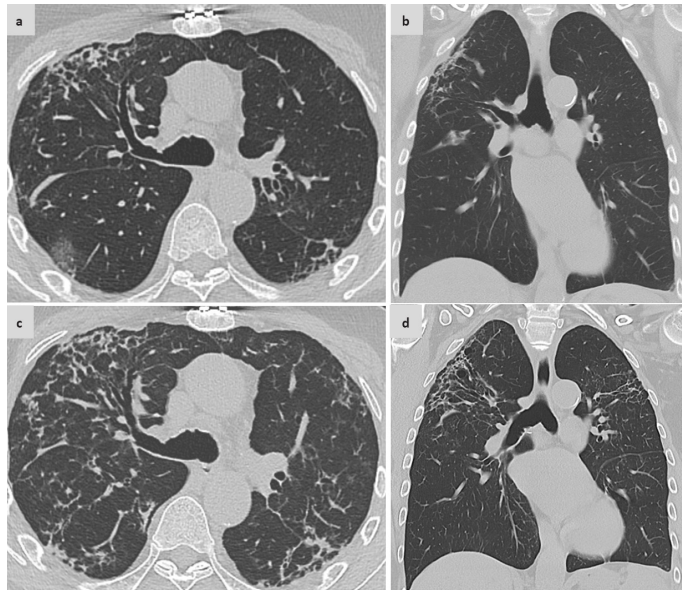

FIG. 18. Restrictive allograft syndrome (RAS) in a patient post-lung transplantation. (a) Axial and (b) coronal chest CT images obtained 1 year after lung transplantation show bilateral upper lobe-predominant subpleural consolidations, reticulation, volume loss, and traction bronchiectasis. (c) Axial and (d) coronal CT images obtained 2 years post-transplant demonstrate progression of these findings, consistent with worsening RAS.

CT, computed tomography.

imaging follow-up is critical since CLAD remains the main cause of late death in lung transplant recipients.[35]

Vascular anastomotic stenosis after lung transplantation

Vascular stenosis or occlusion of the pulmonary arteries or veins following lung transplantation-often occurring at the anastomotic junctions-can result in significant issues such as worsening shortness of breath, decreased exercise capacity, and pulmonary hypertension.[36] Imaging is essential for both diagnosis and treatment planning: although pulmonary angiography is the definitive test, CT angiography offers detailed, high-resolution views of the narrowed segments and any collateral vessel development. These imaging results help determine the appropriate management, which may include balloon angioplasty, stent placement, or surgical correction (Figure 19).[36]

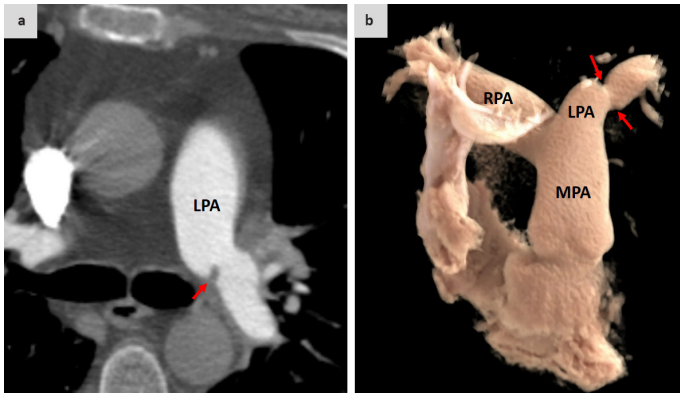

FIG. 19. Pulmonary artery anastomotic stenosis in a patient with a history of lung transplantation. (a) Axial and (b) Three-dimensional volume-rendered CT pulmonary angiography images demonstrate focal narrowing at the left pulmonary artery anastomosis site.

CT, computed tomography; LPA, left pulmonary artery; MPA, main pulmonary artery; RPA, right pulmonary artery.

