Lung/Pleural Injuries

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8.1 Introduction

Trauma is the most common cause of death during the first three decades of life in occidental countries, followed only by malignancies, and deaths for thoracic trauma account for about 25% of all trauma deaths [1]. As thoracic trauma may produce injuries to endothoracic organs that play a vital role in normal physiology and homeostasis, some injuries to the chest and its contents, if unrecognized or untreated, may produce death within minutes [2]. In the diagnostic algorithm of thoracic trauma, clinical data are of extreme importance and must be well recognized by the emergency physician. But, because of the low specificity of most of them consisting mainly in dyspnea, cough, hemoptysis, chest pain and, in very critical cases, in severe hypoxia or shock, imaging plays an essential role in the diagnostic work-up of these events permitting in most cases a fast and definite diagnosis and, therefore, a prompt adequate treatment.

Thoracic traumas are frequently due to blunt traumas (70–90%), generally consequent to road accidents (85%) and, less frequently, to working or domestic accidents [2]; open traumas from wounds, either ballistic or non-ballistic, are rare [3]. Lung and pleural involvement is very frequent in each of these as they are involved in approximately 65–75% [4] in blunt thoracic traumas and in nearly 95% of open traumas [5].

In blunt thoracic trauma, we find at least three different mechanism of action: direct force, rapid deceleration, and barotrauma. Usually, each modality is responsible for different effects on thoracic structures. Direct trauma generally interests structures near the site of impact and, very often, damages the thoracic cage. In deceleration traumas, intrathoracic mobile organs or regions, such as heart, ascending aorta, or pulmonary parenchyma, are thorn off from adjacent organs or structure that are fixed to the thoracic cage (such as descending aorta, trachea, and main bronchi) or may hit nearby bony structures as ribs or spine [2]. Blunt trauma has got a very peculiar

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mechanism of action due to the drastic increment of intrathoracic pressure. In this case, the principal organs involved are lungs and airways. But, very frequently, the three mechanisms coexist in many traumatic events, and the involvement of thoracic organs consists in different associations of injuries [2].

In open trauma, we can find different mechanisms of damage starting from the direct stab of non-ballistic wounds to the complex mechanism of injury of a bullet wound. The resultant injuries are the same as in blunt trauma but, as in open traumas there are imaging peculiarities that should be acknowledged [5], we will discuss them separately at the end of the chapter.

8.2 Imaging Modalities

In an emergency context, three imaging modalities are helpful in the diagnosis of a thoracic trauma: chest X-ray, ultrasound (US) examination, and multidetector computed tomography (MDCT). Chest plain film and US examination play an important role in the initial emergency work-up of post-traumatic thoracic injuries as they are cheap and bedside available [2, 6] and should be considered the first and last imaging approach in unstable patients as MDCT is the technique of choice in stable ones.

Chest radiography is the first-line examination for assessment of thoracic trauma in the emergency setting [7]. Generally, in the emergency room with patients obliged in a supine position, single-view anteroposterior radiographs have many limitations characterized mainly by the incapacity to distinguish alterations in superimposed opacification from bones or damaged lung parenchyma and the impossibility to see air-fluid levels as the XR beam is perpendicular to the air-fluid level surface. Furthermore, the scarce inspiratory status and the heart magnification effect can produce apparent increases in pulmonary vascularity and pseudo-cardiomegaly [7].

Actually, many studies confirmed its lacking in sensibility and specificity compared to chest MDCT [1, 8, 9]. In particular, in 2007 Brink et al. [8] demonstrated the higher accuracy of routine chest CT in detecting lung injuries in post-traumatic patients compared to elective CT acquired only post-positive chest X-ray. More recently, Corbacioglu et al. confirmed this statement [9]. Both the authors, furthermore, proved that the nonselective CT method had changed the course of patient management in 2–22% of the patients [8, 9].

Nevertheless, chest X-ray is a useful tool for the patient evaluation in the emergency setting of thoracic trauma as it should be considered a completion of the history and physical examination [7]. Furthermore, in unstable patient's chest X-ray can rapidly detect tension pneumothorax and massive hemothorax while the patient is still in the emergency room, thus permitting immediate treatment of these life threatening conditions [2].

Chest US examination is a bedside procedure with rapid availability, absence of ionizing radiation (children and pregnant patients can be safely examined), easy repeatability, and lack of contraindications and can be performed at patient supine in the emergency room. Furthermore, US can guide invasive procedures, e.g., drainage of pleural effusions or of tension pneumothorax [10, 11].

Its limitations consist mainly in its dependence on the patient constitution (obese patients cannot be easily explored) and clinical conditions (chest US is not feasible in the presence of extensive subcutaneous emphysema) and in its dependence on the skills of the examiner, which implies that adequate training in US of the chest is a prerequisite for proper results [10]. Another important disadvantage is that pulmonary lesions can only be detected if they are pleura based [10].

Since 2008, the BLUE protocol included chest US for detection of pleuropulmonary emergencies, including pneumothoraces, pleural effusions, and pulmonary consolidations, in patients with respiratory failure admitted to the emergency department, demonstrating that chest US is a rapid and helpful tool in reducing diagnostic time in critical patients [12]. More recently, Zanobetti et al. demonstrated, in a very large cohort of patients, a higher sensitiv-

ity of US, compared to chest X-ray, for pneumothorax, free pleural effusion, and pulmonary consolidations in dyspnoic patients presenting in the emergency department [13].

Regarding post-traumatic patients, Hyacinthe et al. demonstrated that chest US acquired in the emergency room is a better diagnostic test than objective examination plus chest X-ray in comparison with CT scanning, particularly for diagnosing pneumothorax and lung contusion [14].

Actually, US examination of the thorax is strongly suggested in addiction to abdominal examination as part of the extended-FAST (focused assessment with sonography for trauma) [6, 15, 16] which has been developed in order to rule out pleural and pericardial post-traumatic alterations along with the abdominal ones [6].

The US chest examination in post-traumatic patients is generally performed with a linear probe. A range of frequencies (4–12 MHz) can be used to visualize the lungs. High frequencies are useful to look at the periphery of the lung with a high resolution as in looking for signs of pneumothorax. Lower US frequencies help with the imaging of deep lung tissues as in looking at consolidation. The examination consists mainly of longitudinal and oblique scans acquired on anteromedial, anterolateral, and posterolateral thoracic wall areas [6]. In polytraumatized patients, it is targeted to the detection of specific US patterns identified according to the international recommendations on point-of-care lung US for pulmonary contusion, pneumothorax, and pleural effusion (in particular hemothorax) [16, 17]. US signs of normal and injured pleura and lung are based mainly on the high acoustic mismatch between aerated lung tissues and the pleura which casts a total reflection of the sound wave [18]. When air content within the lungs decreases (e.g., in lung contusion), the acoustic mismatch is lowered, and the ultrasound wave can partly demonstrate the deeper pulmonary parenchymal structures. It is important to note that many signs at lung US represent artifacts occurring naturally because of acoustic mismatch of tissues reflecting sound waves [18].

Actually, MDCT has established itself as the preferred imaging method for the evaluation of polytrauma patients [19]. MDCT is widely available, quick, permits a whole-body imaging, offers the possibility of multiplanar and three-dimensional reconstructions, and it has been established as more sensitive and specific than chest radiography [1, 8, 9, 19].

MDCT in a post-traumatic chest should always include a direct acquisition with at least three window levels: one dedicated to the visualization of lung parenchyma; one for soft (mediastinal) structures; and one for the evaluation of bones. These permit us to respectively detect: pulmonary injuries and pneumothoraces, recent hematic extravasations inside (hemothorax) or outside the pleural space (hemopericardium or hematoma), and scheletric disruptions.

Successively, it is mandatory to perform an angio CT of the thoracic aorta in order to rule out large vessels injuries and to depict any contrast media extravasation suggesting arterial active bleeding. A venous and a delayed acquisition are important in order to rule out the non-arterial bleedings [3]. Furthermore, we must not forget that a traumatized patient is often a thoracoabdominal one. This statement is particularly true in open traumas where stab or bullet trajectories often include supra- and infra-diaphragmatic structures [5].

Isotropic acquisition is necessary in order to obtain high quality multiplanar and tridimensional reconstructions, helpful in detecting irregular or complex wound trajectories and in exactly evaluating the site and the extension of post-traumatic injuries.

In this chapter, we will discuss the principal MDCT aspects of lung and pleural injuries following blunt and open thoracic trauma.

8.3 Pleural Injuries

Pleura is frequently involved in chest trauma [2] due to its ubiquitarian localization along the inner surface of the chest wall. Generally, when a mechanical breach to the pleural surface occurs after a penetrating or blunt injury, the collapsed interpleural space may fill with air, blood, or both, forming, respectively, a pneumothorax, a hemothorax, or a

hemopneumothorax [20]. The percentage of their incidence in chest traumas is approximately 28% for hemothorax, 28% for pneumothorax, and 11% for hemopneumothorax [2].

8.3.1 Pneumothorax

Pneumothorax refers to an air collection in the pleural space, and it is found in about 15% to 50% of cases of blunt trauma [2]. The rate of bilateral pneumothorax is reported to be 8–10%. In 52% of these cases, pneumothorax develops in the right hemithorax; in 44%, it develops in the left; and in 4% it developed bilaterally [21].

Following blunt trauma, the main mechanism accountable for a pneumothorax is usually laceration to the visceral pleura from the sharp edges of fractured ribs. It can also be caused, in the absence of rib fractures, by peripheral lung lacerations or by ruptured alveoli and visceral pleura due to the drastic increase of intrapulmonary pressure in barotraumas or even, more rarely, by rupture of the proximal airways [20]. This last rare event is achieved only if the lesion affects the pleura-covered bronchial wall nearby the pulmonary hilum. Otherwise, airways lesions more frequently generate pneumomediastinum [20]. Penetrating injuries are also likely to produce pneumothorax due to direct pleural laceration, with air entering the interpleural space via either the chest wall or the lacerated lung [7].

The diagnosis of pneumothorax with chest X-ray in a supine patient relies on few, scarcely visible, signs. In particular, as in supine patients the free air collects anteriorly, medially, and basally, we should search for one or more of the next signs: hyperlucency at the lung base, a better definition of the mediastinal contour, the "deep sulcus" sign (an apparent deepening of the costophrenic angle) and the "double diaphragm" sign, the latter consisting in the presence of two diaphragm-lung interfaces [7]. Actually, 10–50% of pneumothoraces are not diagnosed on chest radiographs acquired on supine chest X-rays in emergency rooms [19] also if it frequently permits to depict rib fractures (Fig. 8.1).

Due to the lack of sensitivity of chest X-ray in the supine position, US has progressively been taken into account. In 2010, an evidence-based review of chest X-ray and US in post-traumatic pneumothorax, found a US sensitivity ranging from 86 to 98% in the enrolled studies, an US specificity of 97–100%, a supine chest X-ray sensitivity of 28–75%, and a chest X-ray specificity of 100%, suggesting that bedside thoracic US is a more sensitive screening test than supine chest X-ray in detecting pneumothorax in blunt chest trauma [16, 22]. In a recent study, compared to

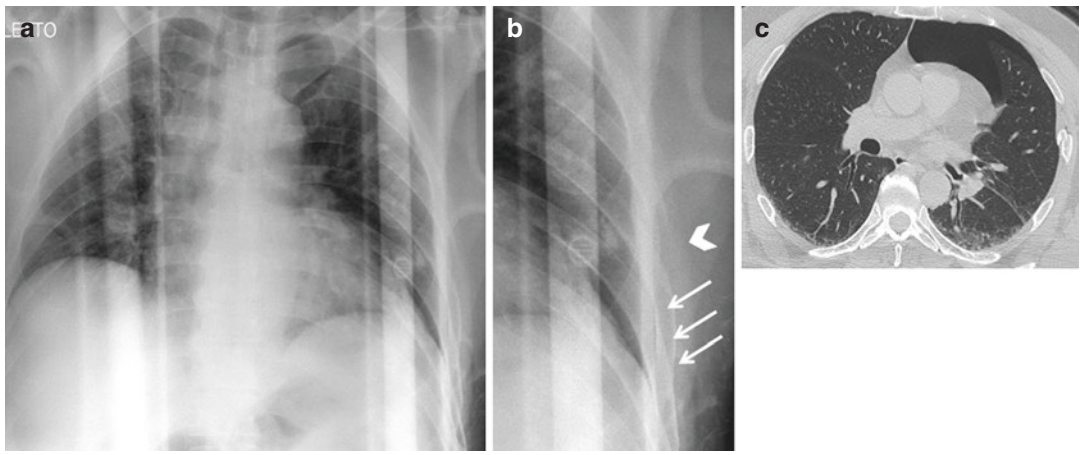

Fig. 8.1 Left pneumothorax. Supine chest X-ray acquired in the emergency room (**a**) (note the opacity of the spinal support over imposed) where is scarcely appreciable a "deep sulcus sign" at the basal left of the radiogram (*arrows*) suggesting a subtle layer of anterobasal left PNX, associated with a rib fracture (*arrowhead*). The signs are better depictable in the magnification (**b**). The chest CT examination in (**c**) confirms the diagnosis

chest CT as gold standard, the sensitivity of bed US to detect pneumothorax was 88% and the specificity 99.5%; these results are very similar to those reported in the previous review [21].

To detect a pneumothorax, the area of interest corresponds to the anterior part of the chest on both sides of the thoracic wall, approximately the third to eighth intercostal space between the parasternal and the midclavicular lines. It is essential that the results obtained have to be compared with the contralateral site. The probe is moved longitudinally in order to study the intercostal tissues on the real-time image. The hyperechogenic pleural line between the shadows of two ribs must be detected. The necessary criteria to diagnose a pneumothorax sonographically have been assessed in the "International evidence-based recommendations for point-of-care lung ultrasound" [17] and consist in absence of respiratory lung movement during dynamic examination (the so-called lung sliding sign), absence of lung pulse at power color Doppler imaging, absence of B-lines (vertical narrow based lines arising from the pleural line to the edge of the ultrasound screen, the so-called comet-tail image), detection of a "lung point" that reflects the border between the movement of the lung during respiration, and the area of absence of the sliding sign due to the pneumothorax [17] (Fig. 8.2).

