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Uncommon causes of noncardiogenic pulmonary edema

Abstract: Although acute respiratory distress syndrome is the most common cause of noncardiogenic pulmonary edema, NPs also should be familiar with several other less common causes, including transfusion-related acute lung injury, neurogenic pulmonary edema, preeclampsia/eclampsia, opioid overdose, high-altitude pulmonary edema, and pulmonary embolism. This article addresses the pathophysiology, clinical presentation, diagnostics, treatment, and nursing considerations associated with each uncommon cause of noncardiogenic pulmonary edema.

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Patients are commonly admitted to the hospital with a general diagnosis of pulmonary edema. However, there are two specific types of pulmonary edema: cardiogenic and noncardiogenic. A similar set of clinical manifestations makes differentiating between the two types of pulmonary edema challenging. This article focuses on noncardiogenic pulmonary edema.

NPs can help improve patient outcomes by making a swift diagnosis, setting appropriate treatment goals, and beginning therapeutic interventions by identifying the specific cause of a patient's noncardiogenic pulmonary edema. The most common cause of noncardiogenic pulmonary edema is acute respiratory distress syndrome (ARDS); however, a thorough exploration of ARDS is beyond the scope of this article.¹ Instead, this article highlights some less common causes of noncardiogenic pulmonary edema, including transfusion-related acute lung injury (TRALI), neurogenic pulmonary edema, preeclampsia/eclampsia,

opioid overdose, high-altitude pulmonary edema, and pulmonary embolism. NPs should know the pathophysiology, clinical presentation, diagnostics, treatment, and nursing considerations associated with each cause of noncardiogenic pulmonary edema to achieve optimal patient outcomes.

■ Cardiogenic vs. noncardiogenic pulmonary edema

In cardiogenic pulmonary edema, high pulmonary capillary pressure estimated by pulmonary artery occlusion pressures is responsible for the abnormal excess fluid movement into the alveoli. Cardiogenic pulmonary edema is frequently caused by acute decompensated heart failure (ADHF).² Clinically, a patient diagnosed with cardiogenic pulmonary edema presents with dyspnea, decreased SaO₂, and alveolar infiltrates on chest imaging. This fluid accumulation is a result of acutely elevated cardiac filling pressures.² ADHF is predominately attributed to left ventricular systolic or diastolic dysfunction, with or without other associated cardiac

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pathology. This can include coronary artery disease or valve abnormalities. In the absence of underlying heart disease, causes of cardiogenic pulmonary edema can include fluid overload, severe hypertension, renal artery stenosis, and severe renal disease.²

In contrast, noncardiogenic pulmonary edema is often clinically recognized by the presence of alveolar fluid accumulation on chest imaging without hemodynamic evidence to suggest a cardiogenic etiology, such as a pulmonary artery occlusion pressure of 18 mm Hg



Noncardiogenic pulmonary edema occurs because of an alteration in the Starling forces.

or less (normal, 4 to 12 mm Hg). Noncardiogenic pulmonary edema occurs because of excessive pulmonary capillary permeability. Causes include excessive renin-angiotensin-aldosterone system activity, impaired nitric oxide synthesis, increased endothelin levels, and/or excessive circulating catecholamines.²

■ The Starling relationship

The Starling relationship describes the fluid balance between the interstitium and pulmonary vascular bed. The Starling forces include capillary surface area, capillary wall permeability, capillary and interstitial fluid hydraulic pressures, capillary and interstitial fluid oncotic pressures, and the pressure differential across the capillary wall. Noncardiogenic pulmonary edema occurs because of an alteration in the Starling forces resulting in increased capillary permeability and fluid and protein accumulation in the alveoli.³ In noncardiogenic pulmonary edema, the concentration of protein in the pulmonary interstitium exceeds 60% of the plasma protein level. By comparison, the concentration of protein in the pulmonary interstitium is typically less than 45% in cardiogenic pulmonary edema.⁴