VASCULAR COMPLICATIONS

Pulmonary embolism (PE)

PE is a potentially fatal complication, and surgical patients face an elevated risk due to immobility, the hypercoagulable state induced by surgery, and endothelial damage.[1,3] Clinically, PE usually presents with sudden shortness of breath, pleuritic chest pain, and an elevated heart rate. CT pulmonary angiography (CTPA) is the gold standard for diagnosis, as it not only confirms PE but also assesses right ventricular strain and can reveal alternative diagnoses.[21] When CTPA cannot be performed, ventilation-perfusion scans or magnetic resonance angiography are alternative options.[37] Treatment requires careful balancing of bleeding risk and the need for anticoagulation; most patients are managed with systemic anticoagulants. In cases of massive PE with hemodynamic instability, thrombolytic therapy or surgical embolectomy may be necessary, while catheter-directed thrombolysis or thrombectomy can also be considered. If anticoagulation is contraindicated, an inferior vena cava filter may be used instead.[1,21]

Pulmonary artery stump thrombosis

Following pneumonectomy, the development of a thrombus in the remaining pulmonary artery stump is relatively common, with studies noting an occurrence rate of about 10%-15% on routine CT follow-up.[38] These thrombi are usually asymptomatic and found incidentally but do carry a slight risk of embolization and can sometimes be mistaken for tumor recurrence. Most patients have no related symptoms, although very large thrombi could theoretically create hemodynamic stress.[21,38,39] CTPA is the preferred diagnostic method, showing a filling defect within the contrast-enhanced pulmonary artery stump (Figure 20). It is important for radiologists to distinguish these from residual tumors or other filling defects-stump thrombi typically do not show PET uptake and tend to remain stable or shrink with treatment on follow-up imaging.[39] Management remains somewhat debated; some clinicians choose anticoagulation to lower embolic risk, while others opt for observation, especially if

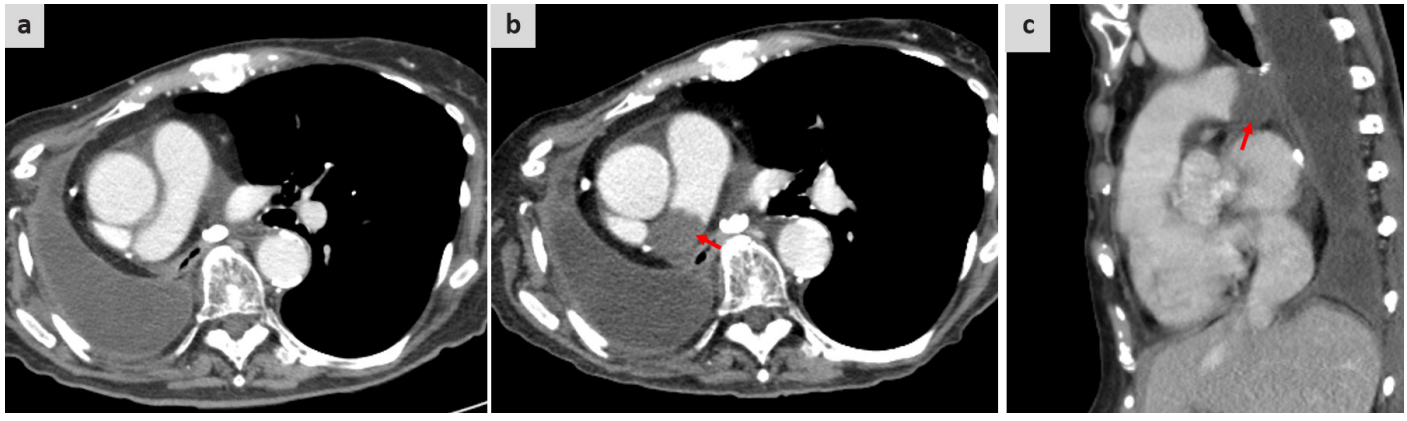

FIG. 20. Pulmonary artery stump thrombosis in a patient status post right pneumonectomy for non-small cell lung cancer. (a) Initial axial chest CT image shows a clear right pulmonary artery stump and post-pneumonectomy changes. (b) Axial and (c) coronal follow-up CT images obtained 6 months later demonstrate interval development of an *in-situ* thrombus within the right main pulmonary artery stump (arrows).

CT, computed tomography.

the clot appears chronic and the patient is no longer in the high-risk window for venous thromboembolism. Though uncommon, large stump thrombi can embolize to the opposite lung, which supports discussing the possible use of preventive anticoagulation.[38,39]

Arteriovenous fistula (AVF)

Pulmonary arteriovenous fistulas (AVFs) are abnormal direct connections that bypass the capillary bed and may rarely develop after surgery due to operative injury, trauma from a chest tube, or lung biopsy.[40] Larger AVFs can cause exercise limitation, cyanosis, digital clubbing, and even heart failure due to significant shunting, while smaller ones are often symptom-free. CT angiography is the diagnostic method of choice, revealing a nodular vascular lesion where a widened feeding artery connects to a draining vein; catheter angiography remains the gold standard when treatment is planned. Management usually involves percutaneous transcatheter embolization, with surgery reserved for very large or complex AVFs.[36,40]