While the absence of lung sliding can be seen in conditions in which there is no lung movement

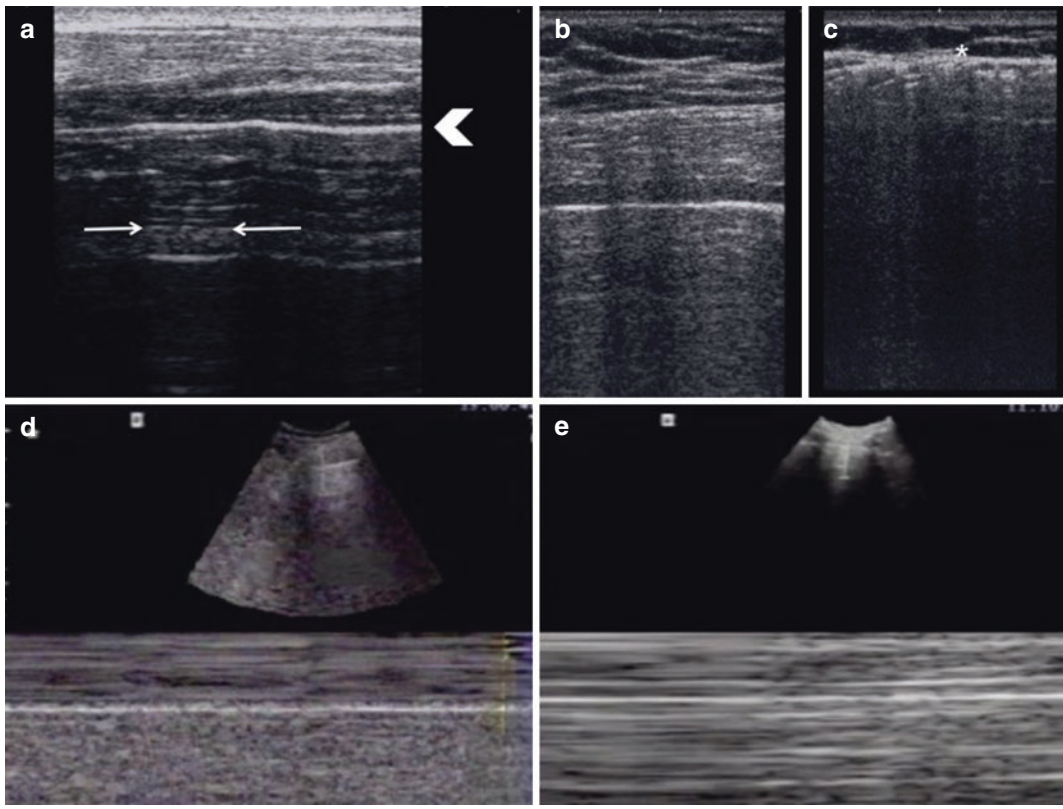

Fig. 8.2 US images to detect a pneumothorax. Despite the fact that diagnosis is purely dynamic, we have to find some basic features: first of all, the hyperechogenic pleural line between the *shadows* of two ribs must be detected (*arrowhead* in **a**). It is crucial to find the "lung point sign" (*arrows* in **a**) that reflects the border between the movement of the lung during respiration and the area of absence of the sliding sign due to the pneumothorax. It is essential that the results obtained have to be compared with the contralateral site: in (**b**) it's possible to detect the hyperechogenic pleural line in the left lung, instead in the right one (**c**) there is subcutaneous emphysema that creates an acoustic barrier (*asterisk*). M-mode on a physiological pleura (**d**), and on a pathological one (**e**). (Courtesy of Dr. G. Volpicelli, Department of Emergency Medicine, San Luigi Gonzaga University Hospital, Turin)

against the thoracic wall, for example, after pneumonectomy, pleuroparenchymal adhesion, or subpleural bullae [23], the "lung point" sign has been shown to be 100% specific for pneumothorax and should routinely be sought in all the patients with loss of lung sliding [24].

In spite of its high sensibility and specificity, we must take into account US limitations. In addition to the above-mentioned referring to the patient constitution, the presence of anterior subcutaneous emphysema and the operator skill, we must not forget that small, apical, and dependent pneumothoraces are poorly detected using thoracic US. A perspective study in 2012 correlated the site of pneumothoraces detected with chest CT with the capability of the previously acquired chest US to detect them. They found that the frequency and the extent of pneumothoraces both increments moving towards the medial and inferior thoracic regions which should always, in the authors opinion, be the first to be scanned [25]. Another limitation of chest US examination is that quantification of the pneumothorax is not easy [10] even if Volpicelli et al. in 2014 demonstrated that in supine patients, the more posterior the lung point is located, the greater the pneumothorax is, as compared to CT volume measurement [26].

MDCT offers confident diagnosis of the pneumothorax itself plus a panoramic view of all thoracic structures allowing the detection of associated injuries and accompanying pathological processes. In particular, it allows to detect the exact number and site of rib fractures, the associated lung injuries and the definition of presence and extent of an eventual subcutaneous emphysema, the latter frequently associated with pneumothorax when rib fractures are involved [11, 19, 27] (Fig. 8.3).

Intrapleural air usually collects in the nondependent regions of the thorax and, in particular, as the patients during CT scan is in a supine position, in the anteromedial basal pleural recesses [27] (Fig. 8.4). Sometimes however, especially in little pneumothorax, air can be detected nearby the rib fracture, wherever the fracture is (also if in a dependent region). This fact may be due to preexisting or trauma-related pleural adhesions which can delimitate small air collections (Fig. 8.5).

A tension pneumothorax is a less common and more dangerous variant of the simple pneumothorax and occurs when air continues to

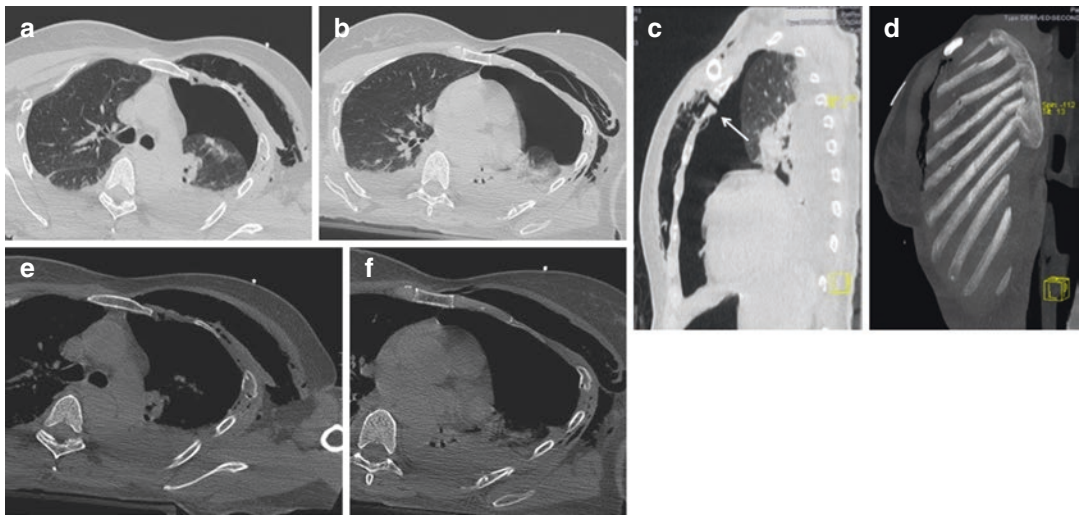

Fig. 8.3 Left pneumothorax. Axial CT-scans in (**a**–**f**) shows large pneumothorax located in the anteromedial regions with extensive chest wall emphysema. Lung parenchyma window (**a**–**c**) best helps to detect the intrapleural and intramural air collections while bone-window (**e**–**f**) permits to determine site and number of rib fractures. Note the panoramic views, often very useful for surgeons or clinicians, offered by sagittal (**c**), and maximum intensity projection (**d**) images. In the sagittal CT-image, you can also depict the mural disruption which permits the passage of the air from the interpleural to the intramural space (*arrow*)

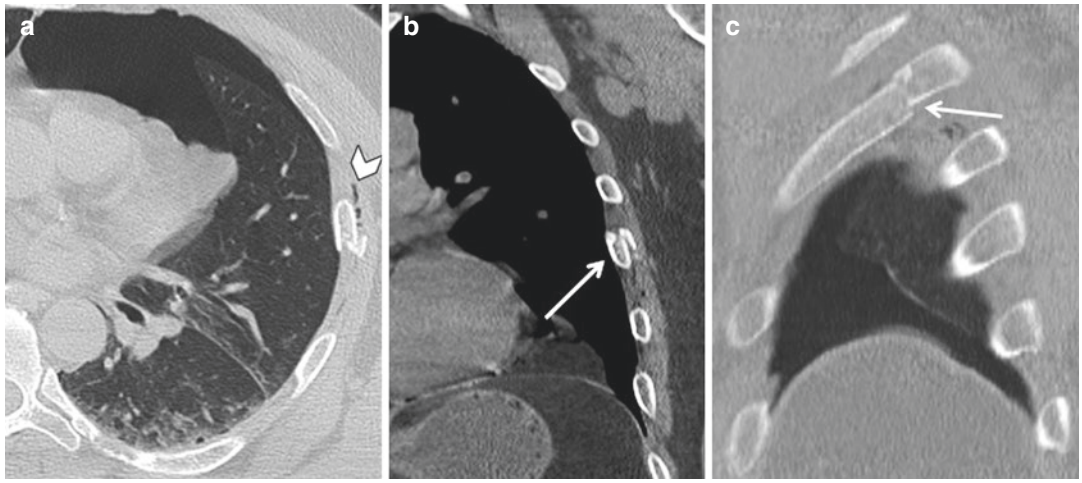

Fig. 8.4 Left pneumothorax. Axial CT-scan (**a**) shows a pneumothorax localized in the anterior-basal site, associated with rib fracture (*arrow*) and subcutaneous emphysema (*arrowhead*), as it is shown in coronal (**b**) and sagittal (**c**) reconstruction

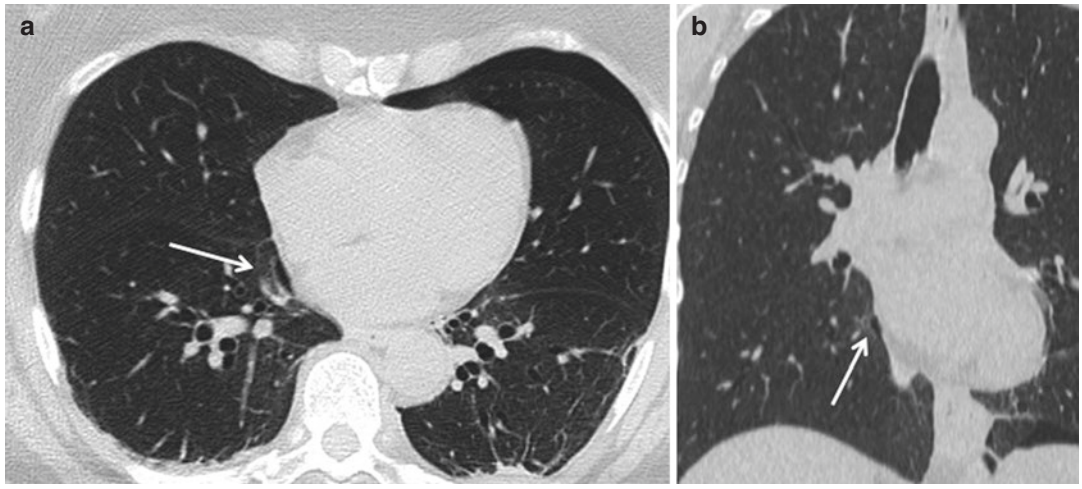

Fig. 8.5 Bilateral pneumothorax. Left pneumothorax located anteromedially and a very small air collection in the right para-mediastinal space (*arrows*): axial (**a**) and coronal (**b**) CT-scan. Note the absence of rib fractures in the right hemithorax: here, the main mechanism involved in the intrapleural air collection may have been parenchymal disruption due to the high increase in intrapulmonary pressure

enter the interpleural space with each breath, either via laceration in the chest wall or via an injured lung, but cannot escape [20]. The resultant progressive increase in volume of trapped air will compress the adjacent lung and diminish its expansion leading initially to respiratory distress and hypoxemia. As the interpleural volume of air continues to increase, the mediastinal structures, including the vena cava, are also displaced and compressed, producing an eventual drop in cardiac preload due to diminished venous cardiac filling pressures. The result is compromised cardiac output which usually rapidly progresses to cardiogenic shock and eventual cardiac arrest [20].

In this life-threatening situation, chest X-ray permits to depict in a few minutes the hyperlucency of a hemithorax plus the opacification of the

contralateral (occupied by the mediastinal structures and by the contralateral collapsed lung) thus permitting immediate pleural drainage which is necessary as promptly as possible in order to save the patient's life [7] (Fig. 8.6). We must remember that only a mediastinal shift from its median localization is not a definite sign of a tension pneumothorax as it can also occur in a massive lung athelectasia. So it is important to look for hyperlucency of a hemithorax associated with the ancillary signs of overinflation: flattened or inverted diaphragm profile and enlarged intercostal spaces [7]. Sudden evacuation of a large pneumothorax with tube drainage can be complicated by re-expansion pulmonary edema [28, 29] which is clearly visible in a chest radiogram, being characterized mainly by immediate appearance of subtle opacifications in the ipsilateral lung that can rapidly progress to the total opacification of the entire hemithorax. The complication is more common in younger patients (20–50 years of age), occurs more often than was previously believed, and it has a reported variable mortality rate reaching 20% [29]. So, a control chest X-ray should always be acquired after positioning a drainage tube in a hypertension pneumothorax as it exactly localizes the tube apex and helps us to rapidly diagnose acute complications of the invasive maneuver [7].

MDCT signs are much the same as in conventional radiography, namely contralateral mediastinal shift and collapsed ipsilateral and contralateral lung in a hyperexpanded ipsilateral hemithorax. Also in MDCT imagines, we can find a flattened or inverted ipsilateral diaphragm [19] (Fig. 8.7). Sometimes, with MDCT we are able to find the exact site of the laceration responsible for the intrapleural air accumulation.

8.3.2 Hemothorax

Hemothorax represents blood in the pleural space. It is seen in 50% of blunt thoracic trauma [19] with many possible bleeding sources including pleura, chest wall, lung, mediastinal structures, and diaphragm [20]. The main reported sources of bleeding are intercostal vessels (in rib fractures) and lung parenchyma which, respectively, represent the 50 and 30% of sources [20]. In the left hemothorax, we must also be aware that the 15% of blood is derived from the aortic isthmus [20].

