

CASE REPORT

ATAK Complex Presenting as Hypertensive Pulmonary Edema Following Meropenem-Induced Hypersensitivity: A Case Report

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ABSTRACT

Background: The ATAK complex is a rare yet clinically significant overlap of allergic and cardiac pathology. It represents a clinical continuum involving Adrenaline, Takotsubo, Anaphylaxis, and Kounis syndrome, resulting in coronary vasomotor dysfunction and transient myocardial impairment. Such presentations often resemble acute coronary syndrome (ACS), creating a diagnostic challenge in an emergency setting.

Case Presentation: We present the case of a 71-year-old male with hypertension, chronic kidney disease, and a permanent pacemaker who developed acute chest tightness, dyspnea, diaphoresis and generalised urticaria immediately following intravenous meropenem administration. Initial evaluation suggested ACS and 2D-echocardiography demonstrated an acute reduction in left ventricular systolic function; however, coronary angiography revealed non-obstructive coronary arteries. Serial echocardiography demonstrated partial recovery of left ventricular systolic function. The combination of an allergic trigger, systemic hypersensitivity, and reversible myocardial dysfunction led to the diagnosis of ATAK complex.

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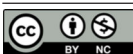
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Conclusion: This case highlights the importance of considering allergic myocardial involvement in patients presenting with ACS-like features following drug exposure. Recognition of ATAK complex is essential when hypersensitivity reactions coexist with reversible cardiac dysfunction, as it prompts a broader diagnostic approach and helps avoid misclassification as obstructive coronary artery disease, ensuring timely and appropriate management.

KEYWORDS

- ATAK Complex • Kounis Syndrome • Takotsubo Cardiomyopathy
- Anaphylaxis • Catecholamine Surge • SCAPE • MINOCA

INTRODUCTION

The ATAK complex is an emerging clinical entity that describes the overlap between adrenaline-mediated effects, Takotsubo cardiomyopathy, anaphylaxis, and Kounis syndrome.^{1,2} It reflects a pathophysiological continuum in which immune activation and sympathetic overdrive simultaneously influence cardiac function. Kounis syndrome represents allergic acute coronary vasospasm resulting from mast cell-mediated release of vasoactive and inflammatory mediators.³ Additionally, administration of exogenous catecholamines during treatment of anaphylaxis may further amplify sympathetic activation and myocardial stress,^{1,4} while Takotsubo cardiomyopathy is characterised by transient left ventricular dysfunction induced by catecholamine surge.⁴

These mechanisms coexist, giving rise to a clinical syndrome characterised by overlapping features of myocardial ischemia and stress induced cardiomyopathy.^{2,4} Patients often present with chest pain, diaphoresis, electrocardiographic abnormalities, and elevated cardiac biomarkers, closely mimicking acute coronary syndrome.^{5,6} However, the absence of obstructive coronary artery disease on angiography distinguishes these cases and places them within the spectrum of myocardial infarction with non-obstructive coronary arteries (MINOCA).⁷

The presentation overlaps with the MINOCA spectrum, particularly when myocardial infarction is established in the absence of obstructive coronary artery disease.

Recognition of this entity is particularly important in emergency medicine, where early differentiation from classical ACS has significant therapeutic implications. We present a case of ATAK complex triggered

by antibiotic administration, presenting as hypertensive pulmonary edema with reversible myocardial dysfunction.

CASE REPORT

A 71-year-old male with a background of hypertension, chronic kidney disease (baseline serum creatinine approximately 3 mg/dL), and prior permanent pacemaker implantation presented to the emergency department for scheduled intravenous meropenem therapy following recent hospitalisation for pyelonephritis.

Within minutes of drug administration, he developed acute chest tightness accompanied by breathlessness, diaphoresis, and diffuse erythematous rash. There was no prior history of hypersensitivity reaction.

On initial evaluation, the airway was patent with no evidence of angioedema. The patient was in significant respiratory distress, with a respiratory rate of 38/min and oxygen saturation of 90% on room air. Auscultation revealed bilateral diffuse crepitations. Hemodynamically, he was markedly hypertensive (190/120 mmHg) and tachycardic (120/min). Neurological examination was unremarkable, with a Glasgow Coma Scale score of 15/15. Dermatological examination revealed generalised urticaria.

Electrocardiography demonstrated sinus tachycardia with diffuse ST-segment depression and nonspecific T-wave abnormalities without a clear territorial ischemic pattern. Arterial blood gas analysis indicated type 1 respiratory failure with elevated lactate, likely reflecting increased sympathetic activity and respiratory distress. High-sensitivity troponin I was elevated at 67.2 ng/L.

Chest radiography was consistent with pulmonary edema. Apriori echocardiogram had shown normal left ventricular systolic function (LVEF 55%) with concentric hypertrophy. However, repeat echocardiography revealed global hypokinesia with a reduced LVEF of approximately 30%.

The patient was managed as anaphylaxis with intramuscular adrenaline, intravenous corticosteroids, and antihistamines. Non-invasive ventilation (BiPAP) and intravenous diuretics were initiated for pulmonary edema. He demonstrated rapid clinical improvement, with normalisation of vital parameters.