■ TRALI

TRALI is defined as noncardiogenic pulmonary edema temporally related to the transfusion of blood products. This is a rare but potentially fatal problem of all plasma-containing blood products, such as packed red blood cells, whole blood, platelets, and fresh-frozen plasma. TRALI is described as a new acute lung injury occurring during or within 6 hours of a transfusion.⁵

Pathophysiology. TRALI is the leading cause of transfusion-related mortality in the US. TRALI is thought to occur based on a “two-hit” mechanism. The first “hit” is the underlying clinical status of the patient, such as shock, advanced age, liver transplantation, current smoking, chronic alcohol abuse, or higher peak airway pressure while being mechanically ventilated.⁵ The second “hit” is the transfusion of injurious blood products, which ensues because of passively transfused antibodies and biologically active lipids. This theory suggests that donor antibodies are transfused with the plasma-containing blood product. These antibodies attach to specific antigens on primed neutrophils, leading to the release of nonoxidative and oxidative products that harm the pulmonary endothelium and proceed to an increased permeability pulmonary edema.⁶

Clinical presentation. The clinical presentation of TRALI is characterized by the sudden onset of hypoxemic respiratory insufficiency shortly after or during the administration of a blood product.⁴ Symptom onset times differ. Most cases occur within minutes of initiating the transfusion. However, signs and symptoms can take 1 to 6 hours to develop.⁴ Common assessment findings in addition to hypoxemic respiratory insufficiency may include fever, chills, pruritus, urticaria, pulmonary infiltrates on chest radiography, pink frothy airway secretions, hypotension, hypovolemia, cyanosis, tachycardia, and tachypnea.⁴

Diagnostics. The National Heart, Lung, and Blood Institute (NHLBI) and the Canadian Consensus Conference have identified criteria that must be met for a diagnosis of TRALI. The signs and symptoms are as follows: 1) a new ARDS occurring during a transfusion or within 6 hours of a completed transfusion; 2) an abnormal chest X-ray with bilateral pulmonary infiltrates; and 3) documented hypoxemia. The hypoxemia must be authenticated by an SpO₂ of less than 90% on room air or a plasma PaO₂/FiO₂ ratio of less than 300 mm Hg (normal, 300 to 500 mm Hg). More recently, a consensus group has proposed subclassifying patients into TRALI Type I and TRALI Type II based on presence or absence of mild ARDS or risk factors for ARDS prior to the transfusion.⁷

Treatment and nursing considerations. The prompt diagnosis of TRALI is essential for patient survival. Immediately stopping the blood product transfusion is

the priority. Next, the clinician must assess the patient's vital signs, determine the extent of hypoxemia present, and obtain a chest X-ray. Pulse oximetry monitoring is adequate in most cases; however, an arterial blood gas analysis may be necessary in more severe cases.⁴ Based on the assessment findings, interventions may include supplemental oxygen in the form of continuous positive airway pressure therapy, bi-level positive airway pressure, or mechanical ventilation in isolated cases based on the degree of hypoxemia. It is important to note that there is little research-based evidence related to the management of TRALI with mechanical ventilation.⁸ In addition, the initiation of fluid resuscitation and/or vasopressor support to treat hypotension and/or hypovolemia may be necessary.^{9,10} The blood bank should be notified to initiate a transfusion reaction evaluation to help prevent further TRALI events.

■ Neurogenic pulmonary edema

Causes of neurogenic pulmonary edema require an injury or event of the central nervous system, including traumatic brain injury, cerebral hemorrhage, and seizure activity, especially status epilepticus.^{11,12}

Pathophysiology. The pathophysiology of neurogenic pulmonary edema alters the Starling forces, as described earlier. The shift of fluid from the capillaries to the pulmonary interstitium and alveoli thus increases the permeability of the pulmonary capillaries.¹²

