



Cardiogenic pulmonary edema: mechanisms and treatment – an intensivist's view

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Purpose of review

This review summarizes current understanding of the pathophysiology of cardiogenic pulmonary edema, its causes and treatment.

Recent findings

The pathobiology and classification of pulmonary edema is more complex than the hydrostatic vs. permeability dichotomy of the past. Mechanisms of alveolar fluid clearance and factors that affect the clearance rate are under intensive study to find therapeutic strategies. Patients need early stabilization of oxygenation and ventilation, preferably with high-flow nasal cannula oxygen or noninvasive ventilation whereas the diagnostic cause is quickly sought with echocardiography and other testing.

Summary

Treatments must be initiated early, whereas evaluation still is occurring and requires multimodality intervention. The general treatment of cardiogenic pulmonary edema includes diuretics, possibly morphine and often nitrates. The appropriate use of newer approaches – such as, nesiritide, high-dose vasodilators, milrinone, and vasopressin receptor antagonists – needs larger clinical trials.

Keywords

acute heart failure, alveolar fluid clearance, flash pulmonary edema, noninvasive ventilation, pulmonary edema

INTRODUCTION

This review updates knowledge about cardiogenic pulmonary edema. Pulmonary edema is defined as increased fluid in the interstitial and/or alveolar spaces of the lung parenchyma. Edema fluid may be transudative – without increased protein, cells or mediators – or exudative or hemorrhagic, with high levels of protein. Pulmonary edema has been of interest to cardiologists, pulmonologists and physiologists since Laennec [1], with summaries in books [2,3] and reviews [4–7], but few in the last decade. This review focuses on acute, rather than chronic, pulmonary edema and primarily on cardiogenic edema.

CLINICAL PRESENTATION

Acute pulmonary edema usually presents with shortness of breath at rest and worsened with exertion, tachypnea, tachycardia and relative hypoxemia. Approximately half of patients with acute heart failure have a history of chronic heart failure. The lung exam may reveal crackles but these are not specific for pulmonary edema or a particular cause. Peribronchial cuffing may cause wheezing ('cardiac asthma'). If the

patient is in cardiogenic shock, then the blood pressure may be low. However, if left ventricular (LV) function is good then hypertension is common because of stress and increased endogenous catecholamine levels. Cough is common and, if the edema is severe, patients may bring up foamy or pink-tinged sputum. Jugular venous pressure often is hard to measure and may be normal or increased. Finding an S3 gallop on exam is relatively specific for cardiogenic edema, but has low sensitivity. Focused exam for cardiac murmurs consistent with valvular stenosis or regurgitation or signs of right heart failure is of high importance. Lactic acidemia may accompany severe hypoxemia, particularly in patients with low blood pressure and poor peripheral perfusion.

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KEY POINTS

- Understanding the mechanism and regulation of alveolar fluid resorption will provide new therapeutic strategies.
- Rapid diagnostic separation of cardiogenic pulmonary edema from ARDS remains a difficult clinical challenge.
- Early stabilization of oxygenation and ventilation should be accomplished by moving rapidly as needed from high-flow nasal cannula oxygen to noninvasive ventilation to intubation and mechanical ventilation. Arterial blood gases are the most reliable way to assess ventilation and acid–base status.
- Multiple new approaches for treatment of cardiogenic pulmonary edema are under active investigation but they have not yet changed the old treatment algorithms.

Pathophysiology and causes of acute pulmonary edema

The normal lung has a very thin layer of alveolar lining fluid that does not impede gas exchange. Starling forces result in small net fluid and solute efflux out of alveolar capillaries into the interstitium as combined effect of capillary hydrostatic and pressure and oncotic gradients. Peribronchovascular lymphatics remove fluid and provide reserve capacity. The primary factor keeping the alveoli relatively dry is the very tight alveolar epithelial cell junctions.