Pseudoaneurysm

A pseudoaneurysm, or false aneurysm, forms when there is a break in the arterial wall that allows blood to leak out, creating a contained hematoma that remains connected to the artery's lumen. Unlike a true aneurysm, where all layers of the arterial wall expand, a pseudoaneurysm is confined by surrounding connective tissue or the adventitia.[41] In the pulmonary vessels, these can arise after surgery due to operative trauma or infection. Some patients have no symptoms, but hemoptysis and chest pain may occur and signal a potential rupture. CTA is the imaging test of choice, showing a clearly defined, contrast-filled bulge next to an artery and helping assess its size, shape, and nearby structures (Figure 21).[1,3,41] Treatment usually involves an endovascular approach such as coil embolization, while surgical repair is done when endovascular methods are not an option.[41]

LYMPHATIC COMPLICATIONS

Chylothorax

Chylothorax refers to the collection of triglyceride-rich lymphatic fluid in the pleural cavity, most often caused by damage to the thoracic duct during surgery or by malignancy.[42,43] Patients usually have shortness of breath, cough, and reduced breath sounds. Chest X-rays generally reveal a unilateral pleural effusion, while CT scans provide detailed images of the fluid (which shows low attenuation), and ultrasound is helpful for detecting the fluid and guiding thoracentesis. Newer techniques now favor contrast-enhanced CT lymphangiography over older methods because of its better accessibility, faster imaging, and lower radiation exposure (Figure 22).[44] Diagnosis is confirmed when the pleural fluid appears milky and has high triglyceride levels (typically above 110 mg/dL). Treatment includes dietary changes, such as a low-fat diet with medium-chain triglycerides, with embolization or surgical ligation of the thoracic duct reserved for cases that do not respond to conservative management. With timely treatment, the outlook is generally good.[43]

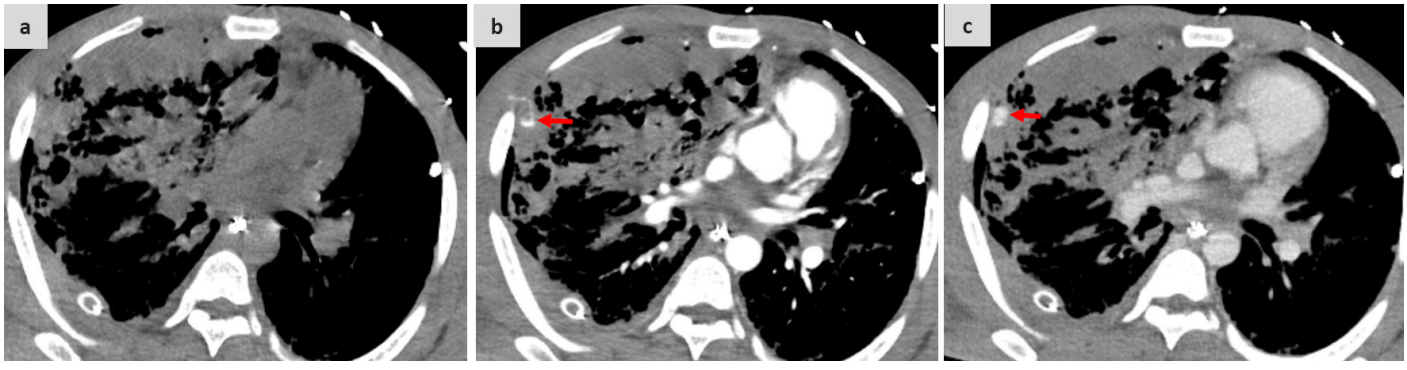

FIG. 21. Intercostal artery pseudoaneurysm in a patient following lung abscess surgery. (a) Axial non-contrast, (b) arterial phase, and (c) venous phase CT images demonstrate extensive right lung consolidation with a small dependent pleural effusion and a chest tube in place. (b) Arterial phase imaging reveals a small focus of contrast extravasation (arrow) within the consolidated lung, originating from a chest wall artery along the intercostal musculature. (c) This focus enlarges on venous phase imaging, consistent with an intercostal artery pseudoaneurysm, likely related to recent thoracotomy in the setting of pneumonia and lung abscess.
CT, computed tomography.

