Emergency Chest Radiology

Tae Jung Kim
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Springer

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ISBN 978-981-33-4395-5 ISBN 978-981-33-4396-2 (eBook)
https://doi.org/10.1007/978-981-33-4396-2

This Springer imprint is published by the registered company Springer Nature Singapore Pte Ltd.
The registered company address is: 152 Beach Road, #21-01/04 Gateway East, Singapore 189721, Singapore

To my beloved parents who taught me faithfulness and dedication, and to my wife, Jeong Soo, and son, Minjune, for their unconditional support and patience.

—T.J.K

To my parents for their endless love, support, and encouragement.

—K.H.L

To my wife, Mi Kyung, and my children, Joo Young and Jooae, for their love and encouragement.

—Y.H.C

To my sons Joo Hwang Lee and Joo Young Lee who are currently shoulder to shoulder with me in medicine, and my beloved wife Kyung Sook Yi. They have always stood by me throughout my medical life.

—K.S.L

Preface

The reliance on imaging for diagnosis and management decisions in various medical disciplines has been continuously evolving, and this is typically reflected in the emergency setting where an accurate and rapid diagnosis is critical for the patients' life. The capability of identifying key imaging features that are crucial for a timely and accurate diagnosis is difficult to achieve and requires a thorough understanding of the pathophysiology and imaging features of the specific diseases.

This book will provide an up-to-date review of every aspect of emergency chest radiology for inpatients as well as for patients admitted to emergency departments with chest trauma, infection, postoperative complications, and cardiovascular emergencies. This comprehensive book is unsurpassed as a valuable source of practical information on the imaging diagnosis of acutely ill and injured patients. To this end, the wide spectrum of chest and cardiovascular emergencies is systematically categorized and typical imaging manifestations of these emergent conditions in the current state-of-the-art imaging modalities are illustrated in detail. In addition, this book contains detailed information on the pathophysiology of diseases and the mechanisms of trauma that are the very basics for imaging diagnosis. This book is ideal for all members of the emergency team, general, thoracic and emergency radiologists, radiology residents, and medical students.

I am immensely grateful to the other three authors, Dr. Kyung Hee Lee, Dr. Yeon Hyeon Choe, and Dr. Kyung Soo Lee, for their motivation, everlasting enthusiasm, wisdom, and dedication throughout the writing and editing of this book. Moreover, I would like to thank the Springer team for their great efforts in the process of planning and producing this book.

Seoul, Korea — Tae Jung Kim

Contents

Traumatic Lung Injuries

1

1.1 Introduction

Blunt thoracic trauma is associated with a high risk of morbidity and mortality. The first description of thoracic trauma appears in the Edwin Smith Papyrus from ancient Egypt, written circa 1600 BC [1]. Hippocrates recognized that hemoptysis after rib fracture was a more severe injury than a simple rib fracture [2]. After head trauma, blunt chest trauma is the second leading cause of morbidity and mortality in patients with trauma [3]. Motor vehicle accidents are the predominant cause of blunt thoracic injury, followed by motorcycle crashes, pedestrian versus auto injuries, and falls from great heights [3, 4]. Thoracic injuries are frequently accompanied by multiple other injuries, including those of the abdomen, head, and extremities. Diagnostic imaging has become a critical component in the diagnosis and treatment of patients with chest trauma. A portable chest radiograph is usually used for initial screening of obvious chest traumas. CT is more sensitive than chest radiography in detecting various thoracic injuries. With the advent of multi-detector CT technologies, routine use of thin-section axial images and multiplanar reformations became possible, and the widespread use of whole-body CT in multi-trauma patients minimizes the rate of missed injuries and decreases the mortality rate.

1.2 Pulmonary Contusion

1.2.1 Mechanism of Injury

Pulmonary contusion is the most common lung injury after blunt thoracic trauma, occurring in approximately 30–70% of patients [5, 6]. Pulmonary contusion is defined as focal parenchymal injury with hemorrhage and edema formation in the alveoli and interstitium, resulting in disruption of alveolar–capillary integrity without accompanying major parenchymal disruption. There are three basic mechanisms of pulmonary contusion [7]: (1) bursting effects at the gas/liquid interfaces of the alveoli, (2) inertial effects that occur when low-density alveolar tissue is stripped from heavier hilar structures as they accelerate at different rates, and (3) implosion effects due to rebound or overexpansion of gas bubbles after passage of a pressure wave. The lung is similar to other air-containing organs such as intestines and eardrums in its vulnerability to blast effects. Contusions can occur when the chest wall is compressed against the lung parenchyma, by shearing of the lung tissue across bony structures, from rib fractures, or by previously formed pleural adhesions tearing the lung tissue. Contusion in the opposite site of the lung may be seen (contrecoup contusion).

Pulmonary contusion leads to pathophysiologic changes depending on the extent and sever-

T. J. Kim et al., *Emergency Chest Radiology*, https://doi.org/10.1007/978-981-33-4396-2_1

ity of injury, such as ventilation/perfusion mismatch, intrapulmonary shunt, increased lung water, and loss of pulmonary compliance, which result clinically in hypoxemia, hypercapia, and increased work of breathing [8]. Patients with pulmonary contusion may present with respiratory symptoms such as dyspnea, tachypnea, and hemoptysis.

Pulmonary hemorrhage is the typical histologic finding in lung contusion. Animal studies have demonstrated that pulmonary contusion shows progressive changes [9, 10]. Interstitial hemorrhage is followed by interstitial edema in 1–2 h. At 24 h, massive edema develops due to extravascular leakage of proteins, red blood cells, and inflammatory cells in the air spaces along with fibrin deposition. At 48 h, more fibrin, cell debris, and many inflammatory cells have accumulated, and the lymphatics are dilated and filled with protein. It has been suggested that the healing process is almost complete 7–10 days after traumatic lung injury, with little residual fibrosis [11].

1.2.2 Imaging Diagnosis

The complex pathophysiology of pulmonary contusion is reflected on chest radiograph and CT as ill-defined, patchy ground-glass opacity in mild contusion, to widespread areas of consolidation in more severe injury. Pulmonary contusion typically presents as a localized increased opacity adjacent to the area of direct trauma with a non-anatomical distribution, which is different from the areas of opacity seen in pneumonia in that they are not confined within the anatomic limits of the various segments and lobes [6] (Figs. 1.1 and 1.2). Contusion frequently accompanies fractures of the adjacent bony thorax (Fig. 1.1c), which are often absent in the pediatric population owing to greater musculoskeletal elasticity [12, 13]. It is important to note that the evidence of contusion may not be radiologically evident within 6 h after trauma. CT is more sensitive in detecting pulmonary contusion compared with chest radiographs. An animal study using a canine model found that 100% of pulmonary contusions were demonstrated on CT, compared with 38% on chest radiograph [14]. On CT, sparing of 1–2 mm of subpleural lung may be seen, especially in children [15]. Contusion develops within 6 h and usually resolves in 7 days (Fig. 1.1c), but severe contusion may persist for up to 14 days [16, 17]. Pulmonary opacities that do not resolve or progress beyond this period more likely indicate pulmonary laceration or complications such as aspiration, pneumonia, or fat embolism, rather than pulmonary contusion [18].

CT may be helpful in predicting clinical course and outcome in patients with pulmonary contusion. Wagner and Jamieson suggested that the percentage of airspace consolidation on CT may predict the need for ventilator support in patients with pulmonary contusion [19]. In that study, all patients required mechanical ventilation when more than 28% of the airspace was involved. Miller et al. measured the volume of contusion on CT and suggested that contusion volume was an independent predictor of development of subsequent acute respiratory distress syndrome (ARDS). They found that 82% of patients with greater than 20% contusion developed ARDS, while only 22% of patients with less than 20% contusion developed ARDS [20].