Acute pulmonary edema traditionally is divided into two categories: high pulmonary capillary pressure (PCP) with normal alveolar–capillary barrier (hydrostatic) and increased alveolar capillary permeability (leak). The latter has a relatively high pleural fluid to serum protein ratio and may be cellular or hemorrhagic. Acute cardiogenic and/or volume overload edema usually have increased pulmonary venous pressure because of elevations in left atrial and LV end diastolic pressure (LAP and LVEDP, respectively). Alveolar flooding occurs with high LAP (>25 mmHg). In chronic left heart failure, an adaptive increase in lymphatic drainage and/or hypertrophy and constriction of precapillary arterioles partially protect against superimposed acute pulmonary edema. Tocolytic drugs (intravenous terbutaline and beta adrenergic agonists) and pheochromocytoma may increase PCP by causing more pulmonary venoconstriction than arteriolar constriction. Permeability edema usually occurs within the spectrum of acute respiratory distress syndrome (ARDS) [8].

Some clinical situations are hard to simply categorize or present a mixed picture. Sudden high PCP may rupture very thin alveolar–capillary walls, resulting in high protein, hemorrhagic edema. This may occur in acute mitral regurgitation with papillary

muscle necrosis or a torn chordae tendinae. Rarely it occurs with very strenuous exercise in a patient with high-grade mitral stenosis or in pregnancy. The acute pulmonary edema with sudden neurologic disasters may be because of neurally mediated severe pulmonary venoconstriction with transient very high PCP that cause ‘blast’ injury. The PCP rapidly returns to normal, but the edema fluid is proteinaceous.

Active epithelial ion transport now is considered the primary mechanism for resolution of alveolar edema [9–11] through the joint action of alveolar epithelial cell apical amiloride-sensitive sodium channels and basolateral sodium pump. Active fluid resorption can be altered in disease and upregulated by a variety of hormones, mediators and cytokines. Alveolar fluid clearance in humans is preserved or increased in most cardiogenic pulmonary edema patients, but decreased in most patients with ARDS. To date, human clinical trials of inhaled or intravenous beta adrenergic agonist therapy in patients with ARDS have not shown benefit.

Diagnostic evaluation

The many cardiogenic causes of acute heart failure and/or pulmonary edema are listed in Table 1.

Table 1. Cardiogenic causes of acute heart failure and/or pulmonary edema

Acute myocardial infarction/ischemia
LV dysfunction or failure
Papillary muscle dysfunction
Ventricular septal rupture
Acute onset cardiomyopathy
Acute fulminant myocarditis
Stress cardiomyopathy/Takotsubo syndrome
Acute valvular dysfunction
Acute severe aortic insufficiency (with or without aortic dissection)
Acute severe mitral regurgitation
Critical valvular dysfunction with volume overload or high cardiac output
Refractory sustained tachyarrhythmias
Atrial fibrillation with a rapid ventricular response
Hypertensive crisis
Myocardial contusion/postcardiac arrest stunning
Dynamic left ventricular outflow tract obstruction
Toxic/metabolic
Beta block or calcium channel antagonist overdose
Scorpion venom
Pheochromocytoma
Cardiac tamponade

LV, left ventricle.

Cardiogenic shock with low cardiac output (CO) typically has high LVEDP and LAP, causing acute pulmonary edema. For patients who present with severe pulmonary edema of uncertain cause, rapidly differentiating cardiogenic from other causes sometimes is very difficult and requires a systematic approach [12^a,13]. The constellation of shortness of breath, hypoxemia and an abnormal chest X ray is common and nonspecific. Rapidly obtaining a high-quality patient history, particularly for cardiopulmonary disorders and risk factors, is very important.