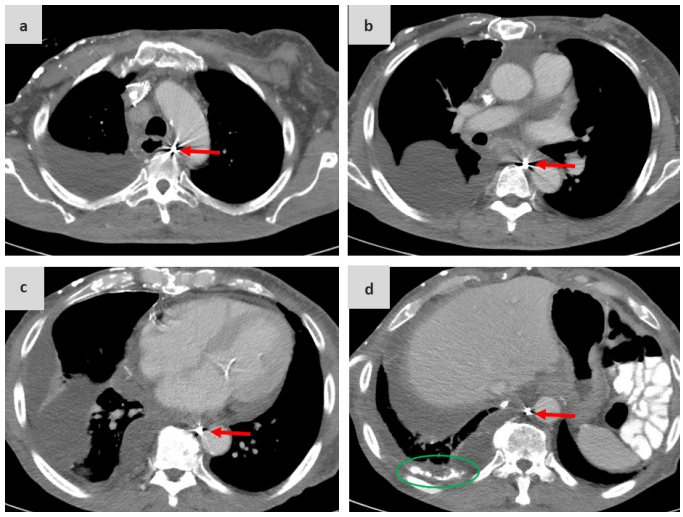

FIG. 22. Chylothorax in a patient following right middle lobectomy for lung adenocarcinoma. (a-d) Axial contrast-enhanced chest CT images demonstrate a moderate to large, partially loculated hypoattenuating right pleural effusion with localized fissural components. Note the pronounced hyperattenuation with streak artifact within the thoracic duct from prior lymphangiography (arrows). Focal hyperattenuation within the dependent basilar pleural fluid (circle) reflects leakage of lymphangiographic contrast, confirming chylous leakage.
CT, computed tomography.

CONCLUSION

In summary, PPCs include various conditions that can greatly impact patient outcomes (Table 1). Timely detection, precise diagnosis, and early treatment are crucial to minimizing morbidity and mortality. Delivering the best care requires a coordinated effort among surgeons, pulmonologists, radiologists, and intensive care specialists. Future studies should aim to advance imaging methods-including artificial intelligence-based image analysis-and conduct prospective trials to assess minimally invasive procedures and targeted treatments designed to lower the occurrence and severity of PPC.

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TABLE 1. The Common Postoperative Pulmonary Complications and Imaging Findings.

Condition	Pathophysiology	Clinical findings	Imaging findings
Pneumonia	Infectious alveolar inflammation from bacteria/viruses	Fever, productive cough, dyspnea	CR: Infiltrates; CT: Consolidation (higher sensitivity)
Empyema	Purulent fluid accumulation in the pleural space from infection/surgery	Fever, chest pain, respiratory distress	CT: Lenticular fluid collection, split pleura sign, air-fluid level
Sepsis	Systemic inflammation from an infection source	Fever, hypotension, tachycardia	CT: Detection of infectious foci (e.g., pneumonia, abscess)
ARDS	Diffuse alveolar damage from inflammation	Rapid-onset dyspnea, hypoxemia	CR: Bilateral infiltrates; CT: Ground-glass opacities (GGO), dependent consolidations
Ischemia-reperfusion injury	Oxidative damage following reperfusion	Acute hypoxemia post-surgery	CT: Ground-glass opacities, septal thickening
Chronic bronchial inflammation	Persistent bronchitis from irritation	Chronic cough, wheezing	CT: Bronchial wall thickening, mosaic attenuation
Atelectasis	Alveolar collapse from obstruction or shallow breathing	Dyspnea, decreased breath sounds	CR: Volume loss; CT: Lobar or segmental collapse without air bronchograms
Pulmonary edema	Fluid accumulation (cardiogenic or ARDS-related)	Dyspnea, orthopnea	CR: Bat-wing appearance; CT: GGO, septal thickening, pleural effusions
Persistent air leak	Alveolar/small airway injury with ongoing air escape	Pneumothorax, subcutaneous emphysema	CR: Visible pneumothorax; CT: Fistula or active air leak detection
Lung torsion	Twisting of a lung lobe causing vascular compromise	Sudden respiratory distress, hypoxemia	CT: Twisted bronchovascular pedicle, poor lobe enhancement, cutoff vessels
Postpneumonectomy syndrome	Extreme mediastinal shift compressing the remaining airways	Progressive dyspnea, positional wheezing, cough	CR: Marked mediastinal deviation; CT: Rotated mediastinum with bronchial compression
Coronary artery compression	Mediastinal shift compressing the coronary arteries	Angina, myocardial ischemia	CT: Direct visualization of coronary artery compression

TABLE 1. Continued.