1.3 Pulmonary Laceration

1.3.1 Mechanism of Injury

Pulmonary laceration is defined as a traumatic disruption of the lung parenchyma that results in the formation of a cavity that is filled with blood or air [19]. A traumatic, blood-filled lung cyst is also called a pulmonary hematoma. Four types of pulmonary laceration have been defined according to the mechanisms of injury. Type I lacerations are the most common; these are caused by rupture or shearing of the lung parenchyma due to sudden compression of the chest wall, are often centrally located, and maybe up to 8 cm in diameter (Fig. 1.3). Type II lacerations are located in a paraspinal area secondary to a traumatic shift of the lower lobes across the spine, and are often

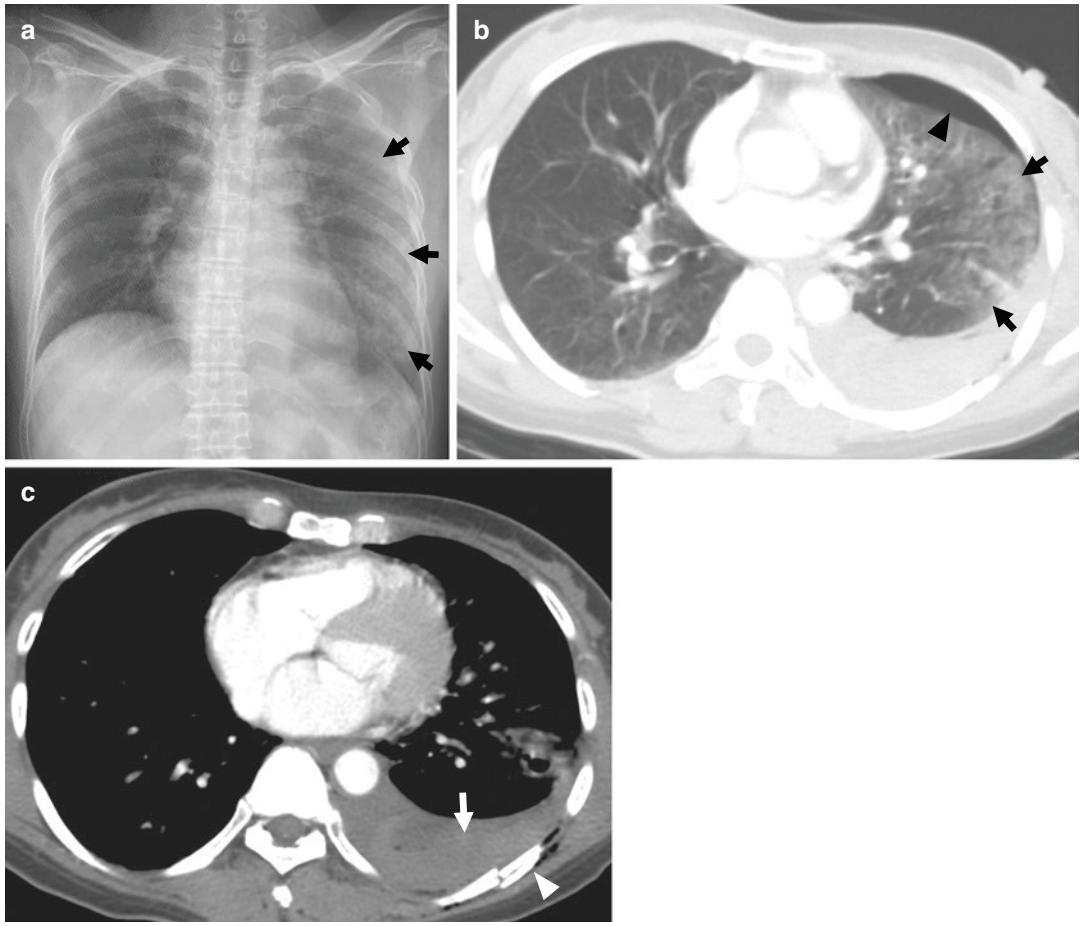

Fig. 1.1 Pulmonary contusion in a 52-year-old woman who suffered a motor vehicle accident. (**a**) Chest radiograph shows diffuse and ill-defined increased opacity in the left lung (arrows). (**b**) Lung window image of a CT scan obtained at the level of the left atrium shows diffuse ill-defined ground-glass opacity in the left upper and lower lobes (arrows). Also note the small pneumothorax (arrowhead). (**c**) Mediastinal window image of an enhanced CT scan depicts hyperdense pleural effusion, suggesting hemothorax (arrow). Also, note the rib fracture (arrowhead) in the left posterior thorax

elongated in shape (Fig. 1.4). Type III lacerations are caused by a direct puncture of the lungs by a fractured rib; these are the second most common type of laceration and usually occur in older patients (Fig. 1.5). Finally, type IV lacerations are tears of the lung adjacent to previously formed dense pleural adhesions [5, 6, 16]. Immediately after trauma, pulmonary laceration is often obscured on CT as well as chest radiograph by an accompanying pulmonary contusion, and becomes apparent over the next 2–3 days as the contusion gradually resolves [21].

1.3.2 Imaging Diagnosis

Pulmonary lacerations are often round or ovoid in shape owing to elastic recoil of the lung parenchyma, and may have a thin rim of hyperdense pseudomembrane, representing adjacent compressed lung parenchyma. Pulmonary laceration may demonstrate an air–fluid level or air meniscus, depending on the time course of blood coagulation and lysis in the cavity. Pulmonary hematoma presents as a well-circumscribed, round area of increased attenuation

