Review Article

Cardiogenic Pulmonary Edema

Abstract

Background: the first manifestations of cardiogenic pulmonary edema include hemodynamic pulmonary congestion and high capillary pressure. This causes fluid to flow from the capillaries into the interstitium and alveolar spaces. High capillary pressure can also cause blockage, increasing the inability to penetrate and transfer fluid to the interstitium and alveoli. The fluids in the alveoli alter the properties of the surfactant and increase local stress. This can lead to the formation of additional edema and atelectasis due to inactive gas exchange. Patients with gait obstruction have elevated levels of surfactant protein B, and these levels often remain high after initial clinical development. Routine clinical examination may not identify patients with increased extravascular fluid in the lungs; Pulmonary ultrasound can easily detect pulmonary edema in patients with severe degeneration and in patients at risk of caries. Serial pulmonary ultrasound studies can help identify patients with cardiogenic pulmonary edema and identify small populations that need additional treatment. Conventional treatment for cardiogenic pulmonary edema generally includes diuresis, post-exercise load reduction, and, in some cases, irregular air supply to reduce respiratory function and improve oxygen uptake. Patients with persistent symptoms, abnormal chest x-rays, and resistance to diuretics may benefit from other treatment options. These may include beta agonists and pentoxifylline, which allow further investigation in patients with cardiogenic pulmonary edema.

Conclusion: Unfortunately, despite the right treatment, the effects of cardiogenic pulmonary edema / heart failure are very serious. There is no cure for this disease, and the important thing is to prevent this condition in the first place.

Keywords: Capillary permeability; Cardiogenic pulmonary edema; Pulmonary surfactant-associated protein B; Ultrasonography.

Introduction

Edema is the accumulation of excess fluid in the joints, under the skin, or within the body cavity due to any of the following and produces vital signs and symptoms. The imbalance between the "starling forces", the injury / obstruction of the lymphatic drainage system, the affected part of the body often becomes inflamed when there is swelling under the skin or vital signs and symptoms related to the body cavity develop. There are different types of edema, and there are peripheral edema, pulmonary edema, cerebral edema, macular edema, and lymphedema. The atypical forms are idiopathic edema and angioneurotic genetic inflammation. Pulmonary edema refers to the accumulation of excess fluid in the alveolar walls and alveolar cavities of the lungs. For some patients, it can be a life-threatening condition. Pulmonary edema can be: cardiogenic (interrupted stellar power including pulmonary vascularization and interstitium), non-cardiogenic (direct injury / damage to the pulmonary parenchyma / vascular) (1).

Causes and Risk Factors

All factors that contribute to increased pressure in the left side of the heart and the formation of blood clots in the left side of the heart can cause cardiogenic pulmonary edema. The result of all these conditions is increased pressure on the left side of the heart: increased pulmonary vein pressure -> increased capillary pressure in the lungs -> pulmonary edema. Coronary artery disease, left ventricular insufficiency (myocardial infarction), recurrent heart failure, cardiomyopathy, heart valve disease of the left side of the heart (stenosis and relapse), cardiac arrhythmias, right-left shunts, cardiogenic pulmonary edema due to high blood pressure. in the heart. This is usually the result of heart failure. When the left or overactive left ventricle cannot pump enough blood out of the lungs, the heart rate increases. The increased pressure pushes fluid through the walls of the blood vessels into the air sacs (2).

Medical conditions that cause heart failure and pulmonary edema include: coronary artery disease. Over time, the arteries that carry blood to the heart muscle can become thin (plaque). A slight narrowing of the coronary arteries weakens the left ventricle. In some cases, blood clots form in one of these small arteries, blocking blood flow and damaging part of the heart muscle, leading to heart attacks. A damaged heart muscle can no longer pump properly. Cardiomyopathy The term refers to myocardial infarction. If you have cardiomyopathy, your heart needs to pump faster and the pressure increases. The heart cannot respond to conditions that require vigorous activity, such as exercise, infections, or high blood pressure. When the left ventricle cannot meet the requirements placed on it, fluid flows back into the lungs. Heart valve problems. A narrowing of the aortic or mitral heart valves (stenosis) or a leaky or poorly closed valve can affect blood flow to the heart. The heart has to work harder and the pressure increases. If valve leaks start suddenly, you can develop sudden and severe pulmonary edema. Hypertension (hypertension). Untreated or uncontrolled hypertension can increase the heart rate. Other heart problems. Inflammation of the heart muscle (myocarditis), congenital heart failure and irregular heartbeat (arrhythmias) may also cause pulmonary edema. Kidney disease. High blood pressure due to renal artery stenosis or fluid accumulation due to kidney disease can cause pulmonary edema. Endless health conditions. Thyroid disease and iron deficiency (hemochromatosis) or protein deficiency (amyloidosis) may also contribute to heart failure and cause pulmonary edema (3).

