European Journal of Radiology Open 7 (2020) 100274

Contents lists available at ScienceDirect

European Journal of Radiology Open

journal homepage: www.elsevier.com/locate/ejro

Pulmonary Edema: A Pictorial Review of Imaging Manifestations and Current Understanding of Mechanisms of Disease

Maria Barile

Department of Radiology at University of Massachusetts Memorial Medical Center, University of Massachusetts Medical School, Worcester, MA, United States

ARTICLE INFO

Keywords:
Pulmonary edema
Chest radiograph
CT
Hydrostatic edema
Permeability edema
ARDS

ABSTRACT

Pulmonary edema is a common clinical entity caused by the extravascular movement of fluid into the pulmonary interstitium and alveoli. The four physiologic categories of edema include hydrostatic pressure edema, permeability edema with and without diffuse alveolar damage (DAD), and mixed edema where there is both an increase in hydrostatic pressure and membrane permeability. As radiographic manifestations and etiologies are varied, an appreciation for both the common and uncommon manifestations and causes of pulmonary edema is essential for accurate diagnosis.

1. Introduction

Pulmonary edema is one of the most common entities encountered on routine chest imaging in both the inpatient and outpatient settings. It is caused by the extravascular movement of fluid into the pulmonary interstitium and alveoli. Radiographic manifestations of this entity are varied, and accurate radiographic diagnosis is essential for proper management of this common condition. Additionally, understanding the cause of these fluid shifts is critical to patient management and radiographic assessment can help in distinguishing among the various causes.

The physiologic determinates of edema include 1) hydrostatic pressure, defined as the pressure within the capillaries driving fluid out of the vessels, 2) oncotic pressure, described as pressure related to the macromolecules in the blood which help to retain fluid in the vessels, and 3) membrane permeability, the ease with which fluid passes through the capillary or alveolar walls (Fig. 1). The relative balance of the hydrostatic and oncotic pressures in addition to the membrane permeability determine the net fluid movement between the vasculature and the pulmonary interstitium and alveolar air spaces. The lymphatic channels are also important in maintaining fluid balance as they allow for return of the extravascular fluid back into the central vasculature. The lymphatic channels run along the bronchovascular bundles and interlobular septa along the periphery of the pulmonary lobule (Fig. 2). The lymphatic flow can increase 3-10x in the setting of hydrostatic pulmonary edema [1], thereby providing an opportunity to re-establish euvolemia.

Pulmonary edema can be classified into four categories based on these physiologic determinates of edema: hydrostatic pressure edema, permeability edema with and without diffuse alveolar damage (DAD), and mixed edema where there is both an increase in hydrostatic pressure and membrane permeability. Left heart failure and volume overload are two common examples of hydrostatic pressure edema. In these settings, the hydrostatic pressure in the capillaries exceeds the oncotic pressure and fluid is driven out of the vasculature into the pulmonary interstitium and eventually into the alveoli through the capillary endothelium and alveolar epithelium. An example of permeability edema with DAD is acute respiratory distress syndrome, a very common entity encountered in the ICU setting in which a robust inflammatory process induces lung injury. In this setting, there is direct injury to the capillary endothelium and alveolar epithelium by numerous different causes including infectious processes, inhaled toxins, or inflammatory mediators arising from a larger systemic insult. Permeability edema without DAD occurs when there are changes in membrane permeability without severe alveolar damage. Examples include "crack-lung" or opioid overdose or, in the setting of high-altitude pulmonary edema, or cytokine administration such as IL-2. Mixed pulmonary edema involves increases in hydrostatic pressure and membrane permeability and can be seen in the setting of severe neurologic injury following abrupt lung re-expansion or lung transplant.

In this article, we will first describe both the common and uncommon radiographic imaging manifestations of pulmonary edema that clinical radiologists should be able to accurately identify on routine CXR and

Abbreviations: CXR, Chest X-Ray; CT, Computed Tomography; DAD, Diffuse alveolar damage; ARDS, Acute respiratory distress syndrome; PE, Pulmonary embolus; PVOD, Pulmonary veno-occlusive disease; PA, Pulmonary artery.

E-mail address: maria.barile@umassmemorial.org.

https://doi.org/10.1016/j.ejro.2020.100274
Received 31 August 2020; Accepted 22 September 2020

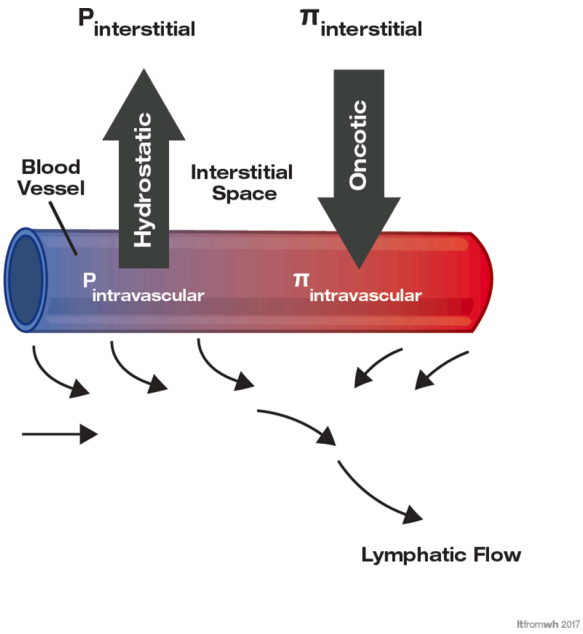


Fig. 1. Schematic representation of the hydrostatic and oncotic pressure gradients which influence fluid movement across the pulmonary blood vessel walls (capillary endothelium).
$P_{intravascular}$: hydrostatic pressure in the vessel.
$P_{interstitial}$: hydrostatic pressure in the interstitium.
$\Pi_{intravascular}$: protein oncotic pressure in the vessel.
$\Pi_{interstitial}$: protein oncotic pressure in the interstitium.

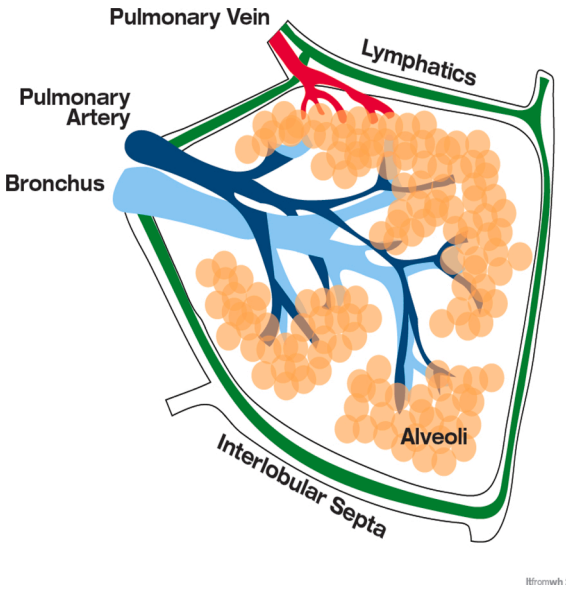


Fig. 2. Drawing of the secondary pulmonary lobule of the lung. The interlobular septa runs along the periphery of the lobule, containing lymphatics. The pulmonary artery, bronchus, and central lymphatics run along the center of the secondary pulmonary lobule.

chest CT. Secondly, we will identify common and uncommon etiologies of pulmonary edema and how to distinguish between these entities. A thorough understanding of the imaging manifestations and causes of pulmonary edema is essential for an accurate diagnosis and subsequent clinical management.

2. Common Radiographic Manifestations of Pulmonary Edema

CXR assessment of pulmonary edema is one of the most commonly performed diagnostic tests and has been shown to correlate with volume status, total blood volume (1–3), and other indicators of heart failure (4). Snashall, et al. demonstrated that changes in water lung volume in animal models as low as 35% can be detected on CXR (5). One of the earliest manifestations of hydrostatic pulmonary edema on CXR is enlargement of the vascular pedicle width (Fig. 3), defined as the superior mediastinum just above the aortic arch, and cephalization of pulmonary vessels [2,3]. Cephalization is defined as a redistribution of blood into the upper lobe vessels and can be diagnosed when the upper lobe veins are the same or larger in diameter relative to the lower lobe veins (Fig. 4). Both cephalization and vascular pedicle enlargement are manifestations of pulmonary venous hypertension. While these findings are commonly seen in hydrostatic pulmonary edema, they not commonly seen in permeability edema.

As the hydrostatic pressure increases to 20-25 mmHg, fluid is driven from the intravascular space into the surrounding interstitium. The pulmonary interstitium expands and has several manifestations on CXR, including thickening of the interlobular fissures, peribronchovascular cuffing and blurring or indistinctness of the pulmonary vessel walls (Fig. 4). Peribronchial cuffing is best seen centrally, where the airways are larger and the cuffing will be more pronounced due to increased compliance of the interstitium around the central vasculature [4]. Flooding of the interstitium affects not only the peribronchial interstitium within the center of the secondary pulmonary lobule, but also the interstitium around the periphery of the secondary pulmonary lobule, generally referred to as the interlobular septal interstitium (Fig. 2). Kerley lines are a manifestation of fluid expanding the interlobular septal interstitium (Fig. 5). Kerley B lines are linear opacities $<$ 2 mm in length, identified peripherally, oriented perpendicular to the pleural surface, representing engorgement of the interlobular septa. Kerley A lines extend obliquely from the periphery towards the hila and are a manifestation of fluid in the anastomotic lymphatics connecting the central peribronchial lymphatics with the peripheral lymphatics running along the interlobular septa.