Chest X-ray is scarcely sensible in the depiction of pleural effusion in the supine position, as it has been demonstrated that between 50 and 100 cm^3 of pleural fluid can be detected on upright chest radiograph while about 170–200 cm^3 are usually neces-

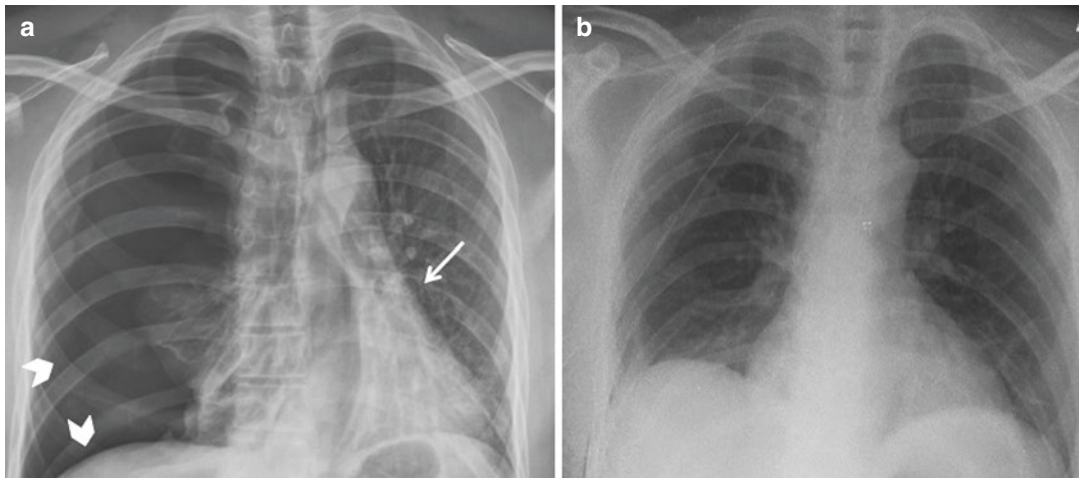

Fig. 8.6 Tension pneumothorax. Chest X-ray (CXR) in (**a**) shows the radiological signs of massive tension pneumothorax: contralateral dislocation of the mediastinum (*arrow*), hyperlucency of the ipsilateral hemithorax associated with flattening of the diaphragm and increment of the intercostal spaces (*arrowheads*). (**b**) CXR control after drainage positioning. Note the complete re-expansion of right lung without any sign of edema

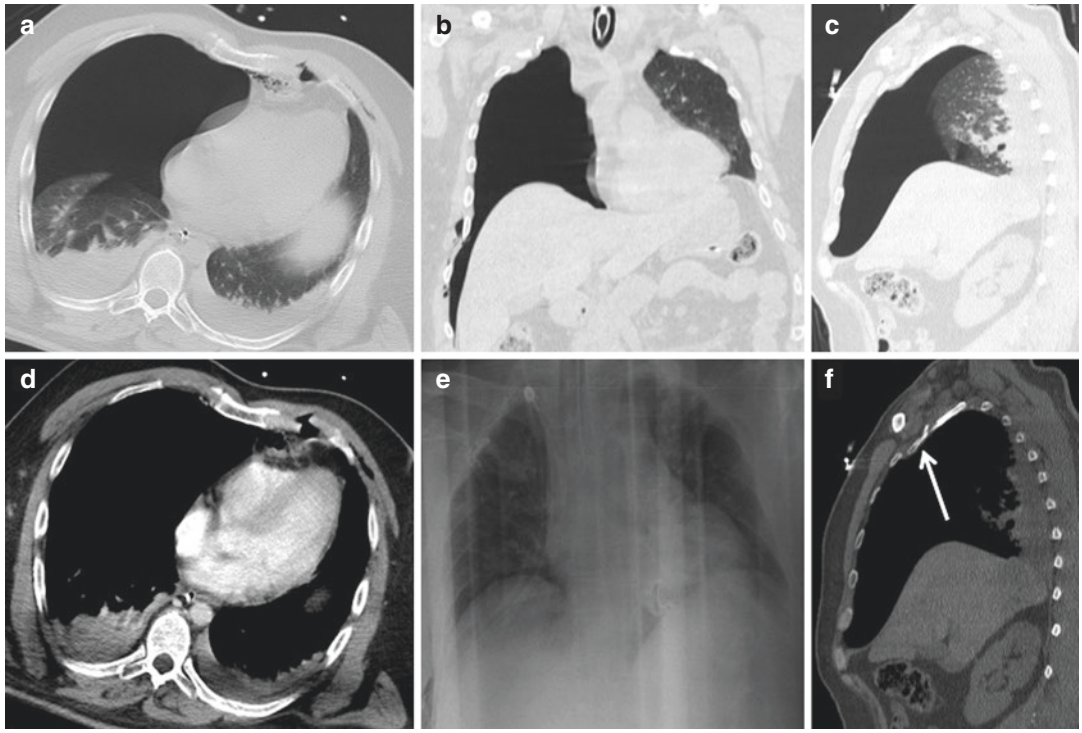

Fig. 8.7 Tension pneumothorax: axial (**a**–**d**), coronal (**b**), and sagittal (**c**) CT-scans show a massive-right pneumothorax associated with contralateral mediastinal shift collapsed ipsilateral and contralateral lung and hyperexpanded ipsilateral hemithorax. It is also visible a rib fracture in (**f**) (*arrow*), sagittal bone-window. Chest X-ray control after drainage positioning (**e**)

sary for detection when the radiograph is taken with the patient supine [30]. Chest X-ray, however, can diagnose complete opacification of a hemithorax that, in unstable post-traumatic patients, is highly suggestive of massive hemothorax, actually defined as a pleural collection of blood exceeding 1 L with clinical signs of shock [31] and so a life-threatening condition [31] (Fig. 8.8).

Following the actual international evidence-based recommendations for point-of-care lung ultrasound [17], the optimal site to detect a free pleural effusion is at the posterior axillary line above the diaphragm and the main standardized US signs to look for are a space (usually anechoic) between the parietal and visceral pleura and respiratory movements of the lung within the anechoic space. It has been demonstrated that US is an accurate methodic not only to detect even small pleural effusions but also to differentiate its nature [32]. In fact, visualization of internal echoes, mobile particles, or septa is highly suggestive of hemothorax. Unfortunately, if depicting anechoic fluid, US alone can't differentiate between pleural effusion and hemothorax (Fig. 8.9) [33].

Although it has been stated that even very small quantities of fluid can be detected with ultrasound, with one study estimating that as little as 20 cm^3 of pleural fluid can be visualized [30], a recent meta-analysis pursued in 2016 [34] showed that the sensitivity of US in detection of hemothorax is higher than radiography but it is still at a moderate level (67%), while the specificity of both imaging modalities was found to be very high. In this review, the performance of US was influenced, in the different studies, mainly by the operator skill and by the transducer frequency [34].

MDCT readily characterizes pleural fluid in the setting of trauma and can show even subtle fluid collections [8]. As blood may not be the only fluid encountered in the injured pleural space, with serous effusion and chyle being other possibilities, attenuation measurement may be

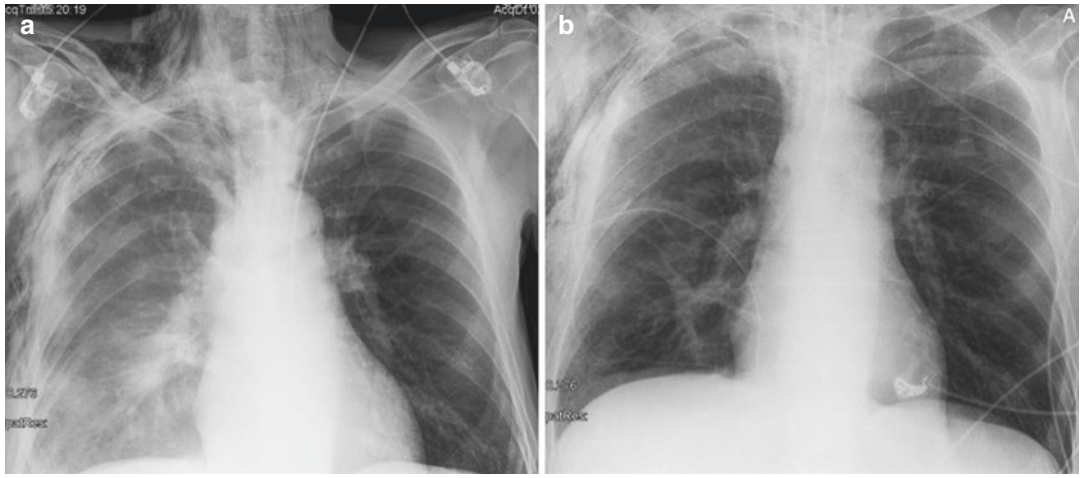

Fig. 8.8 Chest X-ray (CXR) in (**a**) shows an opacification of the right hemithorax caused by a massive hemothorax; subcutaneous emphysema is associated. CXR control after drainage positioning (**b**)

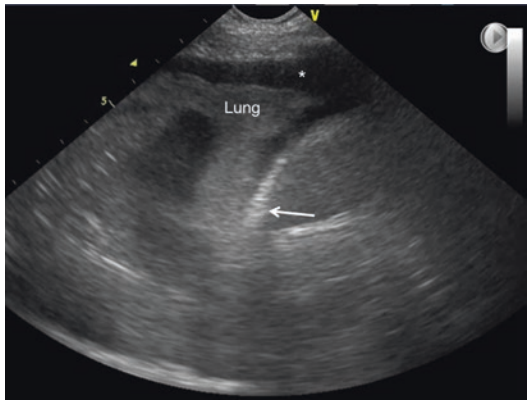

Fig. 8.9 US hemothorax. The optimal site to detect a free pleural effusion is at the posterior axillary line above the diaphragm (*arrow*) and the main standardized US signs to look for are: a space (usually anechoic) between the parietal-visceral pleura and respiratory movements of the lung within the anechoic space (*asterisk*). It has been demonstrated that US is an accurate method not only to detect even small pleural effusions but also to differentiate its nature. In fact, visualization of internal echoes, mobile particles or septa, is highly suggestive of hemothorax. Unfortunately, if depicting anechoic fluid, US alone can't differentiate between pleural effusion and hemothorax

employed to try to differentiate the various entities although this is not a simple effort due to the frequent coexistence of various fluids in the pleural space with different proportion [2]. However, all the authors recommend that measurement of attenuation of pleural space fluid should be routinely performed in the appropriate setting [2]. Usually, a mean density <15 HU is indicative of serous effusion, liquid blood attenuation typically measures 30–45 HU, and clotted blood is 50–90 HU. A mixture of serous and blood fluid can present as a homogeneous fluid with an intermediate density or as a very inhomogeneous one where hyperdense areas or stripes can be seen in the contest of a more hypodense fluid collection, usually the first located in the depending regions of the pleural space [2].

It is mandatory to perform an angio CT of thoracic aorta in order to identify active intrapleural bleeding and to differentiate arterial bleeding from venous one. Arterial hemorrhage is characterized by rapidly increasing hemothorax volume, mainly with hyperdense fluid, with depiction of active arterial bleeding by extravasation of contrast material into the pleural space in the arterial phase, having density similar to that of the contrast-enhanced arterial vessels [3] (Fig. 8.10)**.** In active venous hemorrhage, you can find the presence of contrast media into the pleural space in the venous or delayed phase of the contrast-enhanced examination with the extravasated contrast media showing density values similar or inferior to venous vessels. When an active bleeding is suspected, a delayed acquisition at 5 min is highly recommended, provided that the patient's hemodynamic stability allows for it [3] (Fig. 8.11)**.**

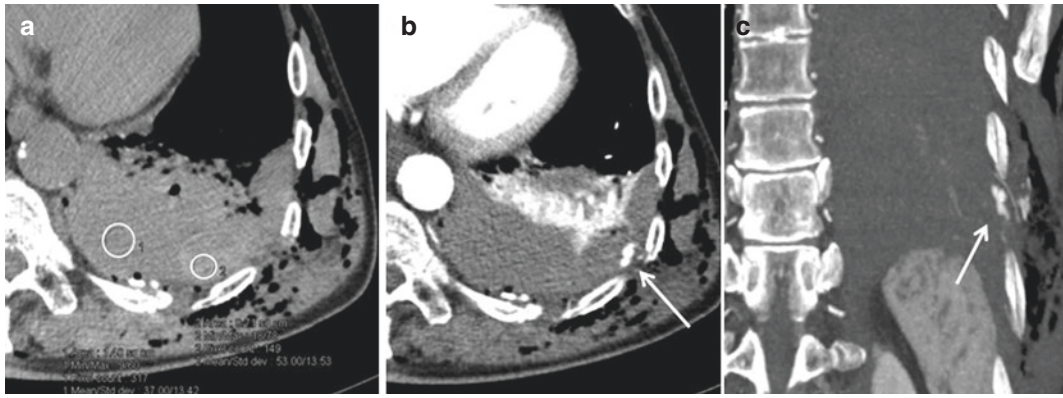

Fig. 8.10 Hemothorax. Axial CT-scan at baseline (**a**) shows a posterior pleural fluid collection. As blood may not be the only fluid encountered in the injured pleural space, measurement of the attenuation of pleural space fluid collection should be performed. In (**a**), the pleural fluid is homogeneous with a marginal hyperdense area: one roi (*red circle*) shows an attenuation of 37 HU (liquid blood attenuation measures 30–45 HU) and another roi (*yellow circle*) has 53 HU (clotted blood is 50–90 HU). Angio-CT is performed (**b**, **c**) and shows an extravasation of contrast material, proving the active arterial bleeding from an intercostal artery (*arrows*)