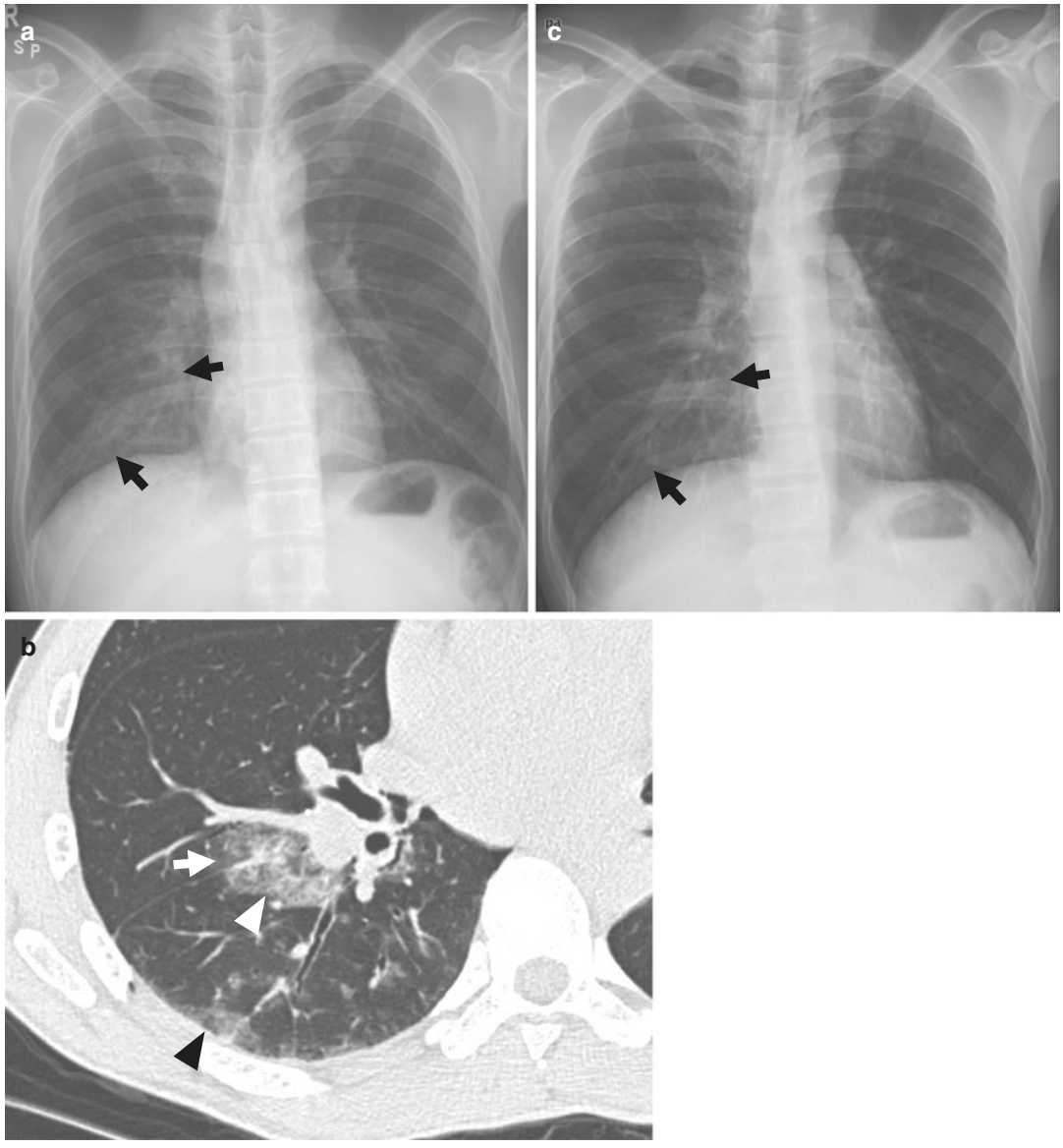

Fig. 1.2 Pulmonary contusion in a 38-year-old man after a fall from a rooftop. (**a**) Chest radiograph shows ground-glass opacities in the right lower lung zone (arrows). (**b**) Lung window image of a CT scan obtained at the level of the right middle lobar bronchus demonstrates ill-defined ground-glass opacities in the right middle (arrow) and lower (arrowheads) lobes showing nonanatomical distribution, which is a means of distinguishing injury from infectious pneumonia. (**c**) Follow-up chest radiograph taken 5 days after the initial event demonstrates resorption of pulmonary contusion in the right lower lung zone (arrows)

on CT (Fig. 1.3c). Typically, pulmonary laceration takes weeks to months to resolve completely. During the resorption process, some lacerations may appear as thin-walled lung cysts, referred to as posttraumatic pneumatoceles, or solid pulmonary nodules. Correlation with the previous radiological studies as well as trauma history aids in accurate diagnosis. Potential complications of pulmonary laceration include lung abscess formation due to secondary infection, enlargement of the cavity due to the ball-valve effect, and formation of a bronchopleural fistula in cases of peripheral laceration [22].

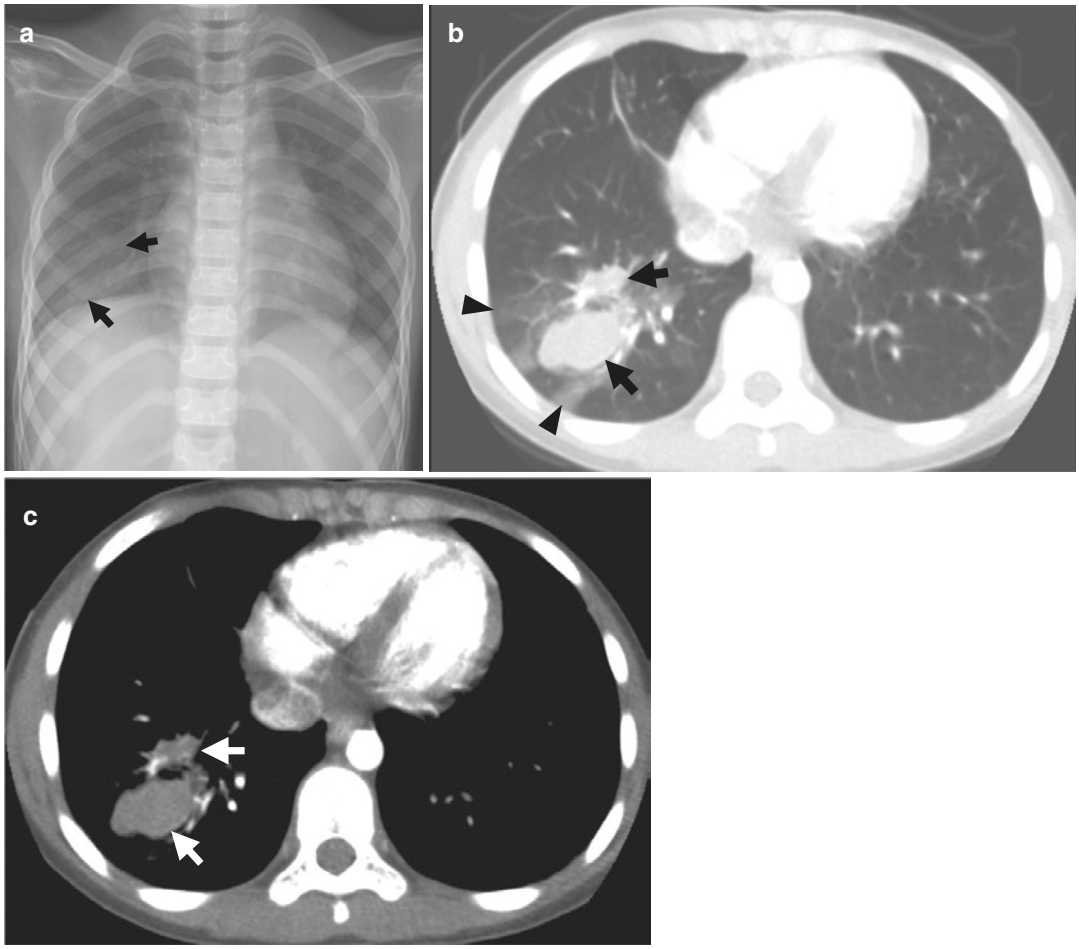

Fig. 1.3 Type I pulmonary laceration in a 9-year-old boy with a history of a fall from a great height. (**a**) Chest radiograph shows a localized area of increased opacity in the right lower lung zone (arrows). (**b**) Lung window image of a CT scan obtained at the level of the left ventricle demonstrates type I pulmonary lacerations in an ovoid shape in the right lower lobe (arrows). Also, note surrounding ground-glass opacities representing either pulmonary contusion or hemorrhage (arrowhead). (**c**) Mediastinal window image of an enhanced CT scan depicts the hyperdense nature of the lesion, representing a hematoma in the lacerated parenchyma (arrows)

1.4 Traumatic Lung Herniation

Herniation of the lung through a traumatically induced thoracic wall defect is a rare occurrence. The anterior thorax is the site of predilection for lung herniation because it lacks muscular support compared to the posterior thorax, which is supported by the trapezius, latissimus dorsi, and rhomboid muscles [23]. Lung herniation is most often asymptomatic. However, surgical correction is necessary if lung herniation is accompanied by incarceration or worsened by positive pressure ventilation. CT can be used to clearly define the extent of lung herniation as well as associated traumatic injuries in the chest wall and pleural space (Fig. 1.6).

