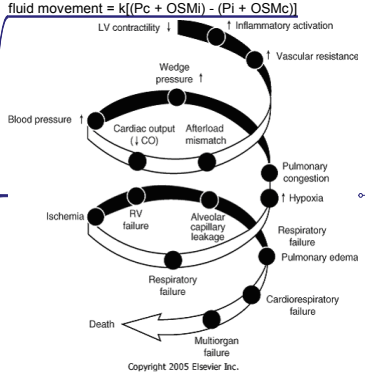
cardiogenic pulmonary oedema

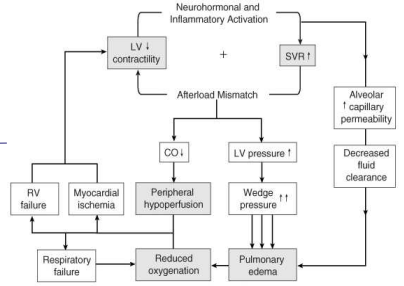
definition

- Pulmonary edema is a life-threatening syndrome caused by accumulation of fluid within the alveoli leading to disruption of the normal gas exchange process, severe hypoxemia, failure of tissue oxygenation, acidosis, and widespread organ failure; if untreated, it rapidly progresses to death.

pathophysiology

fluid movement = k[(Pc + OSMi) - (Pi + OSMc)]





clinical features

- pulmonary oedema presents as shortness of breath, orthopnoea, PND, & a cough productive of pink frothy fluid
- clinical findings are of fine inspiratory crackles on auscultation
- gas exchange is impaired

CLINICAL SYMPTOMS AND SIGNS OF DETERIORATED CHRONIC HEART FAILURE VERSUS "TRANSIENT" PULMONARY EDEMA

	"Transient" Pulmonary Edema	Acute Decompensation of Chronic Heart Failure
Chronic heart failure symptoms (dyspnea/fatigue)	+	++++
Treatment with loop diuretics	+	+++
Peripheral edema	+	+++
Gain in body weight	+	+++
Reduced ejection fraction (echo)	+	+++
Neurohormonal activation (endothelin)	+++	++
Aggravating factor	+	+++

COMMON AGGRAVATING FACTORS LEADING TO DECOMPENSATION OF CHRONIC HEART FAILURE

Acute fluid and/or salt intake (diet noncompliance)
Medical treatment noncompliance
Acute ischemia or myocardial infarction
Sepsis or other infections (mostly upper respiratory tract)
Significant arrhythmias (atrial tachycardia, fibrillation or flutter, ventricular tachycardias, bradyarrhythmias)
Pulmonary embolism
Anemia
Hyperkalemia
Acute renal failure

causes

Reduced Left Ventricular Contractility
Severe acute myocardial ischemia
Valvular or mechanical factors
Cardiomyopathies (dilated, hypertrophic, restrictive)
Myocarditis
Cardiodepressant drugs (beta-adrenergic blockers, calcium channel blockers, antiarrhythmics)
Severe arrhythmias
Increased Systemic Vascular Resistance
"Flush" hypertensive pulmonary edema
Renal artery stenosis
Pheochromocytoma, hypertensive crisis
Neurogenic pulmonary edema?
Cold immersion pulmonary edema?

general approach

1.Establish the diagnosis of pulmonary edema.
2.Determine whether pulmonary edema is the result of acute ischemia.
3.Determine the severity of pulmonary edema.
a.Baseline characteristics:
- Older age, male sex, lower weight, hyponatremia, and reduced hemoglobin and renal function have been correlated with worse outcome.
b.Findings on admission:
- as arterial oxygen saturation decreases, the chances of respiratory failure increase and the patient's prognosis becomes worse.
- Admission blood pressure is also an important sign of disease severity; however, its correlation with outcome is "U"-shaped.
- Higher admission blood pressure is usually correlated with higher vascular resistance and, hence, worse outcome.
- On the other hand, low blood pressure (<120 mm Hg systolic) on admission is correlated with decreased left ventricular contractility and is a negative prognostic sign.
- higher pulse rate and respiratory rate at admission were correlated with increased rate of adverse events in patients admitted with pulmonary edema.
c. Cardiac contractility:
- Reduced echocardiographic ejection fraction can be used as a measure of left ventricular contractility.
d. Measures of neurohormonal activation:
- In patients with chronic heart failure, neurohormonal and inflammatory measures have been shown to be correlated with disease severity and outcome.
- The data on such measures in patients with acute heart failure and pulmonary edema are limited. However, it has been suggested that higher admission endothelin level is the mediator associated with worst outcome, whereas admission BNP is of limited value.
4. Determine whether the patient suffers from chronic heart failure that has deteriorated due to aggravating factors.
5. Determine whether pulmonary edema is related to preserved echocardiographic ejection fraction or low ejection fraction
6. Rule out significant valvular and mechanical cardiac causes of pulmonary oedema.