On CT, vascular engorgement and cephalization are present and may be most apparent on coronal reconstructed images. However, interstitial edema is readily apparent on axial images, manifesting as thickening of the interstitium along the periphery of the secondary pulmonary lobule, also called interlobular septal thickening (Fig. 6). Additionally, the lymphatics running along the bronchovascular bundles also become engorged and manifest as thickening of the bronchovascular bundles (Fig. 8). Eventually, fluid fills the alveoli, which produces ground glass opacity on CT (Figs. 7, 8). As this process continues, frank consolidation may also be seen.

3. Uncommon Radiographic Manifestations of Pulmonary Edema

While the common findings of pulmonary edema are characterized by cephalization of vessels, bilateral symmetric ground-glass opacities, septal thickening, and Kerley lines, other less common manifestations have been described. Asymmetric and/or unilateral pulmonary edema is one such manifestation that has been described in many different settings including decubitus position for long periods of time (Fig. 9), emphysema with bullous disease, severe mitral valve regurgitation, re-expansion pulmonary edema, and pulmonary vein occlusion. In this setting, cross-sectional CT imaging may be helpful to better assess the radiologic abnormalities.

4. Common Etiologies of Pulmonary Edema

The most common cause of pulmonary edema is acute decompensated heart failure causing hydrostatic pressure pulmonary edema.

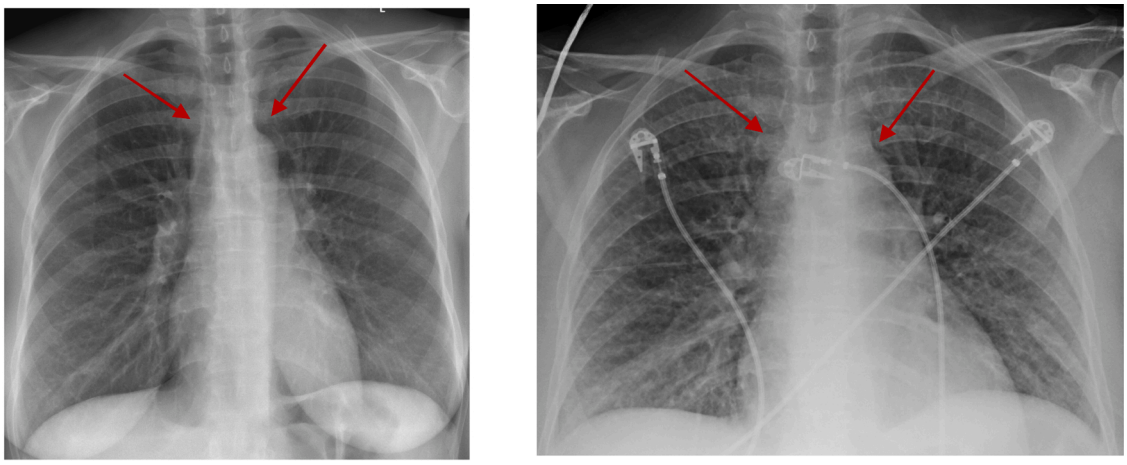


Fig. 3. (a) Euvolemic patient with normal appearance of the vascular pedicle (red arrows). Note distinct vessel margins and smaller caliber vessels in the upper lobe relative to the lower lobes. (b) Same patient 3 months later with new cardiomegaly and mild pulmonary edema with widening of the vascular pedicle (red arrow) and interstitial prominence.

In this form of edema, elevated left ventricular and atrial filling pressures cause enlargement of the vascular pedicle and cephalization of vessels as pulmonary venous pressure increases (Figs. 3,4). Eventually, increases in hydrostatic pressure in the pulmonary vasculature cause fluid to move across the capillary endothelium and alveolar epithelium into the interstitium, causing Kerley lines on CXR and peribronchial cuffing. Finally, in the most severe forms of pulmonary edema, fluid extends into the alveoli (Fig. 7,8) and manifests as increased density on CXR and ground-glass opacity on CT. It is well established that in patients with pulmonary edema, increases in CT Hounsfield unit value correlate with invasive measures of hydrostatic edema such as pulmonary capillary wedge pressures [6]. Pleural effusions and cardiomegaly often accompany the parenchymal manifestations of pulmonary edema secondary to decompensated heart failure.

The rate at which fluid accumulates in the lung interstitium and alveoli is, in part, related to the capacity of the lymphatic vessels to remove the fluid and return it to the central vasculature. This rate varies depending on the acuity of hydrostatic pulmonary edema and the chronicity of elevated intravascular pressures in each particular patient [7]. Because of this, patients with chronic heart failure have chronically

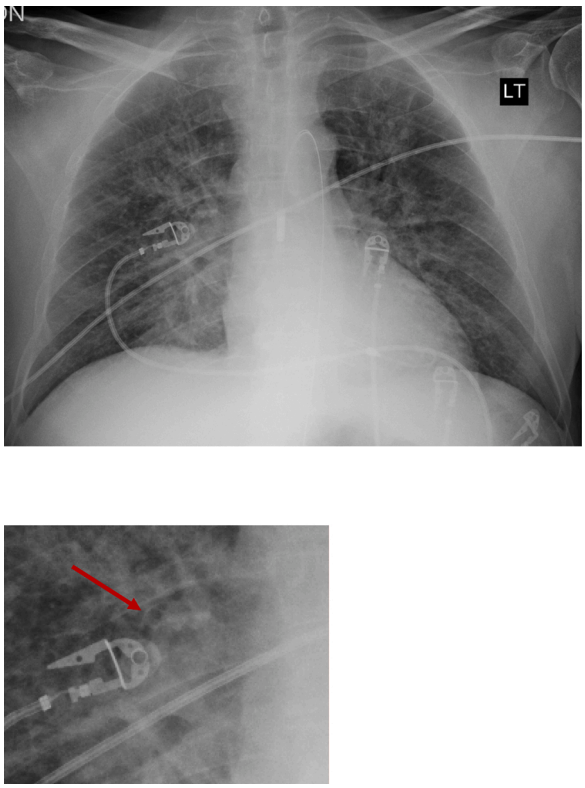


Fig. 4. AP semi-upright portable CXR in a patient with acute onset heart failure secondary to myocardial infarction with a left ventricular assist device in place. There is cephalization and indistinctness of the pulmonary vasculature. Magnified image of the perihilar airways (inset) demonstrates peribronchial cuffing (red arrow).

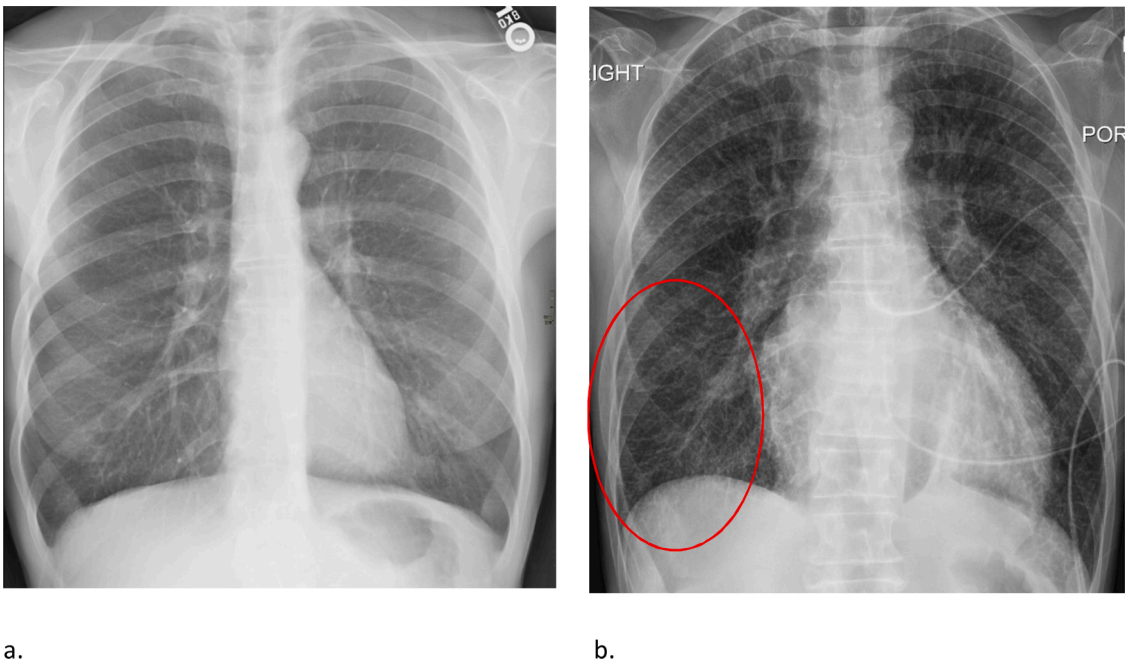


Fig. 5. 59-year-old female with nonischemic cardiomyopathy, EF 10 % in a euvolemic state (a) and with interstitial edema (b) as demonstrated by interstitial prominence, Kerley A and B lines (red circle).