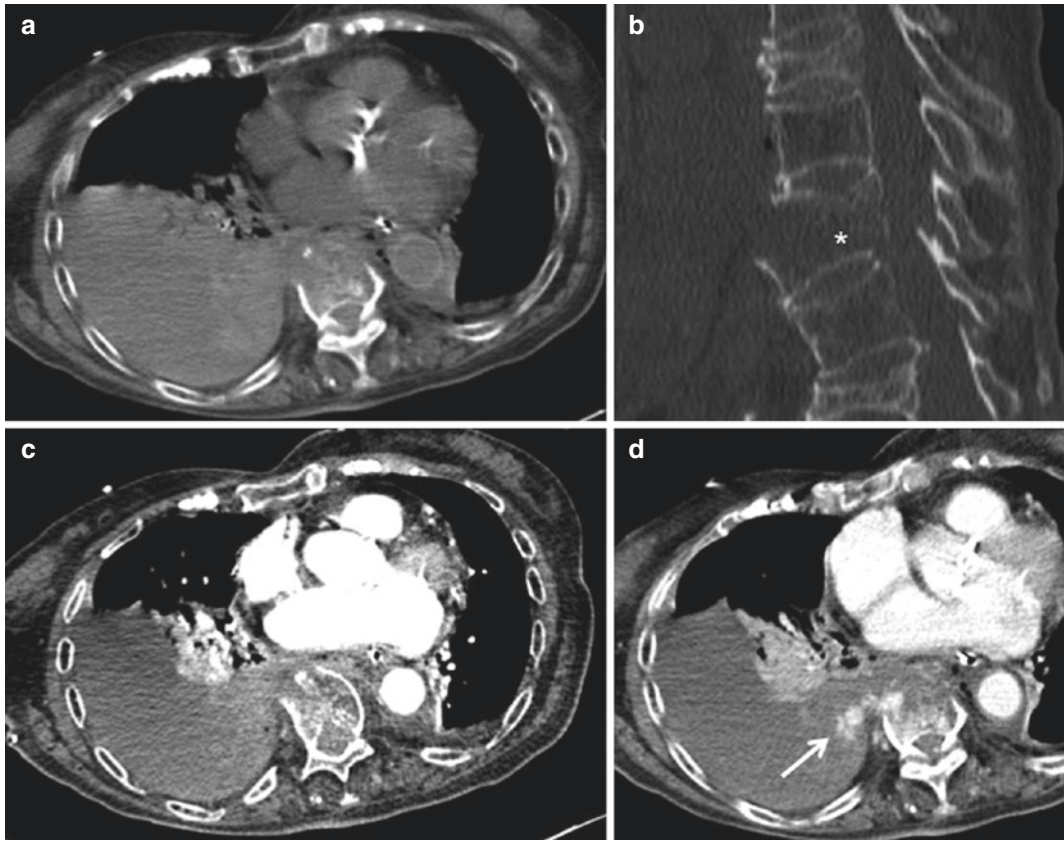

Fig. 8.11 Right hemothorax with active venous bleeding: axial CT-scan at baseline (**a**) shows an inhomogeneous fluid collection with iso- and slightly hyperdense coexisting areas. No extravasation of contrast media can be seen in arterial phase (**c**). The venous phase in (**d**) proves the active blood supply from a lumbar artery (*arrow*), in a vertebral fracture, (**b**) (*asterisk*)

8.4 Lung Injuries

The spectrum of pulmonary injuries consequent to thoracic blunt or open trauma consists in pulmonary contusion, pulmonary laceration, and pulmonary extrathoracic herniation [2].

8.4.1 Lung Contusion

It is the most common type of lung injury in blunt chest trauma with a reported prevalence of 17–70% [35].

Although the pathophysiology of pulmonary contusion is complex and poorly understood, three possible mechanisms have been hypothesized [36, 37]:

1. Inertial effect: the lighter alveolar tissue is sheared from the heavier hilar structures due to different tissue densities and therefore different rates of acceleration or deceleration.
2. Spalling effect: lung tissue bursts or is microsheared where a shock wave meets the lung tissue at interfaces between gas and liquid.
3. Implosion effect: it occurs when a pressure wave passes through a tissue containing bubbles of gas; the overexpansion of gas bubbles stretches and tears alveoli walls [36] with no macroscopic disruption of pulmonary parenchyma.

These injuries damage alveolar capillaries, so blood and other fluids accumulate in the alveolar spaces. Then, the different blood components entering the lung tissue most likely cause the release of multiple inflammatory factors which act as starter for many processes as damage to type II pneumocytes and enrollment of many flogistic cells and molecules into pulmonary parenchyma [36].

Recently, studies on rats demonstrated a rising level in bronchoalveolar lavage (BAL) of inflammatory cellules and molecules. They all decline to baseline in about 1 week [38]. This inflammatory response probably leads to multiple events including endoalveolar and endobronchial mucus production. Aufmkolk et al. in 1999 and Raghavendran et al. in the first 2000s [39, 40] reported some abnormalities in surfactant lipid composition in BAL from trauma patients which reflect type II cell injury or dysfunction. Such surfactant activity deficits were most severe 24 h after contusion and returned to normal state over 48–96 h in parallel with improving lung parameters. Furthermore, tracheal instillations of bovine surfactant in rats with trauma-induced pulmonary contusion improve pulmonary function at 24 h post-injury [40] and surfactant therapy was to be found beneficial in suine with unilateral induced lung contusion [41].

So, contusion has been proved to be sustained by multiple, non-completely known, mechanisms, including alveolar and interstitium filling with hematic and non-hematic fluids, flogistic activation (which can be responsible also for systemic effects as fever or contralateral pulmonary edema), and surfactant abnormalities. All these mechanisms lead to a precocious lung dysfunction which rapidly tends to resolve, usually with complete restitution [36, 37].

The principal complications of lung contusion are ARDS and pneumonia [37]. ARDS develops in 17% of patients with isolated pulmonary contusion, while 78% of contusions with additional injuries develop ARDS [42]. Globally, significant pulmonary contusion leads to ARDS in 50 to 60% of cases while pneumonia develops in about 20% of cases [43].

Systemic activation of neutrophils after trauma is likely to be the leading cause of acute respiratory distress syndrome and multiple organ failure in post-contusion hospitalization [44, 45].

Lung contusion is also a well-known risk factor for development of pneumonia. However, the reason for this increased susceptibility is not clear. It has been supposed that acute lung injuries of lung contusion create in the local pulmonary immune system a sort of vulnerability from a “second” hit bacterial infection [46].

The respiratory impairment of lung contusion includes ventilation/perfusion mismatching, increased intrapulmonary shunting, increased lung water, segmental lung damage, and loss of

compliance [36, 37]. Clinically, patients with pulmonary contusion present very unspecific signs as hypoxemia, hypercapnia, and dyspnea [36, 37].

In lung contusion, findings on chest radiography are nonspecific, varying from irregular, patchy areas of opacification to diffuse and extensive homogeneous hypolucency. These changes are usually evident within 6 h after trauma and resolve rapidly, typically within 3–10 days [2, 7, 37]. The scarce specificity of pulmonary post-contusion changes on chest X-ray suggest clinicians to refer as pulmonary contusion every opacification seen on radiograms in post-traumatic patients with the reported time evolution, until proven otherwise [36, 37].

As already stated, lung pulmonary changes in lung contusion consist mainly in three phases characterized, respectively, by: a precocious hematic or serous flooding of alveolar cavities, an intermediate (1–2 h after the traumatic event) edematous infiltration of lung interstitium and a successive flooding of air spaces with inflammatory cells associated with local surfactant deficiency [47]. This last phase, occurring from 6 to 24 h after the trauma and developing in consolidation, is the one visible on chest X-ray [7]. As it has been demonstrated the US accuracy in diagnosing pulmonary interstitial edema [48], we can assume that US is able to depict pulmonary contusion changes in the interstitial phase, earlier than chest X-ray. A study by Soldati et al. [47] confirmed this statement as demonstrated a sensibility of an admission ultrasound examination of 94.6% and a specificity of 100% (in the selected trauma population) compared to CT as gold standard. This specificity cannot be reached in a clinical population, since there are other lung diseases that show the same US patterns, and although US is able to recognize different alterations in lung parenchyma, its specificity in a general population is not so high [47].

As a bedside technique, lung US is performed focusing on the anterior and lateral walls and on the most posterior accessible region beyond the posterior axillary line, not compromising patient immobilization in supine position [18]. The US signs for lung contusion [17] are: the presence of the gliding sign (that is generated by the lung parenchyma movement under the parietal pleural and that indicates the absence of pneumothorax or pleural effusion) associated with the presence of an alveolo-interstitial syndrome defined as the presence of multiple B-lines, in a patient with no clinical suspicion of cardiogenic pulmonary edema, or peripheral parenchymal lesions defined as subpleural echo-poor (or with tissue-like echotexture) regions, allowing ultrasound transmission, from which B-line-like artifacts arise [17, 47]. Lower frequency ultrasound scanning may allow for better evaluation of the extent of a consolidation [17] (Fig. 8.12).

In a recent article [49], the diagnostic accuracy of US was compared to that of combined clinical examination and chest radiography for pneumothorax, lung contusion, and hemothorax, with thoracic CT-scan as reference. It tested also the ability of an US score, measuring the extent of lung contusions, to predict ARDS. It confirmed the major accuracy of US than that of combined clinical examination and chest radiography for all the three injuries, and it demonstrated that the suggested US score can identify patients at risk of developing ARDS after blunt trauma [49].

The more frequent MDCT findings of contusion consist of poorly defined, generally

Fig. 8.12 Lung contusion: US image shows an early pulmonary contusion, which was not yet detectable with a standard chest X-ray. The interstitial involvement is shown by the vertical hyperechoic lines, the so-called B-lines (*asterisk*)

non-segmental areas of consolidation and ground glass alterations [2] sometimes (especially in children) sparing 1–2 mm of subpleural lung parenchyma adjacent to the injured chest wall [43]. Contusion areas generally do not show air bronchograms as the bronchioles are often filled with fluid. These signs are well visible with MDCT, unlike chest X-ray, immediately after the traumatic event though it reaches the major increment within the first 6–24 h [19, 37, 50] (Fig. 8.13). As in chest X-ray, clearance of an uncomplicated contusion in MDCT begins at 24–48 h with complete resolution after 3–14 days [2] (Fig. 8.14).

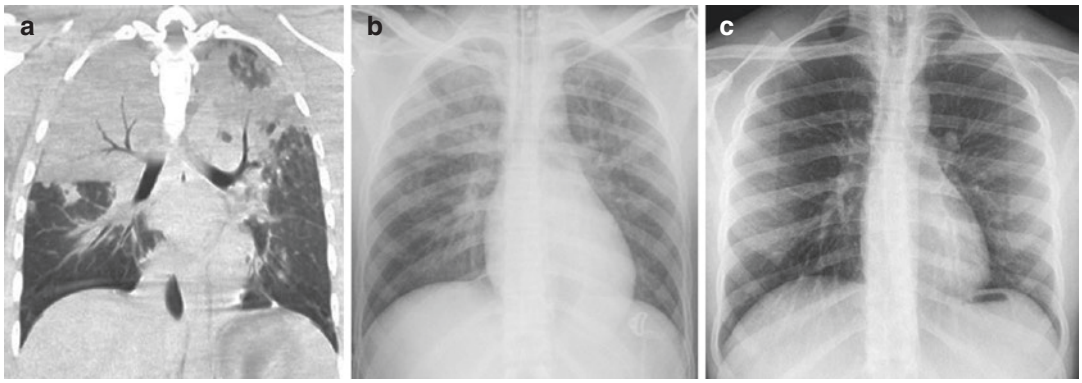

Fig. 8.13 Bilateral parenchymal contusion. Coronal CT-reconstruction immediately after trauma (**a**) shows large parenchymal consolidations. Chest X-ray (CXR) acquired 2 h after the trauma (**b**) shows patchy areas of opacification in the upper lobes. CXR after 14 days shows a complete resolution (**c**)

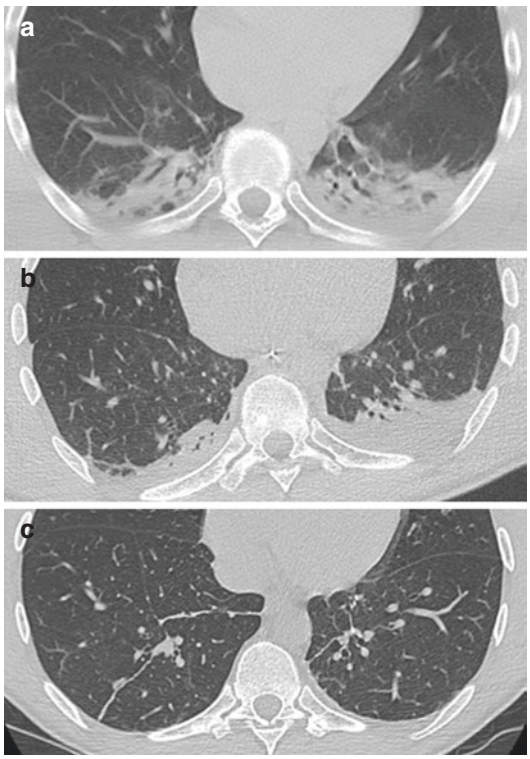

Fig. 8.14 Contusion of the posterior-lower lobes (**a**), that improves in the controls after 5 (**b**), and 10 days (**c**)

Despite the high sensibility, MDCT is poorly specific so in clinical practice, any kind of densitometric increment in lung parenchyma that shows the same temporal pattern is related to contusion damage (Figs. 8.15 and 8.16). At the same time, any kind of parenchymal increase in density arising after 24 h from the traumatic event should be referred to other lung injuries rather than contusion (the more frequents, in a post-traumatic setting, being pneumonia or aspiration) (Fig. 8.17). On the other hand, any kind of lung alterations that do not resolve within a week in a traumatic patient should be regarded as contusion complications, in particular as infection or ARDS [2, 27] (Fig. 8.18).

MDCT is the most useful tool not only in the depiction and in the recognition of contusion injuries but also in its quantification. In fact, it has been proved that, in a large post-traumatic population, the Thoracic Trauma Score (TTS) which considers patients' characteristics as long as CT measurement of contusion extent plus associated thoracic injuries significantly correlates with the occurrence of ARDS.