1.5 The Roles of Imaging Studies and Recommended Protocols in Traumatic Lung Injury

Imaging studies are critical for establishing a diagnosis and treatment plan for patients with traumatic lung injury. Chest radiography is the initial imaging modality for standard workup of

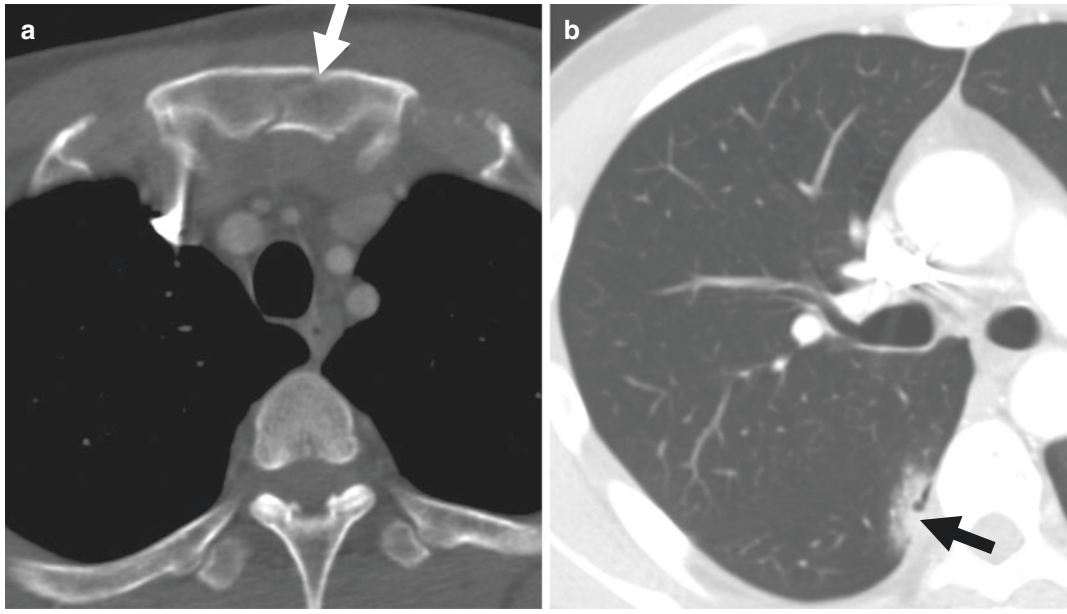

Fig. 1.4 Type II pulmonary laceration in a 30-year-old man with a dashboard injury from a motor vehicle accident. (**a**) Bone window image of a CT scan obtained at the level of the arch vessels shows sternal fracture (arrow), suggesting high-energy trauma. (**b**) Lung window image of a CT scan obtained at the level of the right main stem bronchus demonstrates a type II pulmonary laceration in an elongated shape in the paravertebral area of the right lower lobe (arrow), which occurred due to compression of alveoli against the vertebrae

acute thoracic trauma, and is typically performed as a portable anteroposterior radiograph with the patient in a supine position. Obvious chest injuries such as rib fracture, large pneumothorax, or hemothorax can easily be detected during this initial workup. CT of the chest, either alone or as part of a whole-body scan, is generally the next imaging study if additional workup is required. Small pneumothorax, hemothorax, pulmonary contusion or laceration, and tracheobronchial injuries can be successfully visualized by CT. Scans should be acquired at thin detector configurations so that multiplanar reformations can be reconstructed from the thin axial images. MRI is not commonly used in the acute trauma setting, but can be used in stabilized patients for subsequent detailed evaluation of vascular, cardiac, spinal, and bone and joint injuries. Ultrasound can be used as a complementary tool for detecting pneumothorax, hemothorax, and pericardial effusion.

1.6 Summary

Pulmonary contusion and laceration are the most common lung parenchymal injuries that result from blunt thoracic trauma. Pulmonary contusion represents focal hemorrhage and edema in the lung parenchyma without major parenchymal disruption. Pulmonary contusion typically shows a nonanatomical distribution, not confined within the anatomic limits of the lungs, which differs from pneumonia. Pulmonary laceration is a traumatic disruption of the lung parenchyma, resulting in cavity formation. Four types of pulmonary lacerations have been described according to the mechanism of injury. Imaging plays a vital role in demonstrating pulmonary parenchymal injury and any associated injuries and also provides information that is essential to establishing a treatment plan, as it grants a better understanding of the extent and mechanism of injuries.

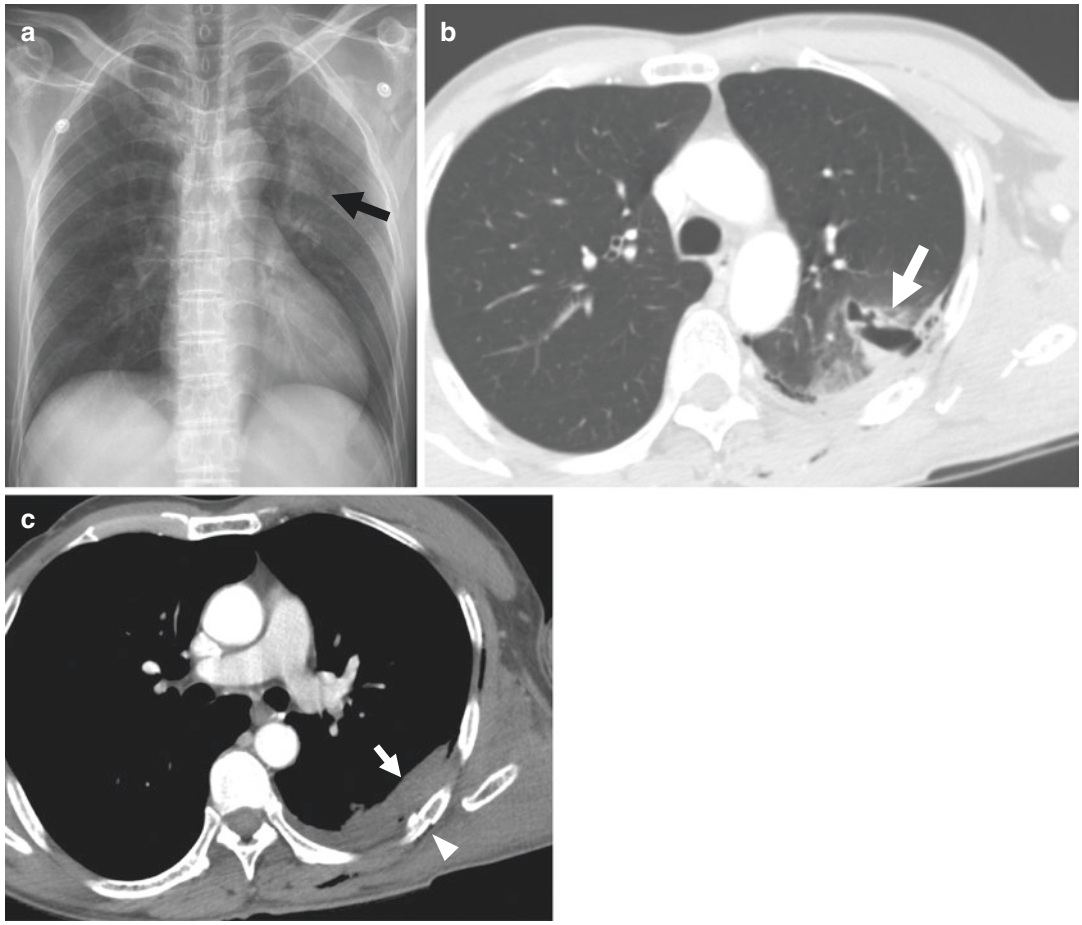

Fig. 1.5 Type III pulmonary laceration in a 38-year-old man who suffered a motor vehicle accident. (**a**) Portable chest radiograph taken with the patient in a supine position shows a localized area of increased opacity in the left upper lung zone (arrow). (**b**) Lung window image of a CT scan obtained at the level of the aortic arch demonstrates a type II pulmonary laceration in a tubular shape (arrow), which was caused by direct puncture of the lung by a fractured rib. (**c**) Mediastinal window image of an enhanced CT scan depicts a hemothorax (arrow) and rib fracture (arrowhead) in the left thorax