Electrocardiograms (EKGs) are essential to look for signs of myocardial infarction, ischemia, atrial abnormalities or pericardial disease. Cardiac rhythm abnormalities also may contribute to pulmonary edema. Atrial fibrillation may lower CO and worsen edema either because of high ventricular rates with inadequate filling and/or the loss of atrial kick. Echocardiography is one of the most helpful early tests, assessing valvular, systolic and diastolic

ventricular function and pericardial abnormalities. Diastolic dysfunction may be difficult to detect. Combined cardiac and thoracic ultrasound can help differentiate cardiogenic edema from ARDS or other causes in ICU patients with acute hypoxic respiratory failure and diffuse chest radiographic abnormalities [14]. Decreased LV function, a large inferior vena cava (IVC) minimal diameter (>23 mm), and a left-sided pleural effusion (>20 mm) favor cardiogenic edema. The presence of B-lines (lung comets or rockets) on chest ultrasound reflects fluid thickened subpleural interlobular septae [15]. Sequential bedside chest ultrasound can assess severity and monitor the resolution of cardiogenic edema, providing prognostic information [16,17].

Chest radiograph findings that favor cardiogenic edema over ARDS include increased size of the heart and/or width of the vascular pedicle, more central or evenly distributed edema opacities, peribronchial cuffing, absence of air bronchograms and presence of pleural effusions. Chest computed tomography (CT) scans in ARDS have more severe ground-glass attenuation with airspace consolidation and decreased lung gas volume without qualitatively discriminating features [18].

Serial measurements of troponin levels are important to assess myocardial ischemia and infarction. Highly elevated levels of brain natriuretic peptide (BNP or NT-ProBNP) support heart failure as the cause of pulmonary edema in noncritically ill patients and a very low level makes heart failure unlikely, but there is an indeterminate middle range of values. If mental status is abnormal, then toxin screens, electrolytes and serum osmolality should be checked.

Thoracentesis can help confirm that pleural effusions are because of heart failure as the causes of transudative effusions are relatively few – heart failure, hypoalbuminemia, nephrotic syndrome, Meigs syndrome, hypothyroidism and sarcoidosis. Effective diuresis of patients with heart failure can convert a transudative effusion into an exudate.

Pulmonary artery catheterization with measurement of the pulmonary artery occlusion pressure used to be a common diagnostic and management tool for patients with acute pulmonary edema, particularly with hypotension. Although now used much less because of lack of demonstrated survival benefit, it can rationalize management of ICU patients with pulmonary edema, based on CO, stroke volume, PCP and vascular resistance. Calculated CO and stroke volume from the Fick equation are often unreliable in unstable ICU patients. Newer semiinvasive methods monitor the extravascular lung water and the pulmonary vascular permeability index [19], without demonstrated survival benefit yet. New techniques to monitor pulmonary edema fluid quantity over time are actively being studied. Bioimpedance uses the frequency-dependent resistance to low-energy electrical flow (better transmission through salt and water than air). Impedance-guided therapy after acute heart failure may reduce complications of chronic heart failure [20,21]. Improved noninvasive monitoring techniques are likely to improve outcomes.

GENERAL TREATMENT OF CARDIOGENIC PULMONARY EDEMA

Acute pulmonary edema is a life-threatening emergency and diagnostic and therapeutic interventions need to occur rapidly and often together. The treatment depends in part on the specific cause. There frequently are comorbid and/or secondary conditions that also require urgent treatment, including infection, acidemia and renal dysfunction.

Support of oxygenation and ventilation

Oxygen therapy

Ensuring adequate oxygenation is essential. The goal should be rapidly achieving an oxygen saturation of 95% and/or an arterial partial pressure of oxygen of more than 70 mmHg to have nearly full saturation of red blood cells. Hypoxia may impair alveolar edema fluid clearance. However, very high oxygen levels also are detrimental. Oxygen can be delivered without intubation or positive pressure by nasal cannula, face mask, or nonrebreathing reservoir masks. For patients with acute hypoxic

respiratory failure without hypercapnia, mortality and intubation rates are improved when using high-flow nasal cannula oxygen compared with face mask oxygen or bi-level positive airway pressure mask ventilation (Bi-PAP) [22]. Arterial blood gas measurement should be done to assess: oxygenation (adjusting for alveolar ventilation and PaCO₂); acid–base disorders; and acidemia, per se.