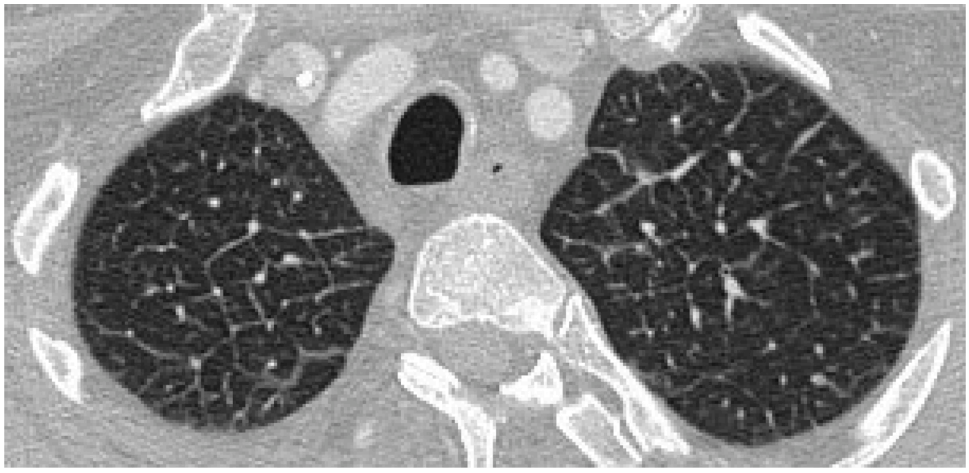
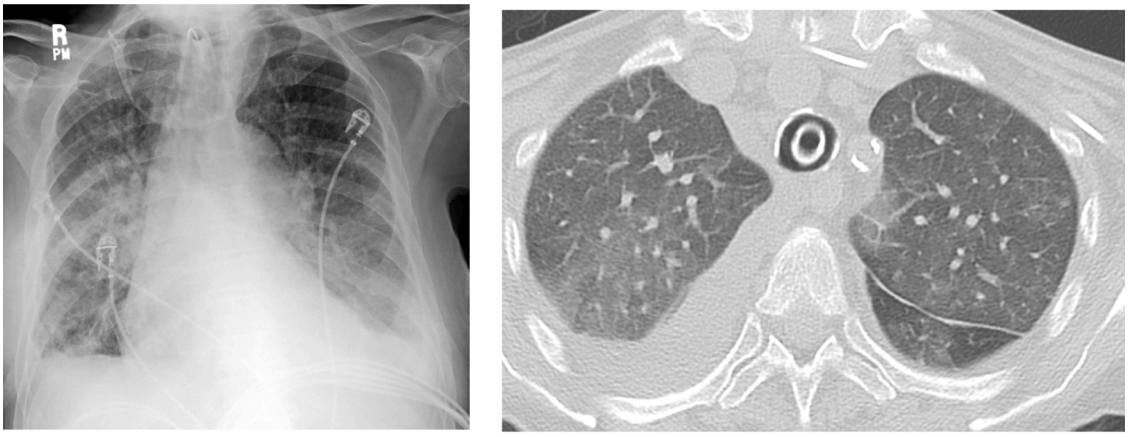


Fig. 6. 71 yo M admitted with sepsis. CT obtained to evaluate for source of infection demonstrates smooth interstitial thickening, ground glass opacities, and pleural effusions (not shown) suggestive of interstitial edema.

elevated cardiac filling pressures and subsequently increased lymphatic capacity and fluid clearance, allowing for the development of pulmonary edema at higher intravascular pressures than patients without chronic heart failure.

Acute respiratory distress syndrome (ARDS) is another common manifestation of pulmonary edema with an estimated incidence of 190,000 cases per year in the United States [8] and many predisposing factors such as trauma or sepsis (Table 1). However, unlike decompensated heart failure, which is a manifestation of hydrostatic edema, ARDS is a manifestation of permeability edema with associated diffuse alveolar damage. ARDS is caused by an acute inflammatory lung injury that allows for increased vascular permeability, causing intravascular fluid to flow through the capillary and alveolar membranes into the pulmonary interstitium and alveoli. Imaging manifestations include ground-glass opacities, consolidation, and septal thickening (Fig. 10). However, cardiomegaly and/or large pleural effusions, commonly seen in the setting of hydrostatic edema, are often absent in ARDS and aid in distinguishing the two etiologies. Additionally, the alveolar ground-glass and consolidative opacities in ARDS often demonstrate a gravitational gradient, with dense consolidation located in the dependent portions of the lower lobes and well aerated lung noted in the anterior/anti-dependent portions of the lung. This gravitational component is attributed in part to atelectasis secondary to the compressive forces of gravity [9] and is helpful in distinguishing ARDS from cardiogenic pulmonary edema.

The permeability edema that is present in ARDS is only one component of the disease. There is also a component of diffuse lung injury at the level of the alveoli mediated by inflammatory cytokines such as IL-1. 6, and 8 [10,11]. These cytokines damage both the capillary endothelium and alveolar epithelium, allowing fluid and proteins to flood the alveoli and cause functional loss of surfactant resulting in alveolar collapse. Therefore, distinguishing between ARDS and pulmonary edema is important as the diffuse alveolar damage component of ARDS can cause permanent fibrosis, including subpleural reticulation



a. b.

Fig. 7. (a) 75 yo M with acute decompensated heart failure with CXR demonstrating cardiomegaly, perihilar opacities and septal thickening with small effusions. (b) CT performed the following day shows ground glass opacities, septal thickening, and increasing pleural effusions, right greater than left.

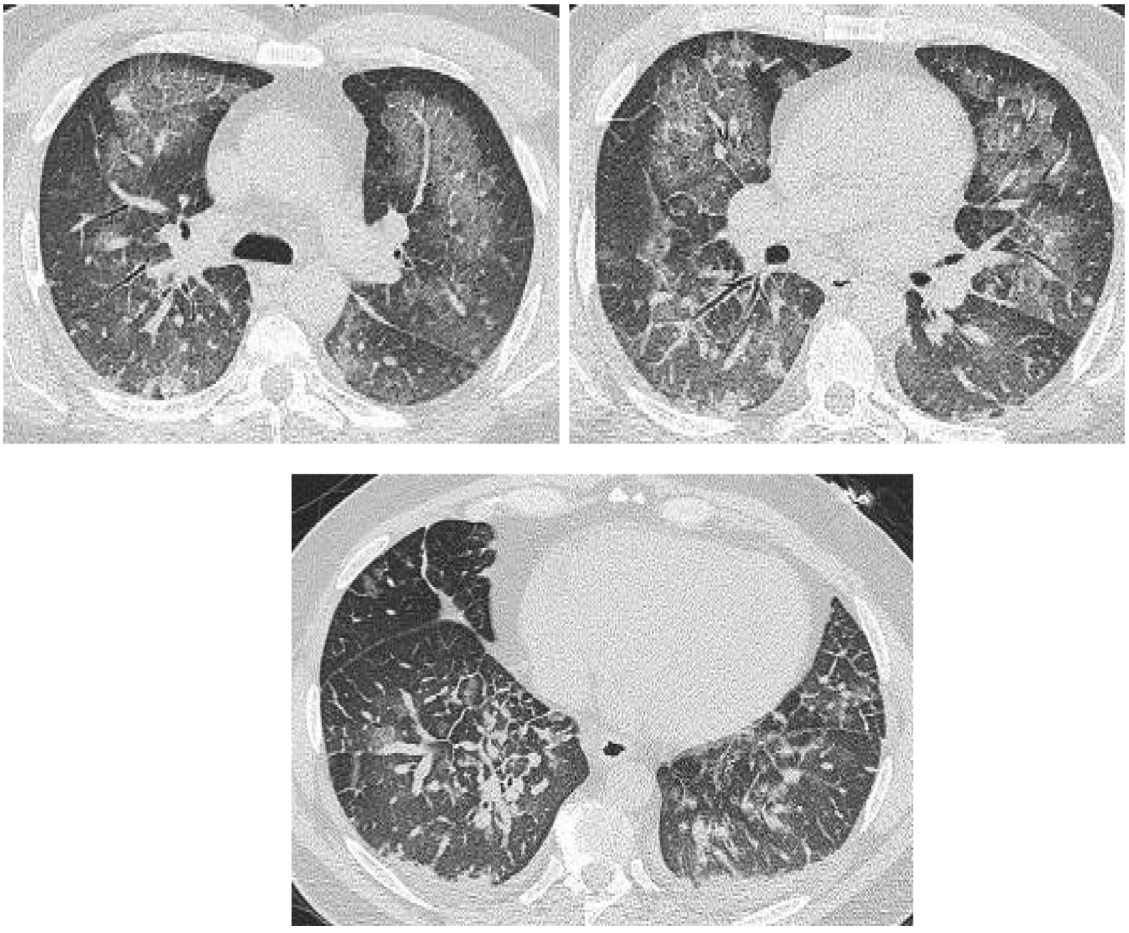


Fig. 8. 65 yo F with CHF and fluid overload following surgery. CT images demonstrate perihilar ground-glass opacities and thickening of the interlobular septa and bronchovascular bundles.

