The chest radiograph is a crucial tool in the care of the critically ill. It serves to diagnose and to monitor a variety of cardiopulmonary disorders. In addition, it is used to evaluate a broad range of monitoring and support equipment, ensure proper positioning, and survey for complications. Daily rounds and prompt communication between the radiologist and the intensivist can help improve diagnostic accuracy and manage potential complications.

**Indications for portable chest radiography**

Indications for portable chest radiography include cardiopulmonary symptoms following cardiacl or thoracic surgery, trauma, patients who have monitoring and life-support devices, and critically ill patients, according to the American College of Radiology (ACR) practice guidelines (revised 2006) [1]. There are no absolute guidelines dictating the frequency of chest radiography for ICU patients. Several studies assessing the benefit of daily chest radiography in the ICU have been performed, with varied findings [2–5]. The ACR recommends daily chest radiography for patients who have acute cardiopulmonary problems [6]. Chest radiographs should also be obtained immediately after the placement of endotracheal tubes, nasogastric tubes, vascular catheters, and chest tubes [6]. Follow-up is warranted when tube or catheter position is suspected to have changed or when otherwise clinically indicated.

**Technical factors**

Inherent challenges exist in ICU chest radiography, all of which limit diagnostic accuracy. Many patients are debilitated and not readily able to cooperate with the examination, precluding optimal upright (posteroanterior) positioning. Radiographs are usually obtained in a semiupright or supine anteroposterior (AP) position. A lateral radiograph is often impractical. External monitoring devices, overlying tubes, and electrocardiographic leads can obscure underlying disease, mimic radiographic pathology, and create ambiguity as to the positioning of other support equipment.

**Role of CT**

CT is another valuable imaging modality in assessing the ICU patient. CT is superior to chest radiography in the detection and characterization of pulmonary, pleural, and mediastinal abnormalities [7]. CT more accurately depicts the pattern and distribution of pulmonary parenchymal abnormalities. Contrast-enhanced CT is useful particularly in assessing pleural fluid collections and potentially for guiding interventional procedures. Pulmonary CT angiography is the primary method of evaluating for pulmonary embolus in the ICU patient.
CT imaging of the ICU patient is not without its logistical limitations. Safe transport of the critically ill, along with support devices, is often difficult. In addition, renal failure may preclude the administration of intravenous contrast, diminishing diagnostic capabilities.

**Monitoring and support devices**

Evaluation of support equipment and monitoring devices is of utmost importance in the imaging of patients in the ICU. Early recognition of malpositioning reduces the likelihood of potentially serious complications. Radiologists often review the position of all support equipment in their initial appraisal of the radiograph. For this reason, and to underline its importance, the evaluation of monitoring and support devices is discussed first.

**Endotracheal and tracheostomy tubes**

Endotracheal tubes are seen in patients requiring short-term respiratory support with mechanical ventilation. With the patient’s head in a neutral position, the endotracheal tube tip should be located 4 to 6 cm above the carina. Neck flexion results in caudal movement of the tube, up to 2 cm. Neck extension can cause 2 cm superior migration.

A malpositioned endotracheal tube is not an uncommon finding. Intubation of the main bronchi can occur when endotracheal tube position is too low, resulting in subsegmental atelectasis, segmental collapse, or complete collapse of the contralateral lung. The ipsilateral lung may be overventilated, increasing the risk of pneumothorax. Main bronchus intubation is most frequently right-sided, owing to a more direct angle of the trachea and right main bronchus (Fig. 1). When the endotracheal tube is too high, there may be inadvertent extubation or damage to the larynx. Esophageal intubation is a severe complication compromising ventilation and introducing excessive amounts of air into the gastrointestinal tract but is typically clinically apparent. Aspiration occurs in up to 8% of intubations [8].

The endotracheal balloon should not be inflated beyond the normal diameter of the trachea. Overinflation to 1.5 times the normal tracheal diameter frequently causes tracheal damage [9]. Tracheal rupture can occur acutely. Tracheal stenosis is a potential chronic complication (Fig. 2).

Tracheostomy tubes are placed when long-term intubation is necessary. The tracheostomy tube tip should be approximately at the T3 level. Position is maintained with neck flexion and extension. Tracheostomy tube diameter should be approximately two thirds that of the trachea’s, and the cuff should not distend the tracheal wall. Mediastinal air can be seen after uncomplicated tube placement.

