INTERNATIONAL SEVEN JOURNAL
OF HEALTH RESEARCH
ISSN: 2764-9415

Acute pulmonary edema secondary to acute coronary syndrome in a patient with intellectual disabilities

Edema agudo de pulmão secundário a síndrome coronariana aguda em paciente com deficiência intelectual

DOI: 10.56238/isevjhv3n1-029
Receipt of originals: 20/01/2024
Publication acceptance: 06/02/2024

Jussara de Almeida Bruno[1], Matheus de Souza Camargo[2], Vitoria Vitor Ortega[3], Maria Laura Sanches Portuguez[4], Bertholina Rosa Lopes Guimarães[5], Ana Luísa Rampazzo Czadotz[6], Luiza Queiroz Combs[7], Matheus Luiz de Oliveira Raymundi[8], Bruno Balisardo Zacharias[9], Bruna Heloísa Ferreira[10], Lorena Queiroz Bordín[11], Isabela Bomediano Malamão[12].

ABSTRACT

Acute pulmonary edema is a clinical entity of varied etiologies and, if not quickly managed, can have negative outcomes. Some of the patients who develop this condition have heart failure, which, once decompensated, leads to ventricular failure and increased pulmonary capillary pressure, causing alveolar flooding. The objective of this article is to report a clinical case of a patient who presented with acute pulmonary edema secondary to an acute myocardial infarction, a condition associated with a communication difficulty between the care team and the patient due to the communication limitation of the patient, who has an intellectual disability.

Keywords: Edema, Ischemia, Coronary artery disease.

INTRODUCTION

Acute pulmonary edema secondary to acute myocardial infarction is a complex clinical syndrome that represents a significant and potentially fatal complication of ischemic cardiac events. This interconnection between these two conditions requires a deep understanding of their pathophysiological mechanisms and therapeutic interventions1.

[1] Cardiologist, Universidade do Oeste Paulista
[2] Medical Student, Universidade do Oeste Paulista
[3] Medical Student, Universidade do Oeste Paulista
[4] Medical Student, Universidade do Oeste Paulista
[5] Medical Student, Universidade do Oeste Paulista
[6] Medical Student, Universidade do Oeste Paulista
[7] Medical Student, Universidade do Oeste Paulista
[8] Medical Student, Universidade do Oeste Paulista
[9] Medical Student, Universidade do Oeste Paulista
[10] Medical Student, Universidade do Oeste Paulista
[11] Medical Student, Universidade do Oeste Paulista
[12] Medical Student, Universidade do Oeste Paulista

Acute myocardial infarction (AMI) is a common manifestation of coronary artery disease and, when not properly treated, can trigger a cascade of events that culminate in the development of pulmonary edema. AMI is one of the leading causes of morbidity and mortality worldwide1,2.

Understanding how this condition can progress to acute pulmonary edema is essential, as this can significantly impact the patient's prognosis. Acute pulmonary edema syndrome (APE) is characterized by a rapid accumulation of fluid in the pulmonary alveolar spaces, resulting in acute respiratory failure. When associated with AMI, PAE often arises as a direct consequence of cardiac involvement2.

Infarctional myocardium can lead to left ventricular systolic or diastolic dysfunction, altering the hydrostatic balance in the lungs and triggering fluid extravasation into the air spaces. This event is widely recognized in the medical literature, but the complexity of the interaction between AMI and EAP remains an active research topic. Several risk factors contribute to the development of PAE after AMI, including the extent of myocardial injury, the time elapsed until coronary reperfusion, and the presence of comorbidities, among others1,3.

Knowledge of these factors is essential to identify patients at higher risk and direct appropriate prevention and management strategies. In addition, understanding the pathophysiology underlying this interaction is crucial for the development of targeted therapies that can improve clinical outcomes2,3.

The objective of this article is to report a complex clinical case of acute pulmonary edema in a patient with special needs, without full cognitive abilities, who was attributed to an acute coronary syndrome of the type of acute myocardial infarction without ST-segment elevation.