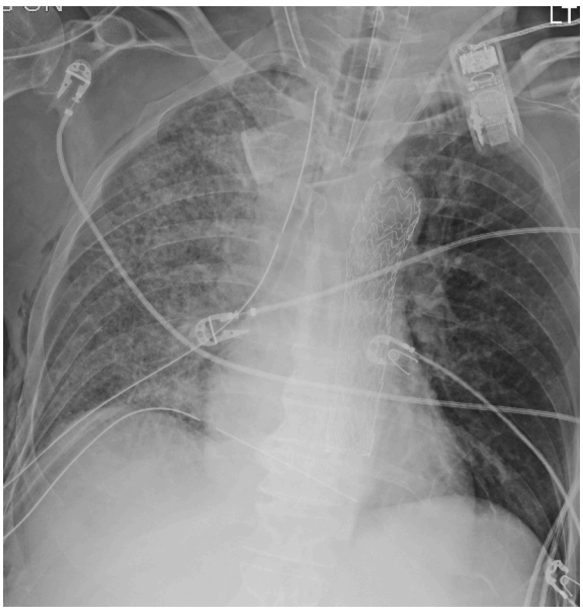


Fig. 9. Asymmetric edema secondary to decubitus positioning after a long surgery characterized by interstitial and alveolar opacities involving the right lung only.

Table 1
Common etiologies of ARDS

Sepsis
Aspiration Pneumonitis
Infectious pneumonia
Trauma
Burns and smoke inhalation
Hematopoietic Stem Cell Transplant
Pancreatitis
Near drowning
Transfusion related acute lung injury
Drugs (chemotherapeutic agents, amiodarone)
Surgery

and bronchiectasis in some patients with severe disease [12–14].

5. Uncommon Etiologies of Pulmonary Edema

Identifying the cause of pulmonary edema is necessary for clinicians to implement the appropriate treatment to ensure a successful recovery. While decompensated heart failure and ARDS are the two most commonly encountered causes of pulmonary edema, there are many other etiologies to be considered (Table 2). Radiographic assessment can often aid in the identification of less common causes of pulmonary edema.

Acute mitral valve insufficiency is one such uncommon cause of pulmonary edema in which imaging plays an important role in diagnosis. In this setting, often seen in acute myocardial infarction or intracardiac tumors, dysfunction of the mitral valve leaflets and/or rupture of the papillary muscles allow for a strong asymmetric regurgitant jet of intracardiac blood to fill the right superior and inferior pulmonary veins. Right-sided pulmonary venous pressures become acutely and asymmetrically elevated, resulting in unilateral or right upper lobe pulmonary edema. As with any type of hydrostatic pressure edema, imaging manifestations range from ground-glass opacity to dense consolidation but are localized in the right upper lobe and remainder of the right lung (Figs. 11,12). The location of the parenchymal manifestations of edema aid in the diagnosis of acute mitral valve dysfunction.

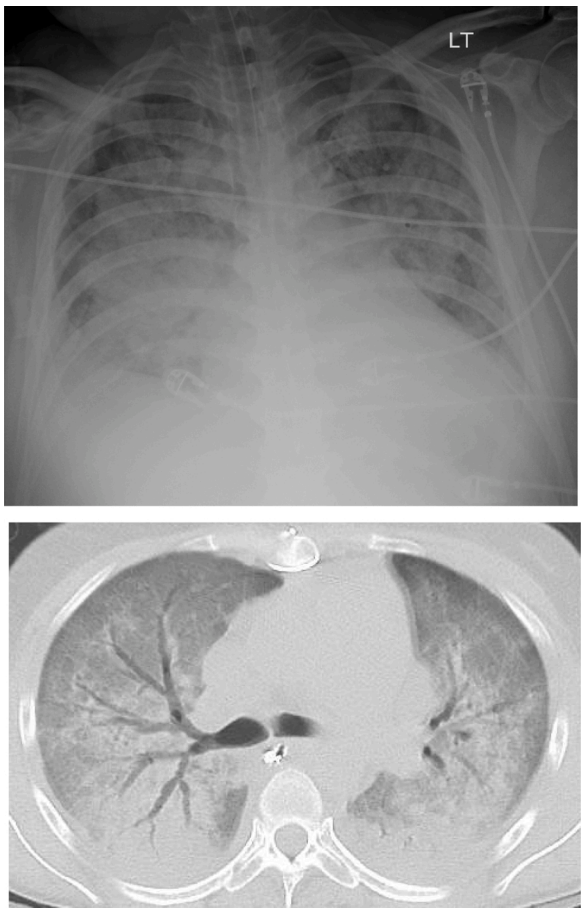
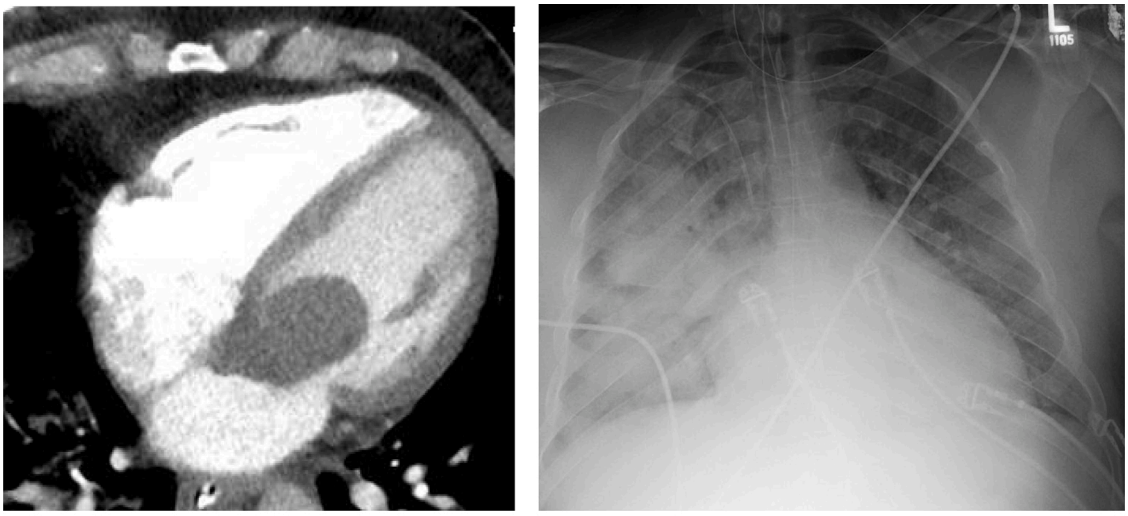


Fig. 10. 31 yo F with MRSA bacteremia and ARDS. (a) Portable CXR demonstrating perihilar consolidative opacities. (b) Chest CT in the same patient demonstrates consolidative and groundglass opacities with air bronchograms most significantly affecting the dependent portion of the lung in keeping with ARDS.

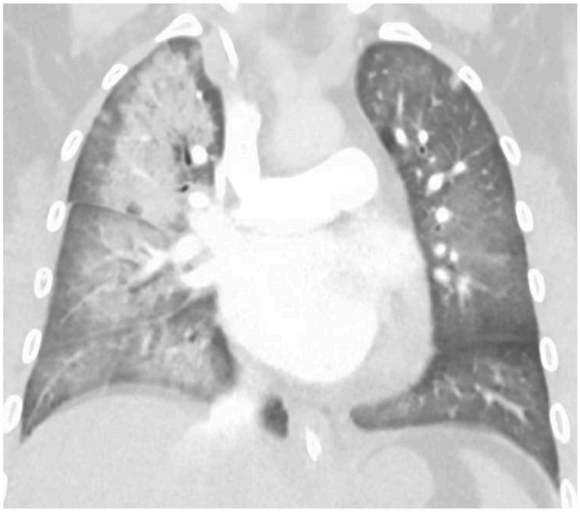
Table 2
Uncommon Etiologies of Pulmonary edema

Mitral Valve Insufficiency
PE
PVOD
Smoke Inhalation
Re-expansion pulmonary edema
Reperfusion edema s/p lung transplantation
Drugs (“Crack Lung”, salicylate toxicity)
High altitude pulmonary edema
Neurogenic pulmonary edema

Pulmonary embolus (PE) is another uncommon cause of pulmonary edema and can be seen in both acute and chronic PE. Following pulmonary arterial occlusion by thrombus, right heart blood volume is shunted entirely to segments of the pulmonary arterial tree that are free of thrombus, causing a relative hyper-perfusion of these unaffected segments of the lung [15]. On CT, this manifests as areas of increased attenuation, usually ground-glass opacity and septal thickening, corresponding to the parenchyma with patent pulmonary arteries. Because of this lobar, segmental, or subsegmental distribution, the parenchymal changes may be sharply demarcated (Fig. 13). Additionally, areas of increased attenuation seen on CT images often correlate with enlarged pulmonary arteries [15,16]. Moreover, in the setting of chronic PE following mechanical thrombectomy, a reperfusion edema has also been identified in areas of the lung to which vascular flow has been restored



a. b.



c.

Fig. 11. 54 yo M with L atrial myxoma. (a) Cardiac gated CT of the heart demonstrating a solid mass in the left atrium attached to the inter-atrial septum, extending through the mitral valve into the left ventricle. Portable CXR (b) and coronal chest CT (c) from the same patient demonstrating asymmetric right upper lobe and perihilar consolidations, consistent with asymmetric pulmonary edema.