In view of elevated cardiac biomarkers and reduced ejection fraction, coronary angiography was performed, which demonstrated non-obstructive coronary arteries with findings suggestive of coronary vasospasm. Repeat echocardiography performed after 48 hours demonstrated partial recovery of left ventricular function (LVEF 45%), indicating reversible myocardial dysfunction.

Given the temporal association with drug exposure, systemic hypersensitivity features, absence of coronary obstruction, and reversible cardiac dysfunction, a diagnosis of ATAK complex was established.

DISCUSSION

The ATAK complex represents a convergence of immunological and neurohormonal mechanisms affecting the cardiovascular system.^{1,2} In this case, antibiotic exposure likely triggered IgE-mediated mast cell activation, resulting in the release of vasoactive mediators such as histamine, leukotrienes, and platelet-activating factor.

These mediators induce coronary vasospasm, endothelial dysfunction, and platelet activation, leading to Kounis syndrome.³ Simultaneously, endogenous stress response and exogenous adrenaline administration contribute to a surge in circulating catecholamines. Excess catecholamines exert direct myocardial toxicity, impairing microvascular perfusion and inducing myocardial stunning, resulting in Takotsubo cardiomyopathy.⁴ The rapid improvement in ventricular function observed in this patient further supports transient catecholamine-mediated myocardial stunning rather than irreversible ischemic injury.

The coexistence of these mechanisms explains the overlapping clinical features observed in ATAK complex, including chest pain, troponin elevation, and transient ventricular dysfunction.^{6,7} The absence of obstructive coronary disease in this patient supports a non-atherosclerotic etiology of myocardial injury.

Differential diagnoses considered in this patient included acute coronary syndrome, sympathetic crashing acute pulmonary edema (SCAPE), hypertensive pulmonary edema, sepsis-related cardiomyopathy, and MINOCA. The presence of generalised urticaria and temporal association with meropenem administration strongly supported a hypersensitivity-mediated process. Additionally, the absence of obstructive coronary artery disease, reversible left ventricular dysfunction, and rapid clinical recovery favoured ATAK complex over isolated ACS or hypertensive pulmonary edema.

Differential Diagnosis	Supporting Features	Features Against
Acute coronary syndrome	Troponin elevation, ECG changes	Non-obstructive coronaries
SCAPE	Severe hypertension, pulmonary edema	Allergic manifestation
MINOCA	Non-obstructive coronaries	Allergic manifestation
ATAK complex	Allergic trigger + reversible LV dysfunction	

MINOCA represents a diagnostic framework encompassing myocardial infarction without significant coronary obstruction. ATAK complex represents a distinct pathophysiological subset within the MINOCA spectrum, characterised by allergic myocardial injury, coronary vasospasm, and catecholamine-mediated myocardial

dysfunction.⁵

Troponin elevation in this patient likely reflects myocardial injury secondary to catecholamine toxicity and microvascular dysfunction. Although chronic kidney disease may contribute to baseline troponin elevation, the dynamic rise in troponin levels accompanied by transient left ventricular

dysfunction strongly suggested acute myocardial involvement rather than chronic elevation alone.⁸

Management of ATAK complex presents a therapeutic challenge. While adrenaline is essential for treating anaphylaxis, it may exacerbate myocardial stress. Nevertheless, it remains the cornerstone of treatment, and its benefits outweigh potential risks.¹⁰ Early use of echocardiography is critical for identifying reversible myocardial dysfunction and guiding further management.

Similar cases of ATAK complex have been described following exposure to antibiotics, chemotherapy agents, vaccines, and other hypersensitivity triggers.^{5,6,9-12} However, presentation with severe hypertension and pulmonary edema resembling sympathetic crashing acute pulmonary edema (SCAPE) physiology remains uncommon. Our case expands the clinical spectrum of ATAK complex by demonstrating hypertensive pulmonary edema with rapidly reversible ventricular dysfunction following meropenem-induced hypersensitivity.

This case highlights the complex interplay between hypersensitivity reactions and acute cardiac dysfunction, emphasising the importance of considering ATAK complex in patients presenting with acute coronary syndrome-like features following drug exposure. Early recognition of this overlap syndrome is essential to avoid misdiagnosis, facilitate appropriate multidisciplinary management, and prevent unnecessary invasive interventions.

Limitations

This report describes a single case, limiting generalisability. Advanced imaging modalities such as cardiac magnetic resonance imaging were not performed, which could have provided further insights into myocardial involvement. Additionally, long-term follow-up data were not available.

CONCLUSION

This case highlights the diagnostic complexity of ATAK complex and underscores the need for heightened clinical awareness in patients presenting with cardiac manifestations following an allergic trigger. It demonstrates how hypersensitivity reactions may simultaneously activate immune-mediated

and catecholamine-driven pathways, resulting in reversible myocardial dysfunction that closely mimics acute coronary syndrome. Early recognition of this overlap syndrome is crucial to facilitate appropriate multidisciplinary management and avoid unnecessary invasive interventions.

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