METHODS

A search was conducted of the clinical data of the patient's care in electronic medical records, which contained the relevant information, in addition to the results of imaging and laboratory tests. In addition, a literature review was carried out with articles less than 5 years old in the PubMed and Scielo databases. To carry out this research, the competent ethics committees approved it, following the protocols of the National Research Council (CONEP).

CASE REPORT

A 58-year-old male patient, smoker (10 pack-years) with heart failure, was admitted to the emergency department in his city of origin with a companion, who reported that the patient

(with intellectual disability and difficulties in verbalizing symptoms) had developed a sudden picture of nonspecific chest pain, associated with severe dyspnea.

The patient was monitored, and his vital signs showed a blood pressure of 200x100 mmHg and oxygen saturation in room air of 85% with bullous rales on pulmonary auscultation, characterizing an acute pulmonary edema as the cause of severe respiratory distress.

Intravenous diuretics and nitrate were administered; There was an improvement in the general condition, with a decrease in the patient's restlessness and a diuresis of 600 ml in the 9 hours following admission. Oxygen support with a mask was also performed at 10L per minute. Parallel to the initial consultation, an electrocardiogram was rotated and showed no ST-segment alterations or other alterations compatible with myocardial ischemia.

Serial myocardial necrosis markers, which were positive, were requested. The patient was transferred to the referral service, where he arrived with a blood pressure of 160x100, a heart rate of 121 and no respiratory distress, with a saturation of 97% while wearing an oxygen mask. The patient was stabilized and admitted to the bed of the coronary care unit (CCU).

A new electrocardiogram was performed at the referral hospital, which showed a repolarization alteration; the markers of myocardial necrosis were also repeated and were positive, concluding the diagnosis of acute myocardial infarction without ST-segment elevation. Transthoracic echocardiography was performed, which showed diffuse hypokinesia of the left ventricle and ejection fraction of only 24%.

Catheterization was performed, which showed coronary artery disease with 50% involvement of the lumen of the anterior descending artery in its distal portion; There was no indication for angioplasty. The cardiology team then requested a cardiac magnetic resonance imaging for a better investigation of the patient's heart failure, however, due to his mental condition, the exam could not be performed, as he presented intense agitation during the procedure.

The patient was hospitalized for 14 days, and after stabilization of the patient's condition, he remained uneventful and with good hemodynamic stability. The patient was discharged with optimization of the medications used for the underlying comorbidities, including heart failure, and was followed up for outpatient follow-up, with a future new attempt to perform cardiac magnetic resonance imaging.

DISCUSSION

Acute pulmonary edema (APE) secondary to acute myocardial infarction (AMI) is a serious complication that requires in-depth discussion. PAE, characterized by the rapid accumulation of fluid in the pulmonary alveolar spaces, can occur as a direct result of cardiac dysfunction triggered by AMI3,4.

The pathophysiology of PAE secondary to AMI is multifaceted. AMI causes damage to the heart muscle, compromising the function of the left ventricle. This can lead to an increase in pressure in the left atrium and pulmonary capillaries, creating a hydrostatic pressure gradient that favors the extravasation of fluids into the air spaces4.

This process is widely recognized, and clinical data demonstrate that left ventricular systolic or diastolic dysfunction is closely associated with the development of PAE after MI. The severity of AMI and the time elapsed until coronary reperfusion are critical factors in determining the risk of PAE. Clinical studies indicate that patients with extensive AMI, particularly those with left ventricular involvement, have a significantly higher risk of developing PAE2,4.

In addition, delays in reperfusion, whether through percutaneous coronary intervention (PCI) or thrombolysis, are also associated with an increased risk of pulmonary complications. These data underscore the importance of AMI treatment strategies that seek to reestablish coronary blood flow as soon as possible. The diagnosis of PAE secondary to AMI is complex due to the overlap of common symptoms, such as dyspnea, with other respiratory and cardiac conditions. Chest X-ray is a useful tool in identifying radiological patterns characteristic of PAE, such as interstitial and alveolar infiltrates2,3.