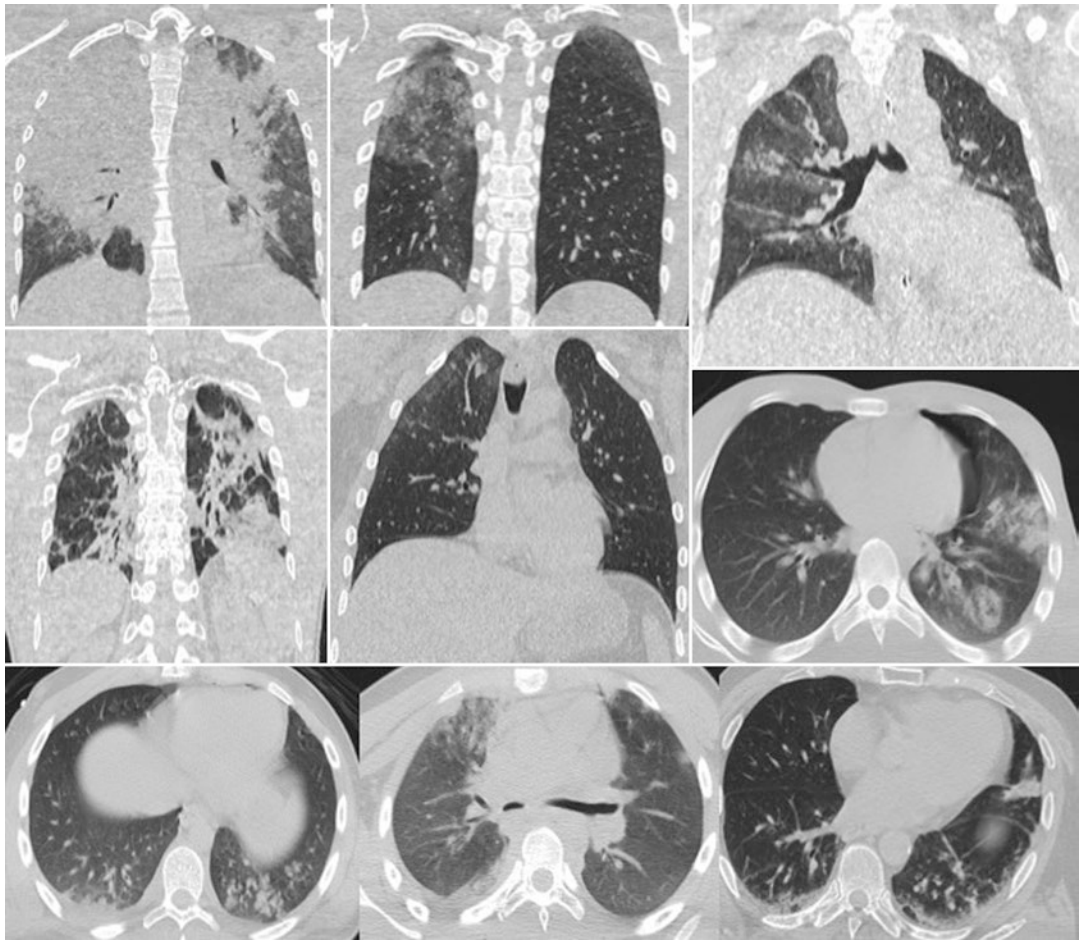

Fig. 8.15 Several examples of pulmonary contusion: it generally consists of poorly defined and non-segmental areas of consolidation and/or ground glass alteration adjacent to the injured chest wall. As we can see, the heterogeneity of lung contusion doesn't permit a differential diagnosis based only upon CT-scan aspect. Thus, every consolidation or ground glass alteration seen on CT-scan after a thoracic trauma should be considered pulmonary contusion until proven otherwise

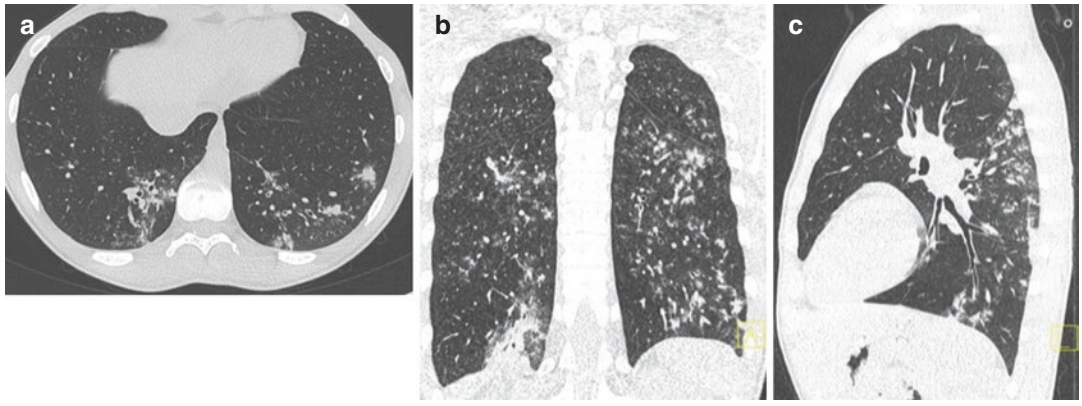

Fig. 8.16 Atypical contusion areas in the posterior segments of the inferior lobes. Post-traumatic CT-scan in axial (**a**), coronal (**b**), and sagittal (**c**) reconstructions. Their clarification in less than 10 days from the traumatic event suggested the hypothesis of pulmonary contusions unless their atypical aspect

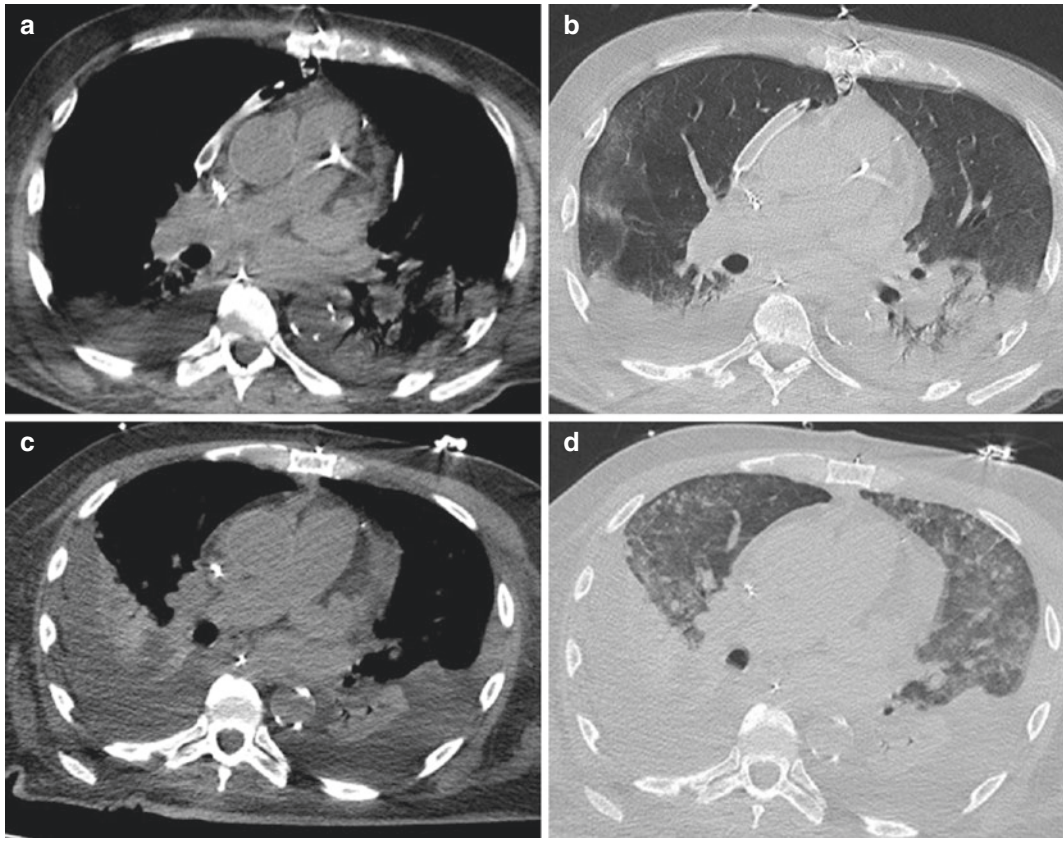

Fig. 8.17 Complication of a pulmonary contusion. (**a**, **b**) Axial CT-scans show the parenchymal areas of consolidation immediately after trauma. Any kind of parenchymal increase in density seen after 24 h should be referred to other causes, such as pneumonia as in this case (**c**, **d**) where pleural effusion and fever appeared

8.4.2 Laceration

Pulmonary laceration occurs when there is a macroscopic disruption of pulmonary parenchyma. Although lung lacerations occur more frequently with penetrating traumas, they may occur also in blunt traumas, caused by much the same mechanisms as pulmonary contusions: the direct effects of pressure on the thorax, the damage of rib fracture, the sudden tears of mobile pulmonary tissue from still structures [19].

The mechanism of laceration consists in lung parenchyma disruption and subsequent filling of intraparenchymal defects with air and/or blood. Due to the elastic recoil properties of lung tissue, normal tissue surrounding pulmonary lacerations recoils to form oval or round defects as opposed to linear or branching defects seen in solid organ tissues [2, 27].

It is difficult to detect lacerations as hyperlucency areas with chest radiography as they usually overlap opacifications due to contusions, hemothorax, or lung collapse [7, 28]. Furthermore, as in their evolution generally fill with fluids and persists for weeks or months, in a later depiction on chest X-ray, they may be misdiagnosed as pulmonary nodules, having the shape of a rounded opacification [28].

CT [35] is significantly superior to chest radiography in detecting even a small laceration and in revealing their overall extent. In the acute setting, it is visualized as a rounded cystic lesion, with air or fluid in its cavity, usually surrounded by ground glass or consolidated area due to surrounding contusion [2, 19, 27]. When filled with fluid and surrounded by consolidation, it is more difficult to detect a laceration even with a CT examination. Lacerations may range from a solitary lesion to multiple confluent small ones; these ones, when

surrounded by consolidated parenchyma, are defined as "swiss cheese appearance" [51].

Lacerations have been classified into four types according to the mechanism of injury, the morphological pattern, and their localization [35]:

Type 1: Compression rupture injury is centrally located, can become very large, and is produced by compressing or tearing off the lung parenchyma from the adjacent tracheobronchial tree (Fig. 8.19).

Type 2: Compression shear injury is produced when the lower lobes are suddenly squeezed against the spine. It is located paraspinally and may be tubular in morphology (Fig. 8.20).

Type 3: Rib penetration tear is peripherally located, is small and round, and is very often multiple and associated with pneumothorax and subcutaneous emphysema (Figs. 8.21 and 8.22).

Type 4: The adhesion tear is seen adjacent to a previous pleuropulmonary adhesion and is

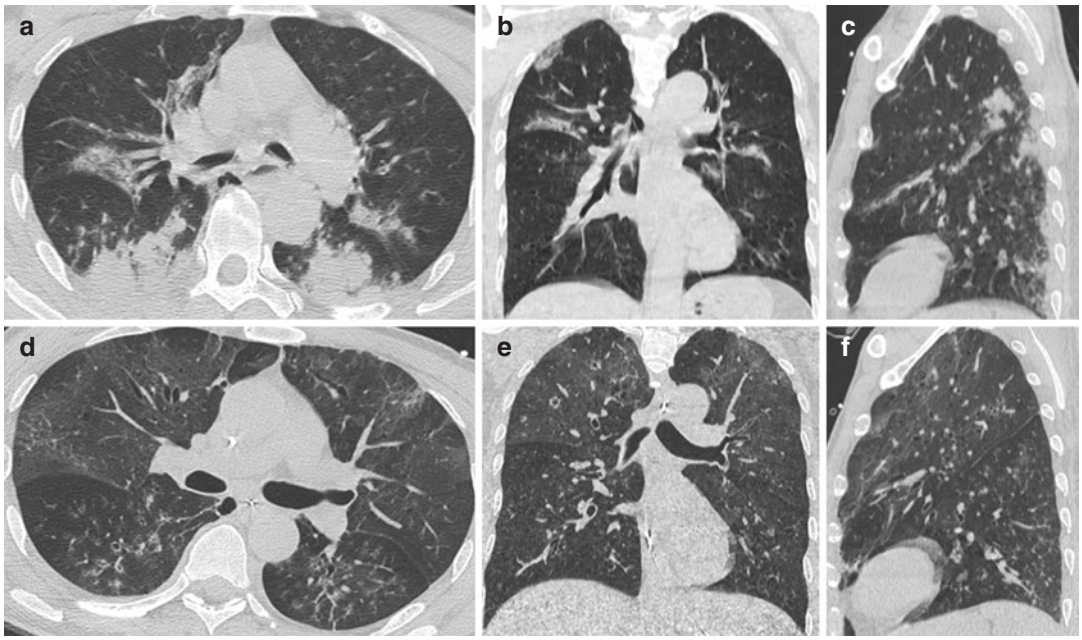

Fig. 8.18 Complication of a pulmonary contusion. (**a**–**c**) Post-traumatic CT-scans show bilateral areas of contusion. Although the relative clarification of contusion areas in the posterior regions, we can appreciate the emerging ground glass alteration of the entire parenchyma at the control CT-scan in (**d**–**f**), acquired 1 week after the hospital admission. The patients experienced a serious ARDS despite the rapid and appropriate respiratory support

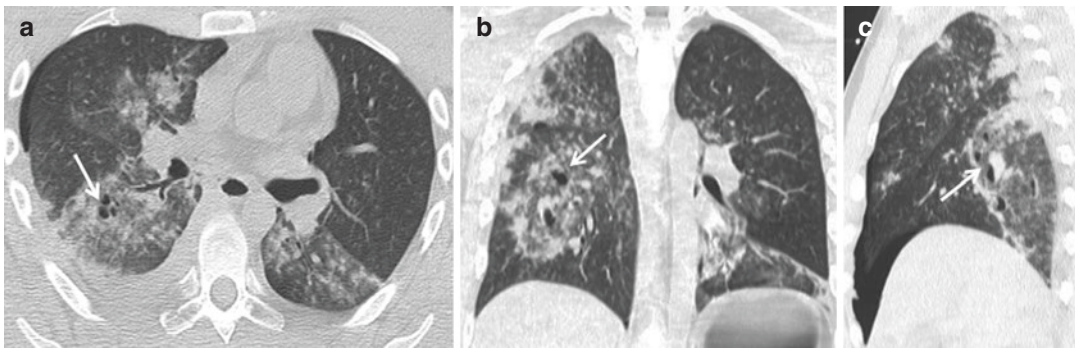

Fig. 8.19 Type 1 lacerations (*arrows*). In (**a**–**c**) are centrally located, surrounded by extensive contusion areas and without any rib fracture visible nearby. Note the ipsilateral hemopneumothorax