specific investigation

- chest x-ray appearances include:
(i) distended upper lobe veins
(ii) Kerley B lines which are short horizontal lines at the periphery of the lower zone
(iii) Kerley A lines are long fine lines in the upper zone radiating from the hilum
(iv) diffuse hazy shadowing from the hilar region ('bat's wing')
(v) small pleural effusions
(vi) cardiac enlargement

INITIAL EVALUATION OF PATIENTS WITH SUSPECTED CARDIOVASCULAR PULMONARY EDEMA

Immediate Work-Up
History of heart failure or regular intake of loop diuretics, previous myocardial infarction, or known significant valvular disease
Physical examination: check blood pressure, temperature, signs of peripheral edema, and cardiac and pulmonary physical findings
Arrhythmia monitoring
Pulse oximetry
12-Lead electrocardiogram
Chest radiograph
Advanced Work-Up
Complete blood gas analysis
Laboratory evaluation (complete blood cell count, electrolytes, urea/creatinine, creatine kinase, troponin)
Brain natriuretic peptide measurement (if available)
Echocardiographic evaluation
Right-sided heart catheterization

immediate stabilisation

1. Improving Systemic Oxygenation
2. Arrhythmia Control
3. Intravenous Furosemide and Morphine
4. Intravenous Nitrates
- Intravenous nitrates should be administered to all patients with cardiogenic pulmonary edema with a systolic blood pressure of greater than 120 mm Hg on admission.
- Intravenous nitrates are the only treatment modality that has been shown in a prospective randomized study to improve the outcome of patients admitted with pulmonary edema by averting respiratory failure and reducing the need for mechanical ventilation.
5. Mechanical Ventilation
- Patients not responding to medical treatment including non invasive ventilation within 10 to 20 minutes, as evident by an increase in arterial oxygen saturation to more than 90%, accompanied by decreased tachypnea and blood pressure, are suffering from refractory pulmonary edema and should be treated by mechanical ventilation.

first 24 hours

General
- After the initial stabilization, as vascular resistance decreases toward normal values, cardiac index increases, and wedge pressure decreases, the main goals of treatment shift from rapid arterial and venous dilatation to preventing recurrence.
- Because decreased left ventricular contractility and increased systemic vascular resistance are important in the pathogenesis of pulmonary edema, traditional treatments employed during this time period are directed toward rapid increase in left ventricular contractility, prevention of recurrent vasoconstriction, and enhancement of diuresis.

1. Increasing Left Ventricular Contractility.
(i) Repair of Significant Valvular and Mechanical Lesions.
(ii) Treatment of Severe Acute Ischemia.
(iii) Positive Inotropes.
- these agents, although effective in the short term in relieving dyspnea and normalizing hemodynamic measurements, may not be effective in the long-term outcome of patients with acute heart failure or may even be harmful).
- Thus, the administration of these agents should be reserved for refractory cases of primary pump failure (refractory heart failure accompanied by low ejection fraction or low cardiac power output) in which recurrent episodes of heart failure exacerbation cannot be prevented by conventional therapy.

2. Preventing Recurrent Vasoconstriction.
(i) vasodilators:
- This goal has been traditionally achieved by the administration of vasodilators. This class of drugs currently includes nitrates and nitroprusside. The main obstacle in the use of such agents for a prolonged period of time is the rapid development of tolerance, limiting their effectiveness to 16 to 24 hours only.
(ii) Diuretics.

after the first 24 hours

- As a patient's condition stabilizes, long-term medical treatment should be established.
- In patients with obvious ischemia, coronary angiography and revascularization should be performed as soon as possible.
- Patients without clinical evidence of ischemia should be scheduled for a noninvasive test to assess the presence of ischemia and viability, either by radionuclide techniques or by dobutamine stress echo.
- oral medical treatment aimed at preventing repeated episodes of vasoconstriction should be administered. These drugs should be initiated at low doses and gradually titrated upward to achieve maximal effect while preventing excessive vasodilatation and hypotension. At present, only angiotensin-converting enzyme (ACE) inhibitors have been shown to be effective in this respect.
- beta-adrenergic blockers, which are extremely beneficial in the long-term treatment of heart failure, should not be administered to patients with acute heart failure until the patient's condition has stabilized.

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