Clinical presentation. Neurogenic pulmonary edema has a rapid onset of acute hypoxemia, tachypnea, and tachycardia. Auscultation of the lungs will reveal diffuse crackles, and the patient will produce large amounts of frothy pink sputum or bloody sputum. Signs and symptoms develop within 4 hours of the neurologic event, with most cases resolving within 48 to 72 hours. Patient outcomes depend on the clinical course of the primary neurologic insult.¹

Diagnostics. A definitive diagnosis of neurogenic pulmonary edema is difficult because the clinical signs and symptoms and routine diagnostic studies are non-specific. Therefore, a diagnosis is established based on the occurrence of pulmonary edema in the appropriate setting and in the absence of a more probable alternative cause. Published criteria for the diagnosis of neurogenic pulmonary edema include bilateral pulmonary opacities, amount of oxygen dissolved in the $\text{PaO}_2/\text{FiO}_2$ ratio less than 200 mm Hg, presence of central nervous system injury (with associated increased intracranial pressure), absence of other common causes of acute respiratory

failure or ARDS (aspiration, massive blood transfusion, sepsis), and no evidence of left atrial hypertension.¹³

Treatment and nursing considerations. As previously noted, outcomes for the patient with neurogenic pulmonary edema are more than often determined by the course of the neurologic insult and not the neurogenic pulmonary edema. As such, treatment interventions are focused on the neurologic disorder, whereas the neurogenic pulmonary edema is managed supportively as indicated. Many episodes of neurogenic pulmonary edema are well tolerated and resolve within 48 to 72 hours.¹ In general, noninvasive supplemental oxygen is required; however, mechanical ventilation may be necessary in some cases. Specific treatment for neurogenic pulmonary edema may also include interventions to decrease intracranial pressure.¹¹

■ Preeclampsia/eclampsia pulmonary edema

Women with preeclampsia are at increased risk for life-threatening events, including pulmonary edema and progression to eclampsia.¹⁴

Pathophysiology. The pathophysiology of noncardiogenic pulmonary edema attributed to preeclampsia and eclampsia is not entirely understood. It is thought to be the result of poor uteroplacental circulation caused by inadequate remodeling of the spiral arteries that happens between weeks 8 and 18 of pregnancy.¹⁵ The predominant pathophysiologic finding in preeclampsia and eclampsia is maternal vasospasm.¹⁶

Clinical presentation. The signs and symptoms of severe preeclampsia- or eclampsia-related pulmonary edema are like other types of pulmonary edema. Tachycardia, hypertension, dyspnea, tachypnea, hypoxemia, scattered crackles, chest pain, and/or cough may be exhibited.¹⁷ The gradual development of proteinuria and hypertension in the last half of pregnancy is typically caused by preeclampsia. The development of noncardiogenic pulmonary edema with the signs and symptoms of chest pain, dyspnea, and/or decreased oxygen saturation (SaO_2 of less than 93%) is prognostic of adverse maternal outcomes including death. Morbidity with renal, hepatic, central nervous system, cardiorespiratory, and hematologic complications may also occur.

Diagnostics. There is not a definitive diagnostic test for this type of noncardiogenic pulmonary edema. As with other types of noncardiogenic pulmonary edema, chest X-rays reveal bilateral pulmonary infiltrates.¹⁷ Diagnosis is made when these findings arise in a woman who has critical preeclampsia or eclampsia

without an alternative diagnosis that would explain the acute respiratory failure.

Treatment and nursing considerations. Supplemental oxygen and fluid restriction are included in the treatment of this type of noncardiogenic pulmonary edema. Diuresis is indicated for volume overload, but this must be done with caution. A decrease in cardiac output can cause inadequate placental perfusion due to intravascular hypovolemia.¹⁷ The goal of managing pulmonary edema in preeclampsia is to balance the management of both mother and fetus and to optimize the outcome for both.¹⁶

■ Opioid overdose

The literature documents that any opioid can cause noncardiogenic pulmonary edema, especially fentanyl mixed with heroin, and methadone overdose. Some investigators believe that noncardiogenic pulmonary edema is caused by naloxone administration; however, there is strong evidence that opioids alone can cause this lung pathology.^{3,18} Risk factors for this type of noncardiogenic pulmonary edema are male gender and a briefer time of heroin use. Most of these cases occur immediately or within hours of drug injection.

