CME LIVE OPEN ACCESS

International Journal of Diabetes & its Complications

Case Report

A Case Report Highlighting Prehospital Recognition and Emergency First-Line Management of Acute Cardiogenic Pulmonary Edema -Cardiac asthma

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Clinical Setting
Initial emergency assessment was performed in a local hospital affiliated with Stara Zagora University Hospital, Bulgaria.

Received: 2026 Feb 09 **Accepted:** 2026 Mar 15 **Published:** 2026 Mar 26

Abstract

Cardiac asthma is not a form of bronchial asthma but a clinical manifestation of acute left ventricular failure characterized by wheezing, dyspnea, and cough secondary to pulmonary congestion. Due to overlapping respiratory symptoms, it is frequently misdiagnosed as bronchial asthma, leading to delayed or inappropriate treatment.

We present the case of a 65-year-old male initially presenting with uncontrolled hypertension who subsequently developed acute cardiogenic pulmonary edema (cardiac asthma) triggered by acute myocardial infarction. During the prehospital evaluation, subtle clinical signs including diaphoresis, agitation, and progressive respiratory discomfort raised suspicion of a developing cardiogenic process despite relatively moderate blood pressure elevation.

Early diuretic therapy, close monitoring, and prompt transfer to hospital enabled rapid diagnosis. Electrocardiography revealed ST-segment elevation, and laboratory testing demonstrated markedly elevated troponin levels. Urgent coronary angiography was performed, and the patient survived following timely intervention. This case highlights the importance of clinical vigilance in prehospital settings and emphasizes that wheezing and respiratory distress in elderly hypertensive patients may represent acute cardiac decompensation rather than primary pulmonary disease.

Keywords: Cardiac asthma, Dyspnea, Electrocardiography, Pulmonary disease

1. Introduction

Cardiac asthma is a clinical syndrome resulting from acute left ventricular dysfunction leading to rapid elevation of pulmonary venous pressure, interstitial pulmonary edema, and bronchial wall edema. The resulting wheezing and dyspnea may closely mimic bronchial asthma. However, unlike bronchial asthma, cardiac asthma is not an immune-mediated inflammatory airway disease but rather a manifestation of acute heart failure [1].

Accurate differentiation between these conditions is crucial, as treatment strategies differ fundamentally. While bronchial asthma requires bronchodilation and anti-inflammatory therapy, cardiac asthma requires rapid decongestion, hemodynamic stabilization, and treatment of the underlying cardiac cause [2]. Misdiagnosis may delay life-saving therapy, particularly in prehospital settings where initial evaluation guides early management decisions.

2. Case Presentation

A 65-year-old male contacted emergency services complaining of elevated blood pressure and complaining severe chest pain headache, dyspnea and it was getting progressively worse. His medical history included poorly controlled hypertension and intermittent non-adherence to antihypertensive medication. According to the patient, previous episodes of elevated blood pressure had been managed by emergency teams using antihypertensive and antitachycardic medication.

Upon arrival at his residence, his blood pressure measured 170/110 mmHg. The patient appeared restless, pale, and diaphoretic [3]. Respiratory rate was 25–27 breaths per minute, and oxygen saturation was 92% on room air. The patient reported that he had taken an oral antitachycardic medication approximately one hour earlier, yet his blood pressure remained elevated.

Although hypertensive urgency was initially suspected, the patient's agitation, sweating, and subtle respiratory discomfort raised concern for an evolving cardiopulmonary condition. Based on clinical judgment, intravenous furosemide (1 ampoule) was administered as an early decongestive intervention [4].

Due to the patient's appearance and clinical uncertainty, hospital observation was recommended rather than leaving the patient at home.

During transport to the hospital, the patient initially remained stable. However, shortly after being placed in the supine position in the emergency department, he developed acute respiratory distress characterized by:
• Severe dyspnea
• Orthopnea
• Dry cough progressing to frothy sputum
• Audible wheezing
• Bilateral pulmonary rales on auscultation
• Progressive oxygen desaturation

Oxygen saturation showed a progressive decline during the prehospital and early hospital phase, indicating clinical deterioration. Initial inhaler therapy was administered before a cardiac etiology was fully recognized.

Electrocardiography subsequently revealed ST-segment elevation, raising immediate suspicion of acute myocardial infarction.

Laboratory analysis demonstrated a troponin level of 8 ng/mL, markedly elevated compared with the expected normal value of approximately 0.02 ng/mL, indicating acute myocardial injury.

The patient was diagnosed with acute coronary syndrome complicated by acute left ventricular failure and cardiogenic pulmonary edema (cardiac asthma).

Urgent coronary angiography was performed, and the patient survived following timely cardiological intervention.

The early decision not to leave the patient at home and to proceed with hospital evaluation likely prevented fatal deterioration.

