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PATHOPHYSIOLOGY OF TUMORS FOUND IN THE LUNGS

Annotatsiya: this article provides information on edema found in the lungs. you can also get enough information about what Origin this condition is and the pathophysiology of flour. The article was written in a state of widespread use of internet resources.

Key words: pulmonary edema, blood, rhythm, cardiomyopathy.

Pulmonary edema can be broadly classified into cardiogenic and noncardiogenic pulmonary edema.

Cardiogenic or volume-overload pulmonary edema arises due to a rapid elevation in the hydrostatic pressure of the pulmonary capillaries. This is typically seen in disorders involving left ventricular systolic and diastolic function (acute myocarditis including other etiologies of non-ischemic cardiomyopathy, acute myocardial infarction), valvular function (aortic/mitral regurgitation and stenosis in the moderate to the severe range), rhythm (atrial fibrillation with a rapid ventricular response, ventricular tachycardia, high degree, and third-degree heart block).

Noncardiogenic pulmonary edema is caused by lung injury with a resultant increase in pulmonary vascular permeability leading to the movement of fluid, rich in proteins, to the alveolar and interstitial compartments. Acute lung injury with severe hypoxemia is referred to as acute respiratory distress syndrome (ARDS) and is seen in various conditions directly affecting the lungs, such as pneumonia, inhalational injury, or indirectly, such as sepsis, acute pancreatitis, severe trauma with shock, multiple blood transfusions.

The resultant pathology of increased extravascular fluid content in the lung remains common to all forms of pulmonary edema. However, the underlying mechanism leading to the edema arises from the disruption of various complex physiologic processes, maintaining a delicate balance of filtration of fluid and solute across the pulmonary capillary membrane. This imbalance can be from one or more of the following factors:

- Increase in intravascular hydrostatic pressure transmitted in a retrograde fashion to the pulmonary microvasculature
- Increase in interstitial hydrostatic pressure
- Endothelial injury and disruption of epithelial barriers
- Decrease in oncotic pressure due to underlying hepatic, renal, malnutrition, and other protein-losing states.
- Lymphatic insufficiency
- Increased negative interstitial pressure

The relationship between hydrostatic and oncotic forces in relation to net fluid filtration is best explained by Ernest Starling's equation. The rate of fluid filtration is determined by the differences in the hydrostatic and oncotic pressures between the pulmonary capillaries and interstitial space.

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Progressively worsening dyspnea, tachypnea, and rales (or crackles) on examination with associated hypoxia are the clinical features common to both cardiogenic and noncardiogenic pulmonary edema.

Cough with pink frothy sputum noted due to hypoxemia from alveolar flooding and auscultation of an S3 gallop could suggest cardiogenic edema. Similarly, the presence of murmurs, elevated jugular venous pressure, peripheral edema may point towards a cardiac etiology.

In patients with non-cardiogenic pulmonary edema, the symptoms of infections such as fever, cough with expectoration, dyspnea pointing to likely pneumonia, recent trauma, blood transfusions should be carefully assessed as these patients may progress to acute respiratory distress syndrome.

Auscultation remains the mainstay of bedside assessment in all patients with respiratory symptoms. More specifically, hearing of either fine or coarse crackles is crucial to determine the next steps in the management. Fine crackles are heard in cardiogenic pulmonary edema. They are exclusively heard in the inspiratory phase when the small airways, which were shut during expiration, open abruptly.

In addition to a thorough history and physical examination, electrocardiogram assists in diagnosing cardiac ischemia or myocardial infarction. It is a quick, inexpensive, and relatively less specialized test that can be done at the bedside.

Following are a variety of diagnostic tools utilized to help diagnose pulmonary edema and, more importantly, differentiate between its different types.

Laboratory Testing

Brain-type natriuretic peptide (BNP) is secreted by the cardiac myocytes of the left ventricles in response to stretching caused by increased ventricular blood volume or increased intracardiac pressures. Elevated BNP levels correlate with left ventricular end-diastolic pressure as well as pulmonary occlusion pressure and can be seen in patients with congestive heart failure. BNP levels less than 100 pg/ml suggest heart failure is less likely, and levels greater than 500 pg/ml suggest a high likelihood of heart failure. Levels between 100 and 500 pg/ml do not help in the diagnosis of heart failure and are often seen in critically ill patients.

Troponin elevation is commonly noted in patients with damage to myocytes, such as acute coronary syndrome. They, however, are also noted to be elevated in patients with severe sepsis.

Hypoalbuminemia (≤3.4 g/dL) is an independent marker of increased in-hospital and post-discharge mortality for patients presenting in acute decompensated heart failure. Low albumin in isolation does not lead to pulmonary edema as there is a concurrent drop in pulmonary interstitial and plasma albumin levels preventing the creation of a transpulmonary oncotic pressure gradient.

Obtaining serum electrolyte levels, including renal function, serum osmolarity, toxicology screening, help in patients with pulmonary edema due to toxic ingestion. Obtaining lipase and amylase levels help diagnose acute pancreatitis.

Radiographic Testing

Both posteroanterior and lateral views in standard imaging or anteroposterior views in portable imaging are utilized. Cardiogenic pulmonary edema is characterized by the presence of central edema, pleural effusions, Kerley B septal lines, peribronchial cuffing, and enlarged heart size. In noncardiogenic etiologies, the edema pattern is typically patchy and peripheral that can demonstrate

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the presence of ground-glass opacities and consolidations with air bronchograms.[10] Pleural effusions are more commonly seen in the cardiogenic type.[1]

Echocardiography

Assists in the diagnosis of left ventricular systolic dysfunction and valvular dysfunction. Through modalities, including tissue Doppler imaging of the mitral annulus, the presence and degree of diastolic dysfunction can be assessed.

Lung Ultrasound

A newer technique that is non-invasive and does not involve radiation exposure. It is most commonly used in intensive care units, emergency rooms, and operating rooms. It helps detect the accumulation of extravascular lung water (EVLW) ahead of the clinical manifestations.

Pulmonary Artery Catheterization

Often considered a gold standard in the determination of the etiology of pulmonary edema, it is an invasive test that helps monitor systemic vascular resistance, cardiac output, and filling pressures. An elevated pulmonary artery occlusion pressure over 18 mm Hg is helpful in the determination of cardiogenic pulmonary edema.

Transpulmonary Thermodilution

It is an invasive testing modality performed in patients typically undergoing major cardiac, vascular, or thoracic surgeries. They are also used in septic shock and monitors several hemodynamic indices such as cardiac index, mixed venous oxygen saturation, stroke volume index, and EVLW.

Pulmonary edema is an acutely decompensated state due to either cardiac or noncardiac etiologies. Temporizing measures such as supplemental oxygenation, diuretics, nitrates, and morphine help manage dyspnea, hypoxemia. However, definitive management of the underlying causes is necessary to prevent its recurrences. Prognostic predictions are difficult to quantify, given the vast number of cardiogenic and non-cardiogenic etiologies of pulmonary edema and their individual mortality data. Pulmonary edema's advanced state in ARDS has had progressively improved outcomes. Hospital mortality has decreased from 60% from 1967 through 1981 to the range of 30% to 40% in the 1990s.. Furthermore, analysis of ARDS mortality studies demonstrated a decline in overall mortality of about 1.1% per year from 1994 to 2006. Prognosis utilizing mortality data is largely variable and depends on the precipitating process of ARDS.

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