Pathophysiology. The pathophysiology of this form of noncardiogenic pulmonary edema is unknown. A combination of direct drug toxicity, hypoxia, and acidosis secondary to hypoventilation and/or cerebral edema has been proposed. The edema contains protein concentrations approximately identical to plasma; the pulmonary artery occlusion pressures are normal and suggest a leaking alveolar-capillary

decreased tidal volume, decreased bowel sounds, and miotic pupils.²⁰

Diagnostics. Acute opioid toxicity is a clinical diagnosis. Patients with pulmonary edema will frequently have a nonuniform distribution of pulmonary edema on chest imaging.²⁰ Urine toxicologic screens should not be routinely obtained and relied on when a patient presents to the ED with signs and symptoms of drug overdose. A positive urine toxicologic screen indicates recent use but does not confirm active toxicity. Conversely, many opioids, particularly the synthetic drugs, will cause false-negative urine screens. The management of a patient with an opioid overdose is unchanged by the results of a urine opioid test.²⁰

Treatment and nursing considerations. Respiratory support and, in most cases, the administration of naloxone are the mainstays of treatment. Normally, this form of pulmonary edema is rapidly corrected after treatment for the hypoventilation and hypoxia. Adjunct therapy may also include the administration of naloxone to reverse the opioid effects. Given the present opioid epidemic, clinicians are likely to witness an increase in the incidence of pulmonary edema secondary to opioid overdose.²¹

■ High-altitude pulmonary edema

High-altitude pulmonary edema (HAPE) generally occurs above 2,500 m (8,000 ft) and is uncommon below 3,000 m (10,000 ft). The risk depends on individual susceptibility, altitude attained, rate of ascent, and time spent at high altitude.²² Individuals usually affected by HAPE include skiers, mountain climbers, soldiers, hikers, sky divers, and rescue workers.²²

Pathophysiology. HAPE is the unusual accumulation of plasma and some red blood cells in the lung due to an interruption in the pulmonary blood-gas barrier. This is activated

by hypobaric hypoxia. This breakdown occurs from numerous maladaptive responses to the hypoxia experienced at higher altitudes. Some of the maladaptive responses include poor ventilatory response, exaggerated and uneven pulmonary vasoconstriction (pulmonary hypertension), increased sympathetic tone, inadequate production of endothelial nitric oxide, and excess of endothelin, many of these maladaptive responses are genetically determined. The consequence is a patchy accumulation of fluid in the alveolar spaces



Distinguishing between cardiogenic and noncardiogenic pulmonary edema is vital because treatment varies significantly.

membrane as the initiating cause. The problem with Starling forces resulting in increased capillary permeability and fluid accumulation in the alveoli is present as with the other types of noncardiogenic pulmonary edema.³

Clinical presentation. Patients with opioid-induced noncardiogenic pulmonary edema present with respiratory depression and/or agonal respirations.¹⁹ The classic signs of opioid intoxication include depressed mental status, decreased respiratory rate,

that impairs oxygen and carbon dioxide diffusion and can, in severe cases, prove fatal.²²