![Fig. 1. Right main bronchus intubation. AP chest radiograph demonstrating endotracheal tube tip in the proximal right main bronchus (arrow) with resultant left upper and lower lobe atelectasis and mild leftward shift of the mediastinum.](image1)

![Fig. 2. Tracheal stenosis. Magnified AP view of the trachea of a 42-year-old man who had a history of prolonged intubation showing focal tracheal stenosis (arrow).](image2)
**Enteric tubes**

Oroenteric and nasoenteric tubes are used for feeding, medication administration, and suction. For feeding, ideal tip position is in the gastric antrum or the duodenum to reduce aspiration risk. When the enteric tube is used exclusively for suction or medication administration, placement within the stomach is adequate. Sideports, when present, should extend beyond the gastroesophageal junction to decrease aspiration risk.

Radiography is important in detecting aberrant tube location and in preventing potentially lethal complications. Tubes can coil within the pharynx or esophagus, creating a high risk of aspiration if nutrition is administered. Pharyngeal and esophageal perforations are rare complications. Occasionally, enteric tubes terminate in the trachea or bronchi, and ectopic feeding can result in direct bronchopulmonary injury and pneumonia. Pneumothorax, pulmonary laceration, and pulmonary contusion may be seen if the lung parenchyma is punctured. If an enteric tube has been placed in the airway and extends to the lung periphery or into the pleural space, then it is essential to obtain a follow-up radiograph because pneumothorax may only be apparent post removal (Fig. 3).

**Venous catheters**

Venous catheters are common in the ICU. Medications and intravenous fluids can be administered, blood withdrawn, and central venous pressure measurements obtained. Catheters may be inserted peripherally in upper-extremity veins (peripherally inserted central catheter [PICC]) or more proximally within the subclavian or internal jugular veins, depending on their intended use. The femoral vein is less commonly accessed. Tunneled catheters are often used for renal dialysis, and ports are placed in the chest wall in patients requiring repeated doses of intravenous pharmacotherapy for extended periods.

The venous catheter tip should be located within the superior vena cava (SVC), beyond venous valves, to reduce the risk of thrombosis. Positioning of the catheter tip in the lower SVC likely results in further reduction of thrombosis around the catheter tip [10]. When catheter position is too caudal, it may enter the right atrium, increasing the risk of dysrhythmia and, rarely, cardiac perforation. Thus, catheters should ideally terminate within the lower SVC or at the cavoatrial junction.

Aberrant positioning of venous catheters is quite common. Usually, the aberrantly located catheter is intravenous or within the right atrium. Peripherally inserted catheters can coil in the veins of the upper extremity, course cephalad within the internal jugular vein (Fig. 4), or traverse midline by way of the contralateral brachiocephalic vein. Catheter location within a persistent left-sided SVC, an anomalous vein occurring in 0.3% of the population [11], is occasionally seen (Fig. 5). When a catheter is located in a left-sided SVC, it may mimic an intra-arterial location on an AP chest radiograph. The catheter may also terminate

Fig. 3. Right lower lobe feeding tube placement. (A) AP chest radiograph of a 75-year-old man demonstrates aberrant enteric tube terminating in the right lower lobe. (B) Follow-up AP chest radiograph after enteric tube removal shows a visceral pleural line (arrows) indicative of right-sided pneumothorax. The left upper lobe mass proved to be poorly differentiated large cell carcinoma.
within smaller venous side branches, including the azygous vein.

Occasionally, arteries are inadvertently accessed, most often the subclavian artery or common carotid artery (Fig. 6). Arterial catheterization is usually clinically apparent with pulsatile flow of bright red oxygenated blood from the catheter. On the AP chest radiograph, subclavian artery placement should be suspected when the catheter travels above the clavicle. If uncertainty remains after radiographic analysis, the determination of wave form (arterial versus venous) can confirm location.

When the catheter tip is directed at and abuts the venous wall, the catheter should be repositioned or withdrawn to reduce the risk of vessel perforation. Vascular perforation causes hematoma in the surrounding soft tissues. Fluid and medications can accumulate in adjacent soft tissues or pleural space if extravascular catheter position (Fig. 7) is unnoticed. The chest radiograph should also be used to evaluate for hemothorax and pneumothorax following line placement. Pneumothorax occurs uncommonly with PICC placement and is most frequently seen when the subclavian vein is accessed.