Mechanism of Cardiogenic Pulmonary Edema

The cardiogenic form of pulmonary edema (pressure-induced) produces a non-inflammatory type of edema with disruption of Starling's energy. The pulmonary capillary pressure is 10 mm Hg (range: 6 to 13) under normal conditions, but anything that increases this pressure can cause pulmonary edema. The alveoli are generally kept dry due to unfavorable pressure in the extraalveolar interstitial spaces, but if applicable: excess pressure / conjunction -> increased pulmonary venous pressure -> increased pulmonary capillary pressure -> fluid in central areas -> increased pressure in the interstitial spaces -> fluid in the alveoli (pulmonary edema). Pulmonary capillary pressure can be measured, adjusted, and will produce multiple radiographs (figure 1) (4).

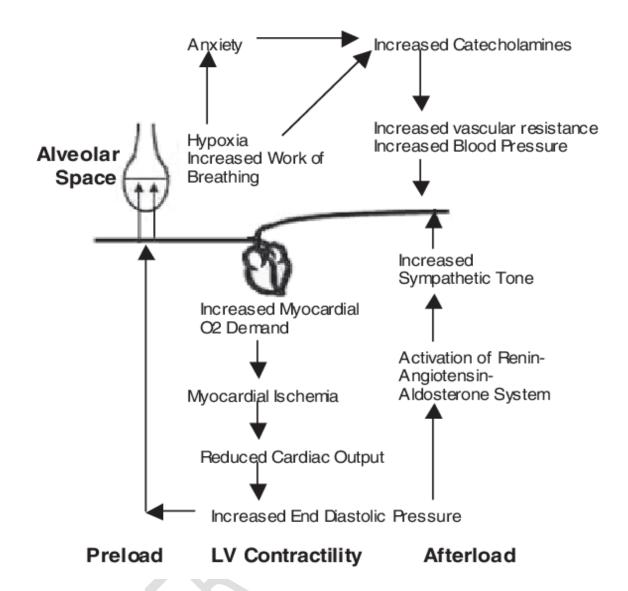


Figure 1 Mechanism of Cardiogenic Pulmonary Edema (4)

Symptoms

Signs and symptoms of pulmonary edema may suddenly appear or improve over time. The signs and symptoms you have will depend on the type of pulmonary edema. Severe pulmonary edema (acute), shortness of breath (dyspnea) or difficulty breathing while working or sleeping, drowsiness or drowsiness during sleep, coughing, cold, shiny skin, anxiety, restless or nervous feeling, blue signs and symptoms Heart rate), chronic pulmonary embolism, shortness of breath or sleeping on the floor, coughing or sneezing at night, difficulty breathing by sitting, excessive breathing, shortness of breath, rapid weight gain, swelling in the lower extremities, fresh or bad cough, Signs and symptoms of lung edema (HAPE), HAPE, may be seen in adults and children. Symptoms and signs are associated with severe lung edema and may include: headache, perhaps the first sign, fatigue, restlessness, reduced ability to exercise, dry cough, early, later, foamy cough, pink sputum, rapid heartbeat (tachycardia), weakness Signs and symptoms of chest pain, low-grade fever, high-altitude pulmonary edema (HAPE), are worse at night (5).

Complications

Many problems with pulmonary edema result from underlying complications. Common problems associated with cardiogenic etiologies include: risk of arrhythmia (atrial fibrillation, ventricular tachycardia), thromboembolism (pulmonary embolism, DVT, stroke), pericarditis, fracture, valvular disease.Pulmonary edema can cause severe hypoxia and hypoxemia leading to endocrine damage and multiple organ failure. Respiratory failure is another common complication of cardiogenic pulmonary edema (6).

Investigations

Respiratory problems require immediate diagnosis and treatment. Your doctor can make an early diagnosis of pulmonary edema based on your signs and symptoms and the results of a physical examination, electrocardiogram, and chest x-ray. Once your condition is stable, your doctor will ask you questions about your medical history, especially if you have heart or lung disease. To diagnose pulmonary edema or determine why fluid builds up in your lungs include (7):

Chest X-ray

The chest x-ray confirms the diagnosis of pulmonary edema and reveals other causes that may be causing your breathing. The first test is usually done when a person has signs or symptoms of pulmonary edema (figure 2) (8).