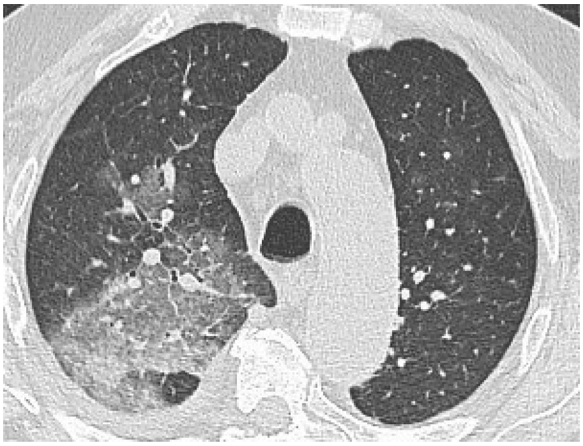
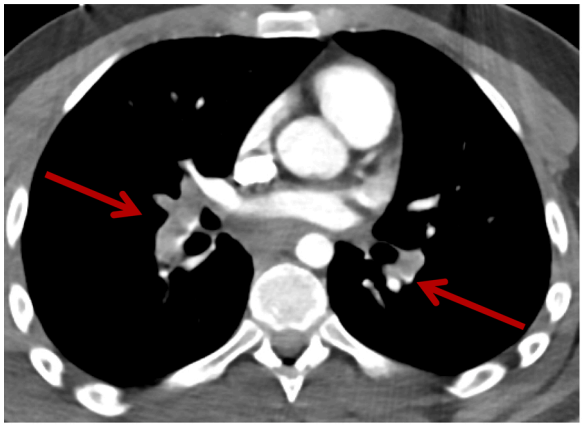


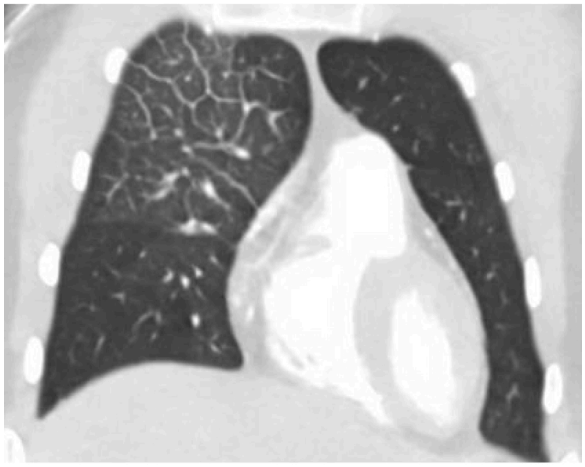
Fig. 12. 89 yo M hx admitted for fatigue and dyspnea on exertion, found to have severe mitral valve prolapse. CT chest demonstrating right upper.

[17].

Pulmonary veno-occlusive disease (PVOD) is a rare form of pulmonary hyptertension that demonstrates imaging manifestations of pulmonary edema [16,18] occurring in only 0.1-0.2 cases per million people [19,20]. The edema in PVOD is a hydrostatic pressure type of edema caused by occlusion of pulmonary venules and small veins by fibrin and proliferation of endothelial cells and connective tissue [18, 21]. The obstruction at the level of the venules causes increases in pulmonary artery pressures. On CXR, there is increase in pulmonary parenchymal opacification with Kerley lines, peribronchial cuffing, enlarged pulmonary arteries, with a normal sized left ventricle, normal pulmonary capillary wedge pressure, enlarged pulmonary artery (PA), and right heart. Pleural and pericardial effusions are usually present. On CT, ground-glass opacity with interlobular septal thickening is often seen with associated pleural effusions, enlargement of the pulmonary arteries and right heart, with a normal size left ventricle, left atrium and pulmonary veins (Fig. 14) [22,23]. The distribution of ground-glass opacities ranges from diffuse to geographic, perihilar, patchy, or centrilobular [22]. These findings in the setting of pulmonary hypertension are highly suggestive of PVOD and can help direct appropriate workup towards confirmation with lung biopsy.



a.



b.

Fig. 13. 23yo F IV drug abuser with multifocal pulmonary emboli in the right middle, right lower, left lower lobes, and left upper lobes (red arrows). No filling defects in the right upper lobe pulmonary artery (not shown). Ground glass opacities, bronchial wall and septal thickening in the right upper lobe, consistent with focal asymmetric edema.

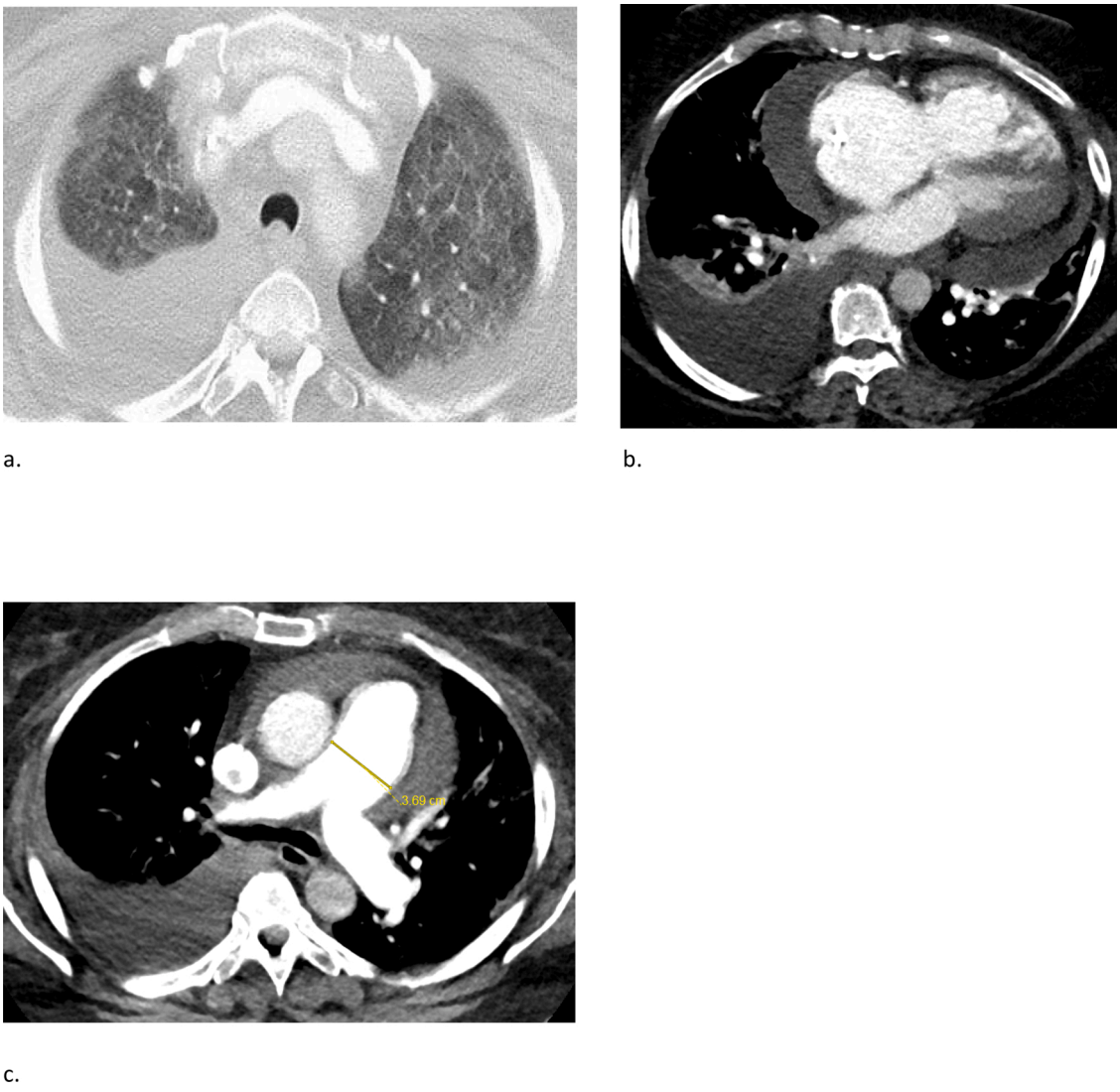


Fig. 14. 58 yo F with severe shortness of breath attributed to PVOD. (a) CT of the chest demonstrating diffuse ground-glass opacities, interlobular septal thickening, and pleural effusions. Note that the right heart (b) and main pulmonary artery (c) are enlarged. The pulmonary capillary wedge pressure (PCWP) was normal.

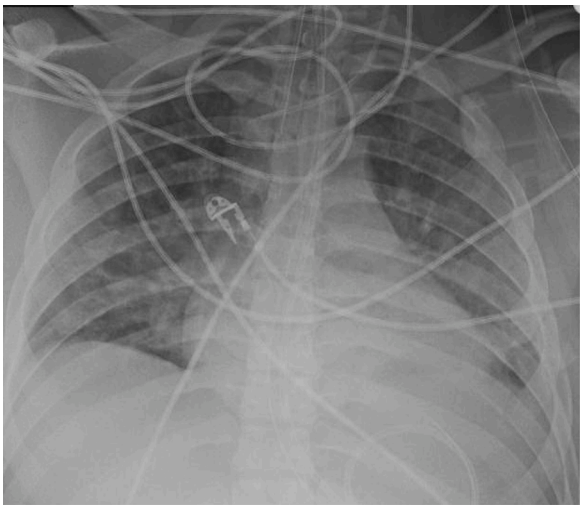
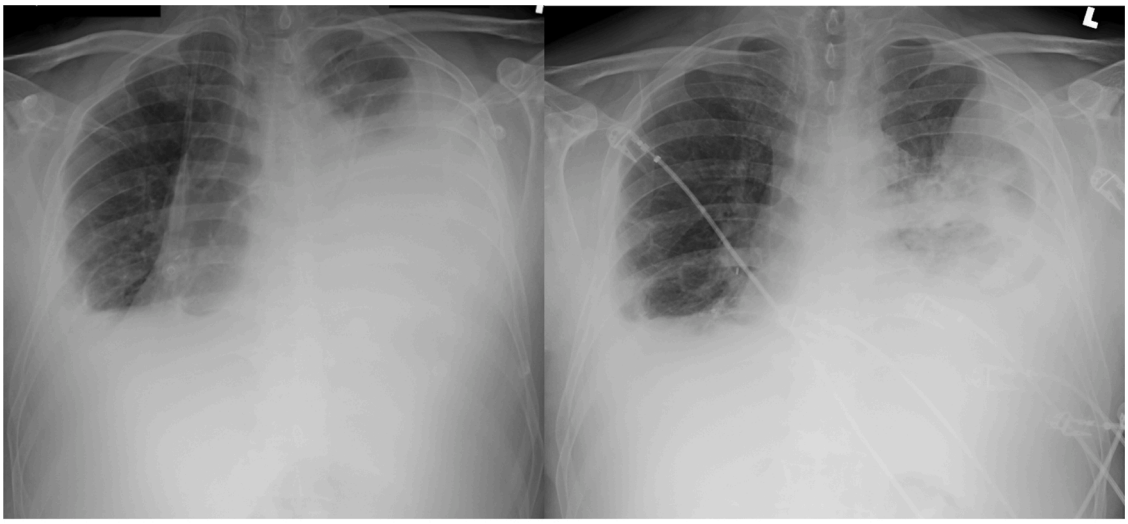