Pulmonary artery catheters

Pulmonary artery catheters, or Swan-Ganz catheters, are used to measure pulmonary artery pressure, pulmonary capillary wedge pressure, and cardiac output. The catheter tip should be within the right main pulmonary artery, left main pulmonary artery, or the proximal interlobar pulmonary artery. When the catheter extends beyond the pulmonary hilum on the chest radiograph, the catheter should be retracted [12]. The pulmonary arteries narrow as they extend from
Intra-aortic balloon pump

The intra-aortic balloon pump (IABP) is a 26 to 28 cm–long balloon device [10] that inflates during systole to assist coronary perfusion, and deflates during diastole to decrease cardiac afterload. It is radiolucent, except for its radio-opaque tip that assists in radiographic localization. The IABP tip should be within the proximal descending thoracic aorta just distal to the origins of the major branch arteries of the aortic arch (Fig. 10). Cerebral or left upper-extremity ischemia may result when the catheter is located too proximally. Too distal a location risks occlusion of the abdominal aortic branch arteries and renal and mesenteric ischemia. Aortic rupture, limb ischemia, and balloon rupture with air embolization are other rare potential complications.

Chest tubes

Chest tube malposition occurs in approximately 10% of placements [6]. Chest tube sideholes—radiographically evident as interruptions
of the tube’s radio-opaque line—should be located within the pleural space. Improper chest tube location may manifest as a poorly functioning or nonfunctioning tube. When a chest tube is inserted into the pulmonary parenchyma (Fig. 11), pulmonary contusion may be seen, manifested as a new opacity adjacent to the chest tube. Abnormal location in the pulmonary fissures may or may not affect tube function. Viscous debris within the chest is easily identified on chest CT but may be occult on conventional radiography. An inappropriately positioned chest tube can injure mediastinal and upper abdominal organs, major blood vessels, and the diaphragm.

Pneumothorax, pneumomediastinum, and pleural fluid

Pleural space abnormalities include pneumothorax and pleural fluid and are extremely common in the ICU setting. Pneumomediastinum is less commonly encountered but important to recognize because it may be indicative of underlying tracheobronchial injury or alveolar rupture in a mechanically ventilated patient.

Pneumothorax

Underlying pulmonary disease, trauma, and iatrogenesis may result in pneumothorax (Fig. 12). The classic sign of a thin radiodense curvilinear pleural line, bordered by lung on one side and pleural air on the other, is often absent in the supine ICU patient. Detection can require a high degree of suspicion. In the ventilated patient, a small pneumothorax can rapidly progress to tension, and recognition is critical.

In the supine patient, pleural air initially accumulates in the anteromedial recess, the least-dependent location in the hemithorax [13]. Abnormal lucency at the lung base or projecting over the upper abdomen is suggestive of pneumothorax.
A lucent deep sulcus may be visualized in the medial or lateral hemithorax (Fig. 13). In addition, the mediastinum may be unusually well outlined [13]. The lateral decubitus position is the most sensitive for detecting pleural air but is often impractical. When pneumothorax is suspected, an upright radiograph should be obtained for confirmation.

Tension pneumothorax occurs when intrathoracic pressure is greater than atmospheric pressure. Radiographically, tension pneumothorax is most reliably diagnosed by inversion or flattening of the hemidiaphragm. Mediastinal shift may also be seen but is less reliable and frequently less pronounced in patients who have acute respiratory distress syndrome (ARDS), because of reduced lung compliance.

Because skin folds can mimic pneumothoraces, important distinguishing features should be recognized. A skin fold is seen as a soft tissue–air interface, with radio-opacity on one side and normal lung on the other. In pneumothorax, a pleural line is often bordered by air on both sides: normal lung and pleural air (Fig. 14). The diagnosis may be more complex when the lung is abnormally opaque, creating the illusion of a soft tissue–air interface. If pulmonary vessels extend peripheral to the interface, then the opacity is a skin fold. If no pulmonary vessels are seen peripherally, then a pneumothorax is present.