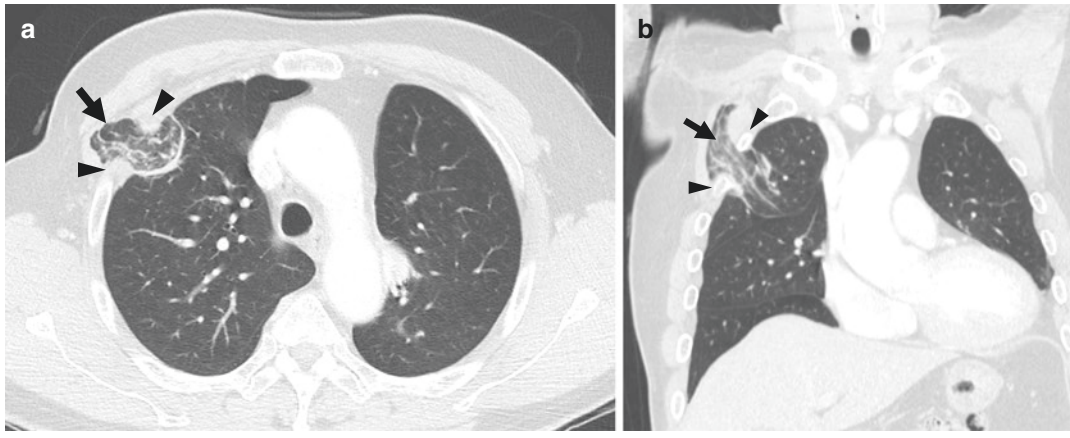

Fig. 1.6 Traumatic lung herniation in a 65-year-old man with a history of rib fracture. (**a**, **b**). Axial (**a**) and coronal (**b**) lung window images of a CT scan obtained at the level of the aortic arch demonstrate a focal herniation of the right upper lobe (arrow) through the chest wall defect (arrowheads) due to a previous rib fracture

Key Points

- On CT, pulmonary contusion manifests as a poorly defined area of consolidation and ground-glass opacity, usually in the lung periphery adjacent to the area of direct trauma with a nonanatomical distribution.
- On CT, pulmonary laceration is characterized by the presence of localized air collection in an area of consolidation.
- Traumatic lung injury typically shows a nonanatomical distribution, not confined within the anatomic limits of the lungs, which differs from pneumonia.

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Traumatic Airway Injuries

2

2.1 Introduction

Tracheobronchial injuries are relatively uncommon, occurring in only 0.5% of all patients with multiple injuries managed in major trauma centers [1]. Tracheobronchial injuries are life threatening, with more than 75% of patients with blunt tracheobronchial trauma dying before they reach the emergency department [2]. There are two types of tracheobronchial injuries. Penetrating injuries can be caused by laceration or from projectile injuries to the neck or chest. Blunt injuries can occur from motor vehicle accidents or fall from great heights. Pneumomediastinum and pulmonary interstitial emphysema are defined as abnormal gas collection in the mediastinum and pulmonary interstitium, respectively, and mainly occur because of alveolar rupture due to increased alveolar pressure. This chapter concentrates on blunt tracheobronchial injuries that occur between the intrathoracic trachea and the main stem bronchi, pneumomediastinum, and pulmonary interstitial emphysema.

2.2 Tracheobronchial Injury

2.2.1 Mechanism of Injury

Three potential mechanisms of blunt tracheobronchial injuries have been proposed [3]. First, sudden compression of the thoracic cage is the most common pattern of injury associated with tracheobronchial disruption. It is postulated that this produces a decrease in the anteroposterior diameter and a widening of the transverse diameter. Then, lateral expansion of the lungs causes traction on the trachea at the carina. Airway injury occurs if this lateral force exceeds the limits of tracheobronchial elasticity. Second, airway rupture may occur as a consequence of high intraluminal airway pressure. Pressure in the trachea suddenly increases due to compression of the lungs, trachea, and main bronchi between the sternum and the spine. If the glottis is closed at the time of impact, rupture may occur if the intraluminal airway pressure exceeds the elasticity of the main airway. These types of airway injuries most commonly occur at the junction between the membranous and the cartilaginous airway or between cartilaginous rings. The third potential mechanism is a rapid deceleration injury that generates shearing forces at the cricoid cartilage and carina, which are relatively well-fixed to surrounding structures [4].

Fractures of the bronchi are more common than those of the trachea, constituting approximately 80% of all airway injuries. The main bronchi 1–2 cm distal to the carina is the most frequently involved location, and the right side is more frequently involved than the left [5, 6].

Symptoms and signs of tracheobronchial injuries include cough, dyspnea, hemoptysis, and shock. Tracheobronchial injuries are most often accompanied by pneumothorax, pneumomediastinum, and subcutaneous emphysema.

T. J. Kim et al., *Emergency Chest Radiology*, https://doi.org/10.1007/978-981-33-4396-2_2

Subcutaneous emphysema in the cervical or thoracic region is the most common finding, seen in 65–87% of patients [7].

Early diagnosis of tracheobronchial injuries is very important because unrecognized injuries may eventually lead to airway stenosis and stricture, for which resection rather than reparative surgery is usually warranted [2].

2.2.2 Imaging Diagnosis

The most common radiographic findings of tracheobronchial injuries are the presence of pneumothorax, pneumomediastinum, and subcutaneous emphysema [8] (Fig. 2.1). Subcutaneous emphysema initially involves the neck and upper thorax and later becomes generalized [9]. Tracheobronchial laceration or fracture may cause bronchial obstruction and atelectasis of the ipsilateral lung. In case of complete bronchial transection, the ipsilateral lung may fall posterolaterally away from the hilum ("fallen lung sign"), which is a pathognomonic but uncommon finding [10–12] (Fig. 2.1). The presence of a persistent pneumothorax, even with chest tube placement and suction, pneumothorax, and pneumomediastinum in the absence of pleural effusion, or mediastinal or cervical subcutaneous emphysema in a patient who is not receiving positive pressure ventilation should raise the concern of possible tracheobronchial injury. Overdistention or herniation of an endotracheal cuff may be seen in tracheal laceration. CT can be used to identify the tear site in tracheobronchial injuries. Disruption of the bronchial wall can be visualized on CT. An endotracheal cuff that has herniated through the tracheal defect may show a "Mickey Mouse head" appearance on CT [13]. An acute change in the caliber or acute angulation of the airways can also suggest tracheobronchial injury (Figs. 2.1b and 2.2). The presence of mediastinal air adjacent to the airways is an indirect sign of tracheobronchial tear [13, 14]. Free air detected at the level of the carina is consistent with main bronchial injury [14, 15]. In case of suspected tracheobronchial injury on CT, immediate bronchoscopy should be attempted to confirm the diagnosis and evaluate the extent of injury.