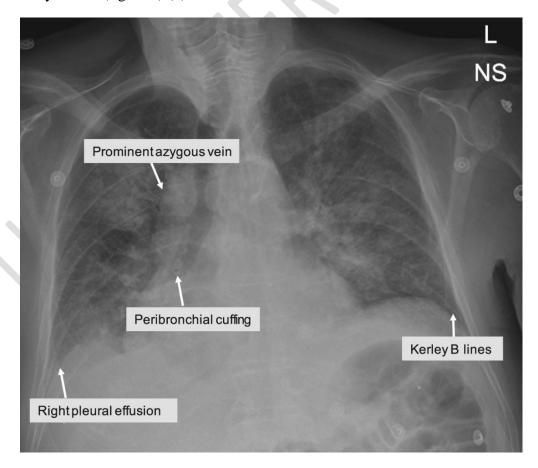


Figure 2 Chest X-ray of Cardiogenic Pulmonary Edema (8)

Chest CT

Computed tomography (CT) chest scan may not give the cause of pulmonary edema, but may give your doctor indirect clues to help diagnose it (figure 3) (9).

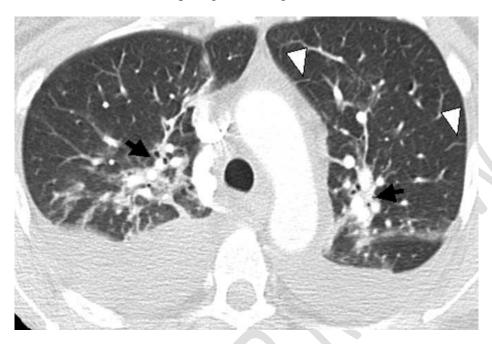


Figure 3 Chest CT of Cardiogenic Pulmonary Edema (9)

Pulse oximetry

The sensor is attached to your finger or ear and uses light to determine the amount of oxygen in your blood (10).

Arterial blood gas test

The blood is drawn, usually from a vein in your wrist, and tested for the amount of oxygen and carbon dioxide in it (concentration of arterial blood gas) (10).

B-type natriuretic peptide (BNP) blood test

An increase in BNP levels may indicate a heart condition (11).

Other blood tests

Blood tests to diagnose pulmonary edema and its causes usually include total blood count, metabolic panel to test kidney function and thyroid function (12).

Electrocardiogram (ECG or EKG)

This painless test uses a small sensor (electrode) attached to the skin of your chest and legs to detect and record the time and energy of your heart signals. Symbols are recorded on the grid paper or on the monitor as waves. ECG may show symptoms of heart failure or previous heart disease. A portable ECG device such as a Holter monitor can be used at home to continuously monitor your heart rate (figure 4) (13).

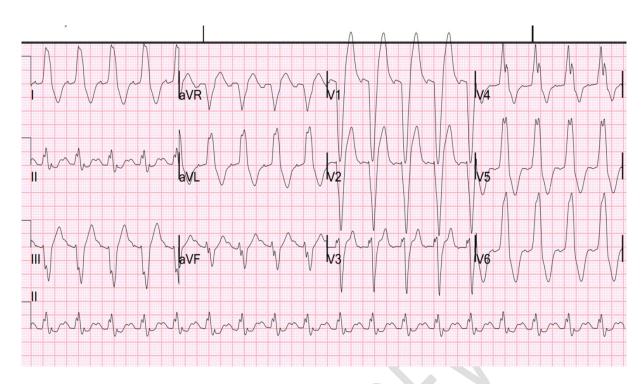


Figure 4 ECG of Acute Pulmonary Edema (13)

Echocardiogram

An echocardiogram uses sound waves (ultrasound) to create a moving image of your heart. It can detect abnormal blood flow, abnormal heart valves, and abnormal heart muscle areas. Your doctor may use this test to help diagnose pericardial fusion (figure 5) (14).