**Pneumomediastinum**

Pneumomediastinum is extraluminal air within the mediastinum. It can be seen in tracheobronchial injury, tracheostomy tube placement, mechanically ventilated patients, asthmatics, and esophageal rupture (although this is a rare cause). Most commonly, pneumomediastinum occurs by way of the Macklin effect. The Macklin effect describes the process by which air from ruptured alveoli dissects along the bronchovascular interstitium to the mediastinum [14]. Pulmonary interstitial emphysema in the mechanically ventilated patient is a sign of alveolar rupture. Air may dissect cephalad to the subcutaneous tissues of the neck (Fig. 15), and caudad to the retroperitoneum.

The Mach band effect can mimic pneumomediastinum on the chest radiograph [15]. The Mach band effect is a perceptual error, creating the appearance of abnormal lucency adjacent to a radiodense convexity such as the heart. When paracardiac lucency is seen in the absence of an adjacent pleural line, the Mach band effect should be suspected.

**Pleural fluid**

Pleural fluid is common in ICU patients and is most frequently transudative. The supine radiograph is relatively insensitive in the detection of pleural fluid and often underestimates the amount of pleural fluid. On the upright lateral radiograph, blunting of the costophrenic angle usually occurs when 200 mL of fluid are present but may be

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**Fig. 12. Pneumothorax.** AP chest radiograph showing large right pneumothorax with retraction of the radioopaque, collapsed right lung toward the right hilum.

**Fig. 13. Pneumothorax, “deep sulcus sign.”** AP chest radiograph in a supine trauma patient demonstrating lucent deep lateral costophrenic sulcus (arrows) and luency of the right hemithoracic base, characteristic of pneumothorax. Left pulmonary contusion, subcutaneous emphysema, multiple displaced rib fractures, and left basilar pneumothorax are also seen.
absent with as much as 500 mL [16]. Layering pleural fluid is more difficult to detect on the supine radiograph. The costophrenic angle is often not blunted, and the supine radiograph may only demonstrate hazy “veil-like” opacification due to layering pleural fluid (Fig. 16). The apex is the most dependent location in the supine patient, and pleural effusion may manifest as an apical cap [16].

Consolidation, atelectasis, and pleural fluid cause opacities on the chest radiograph and frequently coexist, particularly at the thoracic base. CT is useful in differentiating pleural fluid from pulmonary parenchymal disease (Fig. 17). CT also better characterizes loculated pleural fluid collections. Empyema is suggested when pleural fluid is bordered by enhancing, thick pleura. Hemothorax is suggested by relatively high attenuation pleural fluid [17], commonly 35 to 70 Hounsfield units [18].

**Pulmonary parenchymal abnormalities**

Atelectasis, aspiration, pneumonia, hydrostatic pulmonary edema, and noncardiogenic pulmonary edema present as opacities on chest radiography and CT. Although it is often difficult and sometimes impossible to distinguish between these entities, certain radiographic features can aid in their diagnoses.

**Atelectasis**

Atelectasis, a decrease in lung volume, is the most common cause of pulmonary opacities in the ICU population. It is frequently found after general anesthesia and thoracic or upper abdominal surgery, occurring in up to 64% of patients in one surgical investigation [19]. The most common location is the left lower lobe (66%), followed by the right lower lobe (22%), and right upper lobe (11%) [20]. Atelectasis is usually subsegmental and can mimic pneumonia, particularly when signs of volume loss such as crowding of air bronchograms, fissural deviation, mediastinal shift, and diaphragmatic elevation are absent. Flat, platelike opacities are characteristic of discoid atelectasis (Fig. 18). Complete lung collapse, lobar collapse (Fig. 19), or segmental collapse can also be seen. Atelectasis is categorized (according to mechanism) as obstructive, compressive,
cicatricial, or adhesive. Adhesive atelectasis, common in premature neonates secondary to insufficient production of surfactant, is not discussed further.

Obstructive atelectasis is the most common type of atelectasis. Impaired mucociliary function, increased secretions, and altered consciousness are predisposing factors. When only the distal, small...
airways are obstructed, crowded air bronchograms are seen. Air bronchograms are absent when the obstruction is more proximal, in larger airways. Mucous plugging is a common cause of acute segmental, lobar, and complete lung collapse (Fig. 20). The absence of air bronchograms in patients who have acute lobar collapse favors mucoid impaction as the etiology and predicts a higher rate of therapeutic success with bronchoscopy (79%–89% in favorable patients) [21].