Positive pressure ventilation

Pulmonary edema increases work of breathing and oxygen consumption up to 20-fold. Like myocardium, the contracting diaphragm is not perfused; lactic acidosis may result. This major physiologic stress on the heart can be partially relieved by non-invasive ventilation (NIV), improving oxygenation and ventilation. Options for NIV include face or nasal mask ventilation, endotracheal intubation and helmet ventilation. Continuous positive airway pressure (CPAP) provides a steady level of positive pressure without additional inspiratory ‘boost’ to augment tidal volume and lung expansion. In contrast, Bi-PAP provides augmented tidal volume, but many patients are claustrophobic and cannot tolerate the prolonged tight pressure required to seal the mask. It is difficult to cough effectively, clear secretions and avoid aspiration. Bi-PAP is contraindicated in patients who cannot protect their airway and/or with abnormal mental status. Endotracheal intubation is the most secure way to effectively ventilate patients, but intubation decreases cough and increases the risk of nosocomial pneumonia. Helmet ventilation is a new modality that utilizes a clear plastic helmet that is sealed at the neck and chest. Helmet ventilation reduces intubation in ARDS patients [23] and has been used successfully in cardiogenic edema. Positive pressure ventilation can reduce systemic venous return and LV afterload, indirectly helping the pulmonary edema.

NIV can be readily initiated without the stress and additional pharmacologic agents typically used for intubation, retaining endotracheal intubation as a fall back option. Many studies demonstrated that CPAP, Bi-PAP can improve oxygenation and reduce the work of breathing and need for intubation for patients with cardiogenic pulmonary edema [24,25]. However, the most severely ill patients may fail NIV, particularly those with a very low LV ejection fraction, a very high BNP level, elevated lactate levels and already negative fluid balance [26]. The Canadian Critical Care Society does not recommend NIV for cardiogenic edema patients who have shock or acute coronary syndrome requiring acute revascularization. NIV is an effective temporizing measure that reduces or delays the need for intubation, provided other effective treatments can be applied.

Mechanical ventilation with intubation has been the standard of care for severe respiratory failure for decades. For ARDS patients, limiting both tidal volume (V_t) and static (or plateau) airway pressure improves outcomes, but it is uncertain if one needs to reduce only one or both. The usual V_t for most ventilated patients has dropped from 10–15 ml/kg to 8–10 ml/kg of ideal body weight; the latter is a reasonable initial setting in cardiogenic edema.

Positive end expiratory pressure (PEEP) can help cardiac function and typically is used in the 5–10 cm H₂O range. PEEP does not directly reduce lung extravascular water, but improves oxygenation. PEEP maintains alveolar expansion and decreases collapse at lower lung volumes, avoiding atelectasis and resulting shunt hypoxemia. The expanded alveolar and lung volumes increase the size of the interstitial areas between the alveoli, creating a ‘sump pump’ that draws lung fluid to this site of lymphatic origination. The hemodynamic effects of PEEP can decrease the net fluid efflux across the pulmonary capillaries. Too high a PEEP level may overdistend or damage alveoli and may decrease perfusion of well ventilated alveoli, thereby worsening oxygenation.