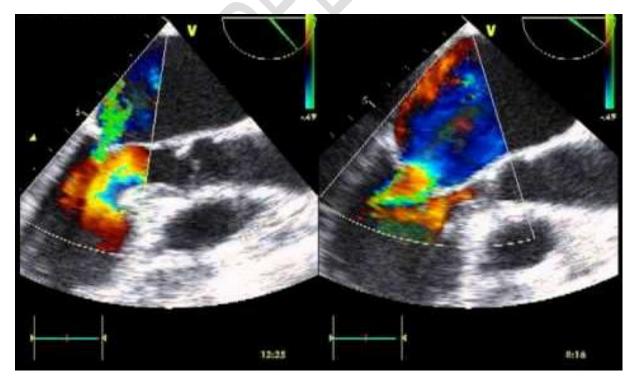


Figure 5 Echocardiogram of Acute Pulmonary Edema on Top of Mitral Regurgitation (14)

Cardiac catheterization and coronary angiogram

This test can be done if an ECG, echocardiogram or other test does not show the cause of pulmonary edema, or if you have chest pain. During cardiac catheterization, the doctor inserts a long, thin tube (catheter) into a vein or vein in the waist, neck, or arm. An X-ray helps direct the catheter from a blood vessel to your heart. During coronary angiogram, dye flows into the catheter, allowing blood vessels to be clearly visible on X-rays. The coronary angiogram can detect any obstruction and measure the pressure in your heart chambers (figure 6) (15).

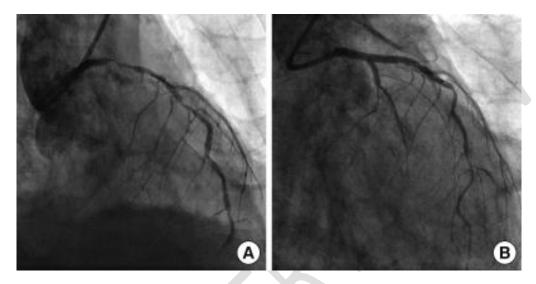


Figure 6 Cardiac Catheterization and Coronary Angiogram (Restoration of Blood Flow)
(15)

Ultrasound of the lungs

This painless test uses sound waves to measure blood flow to the lungs. It quickly shows signs of fluid overload and discharge. Pulmonary ultrasound is now an accurate tool for the diagnosis of pulmonary edema (figure 7) (16).

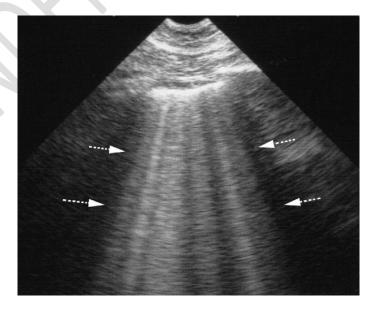


Figure 7 Ultrasound of Pulmonary Edema (16)

Treatment

The first treatment for acute pulmonary edema is oxygen therapy. Oxygen is usually delivered through a facial mask or nasal passage through a flexible plastic tube with two holes through which oxygen enters each nostril. This should reduce some of your symptoms. Your doctor will closely monitor your oxygen levels. Sometimes, you may need to use a device like a ventilator or a ventilator to make breathing easier. You may also take one or more of the following medications depending on the severity of your condition and the cause of the pulmonary edema: Diuretics. Doctors often prescribe sedatives, such as furosemide (Lasix), to relieve the pressure caused by excess fluid in the heart and lungs. Morphine Medication can be taken orally or intravenously to relieve breathing and anxiety. But some doctors believe that the risks and benefits of morphine may outweigh the potential for other drugs. Medications for high blood pressure. If you have high or low blood pressure with pulmonary edema, you will be given medication to help manage the condition. Your doctor may also prescribe medication that lowers the pressure inside or outside your heart. Examples of such drugs are nitroglycerin (nitromist, nitrostate and others) and nitroprusside (nitropress). Anotropes This type of medicine is given intravenously when you are in the hospital with a serious heart condition. Anotropes improve cardiac function and maintain blood pressure. If possible, it is important to diagnose and treat the causes of neurological problems or heart failure (17).

Discussion

Pulmonary edema is a condition caused by excess water in the lungs. This fluid collects in many alveoli of the lungs, making it difficult to breathe. Often, heart problems cause pulmonary edema. However, other causes such as pneumonia, exposure to certain toxins and drugs, trauma to the chest wall, high altitude movement and exercise can cause water to build up in the lungs. Sudden pulmonary edema (acute pulmonary edema) is an emergency disease that requires immediate attention. Pulmonary edema can be fatal. Immediate treatment will improve your eyesight. Treatment of pulmonary edema depends on the cause, but usually requires additional oxygen and medication (18).