Compressive atelectasis is volume loss secondary to mass effect exerted on the lung. In the ICU population, pleural fluid is usually the cause. Other potential causes are thoracic tumor, pulmonary abscess, and severe cardiomegaly. Cicatricial atelectasis is volume loss secondary to pulmonary fibrosis and can be seen in patients who have underlying pulmonary disease or as a complication of ARDS.

On CT, atelectasis can often be identified by signs of volume loss. On contrast-enhanced CT, atelectasis results in relatively high attenuation of the lung parenchyma, a useful feature distinguishing it from relatively lower attenuating consolidative processes such as pneumonia.

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Fig. 17. Pleural effusion versus atelectasis. (A) Supine AP chest radiograph shows hazy, “veil-like” opacity of the right lower hemithorax. (B) Axial CT image through the lower chest in the same patient demonstrates only a small right pleural effusion (small arrow) but significant right lower lobe atelectasis (large arrow). There is also left lower lobe atelectasis.

Fig. 18. Discoid atelectasis. AP chest radiograph of a 54-year-old man demonstrates low lung volumes and linear left basilar opacity characteristic of discoid atelectasis.

Fig. 19. Right upper lobe collapse. AP chest radiograph of a 72-year-old man demonstrates right upper lobe collapse with cephalad deviation of the minor fissure. Subsequent therapeutic bronchoscopy found viscous secretions within the right upper lobe bronchi.
Fig. 20. Atelectasis, mucous plug. (A) AP chest radiograph demonstrates abrupt truncation of the left main bronchus (arrow) and significant left lung atelectasis suggestive of a mucous plug. (B) Coronal CT image through the chest in the same patient confirms presence of a mucous plug in the left main bronchus (arrows) with postobstructive atelectasis.

Fig. 21. Aspiration pneumonitis and aspiration pneumonia. (A) Initial AP chest radiograph in a 38-year-old man shows bilateral perihilar and lower lung nodular and consolidative opacities; (B) follow-up radiograph 1 week later shows marked improvement, consistent with resolving aspiration pneumonitis. (C) AP radiograph in a different patient demonstrates right greater than left perihilar and lower lung nodular and ill-defined consolidative opacities; (D) AP radiograph obtained 1 week later shows progression to dense right lower lobe consolidation, consistent with aspiration pneumonia.
Aspiration

Intubation, diminished cough reflex, sedation, and enteric tube feeds increase aspiration risk. Aspiration can occur in mechanically ventilated patients despite adequate inflation of the endotracheal tube cuff. Clinically, aspiration events may go unnoticed or may be severe, causing respiratory distress. Aspiration can result in airway obstruction, chemical pneumonitis, or infectious pneumonia, depending on the volume and type of aspirate. Small amounts of aspirated saliva may result in no radiographic abnormality, whereas aspiration of large amounts of food substance increases the likelihood of aspiration pneumonia.

Patchy, ill-defined ground-glass, consolidative, and nodular opacities are the most frequently encountered radiographic manifestations of aspiration. Opacities typically appear rapidly and are most commonly located in the dependent regions of the lungs: the posterior segment of the upper lobes and the superior and posterior basal segments of the lower lobes [22]. Opacities may increase in conspicuity over the first 1 to 2 days in aspiration pneumonitis but should resolve relatively rapidly thereafter. When opacities persist or increase over several days, aspiration pneumonia is likely present (Fig. 21).

Patchy, dependent ground-glass and consolidative opacities are also seen on CT. “Tree-in-bud” opacities [23] result from inflammation of the distal airways. Although tree-in-bud opacities are nonspecific, when present in a dependent distribution, they are highly suggestive of aspiration (Fig. 22).

Pneumonia

Pneumonia is another cause of pulmonary opacities in ICU patients. Aspiration and mechanical ventilation [24] are two important risk factors for pneumonia in the ICU population. Ventilator-associated pneumonia occurs in 9% to 24% of patients ventilated for more than 48 hours [25]. Most pneumonias are caused by mixed anaerobic or, more frequently in the ventilated patient, aerobic gram-negative bacteria such as Pseudomonas aeruginosa [26].

Pneumonia may present as a focal consolidation on the chest radiograph; however, it is often multifocal (Fig. 23). Pneumonia can be difficult to differentiate from other causes of pulmonary opacities such as atelectasis, aspiration, and pulmonary edema. Typically, pneumonia changes more slowly than these other entities. In addition, air bronchograms may be seen and can be differentiated from those seen in atelectasis by noting the absence of volume loss and crowding of bronchi.