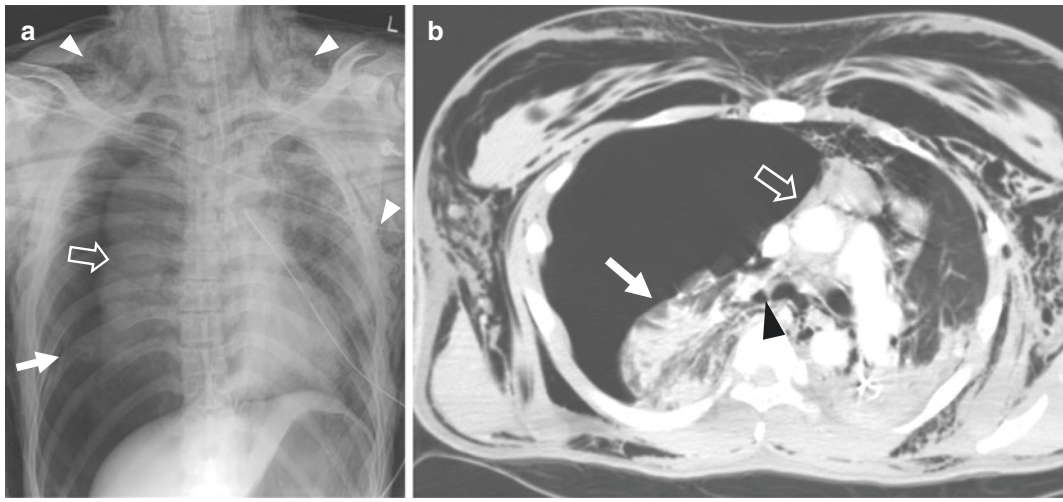

Fig. 2.1 Bronchial laceration in a 50-year-old woman who suffered a motor vehicle accident. (**a**) Portable chest radiograph shows a large pneumothorax (arrow) and extensive subcutaneous emphysema (arrowheads). Note right lung collapse (open arrow). (**b**) Lung window image of a CT scan obtained at the level of the aortic arch shows a large pneumothorax and pneumomediastinum with extensive subcutaneous emphysema. Acute change in the caliber is noted in the right main bronchus, suggesting bronchial laceration (arrowhead). The right lung is collapsed and displaced inferior to the right hilum ("fallen-lung sign") (arrow). Also note mediastinal shifting (open arrow), suggesting tension pneumothorax. Laceration of the right main bronchus was identified during surgery

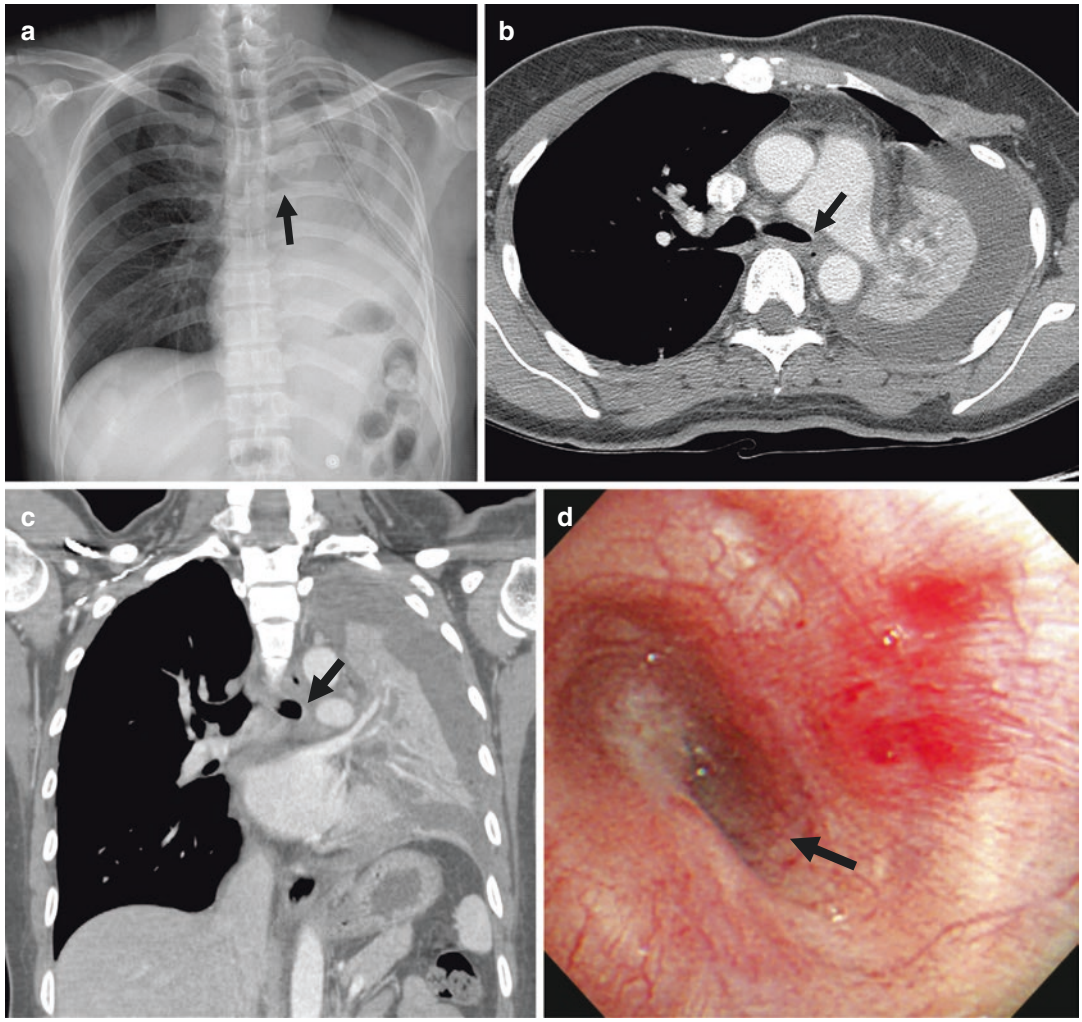

Fig. 2.2 Bronchial laceration in a 45-year-old woman who suffered a motor vehicle accident. (**a**) Chest radiograph shows abrupt cut-off of the left main bronchus (arrow). Total atelectasis of the left lung is persistent although a chest tube is placed in the left hemothorax due to pneumothorax, which strongly suggests bronchial injury. (**b**, **c**) Axial and coronal CT scans demonstrate abrupt cut-off of the left main bronchus (arrow). Also note the total collapse of the left lung with hydropneumothorax. (**d**) Bronchoscopy reveals complete obstruction at the proximal left main bronchus (arrow) due to laceration. Surgery confirmed the bronchial laceration and resection and anastomosis of the left main bronchus was performed

2.3 Pneumomediastinum

2.3.1 Etiology and Pathogenesis

Pneumomediastinum is defined as the presence of gas in the mediastinal space. Gas within the mediastinum may originate from the following sites: the lungs, airways, esophagus, neck, and abdominal cavity [16]. The most common mechanism of pneumomediastinum is the extension of gas from airspaces in the lung parenchyma into the interstitium and thence into the mediastinum. A sudden increase in alveolar pressure with airway closure results in rupture of alveoli adjacent to bronchovascular bundles. Gas moves to the hilum and mediastinum along the peribronchovascular interstitium [16, 17]. Development of pneumomediastinum is closely related to certain conditions that result in a sudden increase in

Table 2.1 Causes of pneumomediastinum

Lungs
Alveolar rupture associated with elevated alveolar pressure
Deep respiratory maneuvers: strenuous exercise, forced vital capacity breaths
Valsalva maneuvers
Weight lifting
Smoking marihuana or cocaine
Asthma
Vomiting
Artificial ventilation
Closed chest trauma
Sudden drop in atmospheric pressure: rapid ascent of a scuba diver or pilot
Infection: tuberculosis, histoplasmosis, dental or retropharyngeal infection
Blunt, or penetrating chest trauma
Airways
Tracheobronchial laceration
Bronchial stump dehiscence
Bronchoscopy
Tracheostomy
Esophagus
Esophageal perforation: Boerhaave syndrome
Esophagoscopy
Esophageal carcinoma
Neck
Surgical procedure in the neck
Dental extraction
Abdominal cavity
Perforation of the hollow viscus
Surgical procedure in the abdomen
Idiopathic

alveolar pressure. Such conditions and diseases are summarized in Table 2.1. Tracheobronchial laceration and esophageal perforation, which can be traumatic, iatrogenic, or spontaneous, can progress to pneumomediastinum. Gas can also enter the mediastinum from the head and neck (e.g., from tracheostomy, laryngeal injury, or facial fracture), the retroperitoneum (e.g., from a perforated duodenal ulcer or perforated diverticulitis), or the chest wall (e.g., from a thoracostomy site). Pneumopericardium can occur by the same mechanism as that which causes pneumomediastinum. Gas probably enters the pericardium along the venous sheath of the pericardial reflections, in which the level of support from the overlying soft tissue is weak. Pneumomediastinum usually has a benign clinical course, and conservative management is indicated. Complications include pneumothorax due to stretching of the mediastinal pleura and hypotension due to impaired venous return to the heart [18].