Conclusion

Cardiogenic pulmonary edema (CPE) is a common and dangerous condition that is common in emergency medicine. There are many diseases that lead directly or indirectly to the development of pulmonary edema. Regardless of the underlying cause of CPE, all patients who develop CPE should be diagnosed and treated immediately. Patients who develop CPE may develop rapid breathing if attention or control is delayed. In fact, mortality rates for patients using KPIs range from 15 to 20%, and mortality can be even higher if the disease is associated with acute myocardial infarction (AMI) or valve dysfunction. Rescue workers must maintain a high level of monitoring of the situation and initiate immediate treatment strategies.

References

- 1) Alwi I. Diagnosis and management of cardiogenic pulmonary edema. Acta Med Indones. 2010 Jul;42(3):176-84.
- 2) Baird A. Acute pulmonary oedema management in general practice. Aust Fam Physician. 2010 Dec;39(12):910-4.
- 3) Chioncel O, Collins SP, Ambrosy AP, Gheorghiade M, Filippatos G. Pulmonary Oedema-Therapeutic Targets. Card Fail Rev. 2015 Apr;1(1):38-45.
- 4) Coraim FI, Wolner E. Continuous hemofiltration for the failing heart. New Horiz. 1995 Nov;3(4):725-31.
- 5) Crane SD. Epidemiology, treatment and outcome of acidotic, acute, cardiogenic pulmonary oedema presenting to an emergency department. Eur J Emerg Med. 2002 Dec;9(4):320-4.
- 6) Fiaccadori E, Regolisti G, Maggiore U, Parenti E, Cremaschi E, Detrenis S, Caiazza A, Cabassi A. Ultrafiltration in heart failure. Am Heart J. 2011 Mar;161(3):439-49.
- 7) Guntupalli KK. Acute pulmonary edema. Cardiol Clin. 1984 May;2(2):183-200.
- 8) Johnson JM. Management of acute cardiogenic pulmonary edema: a literature review. Adv Emerg Nurs J. 2009 Jan-Mar;31(1):36-43.
- 9) Johnson MR. Acute Pulmonary Edema. Curr Treat Options Cardiovasc Med. 1999 Oct;1(3):269-276.
- 10) Komiya K, Akaba T, Kozaki Y, Kadota JI, Rubin BK. A systematic review of diagnostic methods to differentiate acute lung injury/acute respiratory distress syndrome from cardiogenic pulmonary edema. Crit Care. 2017 Aug 25;21(1):228.
- 11) Marenzi G, Grazi S, Giraldi F, Lauri G, Perego G, Guazzi M, Salvioni A, Guazzi MD. Interrelation of humoral factors, hemodynamics, and fluid and salt metabolism in congestive heart failure: effects of extracorporeal ultrafiltration. Am J Med. 1993 Jan;94(1):49-56.
- 12) Matsue Y, Damman K, Voors AA, Kagiyama N, Yamaguchi T, Kuroda S, Okumura T, Kida K, Mizuno A, Oishi S, Inuzuka Y, Akiyama E, Matsukawa R, Kato K, Suzuki S, Naruke T, Yoshioka K, Miyoshi T, Baba Y, Yamamoto M, Murai K, Mizutani K, Yoshida K, Kitai T. Time-to-Furosemide Treatment and Mortality in Patients Hospitalized With Acute Heart Failure. J Am Coll Cardiol. 2017 Jun 27;69(25):3042-3051.
- 13) Matsuyama S, Ootaki M, Saito T, Iino M, Kano M. [Radiographic diagnosis of cardiogenic pulmonary edema]. Nihon Igaku Hoshasen Gakkai Zasshi. 1999 May;59(6):223-30.
- 14) Mattu A, Martinez JP, Kelly BS. Modern management of cardiogenic pulmonary edema. Emerg Med Clin North Am. 2005 Nov;23(4):1105-25.
- 15) Miles WA. Pulmonary edema: an anatomic, pathophysiologic, and roentgenologic analysis. J Natl Med Assoc. 1977 Mar;69(3):179-82.
- 16) Murray JF. Pulmonary edema: pathophysiology and diagnosis. Int J Tuberc Lung Dis. 2011 Feb;15(2):155-60.
- 17) Platz E, Jhund PS, Campbell RT, McMurray JJ. Assessment and prevalence of pulmonary oedema in contemporary acute heart failure trials: a systematic review. Eur J Heart Fail. 2015 Sep;17(9):906-16.

18) Potts JM. Noninvasive positive pressure ventilation: effect on mortality in acute cardiogenic pulmonary edema: a pragmatic meta-analysis. Pol Arch Med Wewn. 2009 Jun;119(6):349-53.