When ARDS is present, the diagnostic accuracy of CT and chest radiography is diminished [27,28]. The presence of underlying consolidation in ARDS limits the ability to exclude the presence of pneumonia. The incidence of pneumonia in patients who have diffuse lung injury at autopsy has been reported to be 58% [29].

Noncardiogenic pulmonary edema

Pulmonary edema can be classified as hydrostatic pulmonary edema or noncardiogenic pulmonary edema, also referred to as increased permeability edema. These entities can be difficult to distinguish radiographically and may coexist, further complicating their diagnoses.

Noncardiogenic pulmonary edema is caused by primary pulmonary pathology such as pneumonia, aspiration, and pulmonary contusion [30]. Extrathoracic causes of increased permeability include drug toxicity, systemic inflammatory response syndrome, sepsis, shock, and extrathoracic trauma. Neurogenic, postpneumonectomy, and re-expansion pulmonary edema demonstrate radiographic features of hydrostasis and capillary leak [31]. Diffuse alveolar damage (DAD) results from injury to the alveolar capillaries and epithelium. The degree of DAD varies from severe (in cases of ARDS) to relatively nonexistent (as in many cases of heroin-induced pulmonary edema)
When DAD is absent or minimal, radiographic abnormalities are likely to be relatively transient. Respiratory symptoms may precede radiographic abnormalities in noncardiogenic pulmonary edema, and the initial radiograph is often normal. Within the first 24 hours, patchy, bilateral ground-glass and consolidative opacities typically appear. These opacities coalesce, forming diffuse pulmonary opacification (Fig. 24) that lasts for days to months depending on etiology, degree of DAD, complications such as aspiration and pneumonia, and treatment. Radiographic features typically associated with hydrostatic pulmonary edema, including septal lines, pleural fluid, and widening of the vascular pedicle, may also be seen with noncardiogenic pulmonary edema. Aberle and colleagues [32] found that a patchy, peripheral distribution is much more commonly seen in noncardiogenic (50%) than in cardiogenic (13%) pulmonary edema and is the best discriminating radiographic feature. Radiographic change is typically slow, and monitoring of ARDS requires the comparison of multiple chest radiographs.

ARDS is a clinical syndrome characterized by hypoxemia resistant to oxygen therapy, the absence of clinically apparent left atrial hypertension, and bilateral pulmonary opacification on the chest radiograph [33]. It was originally described by Ashbaugh and colleagues [34] in 1967 and was previously known as “adult” respiratory distress syndrome. Acute lung injury (ALI) is on the same clinical spectrum as ARDS, and represents a syndrome of respiratory distress due to underlying pulmonary edema and inflammation [35]. The incidence of ARDS/ALI has not been well defined. A recent study conducted in the United States estimated the incidence of severe ARDS/ALI to be 6 per 100,000 hospital admissions [36].

Fig. 23. Multifocal pneumonia. AP chest radiograph (A), coronal CT image through the chest (B), and axial CT image through the chest (C) show bilateral, multifocal consolidative opacities with air bronchograms, consistent with pneumonia. Note the absence of airway crowding that is seen in atelectasis.
States by Rubenfeld and colleagues [36] determined the age-adjusted incidence of ALI to be 86.2 per 100,000 person-years, with an in-hospital mortality rate of 38.5%.

Pulmonary opacities on CT are often more heterogeneous than on the chest radiograph. Goodman and colleagues [37] found that asymmetric ground-glass and consolidative opacities predominate when ARDS is secondary to pulmonary disease. When ARDS is due to extrapulmonary causes, a relatively symmetric ground-glass distribution predominates (Fig. 25). CT patterns in ARDS may be described as typical or atypical. In a typical pattern, dense consolidation involves the posterior lungs in a dependent distribution (Fig. 26). Ground-glass opacities are seen in a non-dependent distribution. In the atypical pattern, dense consolidation is seen in nondependent locations. The atypical distribution of consolidation is more likely to be found when ARDS is incited by pulmonary disease [38]. Air bronchograms are frequently seen in both forms. “Crazy-paving,” a nonspecific CT appearance of interlobular septal thickening in a background of ground-glass attenuation [39], may also be seen.