Fig. 15. 25yo M with a history of smoke inhalation from a kitchen fire. Portable CXR demonstrates hazy perihilar opacities in keeping with pulmonary edema.

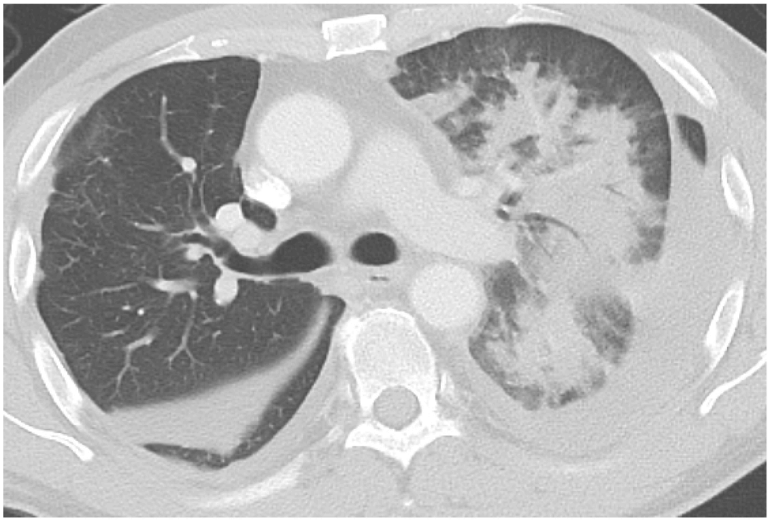
Smoke inhalation is a well-recognized but uncommon cause of pulmonary edema with radiographic manifestations occurring within 24 hours of exposure [24]. Chemicals and particulates in smoke are inhaled and cause irritation and inflammation of the airways. Inflammatory mediators cause vasodilation, increased blood flow, and increased capillary permeability with fluid movement into the alveoli and interstitium [25]. This permeability type of edema manifests as perivascular haziness and peribronchial cuffing with mixed alveolar and interstitial opacities (Fig. 15) [24]. Parenchymal ground-glass opacity and consolidation is often asymmetric in distribution with predominant upper lobe and perihilar involvement [24].

Re-expansion pulmonary edema occurs following the rapid re-expansion following whole lung collapse [26]. The rapid re-expansion is commonly seen following a large volume thoracentesis. The mechanism of the edema is thought to be a combination of both hydrostatic pressure edema and permeability edema. Radiographic manifestations include unilateral alveolar and interstitial opacities in the re-expanded lung (Fig. 16 and 17). These manifestations usually occur within one hour of the re-expansion [27] and increase in severity for 24-48 hours, prior to resolving over 5-7 days.

Reperfusion pulmonary edema following lung transplantation, also called ischemia-reperfusion injury, is an important cause of edema in the

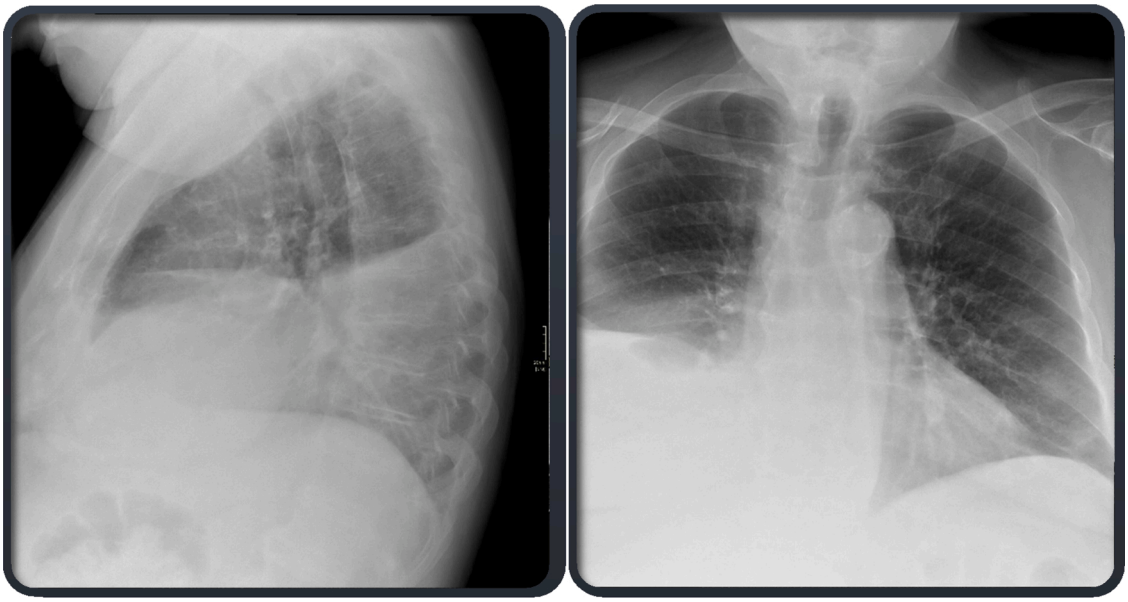


a. b.

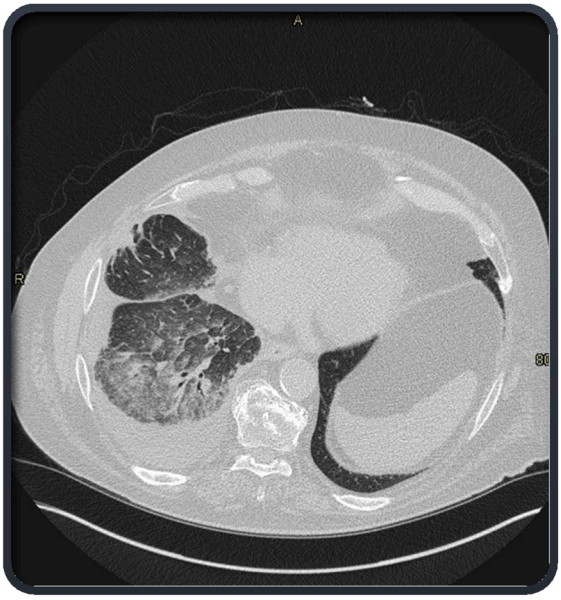


c.

Fig. 16. CXR of a patient with a large left pleural effusion before (a) and after (b) a left thoracentesis with removal of 2 L of fluid. Note that following the thoracentesis (b), the left pleural effusion has decreased in size, the mediastinum is midline, and there is a new left perihilar consolidation. (c) CT following thoracentesis demonstrating central perihilar parenchymal consolidation consistent with re-expansion pulmonary edema.



a. b.



c.

Fig. 17. (a,b) PA and lateral CXR of a 77yo M with recurrent pleural effusion following cardiac arrest s/p 2 L thoracentesis. Chest CT (B) following the thoracentesis demonstrates small residual pleural effusion with ground-glass opacity and septal thickening, consistent with re-expansion pulmonary edema.

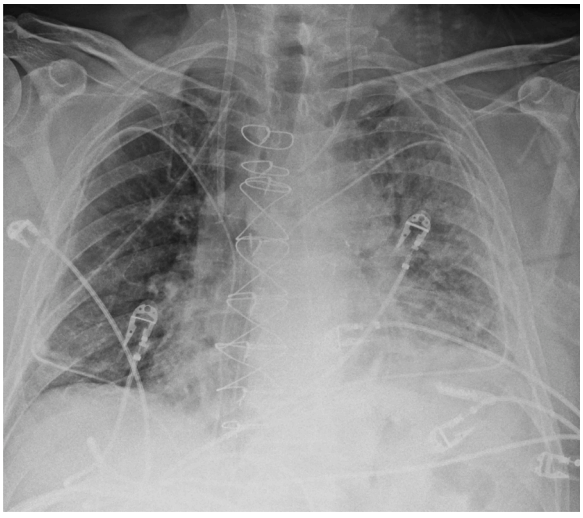


Fig. 18. 64 yo M with severe COPD status post bilateral lung transplantation with asymmetric interstitial opacities in the left lung compatible with reperfusion edema.