Diuretic therapy

Diuresis with preload reduction has been a mainstay of the therapy of patients with pulmonary edema, particularly from decompensated heart failure, as it decreases lung extravascular water and improves oxygenation. Early initiation of intravenous loop diuretics is recommended and early treatment improves mortality. Initial dosing should be based on whether patients are diuretic-naïve or have chronic exposure. Continuous infusion of loop diuretics and diuretic combination therapy have gained recent favor. However, in a large clinical trial in patients with acute decompensated heart failure, continuous infusion or every 12-h bolus furosemide had no significant differences in patients’ symptoms or serum creatinine. Higher dose therapy resulted in greater diuresis and a trend toward greater improvement in global symptom assessment without worsening serum creatinine [27]. A meta-analysis of 10 randomized controlled trials found that continuous diuretic infusion was associated with greater weight loss but without difference in urine output, electrolyte imbalances, ototoxicity or cardiac or all-cause mortality [28]. In the ASCEND-HF study of diuretics in acute heart failure, poor responders to diuretics had more hypotension and worse prognosis and addition of nesiritide did not improve the impact of diuretics [29]. Similarly, adding low-dose dopamine does not improve outcomes or the effect of furosemide infusions [30]. Although there is uncertain benefit for continuous

infusion of diuretics, a Cochrane review found higher urine output and less ototoxicity with continuous infusions than intermittent high-dose bolus injections. The goals of achieving significant net diuresis and weight loss are reasonable and recommended as essential early treatment in most heart failure guidelines. Using infusions or combination therapy is reasonable if required to achieve these intermediate goals.

Furosemide also is a venodilator and can rapidly decrease venous return before any diuresis has occurred. However, sympathetic and renin-angiotensin system activation may counterbalance some of the benefit.

Preload and/or afterload reduction

A number of recent reviews have considered the management of acute heart failure of different causes in detail [31–35,36*,37*,38].

Vasodilators

Nitrates are commonly used in acute heart failure with cardiogenic edema as they reduce preload, and (apart from nitroprusside), to a lesser extent, afterload. Nitrates are particularly useful for patients with hypertension and pulmonary edema. Blood pressure should be carefully monitored because of some risk of hypotension. Some authorities recommend high-dose intravenous nitrates repeated frequently, with sublingual nitrates used an initial treatment before intravenous access is established [39,40]. Others do not feel that outcome benefits have been demonstrated and continue to use intravenous nitrates as adjunctive therapy because improved outcome has not been proven [41,42]. On balance, nitrates have potential benefit as part of combined treatment, particularly in normotensive and hypertensive patients.

Nitroprusside – unlike other nitrates – is a very potent arterial vasodilator, with accompanying venodilation. Benefit of nitroprusside for patients with acute myocardial infarction, low-cardiac output and high LV filling pressures dates back to the 1970s; it still is useful in this situation, but requires continuous arterial pressure monitoring with an arterial catheter.

Vasodilators may decrease pulmonary vascular resistance, interfering with homeostatic ventilation:perfusion-matching mechanisms and worsening oxygenation.

Endothelin antagonists are of interest because endothelin is a potent vasoconstrictor. Antagonists can reduce pulmonary and systemic vascular resistance, improving cardiac output and decreasing pulmonary capillary pressure.

Morphine and opiates

Intravenous morphine has been a standard treatment of acute cardiogenic pulmonary edema. It reduces preload and to some extent, afterload and heart rate. It also reduces anxiety, sympathetic overactivity and serum catecholamine levels, thereby reducing afterload and decreasing myocardial oxygen consumption. However, opiates can decrease the ventilatory drive and should be used cautiously as in the emergency department they may increase the need for ICU admission and/or mechanical ventilation.

Angiotensin-converting enzyme inhibitors

Angiotensin-converting enzyme inhibitors can reduce preload and afterload and are useful in patients with pulmonary edema and hypertension. Initial therapy often is a low intravenous dose of a short-acting agent. If well tolerated, then longer acting and oral agents may be used. Long-term treatment improves mortality for patients with acute myocardial infarction and heart failure.

Physical methods

Rotating tourniquets were used to reduce venous return for patient with acute heart failure and pulmonary edema. Although crude and not studied in controlled trials, some efficacy is likely. For non-hypotensive patients, making patients sit up with legs dangling can be helpful.