2.3.2 Imaging Diagnosis

Pneumomediastinum is manifested by lucent streaks or focal bubble-like or larger collections of gas outlining the mediastinal structures on radiographs (Fig. 2.3). The gas shadow is typically more evident on the left side. On posteroanterior projection, the laterally displaced mediastinal pleura produces a thin longitudinal line parallel to the border of the heart (Figs. 2.3 and 2.4). This line is created by both the parietal pleura and the visceral pleura of the lung, in contrast to the visceral pleural line in cases of pneumothorax. There are several radiographic signs related to pneumomediastinum: continuous diaphragm sign, Naclerio's V sign, ring-around-the-artery sign, thymic spinnaker-sail sign, and extrapleural air sign.

2.3.2.1 Continuous Diaphragm Sign [19] (Fig. 2.3)

This is a radiographic sign of pneumomediastinum or pneumopericardium if lucency is seen above the diaphragm, or of pneumoperitoneum if lucency is seen below the diaphragm. Normally, the central portion of the diaphragm cannot be discretely visualized on chest radiographs as it merges with the cardiac silhouette. If the diaphragm can be seen continuously across the midline, this is highly suggestive of free gas within the mediastinum or pericardium. Compared to pneumomediastinum, pneumopericardium is almost always associated with pericardial fluid, leading to obliteration of the central portion of the diaphragm on radiographs taken with the patient in an erect position. Also, air in the pericardial space demonstrates a change in position on follow-up radiographs taken in different body positions [20].

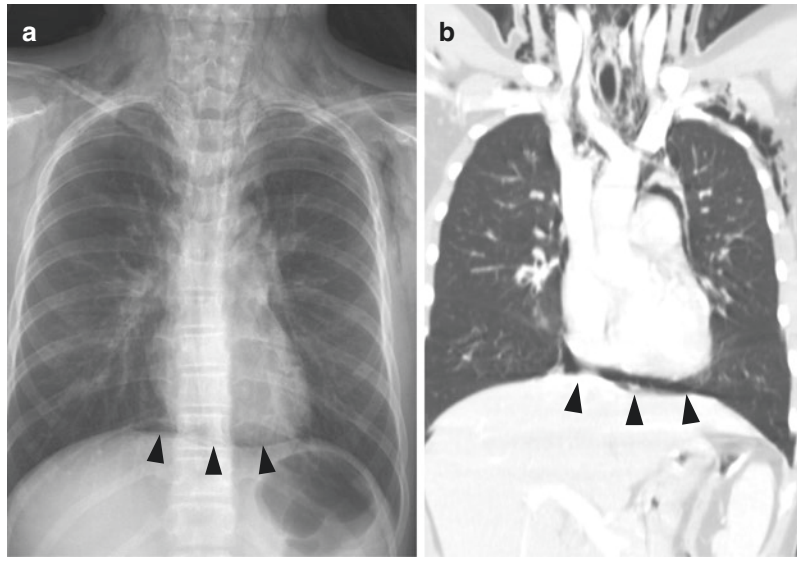

Fig. 2.3 Pneumomediastinum in a 28-year-old woman with abrupt onset of retrosternal pain. (**a**) Chest radiograph shows pneumomediastinum outlining the central portion of the diaphragm (arrowheads), a finding known as the "continuous diaphragm sign." Also note pneumomediastinum outlining the mediastinal structures and extensive subcutaneous emphysema in the neck and axillary area. (**b**) Coronal lung window image of a CT scan demonstrates free gas within the central portion of the lower mediastinum just above the diaphragm (arrow heads)

2.3.2.2 Naclerio's V Sign (Fig. 2.5)

This sign is named after Emil A. Naclerio, an American thoracic surgeon, who first described it in 1957 [21]. It is a V-shaped air collection in the left lower thorax. One limb of the V is produced by mediastinal gas outlining the left lower lateral mediastinal border. The other limb is produced by gas between the parietal pleura and medial left hemidiaphragm. This sign was originally described in patients with esophageal perforation but is not specific to that condition.

2.3.2.3 Ring-Around-the-Artery Sign [22] (Fig. 2.6)

This is seen on lateral chest radiographs; it appears as a well-defined lucency along or surrounding the right pulmonary artery.

2.3.2.4 Extrapleural Air Sign [23]

This is defined as the presence of gas between the parietal pleura and the diaphragm. On a lateral projection, the gas forms a radiolucent pocket of gas posterior to the dome of the hemidiaphragm.

2.3.2.5 Thymic Spinnaker-Sail Sign [24] (Figs. 2.7 and 2.8)

This is a sign of pneumomediastinum present on neonatal chest radiograph. It refers to the thymus being outlined by air with each lobe displaced laterally and appearing like spinnaker sails due to mediastinum. It is distinct from the sail sign of the normal thymus seen in neonates.

2.4 Pulmonary Interstitial Emphysema

2.4.1 Pathophysiology

Pulmonary interstitial emphysema (PIE) refers to collection of gases within the peribronchovascular sheaths, interlobular septa, and visceral pleura [25]. PIE essentially, if not exclusively, occurs in preterm neonates with immature lungs, usually after mechanical ventilation therapy, but it may also occur in adults associated with virtually any phenomenon that increases intrapulmonary pressure or lung volume [26]. One autopsy study demonstrated that PIE is most commonly associated

