Acute Cor Pulmonale

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ABSTRACT

In physiological conditions the left heart ventricle produces a higher pressure which aims to pump blood throughout the body while the right heart ventricle has a lower pressure to pump blood to the lungs. If pulmonary hypertension or increased blood pressure in the pulmonary arteries occurs, it can cause the right ventricle of the heart to work harder to pump blood against the high pressure. If there is pulmonary hypertension in the long term so that the right ventricle of the heart is burdened, it will cause cor pulmonale. Cor pulmonale is hypertrophy or dilatation of the right ventricle caused by pulmonary hypertension. Pulmonary hypertension is caused by diseases that attack the structure, function of the lungs or pulmonary blood vessels that can progress to right heart failure. Cor pulmonale can be acute or chronic. This literature review in made to know the theoretical concept of patients with cor pulmonale disorders including definitions, epidemiology, pathophysiology, clinical features, interventions, and prognosis and complications in acute cor pulmonale patients.

Keywords: Cor Pulmonale, Pulmonary Hypertension, Pulmonary Embolism.

INTRODUCTION

In physiological conditions the left heart ventricle produces higher pressure which aims to pump blood throughout the body while the right heart ventricle has lower pressure to pump blood to the lungs. If pulmonary hypertension or increased blood pressure in the pulmonary arteries occurs, it can cause the right ventricle of the heart to work harder in pumping blood against the high pressure (Bayoumi et al., 2020; Beyls et
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If there is pulmonary hypertension for a long time so that the right ventricle of the heart is burdened, it will cause cor pulmonale (Cavaleiro et al., 2021; Creel-Bulos et al., 2020; Dong et al., 2018). *Cor pulmonale is hypertrophy or dilatation of the right ventricle caused by pulmonary hypertension* (Kuwahata et al., 2023; Sosa-Villarreal & Arce-Carréon, 2020; Zhao et al., 2022). Pulmonary hypertension is caused by diseases affecting the structure, function of the lungs or pulmonary blood vessels that can progress to right heart failure (Charron et al., 2021; Luque et al., 2021; Stein et al., 2020; Zapata et al., 2022). *Cor pulmonale can occur acutely or chronically. In acute cor pulmonale there is dilatation of the right ventricle of the heart, while chronic cor pulmonale generally occurs hypertrophy of the right ventricle of the heart* (Ali et al., 2020; Krishnan et al., 2022; Su et al., 2019) (S. Adiyanti, 2019). This paper is made to find out the theoretical concepts of patients with cor pulmonale disorders including definitions, epidemiology, pathophysiology, clinical features, interventions, and prognosis and complications in patients with acute cor pulmonale.

**RESEARCH METHODOLOGY**

Acute cor pulmonale usually occurs due to pulmonary embolism. Pulmonary embolism is a condition where a deposit of blood clots or fat formed in peripheral blood vessels is released and flows in the blood vessels in the lungs. In acute cor pulmonale, a sudden and large embolism can cause a low output state that occurs due to the inability of the right ventricle to generate the pressure needed to regulate blood flow through the acute compensated pulmonary vascular bed (S. Adiyanti, 2019).

Research conducted in Ethiopia found that the most common causes of cor pulmonale were bronchial asthma, pulmonary tuberculosis, chronic bronchitis, emphysema, pulmonary interstitial disease, bronchiectasis, obesity, and kyphoscoliosis, respectively. The incidence of cor pulmonale of all adult heart disease cases in the United States is about 6-7% with COPD due to bronchitis and emphysema being the cause of more than 50% of cor pulmonale cases (G. Puspitasari).