Natriuretic peptides

Nesiritide, human recombinant BNP, and other drugs of this class combine potent arterial and venous vasodilatory and natriuretic actions. Nesiritide increases CO and decreases PCP and vascular resistance. Patients often have less dyspnea. In a large 2002 study, comparing nesiritide with intravenous nitrates for acute congestive heart failure, nesiritide was efficacious but there was a statistical trend towards increased mortality with nesiritide. At present, nesiritide is not a first line agent and it probably should not be used in patients with acute myocardial ischemia or infarction.

Improved cardiac output

Inotropic and inodilator drugs

Dopamine and dobutamine are positive inotropes that also increase myocardial oxygen demand and the likelihood of arrhythmias. Clinical trials have not established improved outcomes. Milrinone is a phosphodiesterase-3 inhibitor that increases myocardial contractility while vasodilating the systemic and pulmonary vascular beds. Although efficacy has

not been proven, inodilators may theoretically be helpful for pulmonary edema patients with severe LV dysfunction and low cardiac output.

Calcium promoters are a new drug class intended to treat heart failure by enhancing myocardial contractility through increased troponin calcium binding. Levosimendan can increase CO with lower vascular resistance and pulmonary capillary pressures. Most studies have been in chronic heart failure patients with low CO; their role in acute heart failure needs better definition and currently no trial has proven any survival benefit.

Other approaches

Treatment of arrhythmias and/or atrioventricular resynchronization

Tachyarrhythmias decrease time for LV filling, elevating LAP and lowering CO. In turn, atrial fibrillation, atrial tachyarrhythmias or sinus tachycardia can result from high LAP and stretching or from sympathetic stimulation. This downward spiral may be interrupted with intravenous beta-blockers, digoxin or acute cardioversion. In patients with mild hypotension, use of a short-acting, reversible beta-blocker intravenous infusion may test the extent to which the tachycardia is worsening cardiac output and hypotension. For patients in atrial fibrillation, with rapid ventricular response who are relatively stable hemodynamically, digoxin can be very useful [43]. If patients are unstable and hypotensive, then cardioversion should not be much delayed. For selected patients with low LV function and lack of sequenced atrial 'kick,' an atrioventricular sequential pacemaker may be helpful.

Renal replacement therapy

Renal replacement therapy should be considered for patients with pulmonary edema and refractory volume overload, significant metabolic acidemia (pH <7.20), refractory hypoxemia and/or persistent hyperkalemia. Conventional hemodialysis would be used unless patients are hypotensive, on inotropic support or do not tolerate conventional hemodialysis. The latter usually can be accomplished with some net fluid removal.

SPECIFIC PULMONARY EDEMA CONDITIONS

'Flash' pulmonary edema

Patients sometimes present with acute severe pulmonary edema that appears to be of cardiogenic origin, rather than ARDS, anaphylaxis or part of systemic

capillary leak syndrome [44]. The defining characteristic of this dramatic presentation is an acute increase in LVEDP. Sometimes this is the initial manifestation of any cardiopulmonary disorder, but other patients have recurrent episodes that can be puzzling. Patients may improve very quickly, making diagnosis more confusing. There are few large clinical studies, but myocardial ischemia or acute hypertension were the most common precipitants of a first episode of flash pulmonary edema in a small study [45]. The underlying associated entities include: intermittent myocardial ischemia with diastolic dysfunction or transient mitral regurgitation (such as from papillary muscle dysfunction); sympathetic crashing acute pulmonary edema [46]; acute hypertensive cardiogenic pulmonary edema [47]; acute pheochromocytoma [48]; and bilateral atheromatous renovascular disease (Pickering syndrome) [49]. Defining the cause of the flash pulmonary edema is important for specific treatment. For example, revascularization significantly improves survival when atherosclerotic renal artery stenosis is the cause [50]. Obstructive sleep apnea also contributes to episodic nocturnal pulmonary edema [51].

