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Case Report

Anaphylaxis and Cardiogenic Pulmonary Edema due to Non ST Elevation Myocardial Infarction NSTEMI: A Case Report

Pablo Andrés Pérez Giraldo [1,*], Alexander Lopez Villareal [2], Alejandro Cardozo [3], Manuel Alejandro García [3]

[1] Emergency medicine resident, Cooperative university of Colombia, Medellín, Colombia
[2] Emergency medicine resident, CES university, Medellín, Colombia
[3] Emergency physician, Neurological Institute of Colombia, Medellín, Colombia

*Correspondence: Pablo Andrés Pérez Giraldo (pablo.perezg@campusucc.edu.co)

Abstract: Anaphylaxis can be associated with hemodynamic shock, which requires the early initiation of adrenaline as part of its management. Cardiogenic pulmonary edema is a frequent entity in emergency services with increased mortality in patients with acute coronary syndrome. The case report presents the case of a 55-year-old male patient who entered the emergency department with a non-ST-segment elevation myocardial infarction (NSTEMI) associated to pulmonary edema and anaphylaxis. During his stay in the emergency room, he had an anaphylactic reaction to dipyrone (metamizole) used for pain control. The patient presented signs of acute pulmonary edema, a hypertensive urgency after the use of adrenaline for the management of anaphylaxis. There was doubt as to whether the dyspnea was of anaphylactic or cardiogenic origin, so an emergency ultrasound was performed, which suggested a bilateral pattern B. This allowed timely management of ventilatory failure with systemic nitrates, diuretics, and oxygen therapy, which controlled blood pressure and resolved ventilatory failure. Subsequently, he was transferred to an institution with a hemodynamic service for the management of NSTEMI. We highlight the utility of emergency ultrasonography for immediate decision-making and the low prevalence of anaphylactic reaction in a patient with NSTEMI leading to acute pulmonary edema.

Keywords: Anaphylaxis, Non-ST Elevated Myocardial Infarction

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1. Introduction

Anaphylaxis is a life-threatening multisystem allergic reaction that can compromise airway and circulation. In severe ill patients, intramuscular epinephrine is the immediate treatment of choice [1].

Acute heart failure is a frequent complication of NSTEMI and is associated with a 2-to-3-fold increased risk of in-hospital death compared with NSTEMI without acute heart failure [2]. In patients with acute ischemia, pulmonary edema is one of the feared complications in the emergency room because it can lead to respiratory failure. Patients with acute heart failure do not easily tolerate fluctuations in their blood pressure, which leads to volume overload and pulmonary edema [3].

During an allergic reaction, an increase in blood pressure may occur due to anaphylaxis or the use of adrenaline in its treatment [4].

We report a patient with NSTEMI in whom an anaphylactic reaction led to respiratory failure of cardiogenic origin and his management was guided by emergency ultrasonography.

2. Case report

A 55-year-old man with a history of diabetes mellitus without requiring insulin, no history of allergies or other relevant medical history. He was admitted to our emergency department for 1 week of intermittent angina-type chest pain, which had worsened in the last 12 hours. He had no other symptoms such as syncope, dizziness, or dyspnea.

The initial electrocardiogram did not show elevation or depression of the ST segment, with signs of ventricular hypertrophy (see image 1). On admission, his vital signs were: heart rate 100 bpm, blood pressure 170/100 mmHg, oxygen saturation 95% without requiring supplemental oxygen. On physical examination there was no respiratory distress, normal lung auscultation and without signs of fluid overload.