In addition, echocardiography plays a crucial role in assessing heart function and identifying abnormalities in the left ventricle. Treatment of post-AMI PAE is based on clinical data that support pharmacological approaches, including the use of diuretics to reduce fluid load, vasodilators to decrease cardiac preload, and positive inotropic agents to improve myocardial contractility. Optimization of heart failure management and cardiac rehabilitation are essential components of treatment[1-3].

It is important to note that prevention of PAE secondary to AMI is a crucial component of patient care. This involves screening strategies to identify patients at higher risk, such as those with extensive AMI, and the implementation of early interventions, such as immediate coronary reperfusion. In summary, PAE secondary to AMI is a serious and potentially fatal complication that is based on a complex pathophysiology3,4.

Clinical data and scientific evidence highlight the importance of identifying risk factors, accurate diagnosis, and well-defined therapeutic strategies. Effective treatment and appropriate prevention play crucial roles in improving clinical outcomes and quality of life for patients facing this critical complication.

In the context of acute myocardial infarction, it is critical to recognize that patients with intellectual disabilities represent a vulnerable subgroup that requires additional health care. Mental disability can affect a patient's ability to comprehend medical information, adhere to complex treatments, and express symptoms appropriately. Therefore, the diagnosis of AMI in patients with mental disabilities can be challenging5.

Healthcare providers should be aware of these communication difficulties and take steps to simplify information, engage caregivers, and use visual aids whenever possible to improve understanding and adherence to treatment. Additionally, the healthcare team should be sensitive to the specific needs of these patients and seek to create a welcoming and inclusive care environment. Another relevant aspect is that patients with mental disabilities may have a potentially higher risk of developing AMI due to common risk factors, such as lack of physical activity, inadequate diet, and smoking5,6.

Therefore, it is crucial that primary prevention programs are tailored to address the needs of these individuals, promoting awareness of healthy lifestyle habits and providing personalized follow-up strategies to reduce cardiovascular risk. In addition, the medical team should work closely with caregivers and families to ensure that patients with mental disabilities have access to appropriate screening and treatment, with the aim of minimizing the risks of AMI and associated complications5,6.

In view of the above-mentioned data, it is concluded that the relationship between acute pulmonary edema and acute myocardial infarction is a complex and clinically relevant topic. The clinical data and scientific evidence discussed in this section highlight the importance of a multidisciplinary approach and effective prevention strategies. Management of PAE secondary to AMI should involve early identification of risk factors, accurate diagnosis, and targeted therapeutic interventions. Effective treatment and cardiac rehabilitation play key roles in improving clinical outcomes and quality of life for patients.

Conflicts of interest

The authors declare that there is no potential conflict of interest that could interfere with the impartiality of this scientific work.

REFERENCES

Huppert, L., Matthay, M., & Ware, L. (2019). Pathogenesis of acute respiratory distress syndrome. Seminars in Respiratory and Critical Care Medicine, 40(01), 31–39.

Meyer, N. J. (2013). Acute respiratory distress syndrome. The Lancet Respiratory Medicine, 1(10), 793–803.

Del Buono, M. G., Moroni, F., Montone, R. A., Azzalini, L., Sanna, T., & Abbate, A. (2022). Ischemic cardiomyopathy and heart failure after acute myocardial infarction. Current Cardiology Reports, 24(10), 1505–1515.

Henri, C., & Rouleau, J. L. (2016). Acute pulmonary edema and acute coronary syndrome: Mostly a trigger or an associated phenomenon? Canadian Journal of Cardiology.

Chan, J. K. N., Chu, R. S. T., Hung, C., Law, J. W. Y., Wong, C. S. M., & Chang, W. C. (2022). Mortality, revascularization, and cardioprotective pharmacotherapy after acute coronary syndrome in patients with severe mental illness: A systematic review and meta-analysis. Schizophrenia Bulletin, 48(5), 981–998.

Tam To, B., Roy, R., Melikian, N., Gaughran, F. P., & O'Gallagher, K. (2023). Coronary artery disease in patients with severe mental illness. Interventional Cardiology: Reviews, Research, Resources, 18. Available from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10311395/pdf/icr-18-e16.pdf