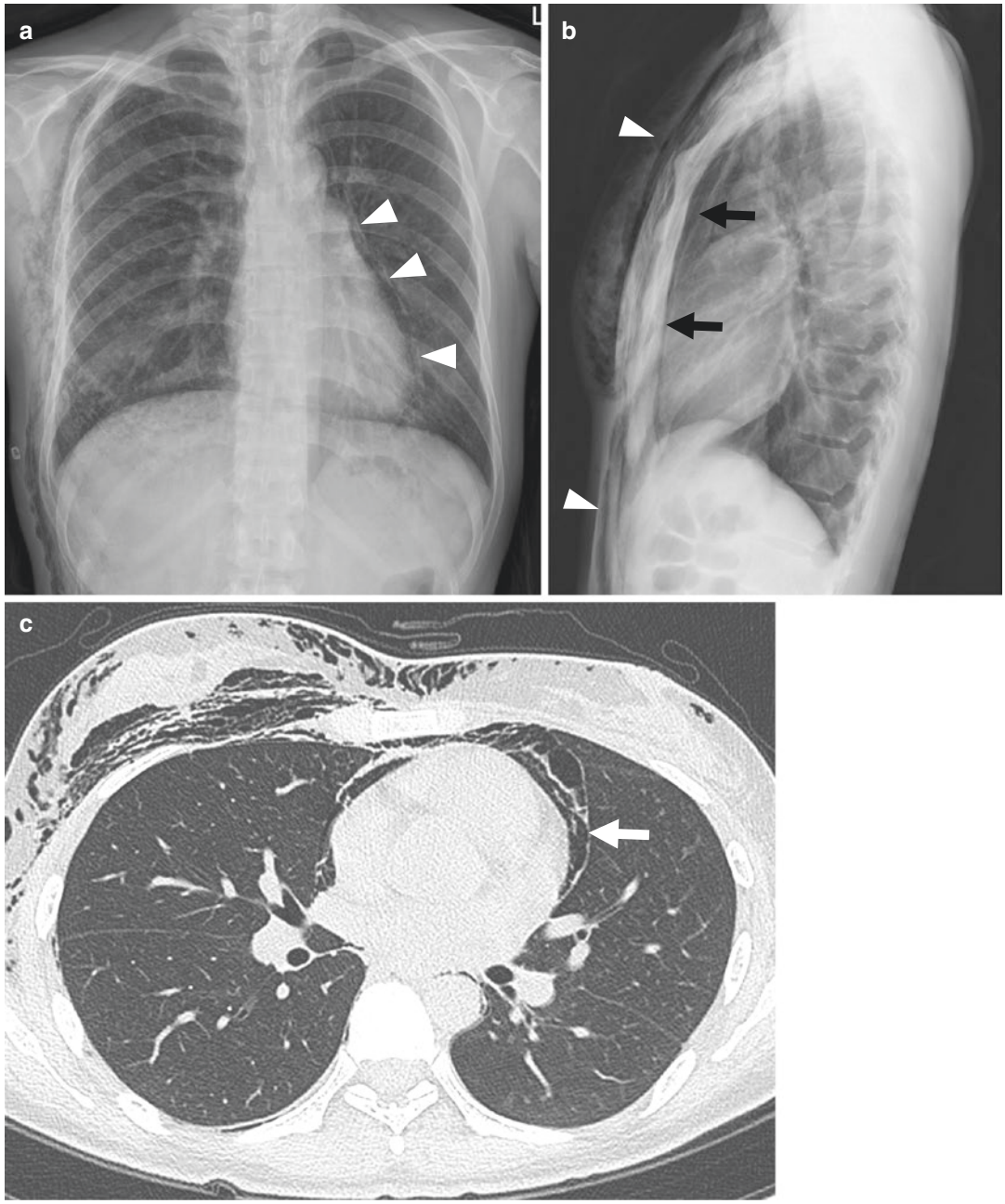

Fig. 2.4 Spontaneous pneumomediastinum in a 23-year-old woman. (**a**) Chest radiograph demonstrates a long linear opacity parallel to the left heart border (arrowheads), representing the laterally displaced mediastinal pleura. Note the mottled lucencies in the right lower thorax, suggesting extensive subcutaneous emphysema. (**b**) Lateral radiograph demonstrates retrosternal pneumomediastinum (arrows) and subcutaneous emphysema in the chest and abdominal walls (arrowheads). (**c**) Lung window image of a CT scan at the level of the aortic root shows pneumomediastinum and subcutaneous emphysema with lateral displacement of the mediastinal pleura (arrow)

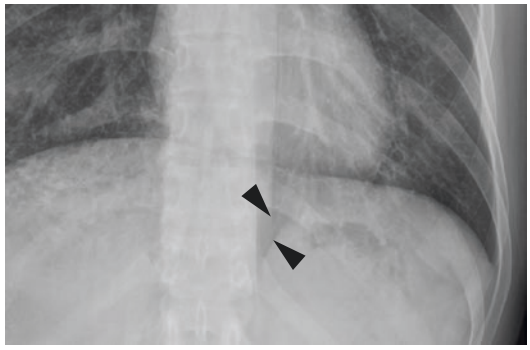

Fig. 2.5 Naclerio's V sign in a 23-year-old woman with pneumomediastinum. Magnified view of a chest radiograph demonstrates an air lucency outlining the medial portion of the left hemidiaphragm and the lower lateral mediastinal border (arrowheads)

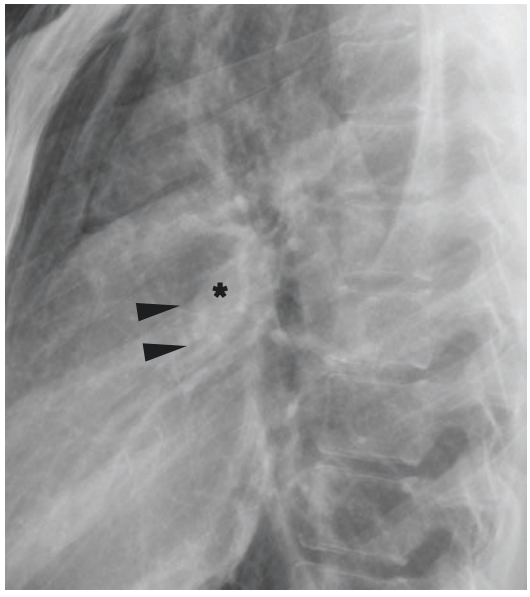

Fig. 2.6 Ring-around-the artery sign in a 32-year-old woman with pneumomediastinum. Magnified view of a lateral radiograph demonstrates a curvilinear air lucency (arrowheads) along the anterior surface of the right pulmonary artery (asterisk)

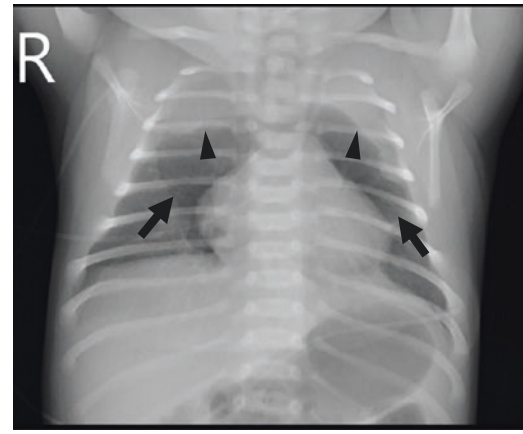

Fig. 2.7 Pneumomediastinum in an infant with chest wall retraction. Supine chest radiograph demonstrates pneumomediastinum outlining the cardiac border (arrows). Both lobes of the thymus (arrowheads) are lifted due to the air in the mediastinum

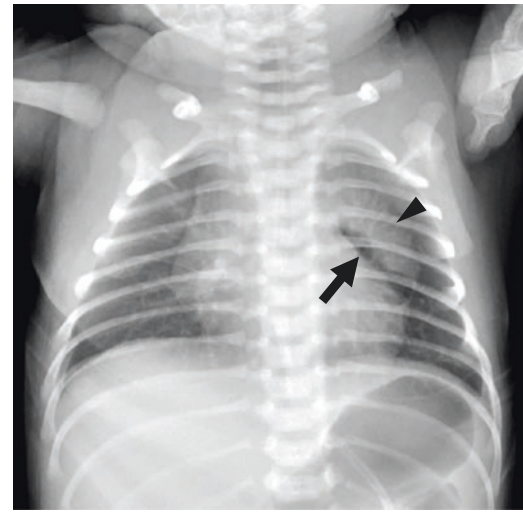

Fig. 2.8 Thymic spinnaker-sail sign in an infant with pneumomediastinum. Supine chest radiograph demonstrates elevation and lateral displacement of the left lobe of the thymus (arrow head) due to air in the mediastinum (arrow), creating a thymic spinnaker-sail sign

with usual interstitial pneumonia and a history of prior mechanical ventilation [27]. In neonate placed on mechanical ventilator support or continuous positive airway pressure, increased alveolar pressure, and poor compliance of the lungs may result in rupture of the alveoli. Air then escapes into the adjacent pulmonary interstitium and lymphatics. Pulmonary interstitial emphysema may resolve spontaneously, but may persist or progress with prolonged air leakage, causing pneumomediastinum, pneumothorax, pneumopericardium, or subcutaneous emphysema.