

## CJON BOOK EXCERPT SERIES

# Pulmonary Edema

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This excerpt, chapter 32 from the book *Clinical Manual for the Oncology Advanced Practice Nurse* (2nd ed.), edited by Dawn Camp-Sorrell, MSN, FNP, AOCN®, and Rebecca A. Hawkins, MSN, ANP, AOCN®, is a part of a series of clinically relevant reprints that appear regularly in the *Clinical Journal of Oncology Nursing*.

- I. Definition: Clinical syndrome resulting in leakage of fluid from the pulmonary capillaries and veins into the interstitium and alveoli of the lungs (Dada & Sznajder, 2003)
- II. Physiology/Pathophysiology (Cotter, Kaluski, & Vered, 2005)
  - A. Normal (Cotter et al., 2005)
    1. According to Starling's Law, the flow of fluid from the pulmonary capillaries to the lungs equals the removal of fluid by pulmonary lymphatics.
    2. To prevent pulmonary edema, an active process of sodium (Na<sup>+</sup>) transport by an osmotic process removes fluid from the alveoli as a protective mechanism.
  - B. Pathophysiology
    1. Pulmonary edema occurs when fluid accumulates in the alveolar spaces.
    2. Cardiogenic pulmonary edema may occur because of an increase in the hydrostatic pressure gradient associated with an altered hemodynamic status, such as in CHF. An elevated PCWP occurs because of left ventricular dysfunction (Cotter et al., 2005).
    3. Noncardiogenic pulmonary edema is caused by leakage of fluid from pulmonary capillaries with a decrease in plasma oncotic pressure and elevation of capillary pressure, producing interstitial and intra-alveolar edema.
      - a) This type of pulmonary edema may be caused by several clinical conditions that result in fluid and protein accumulation in the alveoli.
      - b) May be caused by many clinical conditions that cause injury to the lungs, such as acute respiratory distress syndrome and inflammatory damage to the alveolar capillary membrane (Cotter et al., 2005)
    4. Neurogenic pulmonary edema may occur because of head trauma, intracranial or subarachnoid hemorrhage, and some neurologic disorders (Cotter et al., 2005).
    5. Decreased lung and small airway compliance may result in altered ventilation, hypoxia, and respiratory failure.
    6. Hypoxia reduces the ability to actively transport Na<sup>+</sup>, leading to alveolar edema (Dada & Sznajder, 2003).
- III. Clinical features (Cotter et al., 2005)
  - A. Etiology: See Table 32-1.
  - B. Types of pulmonary edema (see Table 32-1)
  - C. History
    1. History of cancer and cancer treatment
    2. Current medications: Prescribed and over-the-counter
    3. History of presenting symptom(s): Precipitating factors, onset, location, and duration
    4. Changes in ADLs
  - D. Signs and symptoms
    1. Dyspnea, orthopnea
    2. Anxiety or feeling of impending doom
    3. Frothy-pink or salmon-colored sputum
    4. Cough
    5. Cyanosis, pallor
    6. Diaphoresis
    7. Unable to lie flat
    8. Poor prognostic symptoms (Cotter et al., 2005)
      - a) Hypoxia
      - b) High or low blood pressure
      - c) High heart rate
      - d) High respiratory rate
      - e) Increased PCWP
  - E. Physical exam
    1. Vital signs
      - a) Tachypnea
      - b) Tachycardia
      - c) Hypotension
    2. Integument exam: Skin pallor and livedo reticularis (skin discoloration with mottled appearance)
    3. Pulmonary exam

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**Table 32-1. Description of Types of Pulmonary Edema**

TYPE OF PULMONARY EDEMA	ETIOLOGY	CLINICAL FEATURES	HEMODYNAMIC PARAMETERS	DIAGNOSTIC STUDIES
Cardiogenic	Episode of acute heart failure Myocardial infarction Minor ischemia Hypertensive event Cardiomyopathy Severe dysrhythmias Renal artery stenosis Cold immersion	Harsh heart murmurs History of heart failure History of valvular or ischemic heart disease Severe respiratory distress Oxygen saturation < 90% on room air	Increased PCWP Increased BNP plasma levels Increased LV pressures Reduced LV contractility Increased SVR Increase in troponin and creatinine kinase	Chest x-ray—enlarged heart EKG abnormal
Noncardiogenic	ARDS Infection Shock Toxic damage Postoperative Pregnancy related Transfusion associated Excess fluid accumulation Inhaled drugs, substances, and toxins High altitudes Exertional	Hypoxia Severe respiratory distress	Increased PCWP	Chest x-ray—bilateral consolidative pattern, interstitial edema, diffuse infiltrates
Neurogenic	Head injury Intracranial hemorrhage Subarachnoid hemorrhage Seizures Tumors Hydrocephalus Neurosurgical procedures	Highly variable	Increased SVR Decreased LV contractility Increased alveolar capillary leakage	—

ARDS—acute respiratory distress syndrome; BNP—brain natriuretic peptide; EKG—electrocardiogram; LV—left ventricle; PCWP—pulmonary capillary wedge pressure; SVR—systemic vascular resistance

Note. Based on information from Cotter et al., 2005.