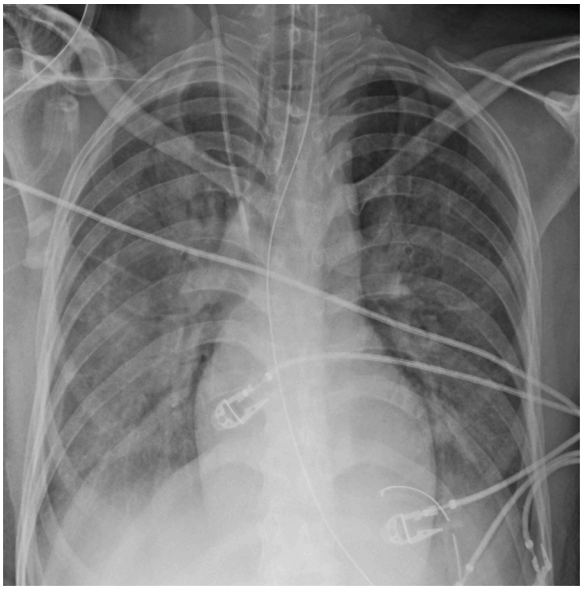


Fig. 19. 34 M following a fall from ladder with a subarachnoid hemorrhage, causing brain dead. Portable CXR demonstrates bilateral hazy and consolidative opacities, with a perihilar predominance on the left and a small layering right pleural effusion. Findings were suggestive of neurogenic pulmonary edema.

newly transplanted patients. The edema is thought to represent a permeability type of edema, caused by combination of donor lung ischemia and subsequent reperfusion following transplantation into the donor, interruption of lymphatic drainage, surfactant deficiency, and denervation of the lung during the course of surgery. Radiologic manifestations are first seen after the first 24 hours following lung transplantation, gradually worsening until day 4 and resolving by day 7 post-operatively [28]. In extreme cases, the edema can continue for longer periods but is usually resolved by 2 months post-operatively. Imaging features include perihilar ground-glass opacities, peribronchial thickening, and interstitial and alveolar opacities in the mid to lower lungs (Fig. 18) [29]. Also, the edema may be asymmetric in up to one-third of patients with bilateral lung transplant [30]. Accurate diagnosis of post-transplant reperfusion edema is important to recognize and treat as severe forms can result in primary graft dysfunction [31–33].

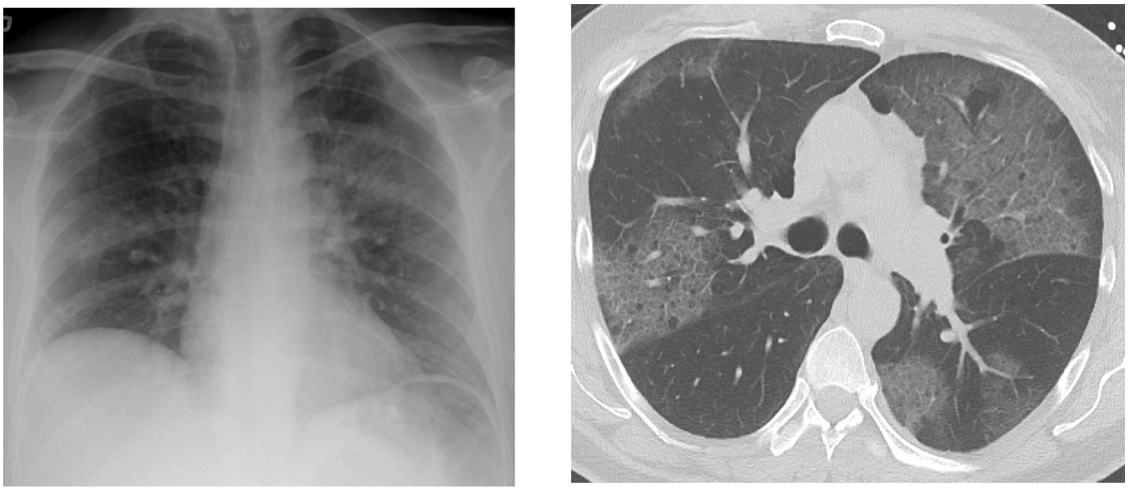
Neurogenic pulmonary edema is a syndrome which occurs acutely after a devastating CNS injury. Injuries range from status epilepticus to spinal cord injuries to traumatic brain injuries including subarachnoid, subdural or intracranial hemorrhage [34–36]. The mechanism of pulmonary edema following neurologic injury is a mixed type of edema, involving hydrostatic and permeability components, both of which may be induced by a transient sympathetic discharge causing pulmonary vasoconstriction and shear stress injury to the capillary membranes [37]. Radiographic appearances include bilateral homogenous alveolar and interstitial opacities and pleural effusions (Fig. 19). Occasionally, the parenchymal opacities demonstrate a predilection for the lung apices. Usually, the opacities resolve after 1-2 days without long-term changes.

High altitude pulmonary edema is another uncommon cause of edema occurring secondary to exposure to low oxygen atmospheric pressures after a rapid ascent to a high altitude, usually higher than 3,000 meters. A study of 150 patients with high altitude pulmonary edema demonstrated a mean onset of symptoms of 3 days with mean arterial oxygen levels of 74%, but falling as low as 38% [38]. Radiographic findings included pulmonary consolidations in 88%, most of which were bilateral and central.

Pulmonary edema can be seen in healthy patients following intravenous cocaine administration and crack cocaine inhalation. Autopsy studies of cocaine-related deaths have demonstrated pulmonary edema in 77-85% of patients [39,40]. The mechanism of edema includes a permeability component with diffuse alveolar damage [41,42], caused by direct damage to the capillary endothelium and a hydrostatic component, caused by vasoconstriction, myocardial ischemia, infarction, and arrhythmias. Radiographic findings include perihilar alveolar opacities, interstitial thickening, and pleural effusions (Fig. 20). Studies of edema in patients smoking free base “crack” cocaine demonstrated these findings, often with a normal sized heart. The radiologic abnormalities resolved within 24-72 hours [43]. The term “crack lung” is used to describe a pulmonary syndrome of edema, hemorrhage, alveolitis, and diffuse alveolar damage [42] demonstrating these perihilar opacities, interstitial thickening, and pleural effusions following free base cocaine inhalation.

6. Conclusions

Pulmonary edema has a range of imaging manifestations that can be easily identified and accurately diagnosed with routine CXR and CT. Identifying the underlying etiology of the edema is crucial to the timely implementation of appropriate therapy. In this article, we have described four types of edema based on mechanism including hydrostatic pressure edema, permeability edema with and without DAD, and mixed hydrostatic and permeability edema. We have demonstrated the common and uncommon radiographic manifestations of edema and various etiologies. With a careful analysis of the radiographic findings and clinical setting, the radiologist can help clinicians accurately diagnosis pulmonary edema and identify a cause of the edema, thereby facilitating appropriate clinical management.



a. b.

Fig. 20. 49 yo M with a history of smoking free-base crack cocaine, presenting with mental status changes and dyspnea. (a) PA CXR demonstrating faint patchy opacities in both lungs. (b) Chest CT in same patient demonstrating geographic areas of ground-glass opacity with associated interlobular septal thickening. The diagnosis of "crack lung".

CRediT authorship contribution statement

Maria Barile: Conceptualization, Project administration, Resources, Writing - original draft, Writing - review & editing.

Acknowledgments

I would like to acknowledge Mark Hammer for his contribution of the CXR of neurogenic edema and Leslie Torre for the artwork in Figs. 1 and 2.