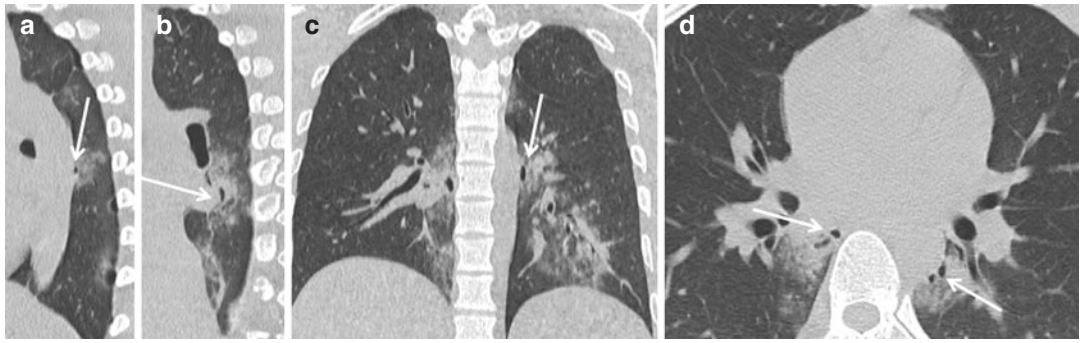

Fig. 8.20 Type 2 lacerations: these injuries (showed in **a**-**b**-**c**-**d**) are produced when the lower lobes are suddenly squeezed against the spine. These lacerations are generally paraspinally located (*arrows*) and bilateral

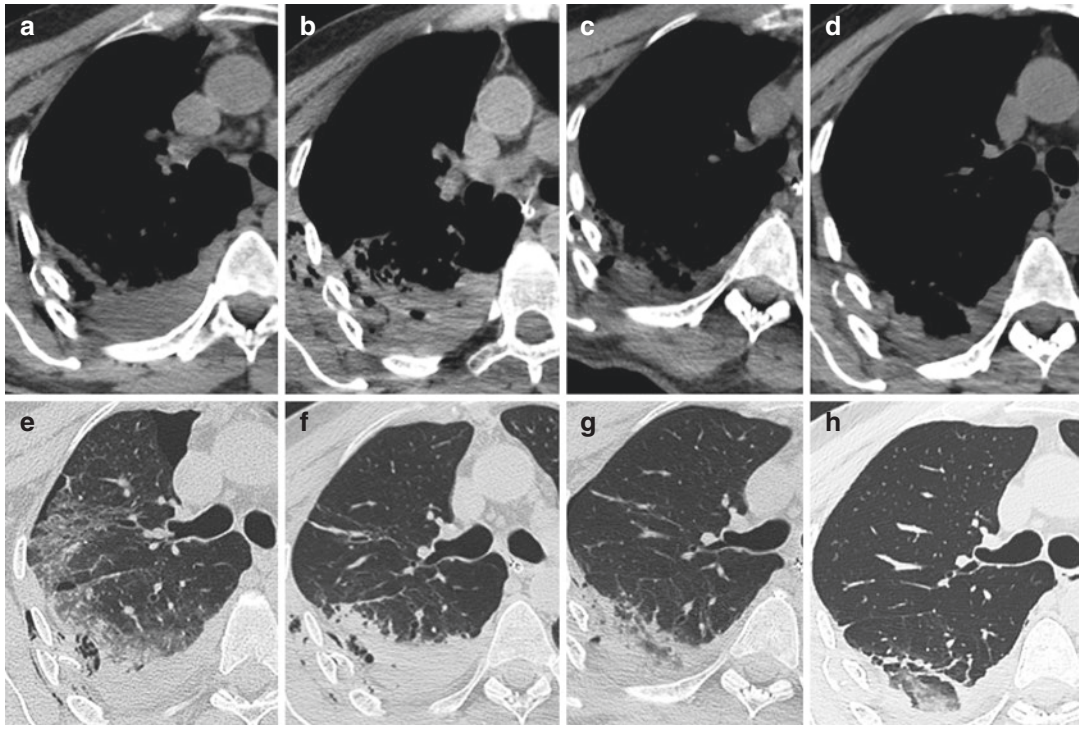

Fig. 8.21 Axial CT-images show contusions and type 3 lacerations (*arrow*). Immediately after trauma (**a**–**e**) the lacerations are small peripheral and associated with local rib fractures, pneumothorax, and chest wall emphysema. In the following controls after 1 (**b**–**f**), 4 (**c**–**g**), and 10 days (**d**–**h**), the lacerations are evolving: become increasingly filled with blood or fluid, so less appreciable (**c**–**g**) and regress slowly, often ending in a residual scar

almost always seen at surgery or at autopsy, not recognizable in MDCT images.

Lacerations resolve more slowly than contusions, and clearance may take months. Over time, they become increasingly filled with blood/fluid and then regress, often ending in residual scarring [52] (Fig. 8.23). Complications of laceration are uncommon and can be: pulmonary abscesses, bronchopleural fistula, or very large pneumatoceles generated by a ball-valve mechanism [19, 28].

Intrapulmonary hematoma is a special kind of laceration consisting in the presence of an

organizing hematoma inside the rounded cavity of lacerated parenchyma. It is well depicted with CT that shows a solid rounded consolidation with high density values at the direct examination and no significant increase in Hounsfield Units after administration of contrast media (Fig. 8.24).

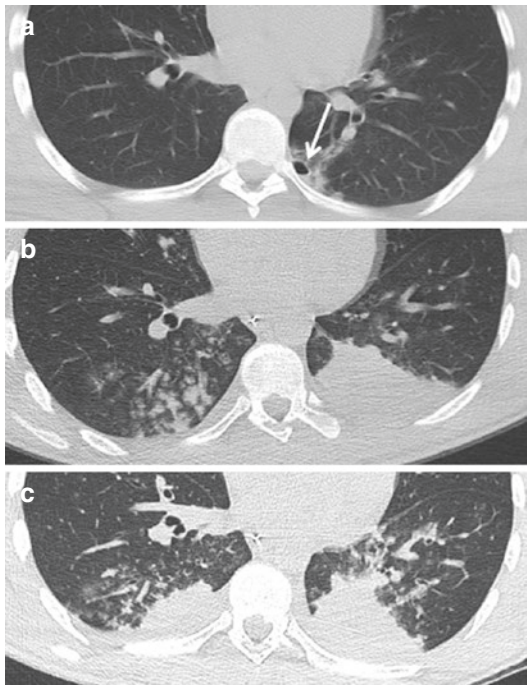

Fig. 8.22 Left lower lobe contusion associated with a small type 3 parenchymal laceration (*arrow*) in a young boy: axial CT-scan immediately after trauma (**a**). Images in (**b**, **c**) show the CT controls after 5 and 10 days: the progressively filling of the small laceration with fluids does not permit its depiction in the successive CT controls

8.4.3 Pulmonary Hernia

Traumatic lung herniation occurs when a pleura-covered part of the lung extrudes through a traumatic defect in the chest wall [2]. It is generally an acquired condition, mainly caused by trauma (52% of cases), which is more often a penetration than a blunt injury [53]. Any chest trauma may cause bones or joint disruption, resulting in acquired chest wall defects and allowing lung herniation; however, lung protrusion is more likely when the trauma causes a sudden marked increase in intrathoracic pressure; many ribs are broken or a focal blunt trauma occurs [53].

Generally, anterolateral chest wall is more susceptible to traumatic lung herniation because of the lesser soft tissue support compared to the posterior wall. The first, in fact, relies only to intercostal muscles while the latter is supported also by the trapezius, latissimus dorsi, rhomboid, and paravertebral muscles [50]. Herniation caused by penetrating trauma occurs, naturally, at the site of the wound [53].

In case of lung hernia, symptoms are aspecific including pain, coughing, hemoptysis, and breathlessness while chest examination may reveal a soft mass that moves paradoxically with respiration and/or is more evident in Valsalva maneuver [53]. Sometimes, lung hernias can remain asymptomatic and uncomplicated and presentation can be delayed. Delays of a few months to 40 years have been reported [54].

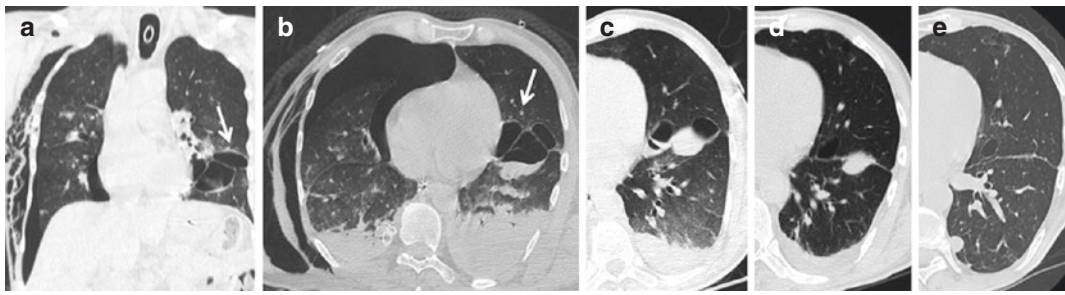

Fig. 8.23 Multiple parenchymal lacerations (*arrows*): coronal (**a**) and Axial (**b**) CT-scans done immediately after trauma; note the large subcutaneous emphysema and ipsilateral pneumothorax. Controls after 10 days (**c**), 3 months (**d**), and 1 year (**e**). Note the progressive filling of the cavities and their slow involution in irregular residual scars

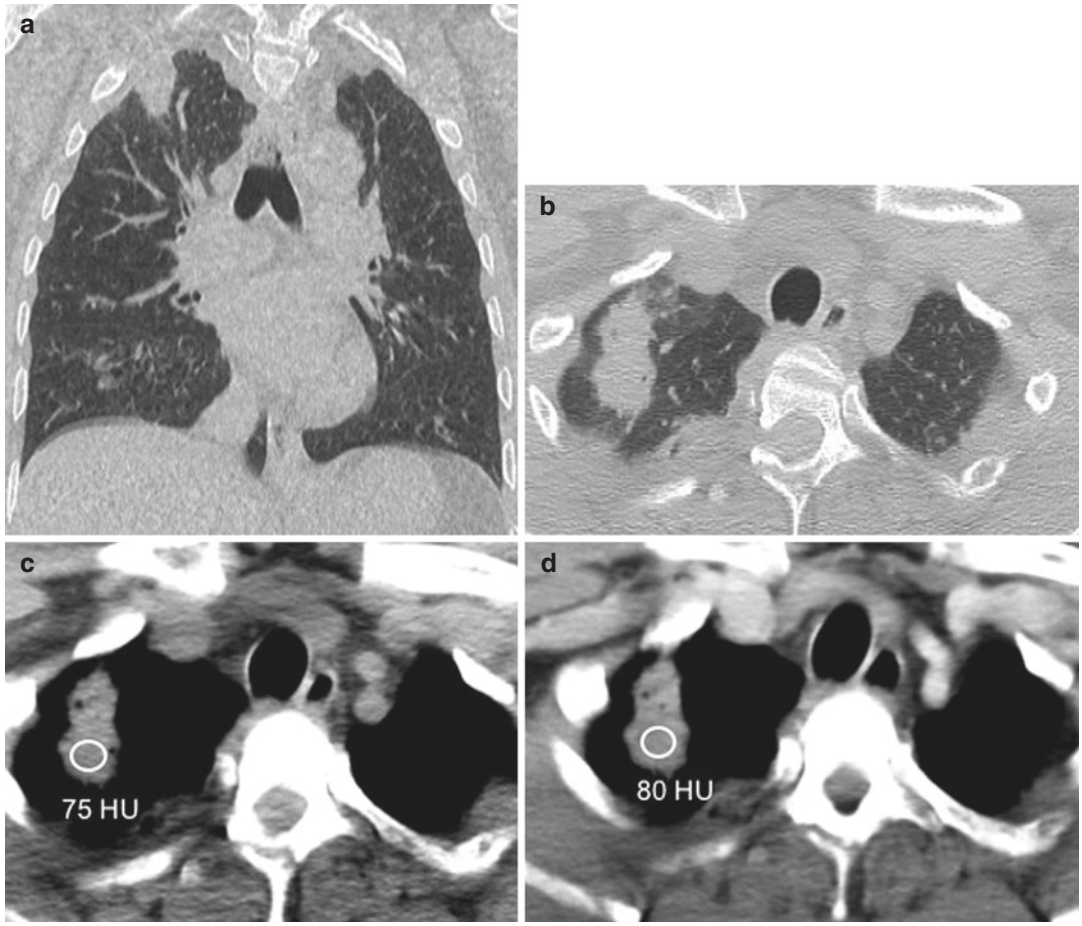

Fig. 8.24 Intrapulmonary hematoma: it is an organizing hematoma inside the rounded cavity of lacerated parenchyma (**a**, **b**). CT-scan shows a solid rounded consolidation with high density at baseline (**c**), and no significant increment after administration of contrast media (**d**)

Lung hernia, when symptomatic, may be a life threatening condition as can be itself complicated with tension pneumothorax or strangulation of the protruding lung and as it is usually associated with other severe injuries of thoracic structures, due to the high energy mechanism that implies this event [50].

Plain chest may see bone or joint rupture [53]. Sometimes, owing to the opacity difference between herniated lung and surrounding soft tissues, the hernia itself may be apparent [7, 53] (Fig. 8.25). Chest X-ray acquired during the Valsalva maneuver have also been demonstrated to be of value for diagnosis because of the increasing hernia volume [54]. Utilization

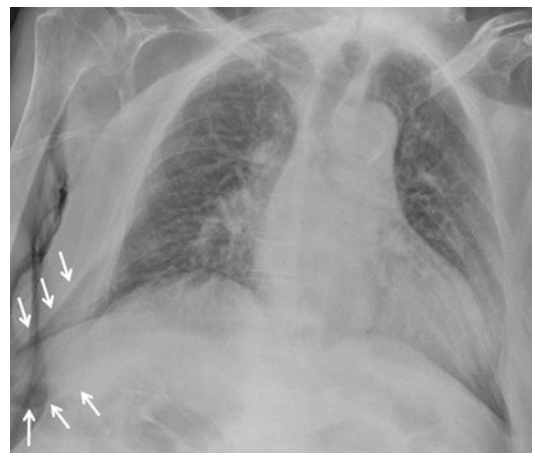

Fig. 8.25 Suspect of right basal lung herniation (*arrows*) in the chest X-ray done immediately after trauma

of plain radiographs is not recommended when a CT-scan can be rapidly performed [53].