- a) Abnormal breath sounds with occasional wheezing
- b) Abnormal breathing pattern with use of accessory muscles
- 4. Cardiac exam
  - a) Pulsus alternans, alternating weak and strong pulse, may be a sign of left ventricular failure in CHF
  - b) Heart sounds with presence of S3, S4, and harsh murmur
  - c) JVD
  - d) Peripheral edema of extremities

IV. Diagnostic tests (see Table 32-1)

A. Laboratory

- 1. ABGs
  - a) Hypoxia (oxygen saturation < 90% and PaO<sub>2</sub> < 60 mm Hg)
  - b) Hypercapnia (CO<sub>2</sub> > 45–55 mm Hg)
  - c) Acidosis (pH < 7.35 nEq/liter). Early findings of pulmonary edema may be respiratory alkalosis because of hyperventilation.
- 2. Serum albumin: May be low (normal = 3.6–5 g/dl)
- 3. Blood urea nitrogen and creatinine to evaluate kidney function to ensure that renal perfusion is occurring

- 4. Liver function tests to evaluate hepatic function (elevation in alanine aminotransferase, aspartate aminotransferase and bilirubin are seen with right ventricular failure and hepatic congestion)
- 5. Plasma brain natriuretic peptide levels may be increased in cardiovascular pulmonary edema (Cotter et al., 2005).

B. Radiography (see Table 32-1): Chest x-ray

- 1. Noncardiogenic: Bilateral consolidative pattern, interstitial edema
- 2. Cardiogenic: Diffuse bilateral infiltrates, cardiomegaly

C. Other: Hemodynamic monitoring

- 1. Pulmonary arterial pressure measurements with elevated PCWP (normal = 6–12 mm Hg)
- 2. Systemic vascular resistance may be elevated (normal = 800–1,200 dynes/sec/m<sup>2</sup>)
- 3. Electrocardiogram (EKG): Abnormal in cardiogenic induced

V. Differential diagnosis: Table 32-1 lists common causes of pulmonary edema.

- A. CHF (see Chapter 40)
- B. Obstruction of pulmonary lymphatics by tumor compression

- C. CLS (see Chapter 27)
  - D. Acute respiratory distress syndrome
  - E. Early phase of septic shock (see Chapters 121 and 140)
- VI. Treatment: Goal is to decrease pulmonary venous and capillary pressure, improve cardiac output, and correct underlying pathology. Patient is hospitalized for management (Cotter et al., 2005).
- A. Drug therapy
    1. Use of loop diuretics (e.g., furosemide, bumetanide, torsemide) causes vasodilation and decreases pulmonary congestion. Doses of 0.5–1 mg/kg may be used.
    2. Administer metolazone (thiazide diuretic) 5–20 mg po once a day for treatment of CHF.
    3. Vasodilators cause vasodilation, therefore decreasing pulmonary vascular pressure.
      - a) Nitroprusside is started at an IV infusion of 0.5 mcg/kg/minute and titrated to achieve the desired effect; average dose is 0.5–0.8 mcg/kg/minute.
      - b) Nitroglycerin is started at an IV infusion of 10–20 mcg/minute, and the dose is increased by 5–10 mcg/minute every 5–10 minutes until the desired effect occurs.
    4. Morphine sulfate may be given to cause venous dilation at doses of 1–3 mg IV push. The dose is repeated every two to three hours as needed up to a total of 10–15 mg.
    5. Aminophylline may be given at 5 mg/kg IV infusion for symptoms of wheezing.
    6. In patients with ventilator-induced lung injury, dopamine and beta-adrenergic agonists (terbutaline and isoproterenol) improved clearance of pulmonary edema (Dada & Sznajder, 2003).
    7. Dysrhythmia control
  - B. Oxygen therapy
    1. Oxygen therapy often is used, and dose is titrated

to patient response. Intubation and mechanical ventilation may be necessary.

2. Continuous positive airway pressure (PAP) and bilevel-PAP have been found to be superior to oxygen therapy in decreasing the need for mechanical ventilation (Park et al., 2004).
- C. Swan Ganz catheter may be placed to evaluate the cause of pulmonary edema.
  - D. Position patient in semi-Fowler position.
  - E. Obtain daily weight to monitor fluid status.
  - F. Obtain frequent intake and output measurements.
- VII. Follow-up
- A. Daily physical assessment and evaluation of response to treatment is necessary.
  - B. Diagnostic studies, such as chest x-ray and ABGs, may be done to evaluate patient condition.
- VIII. Referrals
- A. Pulmonary consult: To evaluate lung status and assist with medical management as needed
  - B. Respiratory therapy: May be needed for oxygen therapy, ventilatory management, percussion, or respiratory treatments

## References

- Cotter, G., Kaluski, E., & Vered, Z. (2005). Pulmonary edema. In M.P. Fink, E. Abraham, J.L. Vincent, & P.M. Kochanek (Eds.), *Textbook of critical care* (5th ed., pp. 719–734). Philadelphia: Elsevier.
- Dada, L.A., & Sznajder, J.I. (2003). Mechanisms of pulmonary edema clearance during acute hypoxemia respiratory failure: Role of the Na,K-ATPase. *Critical Care Medicine*, 31(Suppl.), S248–S252.
- Park, M., Sangean, M., Volpe, M.S., Feltrim, M., Nozawa, E., Leite, P., et al. (2004). Randomized, prospective trial of oxygen, continuous positive airway pressure, and bilevel positive airway pressure by face mask in acute cardiogenic pulmonary edema. *Critical Care Medicine*, 32, 2407–2415.