**RESULT AND DISCUSSION**

**Pathogenesis**

Cor pulmonale is a condition where the right side of the heart has increased filling pressure and pulmonary hypertension causing pulmonary manifestations. Under normal circumstances, the vascular resistance to the lungs tends to be low at about one-tenth of the vascular resistance of the systemic arterial circulation. Chronic hypoxemia underlies this condition, causing chronic vasoconstriction and proliferation of pulmonary smooth muscle that can increase pulmonary vascular resistance. Hypoxemia causes the release of various vascular mediators such as nitric oxide, endothelin (ET 1), platelet-derived growth factor (PDGFG A and B). Nitric oxide acts as a vasodilator which in the state of hypoxemia the production of nitric oxide is suppressed, causing impaired vascular smooth muscle relaxation and causing an increase in pulmonary vascular resistance.
When there is an increase in resistance, pulmonary artery pressure increases and the work of the right ventricle of the heart increases, causing enlargement or dilatation of the right ventricle (Angriman et al., 2020; Kir et al., 2021; Mohan et al., 2019; Osakwe & B. Das, 2022) (A. Aubry & A. Vieillard, 2020).

Acute cor pulmonale is usually secondary to acute respiratory distress syndrome or pulmonary embolism, primary lactic acidosis, vasocclusion crisis in patients with sickle cell anemia, severe acute asthma, air ingress or other particles entering the circulation. In addition, infection has also been reported to cause acute cor pulmonale with a high mortality rate (D.M. Garrison & J. Memon, 2017).

Clinical Features

Shortness of breath (dyspnea) on exertion or dizziness on exertion are often the first symptoms of cor pulmonale. There may be symptoms of a rapid heartbeat or palpitations. Over time, symptoms appear with lighter activities or even at rest (D. Leong, 2017; D.M. Garrison & J. Memon, 2017).

Symptoms include:
1. Fainting with exertion
2. Chest discomfort, usually in the front of the chest
3. Chest pain on exertion
4. Syncope on exertion
5. Swelling (edema) of the feet or ankles
6. Symptoms of lung problems, such as wheezing or coughing or sputum production
7. Bluish lips and fingers (cyanosis)
8. Fatigue
9. Lethargy and tiredness

Usually clinical signs appear late, observable at an advanced stage after the development of pulmonary hypertension.

Physical Examination:
1. Juguar vein distension: The jugular V wave is prominent, suggesting tricuspid regurgitation.
2. Peripheral edema (ankles)
3. Cardiovascular: found left parasternal systolic lift; hardened S2 sound; right ventricle can be palpated on the right parasternal, there is a holosystolic murmur of tricuspid regurgitation at the lower left sternal border; right-sided S4 heart sound
4. Abdomen: hepatomegaly; ascites

Supporting examination:
1. Chest X-ray: Pulmonary artery enlargement may be seen, cardiomegaly
2. CT scan of the chest, with or without injection of contrast fluid (dye): CT angiography of the chest to rule out pulmonary thromboembolism as the cause. Measurement of main pulmonary artery diameter greater than 29 mm has sensitivity
3. 84% and specificity 75% for the diagnosis of pulmonary hypertension.
4. ECG: Shows a picture of right ventricular hypertrophy/enlargement. In the electrocardiogram, abnormal features of cor pulmonale on examination can be: Axis deviation to the right; P wave axis +90° or more; There is S1 S2 S3 pattern; R/S amplitude ratio in V1 is greater than lead 1; R/S amplitude ratio in V6 is smaller than lead 1; There is a pulmonary p pattern in leads 2, 3, and aVF; There is S1 Q3 T3 pattern and complete or incomplete right bundle branch block; There are inverted, horizontal, or biphasic T waves in the precordial leads; QRS waves with lower voltage especially in COPD due to hyperinflation; Advanced right ventricular hypertrophy may give Q wave features in the precordial leads that can be confused with myocardial infarction; Heart rhythm abnormalities ranging from isolated atrial premature depolarization to supraventricular tachycardia, including paroxysmal atrial tachycardia, multifocal atrial tachycardia, atrial fibrillation, and atrial flutter are sometimes encountered; These dysrhythmias can be triggered due to underlying disease states (anxiety, hypoxemia, acid-base balance disorders, electrolyte disturbances, and excessive bronchodilator use).
5. Lung ventilation and perfusion scan (V/Q scan): A ventilation/perfusion (V/Q) scan can be very useful in evaluating patients with pulmonary cor, especially if the pulmonary hypertension is due to chronic thromboembolic pulmonary hypertension.
6. Pulmonary function tests: Pulmonary function test and 6-minute walk test to assess the severity of pulmonary disease and exercise capacity.
7. Right heart catheterization: Right heart catheterization is the gold standard for diagnosis, assessment of severity of pulmonary hypertension. Right heart catheterization shows evidence of right ventricular (RV) dysfunction mean pulmonary artery pressure (PAP) above 25 mmHg) without left ventricular (LV) dysfunction. Differentiating left-sided disease from right-sided disease involves measuring pulmonary capillary wedge pressure (PCWP), which is an estimate of left atrial pressure. Thus, right ventricular (RV) dysfunction is also defined as having a pulmonary capillary wedge pressure below 15 mmHg.
8. Blood oxygen measurement by checking arterial blood gas (ABG)
9. Test for autoimmune lung disease
10. Blood antibody test
11. Blood test to check for a substance called brain natriuretic peptide (BNP)
12. Lung biopsy (rarely done)

