Pulmonary Edema

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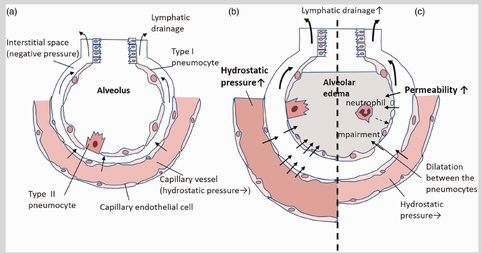
Introduction

Pulmonary edema, characterized by the abnormal accumulation of fluid in the lungs, poses a significant threat to respiratory function. This particular case explores the aftermath of negative pressure pulmonary edema, shedding light on its clinical significance and the typical demographic of patients susceptible to this condition. Understanding the complexities of such cases is paramount for effective clinical management and underscores the need for heightened awareness within the medical community.

Learning Objectives

- Review Basic Alveolar Anatomy
- Recognize signs and symptoms of Pulmonary Edema
- Identify treatment options for Negative Pressure Pulmonary Edema
- Differentiate between Cardiogenic Pulmonary Edema and Non-Cardiogenic Pulmonary Edema

Pulmonary Anatomy



Oxygen-depleted blood from the pulmonary arteries is directed to the alveoli, where it undergoes gas exchange—carbon dioxide is released, and oxygen is absorbed. Oxygenated blood then returns to the heart through the pulmonary veins, facilitating systemic distribution of oxygen-rich blood throughout the body

Types of Pulmonary Edema

Cardiogenic Pulmonary Edema:

- **Heart Failure-Related Edema:** The most common cause of pulmonary edema is heart failure, where the heart's pumping ability is compromised. This can lead to increased pressure in the pulmonary veins, resulting in the leakage of fluid into the lung tissues.
- **Valvular Heart Disease:** Conditions such as mitral valve stenosis or regurgitation can contribute to increased pressure in the pulmonary vasculature, causing pulmonary edema.
- **Ischemic Heart Disease:** Myocardial infarction or coronary artery disease may weaken the heart muscle, leading to fluid accumulation in the lungs.

Non-Cardiogenic Pulmonary Edema:

- **Acute Respiratory Distress Syndrome (ARDS):** Often caused by severe infections, trauma, or inhalation injuries, ARDS results in inflammation and increased permeability of the lung capillaries, leading to non-cardiogenic pulmonary edema.
- **High-Altitude Pulmonary Edema (HAPE):** Exposure to high altitudes can trigger HAPE, characterized by fluid accumulation in the lungs due to increased pulmonary artery pressure.
- **Neurogenic Pulmonary Edema:** Certain neurological events, such as seizures, head trauma, or brain hemorrhage, can induce sudden increases in sympathetic activity, resulting in non-cardiogenic pulmonary edema.
- **Negative Pressure Pulmonary Edema (NPPE):** Caused by upper airway obstruction or inspiratory effort against a closed glottis, NPPE results in a vacuum effect leading to fluid transudation into the pulmonary interstitium.

Case: Total Shoulder Arthroplasty

Total Shoulder Arthroplasty
Plan: General Anesthesia with Interscalene Block
Case Proceeded unremarkably

- Height: 5 ft 11 in
- Weight: 98 kg
- BP: 145/95 HR 67bpm RR: 20
- Male
- Age: 36
- Medical history:
  - HTN
  - OSA
  - Red hair

Emergence

- Patient prior to NMB reversal recorded ¾ TOF
- Patient was reversed with 5 mg Neostigmine and 1mg of Glycopyrrolate
- Post reversal patient recorded 4/4 TOF
- Tidal volumes: 400-500 ml
- Respiratory rate: 12 bpm
- HR: 65 bpm
- spO2: 100% at FiO2 of 100%
- etSEV: 0.1 MAC
- Patient able to open eyes and make movements on command
- Suction occurred and ETT removed
  - Small laryngospasm broken with positive pressure
- spO2: 95% at FiO2 via Simple Facemask at 100%, 8 L/min fresh gas flows

Transport/PACU arrival

Transport:

- Patient SVing
- Patient talking

PACU

- Difficulty holding saturation
- Talking became labored as well as breathing
- Saturation decreased to 85% from 92% on arrival to PACU
- Saturation dropped to 70%
- Lungs positive for crackles

Treatment

- ETT intubation with 100 mg Propofol and 100 mg anectine
  - Foam emerged from tube
- Ambu Bag circuit with 10 L/min 100% O2
- 20mg Lasix IV
- Foley cather Placed
- X-ray ordered

Response to Treatment

- 10 minutes post intubation:
- spO2 90%
- Urine output: 40 mL
- X-ray: reveal fluid in lungs
- 120 minutes later patient was extubated
- 240 minutes later patient was discharged

Discussion

Negative pressure pulmonary edema presents with a respiratory distress, pink frothy sputum, and audible inspiratory efforts. They can result after an episode of airway obstruction. Prompt recognition and treatment is essential for safe management of the patient.

In this case, the patient experience a brief laryngospasm post extubation. Though the spasm was treated the effects were profound as in their effort to breath during the spasm fluid entered the lungs decreasing the ability of the patient to properly exchange gases.

Treatment included re-intubation, increased fresh gas flows, and diuretics (Lasix). Chest X-ray confirmed our diagnosis. Patient's symptoms subsided and was safely discharged later that day.

Understanding the signs and symptoms allowed for the anesthesia team to act swiftly and appropriately.

Take Away Points

- Laryngospasm can cause NPPE
- Cardiogenic and Non-cardiogenic PE have different etiologies
- Quick and appropriate management is important for positive outcomes

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