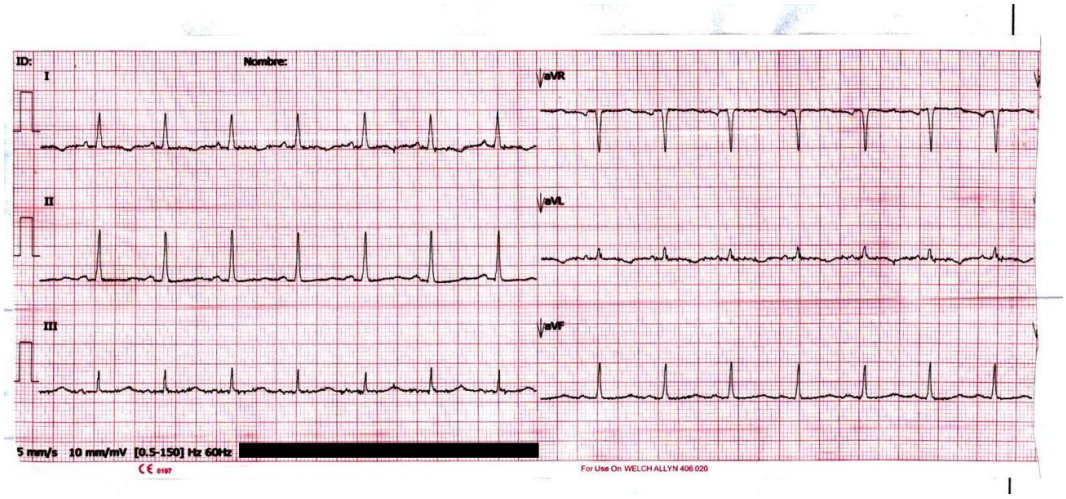

Figure 1. This is an Electrocardiogram at admission. (Absence of ST-segment depression or elevation or its equivalent in high-risk patterns)

The initial working diagnosis was chest pain, high probability for STEMI, initially requesting troponin, complete blood count, renal function, ions, and transthoracic echocardiography. Initial therapy was 250cc of saline solution 0.9%, losartan 50 mg PO and dipyrone 1gr IV single dose. The patient was located at the monitoring room.

After the application of dipyrone, the patient presented sudden dyspnea, chest discomfort, diaphoresis, mottled skin changes, wheezing, crackles, decreased breath sounds, and respiratory distress. His blood pressure 185/110mmHg, ambient oxygen saturation 87%, heart rate 110, respiratory rate 30 per minute. The results of tests requested before the respiratory distress reported complete blood count, ions and creatinine at normal range, troponin I 67 ng/ml (positive), considered a NSTEMI.

An anaphylactic reaction to dipyrone is suspected, 1 dose of adrenaline 0.5 mg IM, hydrocortisone 200 mg IV and diphenhydramine 10 mg IV, high-flow oxygen with a non-rebreathing mask were administered, with no evidence of improvement, worsening his hypertension and tachycardia. At this time, it was decided to perform emergency ultrasonography focused on the lung and heart, evidencing a diffuse bilateral B pattern and moderate compromise of cardiac contractility. The portable chest X-ray suggested pulmonary edema (See figure N° 2.). With this results management for pulmonary edema of cardiogenic origin was initiated, nitroglycerin and furosemide were administered, non-invasive mechanical ventilation and invasive monitoring of blood pressure was provided. His blood glucose was 386 mg/dl, for which an insulin infusion was started. His subsequent paraclinical tests were negative for ketoacidosis and arterial gases without metabolic acidosis and with a mild oxygenation disorder (PaFi 254). With established management, clinical improvement, decreased respiratory effort, and normalization of blood pressure levels occur.

The official thoracic echocardiogram documented a severe diffuse disorder of left ventricular contractility with akinesia in the posterior septum, inferior and inferolateral wall, with left ventricular ejection fraction (LVEF) of 26%. Finally, the patient is transferred to an institution with a hemodynamic service.