2.1. Pathophysiology of Cardiac Asthma

Cardiac asthma represents a manifestation of acute cardiogenic pulmonary edema. Rapid elevation in pulmonary venous pressure leads to:
• Interstitial and alveolar fluid accumulation
• Thickening of interalveolar septa
• Bronchiolar wall edema
• Reflex bronchoconstriction

These mechanisms produce wheezing and cough that mimic bronchial asthma, although the underlying mechanism is hydrostatic pulmonary congestion rather than airway inflammation [5].This explains why bronchodilator therapy alone is often ineffective in true cardiac asthma.

2.2. Emergency and Prehospital First-Line Management

2.2.1. Recognition in ambulance and emergency settings is critical for patient survival

I. Immediate priorities include
• Oxygen supplementation (target SpO_2 > 94%)
• Upright positioning (avoid supine positioning)
• Rapid ECG screening
• Continuous blood pressure and oxygen monitoring
• Early diuretic therapy (intravenous furosemide)
• Consider nitrates in hypertensive pulmonary edema
• Non-invasive ventilation (CPAP or BiPAP) if respiratory distress progresses
• Cardiac biomarker assessment (troponin, BNP)
• Urgent echocardiography
• Early cardiology consultation
• At dose of 4000 ıu iv. Heparin administired
In suspected acute coronary syndrome:
Immediate reperfusion strategy (primary PCI) is required.

II. Differential Diagnosis
Cardiac asthma must be differentiated from several conditions presenting with acute dyspnea:
• Bronchial asthma
• COPD exacerbation
• Pulmonary embolism
• Pneumonia
• Anaphylaxis

III. Key distinguishing features suggesting cardiac asthma include:
• Older patient age
• History of hypertension or coronary artery disease
• Orthopnea
• Frothy sputum
• Pulmonary crackles
• Elevated cardiac biomarkers
• Radiographic pulmonary congestion

2.3. Clinical Lessons from This Case

2.3.1. This case illustrates several important clinical lessons:

• Wheezing does not always indicate bronchial asthma.
• Supine positioning may precipitate acute decompensation in cardiac asthma.
• Diaphoresis and agitation may indicate sympathetic activation during acute heart failure.
• Oxygen saturation decline during monitoring may signal rapid deterioration.
• Early transport and observation may be lifesaving even when initial symptoms appear mild.
• ST-segment elevation combined with respiratory distress should immediately raise suspicion of cardiogenic pulmonary edema.

3. Discussion

Cardiac asthma is frequently misdiagnosed due to

overlapping respiratory symptoms. In elderly patients with hypertension, elevated blood pressure may mask underlying acute myocardial ischemia. The term "asthma" in this context is descriptive and does not represent allergic airway disease [6]. Understanding this distinction is essential to avoid therapeutic errors, such as overreliance on bronchodilators while delaying diuretic or vasodilator therapy.

In the present case, clinical intuition based on the patient's appearance, agitation, pallor, and respiratory pattern prompted early intervention and hospital transfer, which proved crucial for survival.Standardized emergency evaluation including rapid ECG screening, cardiac biomarkers, and early cardiology consultation significantly reduces the risk of missed acute coronary syndrome.

4. Conclusion

Cardiac asthma represents a manifestation of acute left ventricular failure and pulmonary congestion rather than primary airway disease. Early recognition in prehospital settings is essential, as prompt treatment and rapid referral for definitive cardiac care can be lifesaving.

This case demonstrates how careful clinical observation, early suspicion, and timely intervention allowed rapid diagnosis of acute myocardial infarction complicated by cardiogenic pulmonary edema, ultimately contributing to a favorable outcome.

Cardiac Asthma vs Bronchial Asthma: Key Clinical Differences Accurate differentiation between cardiac asthma and bronchial asthma is essential in emergency and prehospital settings because management strategies differ significantly. Cardiac asthma results from pulmonary congestion due to left ventricular failure, whereas bronchial asthma is an inflammatory airway

Feature	Cardiac Asthma	Bronchial Asthma
Underlying mechanism	Left ventricular failure leading to pulmonary congestion	Immune-mediated airway inflammation
Typical patient age	Usually elderly	Often younger patients
Medical history	Hypertension, coronary artery disease, heart failure	Allergies, atopy, previous asthma
Onset of symptoms	Often sudden, may occur at night	Often triggered by allergens, infections, exercise
Dyspnea	Severe, often with orthopnea	Episodic
Wheezing	Present due to bronchial edema	Present due to airway constriction
Cough	Often with frothy sputum	Usually dry or mucus-producing
Lung auscultation	Crackles (rales) often present	Mainly wheezing
Response to bronchodilators	Limited or minimal	Usually good response
Response to diuretics	Significant improvement	No effect

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