Management

Cor pulmonale can be caused by pulmonary hypertension. Pulmonary hypertension can occur due to changes in the structure and function of the heart caused by diseases of the respiratory system, causing pulmonary hypertension. Therapy of cor pulmonale aims to treat or overcome the underlying disease that causes cor pulmonale. The goal of the therapy itself is to increase oxygenation and improve the function of the
right ventricle of the heart by increasing contractility and decreasing vasoconstriction in the lungs. Oxygen therapy can treat pulmonary vasoconstriction and thus increase cardiac output (CO). In addition to increasing CO, oxygen therapy can also improve renal perfusion. Diuretic drugs can be used to manage right ventricular filling volume in patients with chronic cor pulmonale (DM Garrison & J. Memon, 2017).

Prognosis & Complications

Prognosis

The prognosis of cor pulmonale depends on the underlying pathology. Cor pulmonale as a result of primary lung disease usually shows a worse prognosis. For example, patients with chronic obstructive pulmonary disease (COPD) who progress to cor pulmonale have a 30% survival rate within 5 years. The prognosis is still unknown whether cor pulmonale carries independent prognostic value or refers to the severity of COPD as the underlying disease. The prognosis of acute cor pulmonale due to pulmonary embolism or acute respiratory distress syndrome (ARDS) has not been shown to depend on the presence or absence of cor pulmonale. However, a prospective cohort study by Volschan et al showed that in cases of pulmonary embolism, cor pulmonale could predispose to death (Leong D. 2017).

Complications

Complications that can arise from cor pulmonale include right heart failure, CHF (chronic heart failure), respiratory failure, acute kidney injury, hemoptosis and deep vein thrombosis (A. Aubry & A. Vieillard, 2020).

Education

Some education that can be given to patients with cor pulmonale is as follows:

1. Limiting fluid consumption. If too much fluid is drunk, it can risk swelling and aggravate cor pulmonale.
2. Eat a healthy diet. It is recommended to consume foods that are low in salt.
3. Maintain an ideal weight and consult a healthcare provider on the best plan as cor pulmonale patients can only do certain exercises.
4. Prevent pregnancy in women. During pregnancy the heart works harder than usual.
5. Do not drink alcohol. Alcohol can cause breathing difficulties and can worsen cor pulmonale.
6. Do not smoke. Smoking can cause heart and lung disease and worsen symptoms associated with the heart and lungs.

CONCLUSION

Cor pulmonale is hypertrophy or dilatation of the right ventricle caused by pulmonary hypertension. Acute cor pulmonale or acute cor pulmonale is a condition that usually occurs due to pulmonary embolism. In acute cor pulmonale there is a sudden and large embolism, so that it can cause a low output state that occurs due to the
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inability of the right ventricle to produce the pressure needed to regulate blood flow through the acute compensated pulmonary vascular bed. Research says the most common cause of cor pulmonale is bronchial asthma. While research in America says COPD due to bronchitis and emphysema is the most common cause of cor pulmonale. Shortness of breath (dyspnea) on exertion or dizziness on exertion are often the first symptoms of cor pulmonale. The condition of acute cor pulmonale can be identified based on signs and symptoms from history taking, physical examination, and supporting examination.

REFERENCES