References

[1] L. Ketai, D. Godwin, A New View of Pulmonary Edema and Respiratory Distress Syndrome, J. Thorac. Imaging (1998) 147–171.
[2] E.W. Ely, E.F. Haponik, Using the Chest Radiograph To Determine Intravascular Volume Status: The Role of Vascular Pedicle Width, Chest. 121 (2002) 942–950, https://doi.org/10.1378/CHEST.121.3.942.
[3] E. Milne, M. Pistolesi, M. Miniati, C. Giuntini, The radiologic distinction of cardiogenic and noncardiogenic edema, Am. J. Roentgenol. 144 (1985) 879–894, https://doi.org/10.2214/ajr.144.5.879.
[4] J. Bhattacharya, M.A. Gropper, N.C. Staub, Interstitial fluid pressure gradient measured by micropuncture in excised dog lung, J. Appl. Physiol. Respir. Environ. Exerc. Physiol. 56 (1984) 271–277, https://doi.org/10.1152/jappl.1984.56.2.271.
[6] N. Morooka, S. Watanabe, Y. Masuda, Y. Inagaki, Estimation of Pulmonary Water Distribution and Pulmonary Congestion by Computed Tomography, Jpn. Heart J. 23 (1982) 697–709 (accessed January 29, 2018), https://www.jstage.jst.go.jp/article/ihj1960/23/5/23_5_697/_pdf/-char/en.
[7] J.P. Szidon, Pathophysiology of the congested lung, Cardiol. Clin. 7 (1989) 39–48, https://doi.org/10.1016/S0733-8651(18)30455-7.
[8] G.D. Rubenfeld, E. Caldwell, E. Peabody, J. Weaver, D.P. Martin, M. Neff, E. J. Stern, L.D. Hudson, Incidence and Outcomes of Acute Lung Injury, N. Engl. J. Med. 353 (2005) 1685–1693, https://doi.org/10.1056/NEJMoa050333.
[9] L. Gattinoni, A. Pesenti, L. Gattinoni, A. Pesenti, The concept of "baby lung,", Intensive Care Med. 31 (2005) 776–784, https://doi.org/10.1007/s00134-005-2627-z.
[10] T.R. Martin, Lung cytokines and ARDS: Roger S. Mitchell lecture, Chest 116 (1999) 25–85, https://doi.org/10.1378/chest.116.suppl_1.2S.
[11] M.A.M. Miller, Edmund J. Cohen, Allen B. Matthay, Increased interleukin-8 concentrations in the pulmonary edema fluid of patients with acute respiratory distress syndrome from sepsis, Crit. Care Med. 24 (1996) 1448–1454.
[12] F. Sujal, R. Desai, F. Athol, U. Wells, F. Michael, B. Rubens, M. Timothy, W. Evans, F.M. David, M. Hansell, Acute Respiratory Distress Syndrome: CT Abnormalities at Long-term Follow-up 1, 1999.
[13] G. Mineo, F. Ciccarese, C. Modolon, M.P. Landini, M. Valentino, M. Zompatori, Fibrosi polmonare post-ARDS in pazienti con polmonite da H1N1: Ruolo della TC nel follow-up, Radiol. Medica. 117 (2012) 185–200, https://doi.org/10.1007/s11547-011-0740-3.
[14] I.M. Nöbauer-Huhmann, K. Eibenberger, C. Schaefer-Prokop, H. Steltzer, W. Schlick, K. Strasser, P. Fridrich, C.J. Herold, Changes in lung parenchyma after acute respiratory distress syndrome (ARDS): Assessment with high-resolution computed tomography, Eur. Radiol. 11 (2001) 2436–2443, https://doi.org/10.1007/s003300101103.
[15] H.C. Schwickert, F. Schweden, H. Schild, R. Piependburg, C. Duuber, H.-U. Kauczor, C. Renner, S. Iversen, M. Thelen, Pulmonary Arteries and Lung Parenchyma in Chronic Pulmonary Embolism: Preoperative and Postoperative CT Findings, Radiology (1994).
[16] S.L. Primack, N.L. Müller, J.R. Mayo, M. Remy-Jardin, J. Remy, Pulmonary parenchymal abnormalities of vascular origin: high-resolution CT findings, Radiographics. 14 (1994) 739–746, https://doi.org/10.1148/radiographics.14.4.7938765.
[17] T. Inami, M. Kataoka, N. Shimura, H. Ishiguro, R. Yanagisawa, H. Taguchi, K. Fududa, Pulmonary Edema Predictive Scoring Index(PEPSI), a New Index to Predict Risk ofReperfusion Pulmonary Edema and Improvementof Hemodynamics in Percutaneous TransluminalPulmonary Angioplasty, JACC Cardiovasc. Interv. 6 (2013) 725–736.
[18] J.D. Maltby, M.L. Gouverne, CT Findings in Pulmonary Venoocclusive Disease, J. Comput, Assist. Tomogr. 8 (1984) 758–761.
[19] J. Mandel, M. Eugene, C. Hales, PulmonaryVeno-occlusive Disease, Am J Respir Crit Care Med. 162 (2000) 1964–1973, https://doi.org/10.1164/rccm.200408-1109SO.
[20] L. Rubin, Primary Pulmonary Hypertension, N. Engl. J. Med. 336 (1997) 111–117.
[21] D. Montani, E.M. Lau, P. Dorfmüller, B. Girerd, X. Jaïs, L. Savale, F. Perros, E. Nossent, G. Garcia, F. Parent, E. Fadel, F. Soubrier, O. Sitbon, G. Simonneau, M. Humbert, Pulmonary veno-occlusive disease, Eur. Respir. J. 47 (2016) 1518–1534, https://doi.org/10.1183/13993003.00026-2016.
[22] A. Resten, S. Maitre, M. Humbert, A. Rabiller, O. Sitbon, F. Capron, G. Simonneau, D. Musset, Pulmonary Hypertension: CT of the Chest in Pulmonary Venoocclusive Disease, Am. J. Roentgenol. 183 (2004) 65–70.
[23] S.J. Swensen, J.H. Tashjian, J.L. Myers, C.E. Engeler, E.F. Patz, W.D. Edwards, W. W. Douglas, Pulmonary venoocclusive disease: CT findings in eight patients, Am. J. Roentgenol. 167 (1996) 937–940.
[24] H.S. Teixidor, E. Rubin, G.S. Novick, D.R. Alonso, Smoke inhalation: Radiologic manifestations, Radiology. 149 (1983) 383–387, https://doi.org/10.1148/radiology.149.2.6622680.
[25] M.L. Witten, S.F. Quan, R. Sobonya, R.J. Lemen, New developments in the pathogenesis of smoke inhalation-induced pulmonary edema, West. J. Med. 148 (1988) 33–36.
[26] O.M. Dias, L.R. Teixeira, F.S. Vargas, Reexpansion pulmonary edema after herapeutic thoracentesis, Clinics. 65 (2010) 1387–1389, https://doi.org/10.1590/S1807-59322010001200026.
[27] S. Mahfood, W.R. Hix, B.L. Aaron, P. Blaes, D.C. Watson, Reexpansion Pulmonary Edema, Ann. Thorac. Surg. 45 (1988) 340–345, https://doi.org/10.1016/S0003-4975(10)62480-0.
[28] M.S. Krishnam, R.D. Suh, A. Tomasian, J.G. Goldin, C. Lai, K. Brown, P. Batra, D. R. Aberle, Postoperative Compli- cations of Lung Trans- plantation: Radiologic Findings along a Time Continuum 1 LEARNING OBJECTIVES FOR TEST 3 CME FEATURE, 2007, pp. 957–975, https://doi.org/10.1148/rg.274065141.
[29] K. Pilgram, S. Glazer, P. Trulock, J.W. Semenkovich, J.D. Cooper, G. Alexander, Lung Transplant Edema: Chest Radiography after Lung Transplantation- The First 10 Days, Radiology. 195 (1995) 275–281.
[30] J. Collins, Imaging of the chest after lung transplantation, J. Thorac. Imaging 17 (2002) 102–112, https://doi.org/10.1097/00005382-200204000-00002.
[31] M.K. Porteous, J.C. Lee, Primary Graft Dysfunction After Lung Transplantation, Clin. Chest Med. 38 (2017) 641–654, https://doi.org/10.1016/j.ccm.2017.07.005.

[32] A.E. Gelman, A.J. Fisher, H.J. Huang, M.A. Baz, C.M. Shaver, T.M. Egan, M. S. Mulligan, Report of the ISHLT Working Group on Primary Lung Graft Dysfunction Part III: Mechanisms: A 2016 Consensus Group Statement of the International Society for Heart and Lung Transplantation, J Hear. Lung Transpl. 36 (2017) 1114–1120, https://doi.org/10.1097/CCM.0b013e31823da96d.Hydrogen.
[33] V.E. Laubach, A.K. Sharma, Mechanisms of Lung Ischemia-Reperfusion Injury, Physiol. Behav. 176 (2017) 139–148, https://doi.org/10.1016/j.physbeh.2017.03.040.
[34] S. Rogers, G. Shackford, J. Trevisani, R. Davis, D. Hoyt Mackersie, Neurogenic Pulmonary Edema in Fatal and Nonfatal Head Injuries, J. Trauma Inj. Infect. Crit. Care. 39 (1995) 860–868.
[35] G. Colice, M. Matthay, E. Bass, R. Matthay, Neurogenic Pulmonary Edema, Am. Rev. Respir. Dis. 130 (1984) 941–948, https://doi.org/10.1080/00754170902750198.
[36] D. Davison, M. Terek, L. Chawla, Neurogenic Pulmonary Edema, Crit. Care. 16 (2012) 212, https://doi.org/10.1007/978-3-642-25716-2.
[37] A.B. Malik, Mechanisms of neurogenic pulmonary edema, Circ. Res. 57 (1985) 1–18, https://doi.org/10.1161/01.RES.57.1.1.
[38] H.N. Hultgren, B. Honigman, K. Theis, D. Nicholas, High-altitude pulmonary edema at a ski resort, West. J. Med. 164 (1996) 222–227.
[39] R.J. Murray, J.E. Smialek, M. Golle, R.J. Albin, Pulmonary artery medial hypertrophy in cocaine users without foreign particle microembolization, Chest. 96 (1989) 1050–1053, https://doi.org/10.1378/chest.96.5.1050.
[40] M.E. Bailey, A.E. Fraire, S. Donald Greenberg, J. Barnard, P.T. Cagle, Pulmonary histopathology in cocaine abusers, Hum. Pathol. 25 (1994) 203–207, https://doi.org/10.1016/0046-8177(94)90279-8.
[41] B. Mégarbane, L. Chevillard, The large spectrum of pulmonary complications following illicit drug use: Features and mechanisms, Chem. Biol. Interact. 206 (2013) 444–451, https://doi.org/10.1016/j.cbi.2013.10.011.
[42] J.M. Forrester, A.W. Steele, J.A. Waldron, P.E. Parsons, Crack lung: An acute pulmonary syndrome with a spectrum of clinical and histopathologic findings, Am. Rev. Respir. Dis. 142 (1990) 462–467, https://doi.org/10.1164/ajrccm/142.2.462.
[43] C.K. Hoffman, P.C. Goodman, Pulmonary edema in cocaine smokers, Radiology 172 (1989) 463–465, https://doi.org/10.1148/radiology.172.2.2748827.