Acute diastolic dysfunction with preserved ejection fraction

Diastolic dysfunction with decreased LV relaxation and filling is a long-recognized contributing factor in cardiogenic pulmonary edema and is the primary cause in a significant fraction [52]. The latter present with acute pulmonary edema with preserved ejection fraction. Diastolic dysfunction usually includes prolonged LV relaxation, increased LV stiffness and increased LVEDP. Chronic diastolic dysfunction can be because of structural changes including myocardial hypertrophy from chronic hypertension, hypertrophic cardiomyopathy or remodeling of remaining normal myocardium after myocardial infarction. Less common causes are constrictive pericarditis, restrictive cardiomyopathy or valvular insufficiency. Myocardial ischemia can cause rapid-onset diastolic dysfunction with potential to cause acute pulmonary edema. In patients with chronic hypertension, this can present as flash pulmonary edema that may recur [53]. Common precipitants are acute hypertension, intravenous fluid overload and/or atrial fibrillation. Patients with chronic renal failure are at high risk because of high prevalence of chronic hypertension and volume overload. Echocardiographic Doppler is the key diagnostic technique [54].

Takotsubo syndrome

This acute onset, reversible syndrome with chest pain and often dyspnea is triggered by acute emotional

or physiologic stresses. Echocardiogram shows decreased function of the apical and mid-ventricular regions with apical ballooning, often resulting in severe decreases in LV ejection fraction. Abnormal EKGs with precordial ST elevations and increased troponin levels are typical. Coronary artery disease, pheochromocytoma, intracranial hemorrhage, recent acute head trauma or other myocardial disease should be excluded. A small fraction of patients has cardiogenic shock or ventricular arrhythmias. Elevated catecholamine levels and neurogenic sympathetic hyperactivity are potential contributors to this disorder [55,56]. Relatively stable patients should be treated with beta blockers for hypertension and/or tachycardia. For unstable patients, milrinone may prove helpful and for cardiogenic shock temporary use of extracorporeal membrane oxygenation (ECMO) or other percutaneous mechanical circulatory support devices may be considered [57].

Acute toxin and drug-related cardiomyopathy

Rarely acute pulmonary edema results from drugs or toxins. Examples include ketamine [58] and scorpion envenomation [59,60]. Treatment of the latter includes antivenom, ventilatory and hemodynamic support and correction of hypertension if present.

Acute pulmonary edema in pregnancy

Pulmonary edema is a common cause of maternal mortality, occurring in 0.1–0.5% of pregnancies [61]. The majority occur prepartum and mortality can be as high as 8% if recurrent [62]. Fluid overload in the preceding 24 h was an important trigger, occurring in approximately a third of the patients. The physiology of normal pregnancy includes increases in CO, blood volume, heart rate and lower vascular resistance and oncotic pressure – increasing susceptibility to pulmonary edema. Pulmonary edema without maternal hypertension can result from: tocolytic drugs, iatrogenic intravenous fluid administration, underlying cardiac disease, sepsis, pregnancy-associated cardiomyopathy or amniotic fluid embolism. Magnesium sulfate, intravenous fluids and corticosteroid administration have been associated as pulmonary edema precipitants.

Pulmonary edema with hypertension can be related to gestational hypertension, preeclampsia or preeclampsia occurring in women with chronic hypertension. Acute pulmonary edema occurs in up to 3% of women with preeclampsia, usually postpartum. Fluids should be administered cautiously unless there are concerns about placental perfusion. If pulmonary edema occurs with prepartum eclampsia, then urgent fetal delivery should be considered.

In this group, reduction of the blood pressure and treatment of the pulmonary edema with diuretics and nitrates are high priorities. For severe hypertension and pulmonary edema, nitroprusside may need to be considered.

CONCLUSION

Acute cardiogenic edema is a relatively common life-threatening condition that remains a challenge condition because of many causes and ongoing uncertainties about the best use of new therapies.

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Conflicts of interest

There are no conflicts of interest.

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- of special interest
- of outstanding interest

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