MDCT can easily depict the herniated lung portion and evaluate the adjacent altered thoracic wall structures [50] (Fig. 8.26). Furthermore, during the contrast-enhanced phase, MDCT enables to suspect a strangulation of the herniated region when we do not find any opacification of the herniated lung vessels [53] (Fig. 8.27). Owing to the panoramic visualization of all thoracic structures, MDCT can also depict pneumothorax, pneumomediastinum, and hemothorax, which are often associated, and permits to evaluate any other injured thoracic structure. Minimum intensity projection reconstructions may be of value to the cardiothoracic surgeon managing the patient, particularly with regard to their surgical strategy [53] (Fig. 8.28).

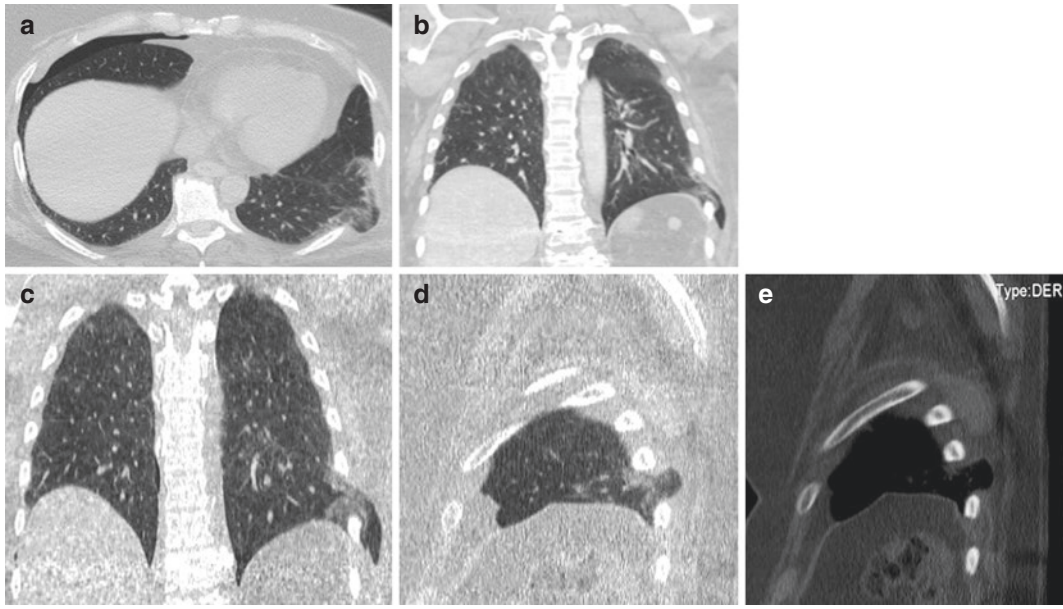

Fig. 8.26 Lung herniation of the posterolateral chest wall. Axial (**a**), coronal (**b**, **c**) and sagittal reconstructions, the latter with both lung (**d**), and bone (**e**) window. While the lung window allows to depict and localize the herniated lung, the bone window provides optimal visualization of the rib cage. As we can see, the hernia wasn't associated to any rib fracture

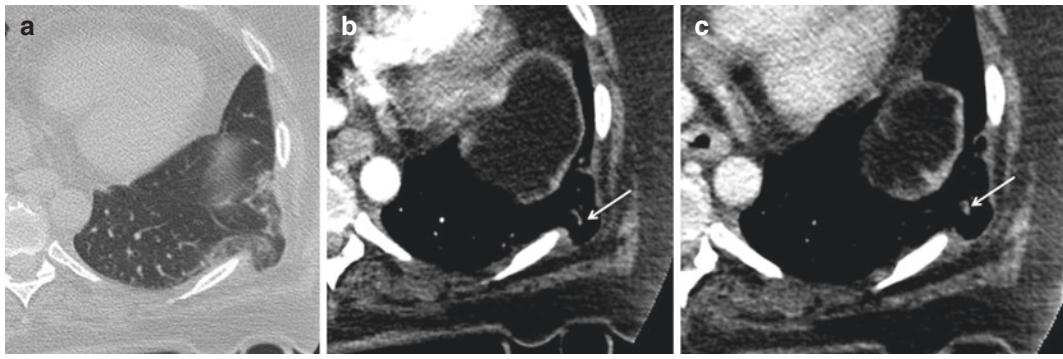

Fig. 8.27 Same patient as Fig. 8.25. Axial CT-scans after administration of contrast media (**a**–**c**). We can appreciate the lack if opacification on arterial (**b**) and venous (**c**) vessels of the herniated portion of the lung, suggesting the possibility of its strangulation (*arrows*). In the lung window image in (**a**), the protruding lung shows subtle increment in density as in aspecific suffering

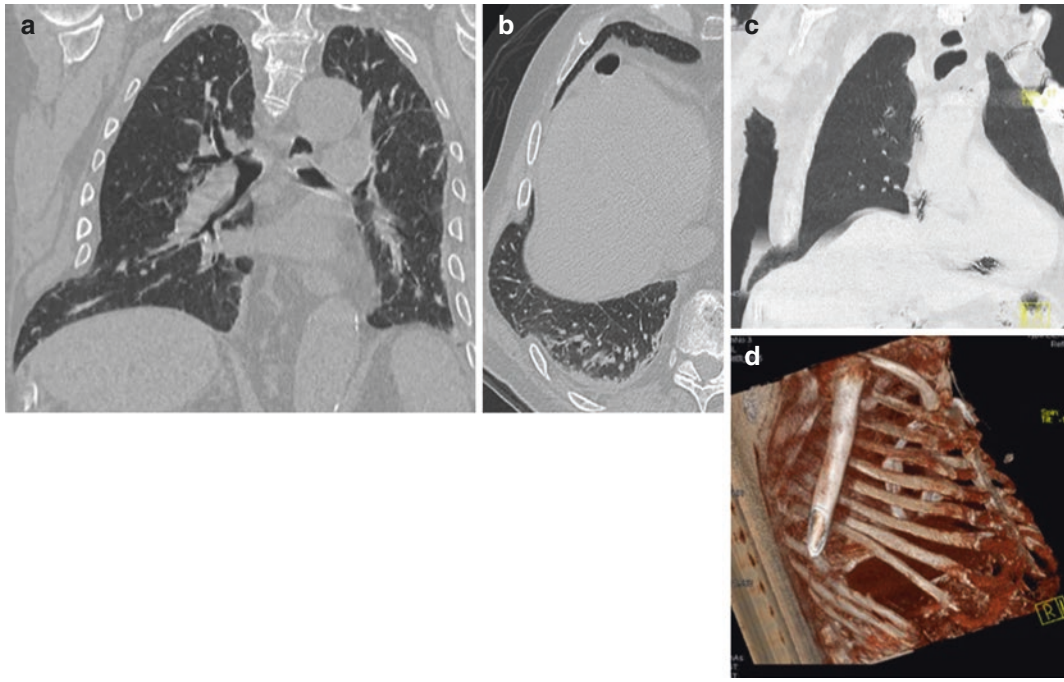

Fig. 8.28 Same patient as in Fig. 8.26: CT confirms the herniation and enables to evaluate the chest wall disruption: coronal (**a**), axial (**b**), minimum intensity projection (**c**), and 3D reconstructions (**d**)

As lung herniation is a rare condition with a wide spectrum of causes, severities, associations, and complications, some of which are life threatening, it is important for the radiologist to recognize the condition as fast as possible [27]. This is particularly true in dyspnoic or hypoxic patients as herniation may increase with positive-pressure ventilation [50, 53].

8.4.4 Open Trauma

Although blunt trauma accounts for approximately 70% of trauma cases, penetrating trauma should not be overlooked as a cause of significant morbidity and mortality. In fact, gunshot wounds are the second leading cause of injury death after motor vehicle crashes in the United States [55]. Overall, the exact incidence of penetrating chest injury varies according to the geographical location with incidence ranging from 1 to 13% of all trauma admissions [56]. These injuries are frequently serious and, particularly in gunshot wounds, any region of the chest may be involved. Also, as many as 20% of open thoracic trauma will have associated abdominal injuries [56]. Generally, less than one-third of knife wounds and approximately one half to two-thirds of gunshot wounds in stable patients will require surgical repair [5] and, in unstable patients (event not rare due to the frequent involvement of vascular structures along the stab trajectory), an acute exploration is required in 5–15% of cases [56].

The lung parenchyma and the pleural sheets are involved in the great majority of open thoracic traumas. From 88 to 97% of penetrating chest injuries involve the chest wall, pleura, or lung [5] and the tissue damage has the same anatomo-pathological characteristics as in blunt trauma. There are, however, peculiarities in imaging of penetrating trauma, especially in gunshot injuries, that are worth considering by the emergency radiologist.

Penetrating trauma can be divided into ballistic and non-ballistic. The first cause tissue disruption and laceration along their trajectory and damage tissue nearby their entrance [55]. In the second, we can identify at least three major

mechanism of disruption: the direct tissue laceration occurring along the bullet trajectory, as in non-ballistic wounds (called permanent cavity); the damage caused by the pressure gradients resulting in tissue separation in a radial direction along the bullet trajectory (called "temporary cavity"); and the damage obtained by the ballistic pressure preceding the bullet [55]. While the term "temporary cavity" refers to the effect of bullet shockwaves on gelatin in experimental studies, in vivo it is usually referred to the "concussion zone" located radially with respect to the bullet path [55]. So, the wound is not in a linear shape (as in stab wounds) but involves also tissues around the trajectory. The degree of injury will depend on how the projectile transfers its kinetic energy into the target tissues. In vital organs, the transfer of energy is influenced by the velocity of the projectile, the bullet shape and design, and the intrinsic tissue characteristics. Experimental studies demonstrated that the temporary cavitation size becomes more significant at higher velocities with almost an exponential relationship between the bullet velocity and the cavity size [55]. Regarding tissue characteristics, damage increases with tissue density and decreases with tissue elasticity [5]. As lung has got more elasticity than abdominal organs (as liver, kidney, or spleen), it can better absorb the perforating forces thus experiencing less damage from the same ballistic trauma [55]. So, while renal, hepatic, and splenic parenchyma show greater degrees of tissue destruction and fragmentation, a surrounding contusion around the bullet path in the highly elastic lung is often the most commonly visible injury of the "concussion zone" [5].

The clinical presentation of a patient with penetrating trauma is highly variable depending mainly on two interrelated factors: the hemodynamic stability and the mechanism and location of the wound and can range from a stable to a comatose condition [55].

Although from 88 to 97% of patients who are admitted with penetrating injuries to the chest have involvement of the chest wall, pleura, or lung, it has been demonstrated that up to 62% of patients are asymptomatic and have normal chest radiographs [57]. Furthermore, other studies and case series reported rates of delayed pneumo- or hemothorax of 8–12%, after a negative admission chest X-ray in asymptomatic patients with penetrating thoracic wounds [58]. In order to prevent this, many studies explored the utility of sequential chest X-rays acquired during the first hours after the patient's admission. In particular, a 2013 study based on a population of 88 patients with thoracic penetrating injuries suggested a hospitalized observational period in asymptomatic patients with normal initial chest radiographs, with repeat chest X-ray, at intervals approaching 1 h, for at least 3–6 h [58]. As a matter of fact, already in 2007, Magnotti et al. [59] demonstrated that after an initial chest CT there was no need for repeat chest radiograph after penetrating thoracic trauma. Actually, chest X-ray in penetrating thoracic traumas, as in blunt trauma, should be considered a helpful tool in the prior evaluation of patients and provides, along with bedside US, immediate useful information in the unstable patient as it rapidly recognizes hemothorax and pneumothorax [3].

The evidence for the use of chest US in the evaluation of thoracic penetrating trauma is more limited than that for blunt trauma [60]. Current literature, however, demonstrates that US is a valid screening tool for penetrating thoracic injuries with a high sensitivity for detecting injury requiring acute intervention [60] as hemothorax, pneumothorax, and pericardial effusion.

In particular, since 1997, it has been demonstrated the high sensitivity of US in detecting hemothorax in penetrating thoracic injury compared to CT and tube thoracostomy as gold standard [30]. In this study was found a sensitivity and specificity of US of 100% in detecting hemothorax in 18 patients with thoracic penetrating trauma [30].

Other studies have also evaluated US for the depiction of pneumothorax in penetrating trauma, and recently Ku et al. [61] compared, in 47 patients, thoracic US for pneumothorax with computed tomography, tube thoracostomy, and supine chest radiograph followed by clinical observation. In this case report, US demonstrated a sensitivity of 57% and a specificity of 99% with

respect to the CT examination [61]. The lack in sensitivity may be explained by the high "operator dependence" of US as this study was performed with many operators, each of them with different experience [61].

Finally, we must not forget that, in penetrating traumas, US is a valuable instrument to detect also a pericardial effusion. In 2015, in a large cohort of injured patients, US demonstrated a sensitivity of about 86% [62].

MDCT has certainly become the modality of choice in stable patients with penetrating torso trauma [63]. The mayor utility of MDCT in penetrating trauma relies in its capability to detect the trajectory of non-ballistic and ballistic wounds, the so-called CT trajectogram [5]. This is particularly true since the advent of multidetector technology which permits to achieve rapid isotropic acquisitions and, thus, to perform multiplanar and 3D reconstructions,

It has been demonstrated that the depiction of the wound trajectory allows a better management of the patient in the acute setting, reducing the frequency of diagnostic surgery, and has got forensic utility as it provides valuable data regarding the dynamic of the traumatic event and the nature of the injuring object [5].