Clinical presentation. The clinical signs and symptoms associated with HAPE are subtle and appear gradually, usually over the course of 2 to 4 days after being introduced to the higher altitude. Initially, the patient will experience a nonproductive cough, shortness of breath with exertion, and difficulty walking uphill. Next, dyspnea at rest will become severe with any type of exertion. It is important to recognize that a cardinal clinical sign of HAPE is the early progression from dyspnea with exertion to dyspnea at rest. As symptoms progress, the patient's cough may be productive for pink, frothy sputum, as well as frank blood. At this point, the patient will be lethargic and severely hypoxemic. Other common assessment findings associated with HAPE are tachypnea, low-grade fever (up to 100.4° F [38° C]), tachycardia, and inspiratory crackles in the right middle lobe that become bilateral and diffuse as HAPE advances.²² Pulse oximetry is useful when assessing a patient for HAPE. For example, a patient may present with an SpO₂ of 40% to 50% and, surprisingly, not appear as debilitated as expected. Under these circumstances, once supplemental oxygen is administered, the SpO₂ often increases quickly, after only about 10 to 15 minutes. Of note, in most normal individuals, SpO₂ values are at their lowest point on the first day at high altitudes but increase over 4 days, usually 3 to 5 points higher than day 1 values.²²

Diagnostics. HAPE is typically diagnosed clinically based on the history and physical assessment findings. However, chest radiography will demonstrate characteristic patchy alveolar infiltrates predominantly in the right central hemithorax, which become more confluent and bilateral as the illness evolves.²²

Treatment and nursing considerations. Contrary to general knowledge, immediate descent is not mandatory in all situations. Descent is, however, highly recommended for patients experiencing HAPE at elevations higher than 4,000 m (13,123 ft). Alternatively, treatment of HAPE varies depending on several key factors, including setting, clinician experience, severity of illness, available treatments, and patient preference. Typical strategies used to address HAPE involve measures to decrease pulmonary artery pressures, improve oxygenation (supplemental oxygen or hyperbaric therapy), reduction of physical activity, decreasing cold exposure, and descent.²² The use of a portable hyperbaric chamber or administration

of supplemental oxygen immediately increases SaO₂ levels and reduces pulmonary artery pressures, heart rate, respiratory rate, and other associated signs and symptoms.²³

■ Pulmonary embolism (PE)

Acute pulmonary edema can be caused by a massive PE, as well as multiple smaller emboli.³

Pathophysiology. PE can cause noncardiogenic pulmonary edema by decreasing the pulmonary and adjacent pleural systemic circulations, raising hydrostatic pressures in pulmonary and systemic veins, and also decreasing pleural pressure due to airway collapse. PE also reduces the exit rates of pleural fluid by rising the systemic venous pressure thus limiting lymphatic drainage. Decreasing lymphatic filling can also occur with a PE by diminishing pleural pressure.³

Clinical presentation. Clinical assessment findings in a patient experiencing PE are varied, ranging from nonspecific symptoms, to no symptoms, to shock or sudden death. However, patients will frequently initially present with dyspnea at rest or with exertion, followed by chest pain and a cough.²⁴

Diagnostics. In most cases of PE, the chest X-ray is normal or nonspecific, including infiltrates, pleural effusion, atelectasis, and consolidation. However, in PE-associated pulmonary edema, chest imaging demonstrates effusions that are typically small and unilateral and may become loculated if the diagnosis is delayed. If PE is suspected, further work-up must be undertaken, such as use of clinical prediction rules to determine the probability of a PE, potentially followed by D-dimer testing and computed tomography pulmonary angiography or V/Q scanning.²⁵

Treatment and nursing considerations. The mainstay of treatment for PE is anticoagulation. Massive pulmonary embolus may warrant fibrinolytic therapy or thrombectomy. Treating the pulmonary embolus will also treat the noncardiogenic pulmonary edema.²⁶ If the patient is unstable, the initial emphasis of treatment is stabilization, which often requires respiratory and hemodynamic support. The patient may require mechanical ventilation, fluid resuscitation, I.V. vasopressor therapy, and inotropic support.²⁷

■ Conclusion

The distinction between noncardiogenic and cardiogenic pulmonary edema is, at times, difficult. The clinical syndrome, along with signs and symptoms of pulmonary

edema, may represent several different disorders.³ Distinguishing between cardiogenic and noncardiogenic pulmonary edema is vital because treatment varies significantly depending on the fundamental pathophysiologic mechanisms. **NP**

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