Condition	Pathophysiology	Clinical findings	Imaging findings
Pleural effusion	Fluid accumulation in the pleural space post-surgery	Dyspnea, pleuritic chest pain	CR: Blunting of costophrenic angle; US: Fluid detection; CT: Differentiation from lung consolidation
Pneumothorax	Air accumulation in the pleural space following procedures	Chest pain, shortness of breath (SOB)	CR: Visible pleural line; CT: Detection of subtle pneumothorax or bullae
Hemothorax	Blood accumulation in the pleural space from trauma or surgery	Chest pain, signs of hypovolemia	CT: Differentiation of blood from simple fluid; identification of active bleeding
Anastomotic dehiscence	Disruption of a surgical airway anastomosis	Subcutaneous emphysema, sepsis	CT: Air outside the airway lumen, wall defect, extraluminal air collections
Bronchomalacia	Weakening of airway cartilage causing dynamic airway collapse	Expiratory wheezing, cough	Expiratory CT: > 50% reduction in airway caliber during expiration
Tracheoesophageal/ bronchopleural fistula	Abnormal communication between the trachea/esophagus or bronchial tree and the pleural space	Cough during swallowing, recurrent aspiration, or persistent air leak	CT: Direct visualization of a fistula tract or indirect signs (mediastinal air, focal thickening); contrast esophagogram (for TEF); bronchoscopy/endoscopy for direct assessment
Chronic respiratory failure	Prolonged gas exchange failure due to progressive lung disease or neuromuscular weakness	Persistent hypoxemia requiring supplemental oxygen or ventilatory support	CT: Emphysema, fibrosis, and interstitial changes
Disease recurrence	Return of malignancy, infection, or underlying disorder	Cough, hemoptysis, dyspnea	CT: New nodules or masses; PET-CT: Increased metabolic activity indicating active tumor
Primary graft dysfunction	Transplant ischemia–reperfusion injury leading to diffuse lung injury	Hypoxemia within 72 hours post-transplant	CT: Bilateral GGO and dependent consolidations resembling ARDS
CLAD	Chronic lung transplant rejection (BOS or RAS)	Progressive dyspnea, decline in lung function	CT: Air trapping (BOS) or fibrosis with honeycombing (RAS)
Vascular anastomotic stenosis	Narrowing of pulmonary vessels at anastomotic sites post-transplant	Dyspnea, pulmonary hypertension	CT angiography: Focal stenosis with collateral vessel formation
Pulmonary embolism	Vascular occlusion due to thrombus formation	Sudden dyspnea, chest pain	CTPA: Filling defects in pulmonary arteries, signs of right ventricular (RV) strain
PA stump thrombosis	Thrombus formation in the residual pulmonary artery stump post-pneumonectomy	Often asymptomatic; may be incidental	CTPA: Filling defect in the PA remnant, with chronic thrombi showing concave margins
AV fistula	Abnormal direct connection between a pulmonary artery and vein	Cyanosis, clubbing, dyspnea	CTA: Vascular nodule where an enlarged feeding artery meets a draining vein
Pseudoaneurysm	Contained arterial wall breach forming a hematoma communicating with the arterial lumen	Hemoptysis, chest pain	CTA: Well-defined, contrast-filled outpouching adjacent to an artery
SVC thrombosis	Clot formation in the superior vena cava post-central line placement or thoracic surgery	Facial and upper extremity swelling	CT venography: Intraluminal thrombus with collateral circulation
Chylothorax	Accumulation of chyle due to thoracic duct injury (surgical or malignant)	Dyspnea, cough	CR: Unilateral effusion; CT: Detailed fluid collection; contrast-enhanced CT lymphangiography offers rapid acquisition; confirmed by milky fluid with elevated TGs (> 110 mg/dL)

CR, chest radiograph; CT, computed tomography; CTPA, computed tomography pulmonary angiography; CTA, computed tomography angiography; US, ultrasound; SOB, shortness of breath; GGO, ground-glass opacities; TEF, tracheoesophageal fistula; PET-CT, positron emission tomography-computed tomography; ARDS, acute respiratory distress syndrome; CLAD, chronic lung allograft dysfunction; BOS, bronchiolitis obliterans syndrome; RAS, restrictive allograft syndrome; PA, pulmonary artery; AV, arteriovenous; SVC, superior vena cava; TGs, triglycerides.

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