Non-ballistic wound trajectories are often not entirely evaluable, even with MDCT, as tissues tend to return to the one adjacent to the other after releasing the stab [5] (Fig. 8.29). Ballistic wounds path, on the other hand, are better depicted as

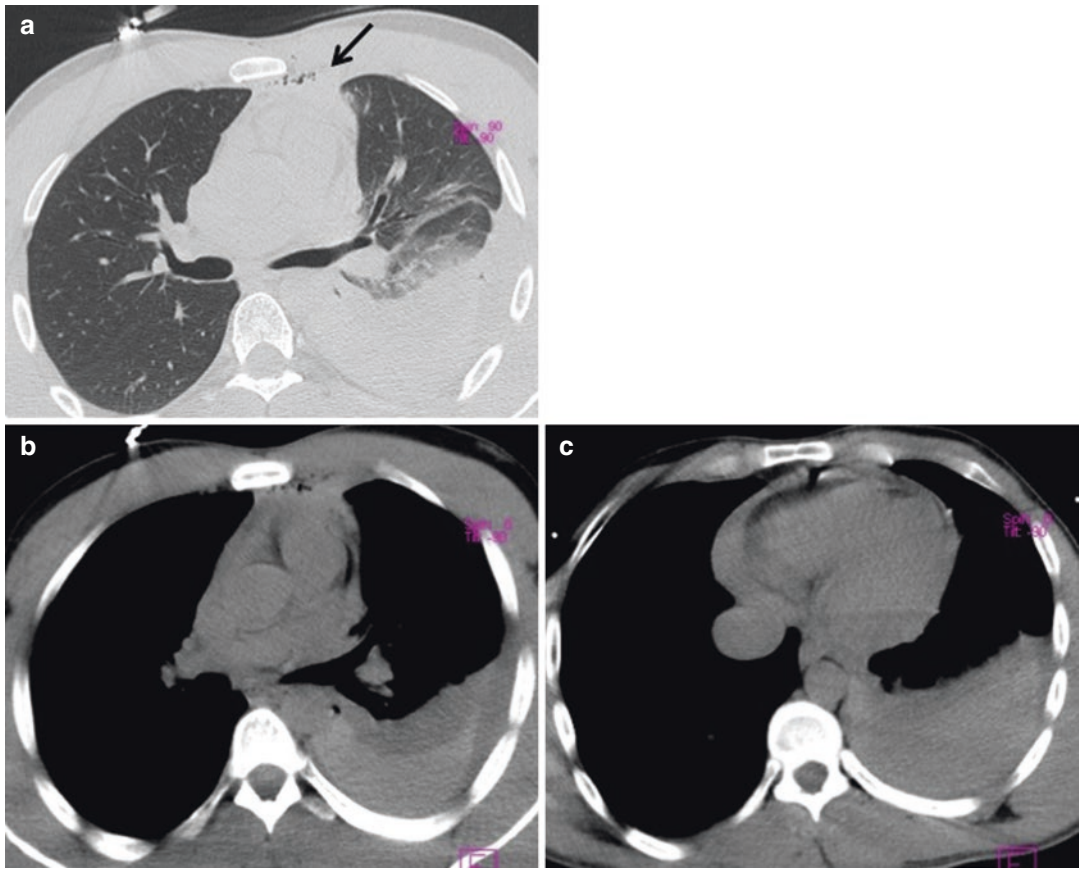

Fig. 8.29 Young man stabbed to the anterior left chest wall. In (**a**) (lung parenchyma window), it is possible to notice the small air bubbles behind the sternum (*arrow*), an indirect knife-penetration sign. We have no more appreciation of the stab trajectory but have indirect signs of pericardial and pleural disruption in the axial CT-scans with smooth tissues window (**b**, **c**) that show left hyperdense pleural and pericardial effusion, signs of left hemothorax and hemopericardium

there is a greater tissue disruption as the damage is not only along the wound tract but also in surrounding tissues. Furthermore, since MDCT has high sensitivity in finding and exactly localizing metallic external fragments, air, hemorrhage, and small bone or bullet fragments (lead dust) along the wound track, it allows better identification of the course of the bullet compared to a stab wound [28] (Fig. 8.30). Unfortunately, pitfalls may occur, especially in cases of multiple penetrating injuries, or when there is not a linear trajectory, for example when projectiles or stab are rebounded off or deviated by bony structures [5]. It is important, in order to reduce pitfalls, to use more than one window setting in order to adequately identify CT trajectograms in the thorax. In fact, it is suggested to use wide CT windows in order to identify the exact wound entry site in the subcutaneous fat, as well as the intrapulmonary path and the bone damage, while we will need a "soft tissues" CT window to identify the relationship of the bullet path with the mediastinal structures [28] (Fig. 8.31).

As just said, since 88–97% of penetrating wounds involve the chest wall, pneumothorax, and hemothorax are the most common complications following these events. In particular, in penetrating traumas, open or communicating pneumothorax occurs, also called "sucking chest wound," which develops when the skin and pleura are both injured [7]. This kind of pneumothorax may more easily develop in tension pneumothorax (up to one-third of patients) [5]. Air location in the pleural space, as in blunt traumas, depends mainly on the patients' position and only rarely on the site of laceration or on previous or newly generated pleural adhesions.

Hemothorax is more frequently seen in open injuries than in blunt traumas [5]. It is usually the result of a laceration or contusion of lung parenchyma (associated to a disruption of the visceral pleura), or of diaphragm, internal mammary, and intercostal vessels, heart or great vessels. Also, more frequently than in blunt trauma, there can be active bleeding that can became rapidly massive and life threatening [5].

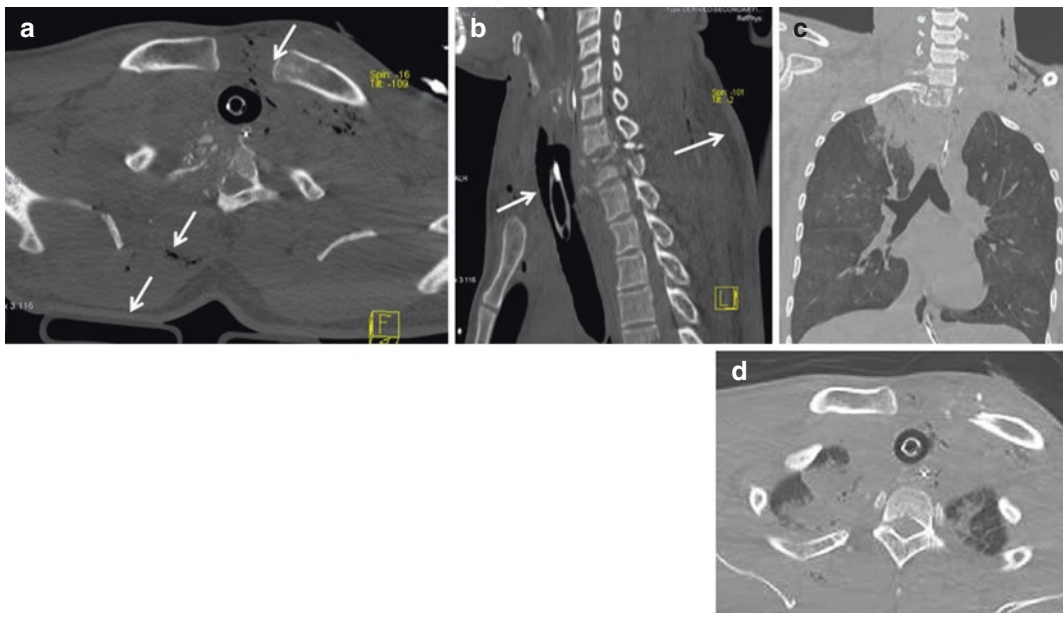

Fig. 8.30 The wide disruption of soft tissues and of bones permits to depict quite exactly the projectile trajectory along the cervicothoracic outlet (*arrows*). In these axial (**a**–**d**), sagittal (**b**), and coronal (**c**), CT-scans we appreciate that the bullet entered from the anterior left jugular region and emerged from the right suprascapular region, crossing the first thoracic vertebral body. Note that, although not directly involved, right lung apex shows a wide contusion area related to the "concussion zone" (**c**, **d**)

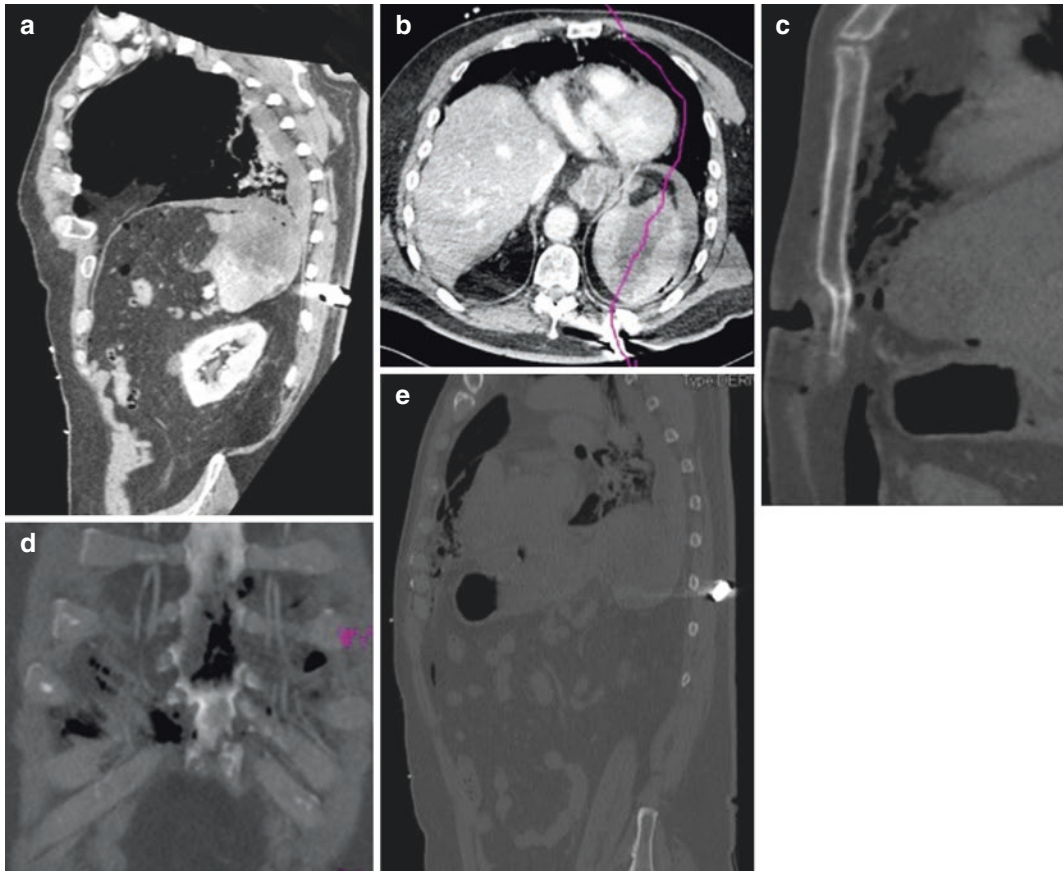

Fig. 8.31 CT trajectogram. CT allows to depict the trajectogram using multiple window levels and multiplanar reconstruction (**a**–**e**). If the trajectory is not linear, as often occurs due to the different densities of crossed tissues, curved reconstruction along the bullet pathway (**a**, **b**) allows to evaluate correctly all the involved organs in just one image. More wide windows width allows to highlight the bullet entry hole in the subcutaneous fat and in the sternum and the bullet itself, in this case, stopped in the posterior chest wall (**e**)

As already stated, compared to many solid organs, lung is more elastic and is less damaged by bullet wounds and the extent of parenchymal damage depends on the energy of the projectile.

Lung contusion is the main injury after penetrating chest trauma as it occurs along the wound track and in the concussion zone as a result of microscopic disruption of small vessels and alveolar walls which lead to hemorrhage into the parenchyma at the time of trauma, followed in 1–2 h by interstitial edema, which peaks 24 h after injury [28].

Laceration are the most frequent lung injury seen along the wound trajectory and, in penetrating traumas lacerations are much more frequent than in blunt ones [5].

Due to the increase in temperature in lung parenchyma along the bullet path, caused by the high energy released, blood in lacerated parenchymal cavities may tend more frequently to coagulate and pulmonary hematomas are more easily seen.

In all these injuries, MDCT multiplanar images are very useful not only to depict the wound trajectory and to evaluate the site and extent of lung damage [28].

Finally, we must not forget that in thoracic penetrating wound, very often is involved the abdomen and MDCT permits, in a single acquisition, a view of the whole body [5] (Fig. 8.32).

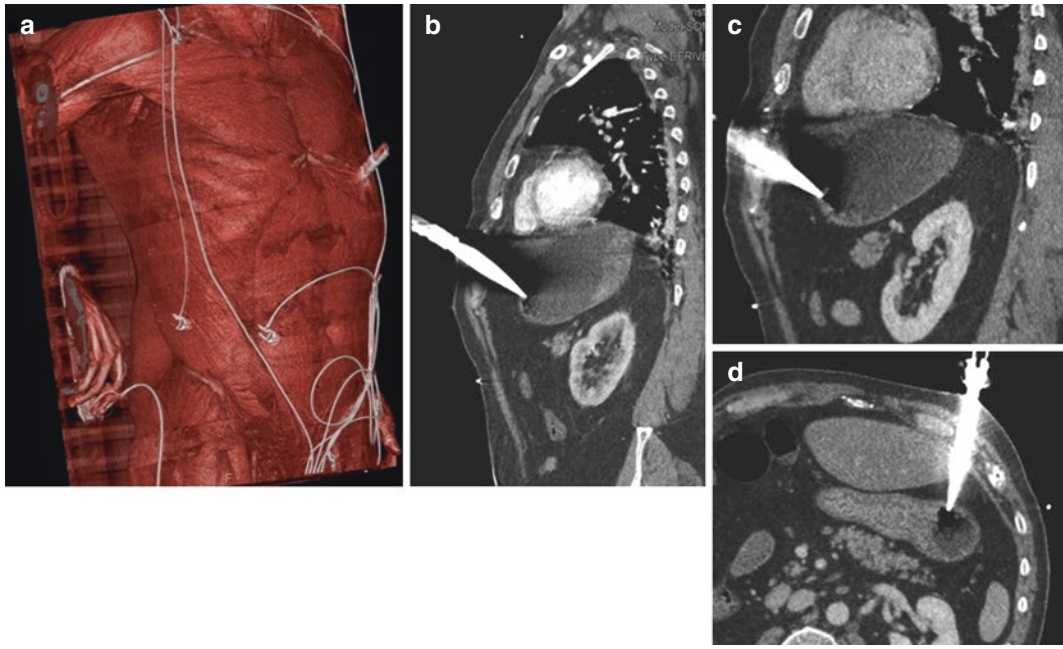

Fig. 8.32 Computed tomography allows to have multiplanar reconstruction to better visualize the penetrating trauma. More than the 3D spectacular reconstruction (**a**), are useful the sagittal (**b**), and the oblique (**c**, **d**) multiplanar imagines that allow to depict the transdiafragmatic trajectory of the stab and the small lesion of the gastric wall and of the left hepatic lobe

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