DAD can be categorized into exudative, proliferative, and fibrotic phases on pathologic findings, although varying degrees of these phases may be occurring at any one time. The early exudative phase cannot be reliably identified using CT [40]; however, in the proliferative and fibrotic phases, traction bronchiectasis and bronchiolectasis may be seen [40]. Ichikado and colleagues [41] found that the presence of extensive fibroproliferative change early in the clinical course of ARDS is predictive of poor prognosis.

Fig. 24. Progression of noncardiogenic pulmonary edema. (A) Initial AP chest radiograph of a 53-year-old woman who had urosepsis is normal. (B, C) Follow-up radiographs over the next 2 days demonstrate progressive diffuse bilateral ground-glass and consolidative opacities, consistent with noncardiogenic pulmonary edema. Note the diminishing lung volumes, a feature frequently seen in ARDS.
Survivors of ARDS often show marked improvement over the first 6 months, with normal spirometric findings, although diffusion capacity often remains low after 1 year [42]. Anterior reticular opacities are the most frequent finding on follow-up CT in survivors of ARDS [43].

Hydrostatic pulmonary edema

Hydrostatic pulmonary edema may be due to cardiac disease, renal failure, or overhydration. The radiographic findings may not be temporally synchronous with clinical disease. Characteristic radiographic findings of hydrostatic pulmonary edema include interlobular septal thickening, manifested as Kerley B lines (1- to 2-cm linear opacities projecting horizontally from the lung periphery), and Kerley A lines (2–6-cm linear opacities projecting horizontally from the mediastinum). Pleural fluid and a widened vascular pedicle are also characteristically seen. Pleural effusions may be bilateral or unilateral. When unilateral, right-sided pleural effusions are more common. Indistinctness of the pulmonary vessels is often subtle but useful in diagnosing pulmonary edema.

Fig. 25. ARDS secondary to extrathoracic disease. (A) AP chest radiograph of an 81-year-old woman who had sepsis shows diffuse ground-glass opacities with relative sparing of the left upper lobe. In addition, lung volumes are low. (B) Axial CT image through the lower chest demonstrates symmetric ground-glass opacities. Dependent atelectasis is also present.

Fig. 26. “Typical” appearance of ARDS on CT. Axial CT image through the lower chest in a 48 year-old postoperative patient demonstrates the typical pattern of ARDS, manifested as dependent opacities with relative sparing of the nondependent regions. Small bilateral pleural effusions are also seen.

Fig. 27. Hydrostatic pulmonary edema. AP chest radiograph of an 87-year-old man shows airway thickening and pulmonary vascular indistinctness, consistent with hydrostatic pulmonary edema.
edema (Fig. 27). Ground-glass opacities may be seen, and consolidative opacities are present in more advanced cases. Distribution is gravity dependent, and abnormalities are most notable at the lung bases; however, this gradient may be absent in the supine ICU patient. Radiographic changes typically occur much more rapidly than those of noncardiogenic pulmonary edema.

Cardiomegaly, with other findings of hydrostatic pulmonary edema, is suggestive of cardiogenic edema. Renal failure may present with similar findings in addition to characteristic perihilar opacities, sometimes referred to as “batwing edema.” Aggressive hydration is often seen in settings of trauma and postoperative patients and may coincide with noncardiogenic pulmonary edema.

CT findings of hydrostatic pulmonary edema include smooth interlobular septal thickening, ground-glass and consolidative opacities, and pleural fluid (Fig. 28). When underlying pulmonary disease such as emphysema is present, hydrostatic pulmonary edema may have an atypical appearance and mimic other pathology such as aspiration pneumonitis or pneumonia. Mitral regurgitation can present as asymmetric opacification of the right upper lobe.

Summary

Chest radiography is a critical component in the evaluation of the ICU patient. Daily chest radiography is typically used in patients who have severe cardiopulmonary compromise and are mechanically ventilated. Atelectasis, aspiration, hydrostatic and noncardiogenic pulmonary edema, pneumonia, pneumothorax, and pleural fluid are frequently encountered abnormalities. Chest radiography is useful in diagnosing and evaluating the progression of these entities. Chest radiography is also paramount in ensuring the proper positioning of support and monitoring equipment and in evaluating potential complications. CT can be useful when clinical and radiologic presentations are discrepant, when the patient is not responding to therapy, and in further assessing pleural fluid collections.

Daily rounds involving critical care physicians and radiologists can assist in more accurate and expedient diagnoses.

References