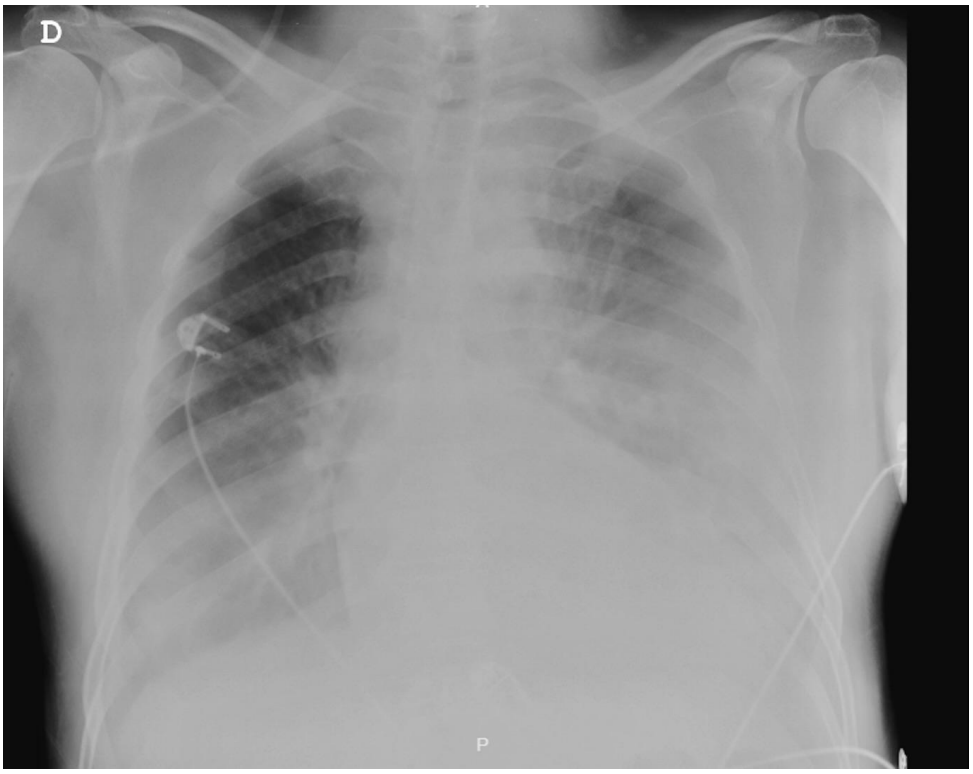

Figure 2. This is a figure. Portable chest x-ray. (Interstitial radiopacities and increased peribronchovascular pattern)

3. Discussion

Anaphylaxis has a range of clinical presentation that can include skin and mucosal changes, gastrointestinal, respiratory, cardiovascular symptoms, and decreased blood pressure [5]. The cardiovascular manifestations increase mortality, especially when hypotension leads to anaphylactic shock [6].

In the anaphylactic reaction, mediators such as histamines and leukotrienes are released, causing increased capillary permeability and reduced vascular tone. Due to this catecholamines are released, which seek to increase peripheral vascular resistance, which can lead to an increase in systemic blood pressure [4]. The hypertension in the presentation of anaphylaxis makes the diagnosis difficult and it generates a dilemma in the decision to use adrenaline in the management of this entity.

Anaphylaxis presenting with hypertension and acute pulmonary edema is rare and rarely published [4]. No current guidelines describe hypertension within the diagnostic criteria for anaphylaxis, even though there are cases reported in the medical literature [7]. Regarding patients with coronary disease, stroke or heart failure, 70% of them do not have their blood pressure in optimal ranges [3].

In patients entering the emergency room with myocardial infarction, it is important to control the factors that can trigger or aggravate acute heart failure, which can occur in 15% of them [2, 8]. In patients with pulmonary congestion and symptoms of respiratory distress, lung-focused ultrasonography contributes to the diagnosis and monitoring of these patients [9]. The finding of diffuse and bilateral b-lines reduces the range of diagnostic possibilities in patients with acute ventilatory failure. [10]

Regarding the association of pulmonary edema and anaphylaxis, there are few studies on its concomitant presentation. However, it may be the consequence of a hyperdynamic reaction in the context of hypertension triggered by anaphylaxis or as a result of the treatment with adrenaline. It is important to have a high clinical suspicion, perform a detailed physical examination and, if the tool it's available, rely on ultrasonography to focus the diagnosis and treatment of these patients [11].

4. Conclusions

Anaphylaxis is a condition that can aggravate cardiovascular pathologies, especially those that require optimal control of blood pressure. This can occur due to the pathophysiology of anaphylaxis and/or treatment with adrenaline, although infrequent anaphylaxis can be hypertensive, this can trigger pulmonary edema in patients susceptible to it. Ultrasonography at the patient's bedside can contribute to the approach of these patients since the bilateral B lines suggest an increase in the water/air ratio at the pulmonary level. In this case decongestion therapies can be indicated avoiding unnecessary orotracheal intubations.

5. Conflict of interests

The authors have no conflicts of interest to declare.

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