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Anterior ST-Segment Elevation Myocardial Infarction Shortly After Cefaclor Exposure: A Case Report Highlighting Kounis Syndrome as a Differential Diagnosis

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Data Interpretation D
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Conflict of interest: None declared

Patient: Male, 61-year-old

Final Diagnosis: Myocardial infarction

Symptoms: Chest pain

Clinical Procedure: —

Specialty: Cardiology

Objective: Unusual clinical course

Background: Kounis syndrome is an acute coronary syndrome associated with allergic or hypersensitivity reactions, but establishment of causality is difficult when objective allergy-related tests are unavailable during emergency treatment. This report describes anterior ST-segment elevation myocardial infarction (STEMI) occurring shortly after cefaclor exposure and highlights the diagnostic limitations of attributing the event to hypersensitivity in routine emergency clinical practice.

Case Report: A 61-year-old man with a history of positive penicillin skin testing, but no prior penicillin administration, developed severe chest pain approximately 1 hour after self-administration of oral cefaclor for respiratory symptoms. He had no rash, urticaria, wheezing, angioedema, oropharyngeal edema, hypotension, or dyspnea; no prehospital antihistamines, corticosteroids, or epinephrine were administered. Electrocardiography showed ST-segment elevation in V2 to V6, and emergency coronary angiography demonstrated 90% proximal left anterior descending artery stenosis with Thrombolysis in Myocardial Infarction grade II flow. Primary percutaneous coronary intervention restored grade III flow and relieved symptoms. High-sensitivity cardiac troponin I increased from 0.57 to 1.10 ng/mL and peaked at 3.92 ng/mL at 15 hours. Serum tryptase, histamine, total and specific IgE, and intracoronary imaging were not obtained. The Naranjo score was 2, indicating a possible adverse drug reaction.

Conclusions: The temporal association prompted consideration of Kounis syndrome, but severe fixed coronary stenosis makes coincidental plaque-related STEMI a major alternative diagnosis. Early allergy biomarker sampling and careful causal assessment are important in similar cases.

Keywords: Myocardial Infarction • Cephalosporins • Drug Hypersensitivity • Percutaneous Coronary Intervention • Case Reports

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Introduction

Kounis syndrome (KS) is generally defined as acute coronary syndrome (ACS) occurring in association with allergic, hypersensitivity, anaphylactic, or anaphylactoid reactions [1,2]. KS is commonly classified into 3 variants: type I, which occurs in individuals without pre-existing coronary artery disease and is primarily mediated by coronary spasm; type II, which occurs in patients with pre-existing coronary atherosclerosis, in whom allergic mediators may contribute to plaque erosion or rupture; and type III, which is associated with coronary stent thrombosis accompanied by hypersensitivity features [2,3]. Because KS can mimic conventional ACS and allergic manifestations may be absent or subtle, diagnosis can be challenging in the emergency setting [2,3]. Cephalosporin-associated KS has been reported, with heterogeneous presentations ranging from unstable angina to STEMI and shock [4-6]. The aims of this report are to describe an anterior STEMI occurring shortly after cefaclor exposure, clarify why KS was considered in the differential diagnosis, and emphasize the limitations of causal inference when allergy biomarkers and intracoronary imaging are unavailable.

Case Report

A 61-year-old man presented with severe substernal chest pain lasting 3 hours. He denied a history of hypertension, diabetes mellitus, dyslipidemia, tobacco smoking, alcohol use, or substance abuse. Previous health examinations had shown blood pressure around 120/75 mm Hg, although he did not regularly monitor his blood pressure at home. He reported a history of positive penicillin skin testing and had avoided penicillin administration thereafter. He had no clinically documented allergic reactions to cephalosporins, foods, or environmental allergens. Because he had self-administered cefaclor before presentation, a reliable history of previous exposure to cefaclor or other cephalosporins could not be definitively established.

The patient developed cough with white sputum the day before admission. On the day of admission, he self-administered oral cefaclor for these respiratory symptoms. The exact cefaclor dose could not be reliably confirmed. Approximately 1 hour after ingestion, he developed severe chest pain accompanied by profuse diaphoresis that persisted despite rest for approximately 3 hours before presentation to the hospital. He did not receive prehospital antihistamines, corticosteroids, epinephrine, or other allergy-directed rescue medications.

On admission, blood pressure was 110/72 mm Hg, heart rate was 77 beats/min, oxygen saturation was 98% on room air, and Killip class was I. He had no urticaria, rash, wheezing, dyspnea, angioedema, or oropharyngeal edema. Electrocardiography (ECG) showed ST-segment elevation in leads V2 to V6 (**Figure 1A**). The working diagnosis was anterior STEMI; the patient received oral aspirin 300 mg and ticagrelor 180 mg before undergoing emergency coronary angiography.

Coronary angiography demonstrated 90% stenosis of the proximal left anterior descending (LAD) artery with Thrombolysis in Myocardial Infarction (TIMI) grade II flow (**Figure 2A**). A 2.0 × 15 mm balloon was used for predilation, followed by implantation of a 3.5 × 23 mm Firebird drug-eluting stent and postdilation with a 3.75 × 10 mm noncompliant balloon. Final angiography demonstrated TIMI grade III flow without clinically significant residual stenosis (**Figure 2B**). Chest pain immediately resolved, and ST-segment elevation regressed (**Figure 1B**). No procedural complications occurred. No antiallergic medication was administered during acute treatment because systemic allergic manifestations were absent.

High-sensitivity cardiac troponin I (hs-cTnI) showed a rising pattern (0 hours: 0.57 ng/mL; 3 hours: 1.10 ng/mL), peaked at 15 hours (3.92 ng/mL), and then gradually declined. Peak creatine kinase-MB was 37 U/L. Laboratory tests showed low-density lipoprotein cholesterol of 2.07 mmol/L (desirable, < 2.6 mmol/L), hemoglobin A1c of 5.3% (reference range, approximately 4.0%-6.0%), white blood cell count of 3.8×10^9/L (reference range, approximately 3.5-9.5 × 10^9/L), and eosinophil count of 0.5×10^9/L (near the upper reference limit in many laboratories). The institutional 99th-percentile upper reference limit for the hs-cTnI assay was not available in the archived record.

Transthoracic echocardiography demonstrated mild anterior wall hypokinesia with a left ventricular ejection fraction of 55%. The patient recovered uneventfully and remained asymptomatic, with normal ECG and echocardiographic findings at the 1- and 2-month follow-up visits. He did not undergo outpatient allergy/immunology consultation, skin prick testing, patch testing, total or specific IgE testing, or baseline tryptase measurement during follow-up.

Discussion

The main lesson from this case is that KS can be considered in the differential diagnosis when ACS develops soon after exposure to a potential allergen, but this possibility should be distinguished from a confirmed diagnosis. In our patient, the established diagnosis was anterior STEMI caused by severe obstructive proximal LAD disease. The temporal relationship with cefaclor exposure and the history of positive penicillin skin testing implied the possibility of a drug-associated hypersensitivity trigger, but the absence of objective allergy-related data precluded confirmation of KS.

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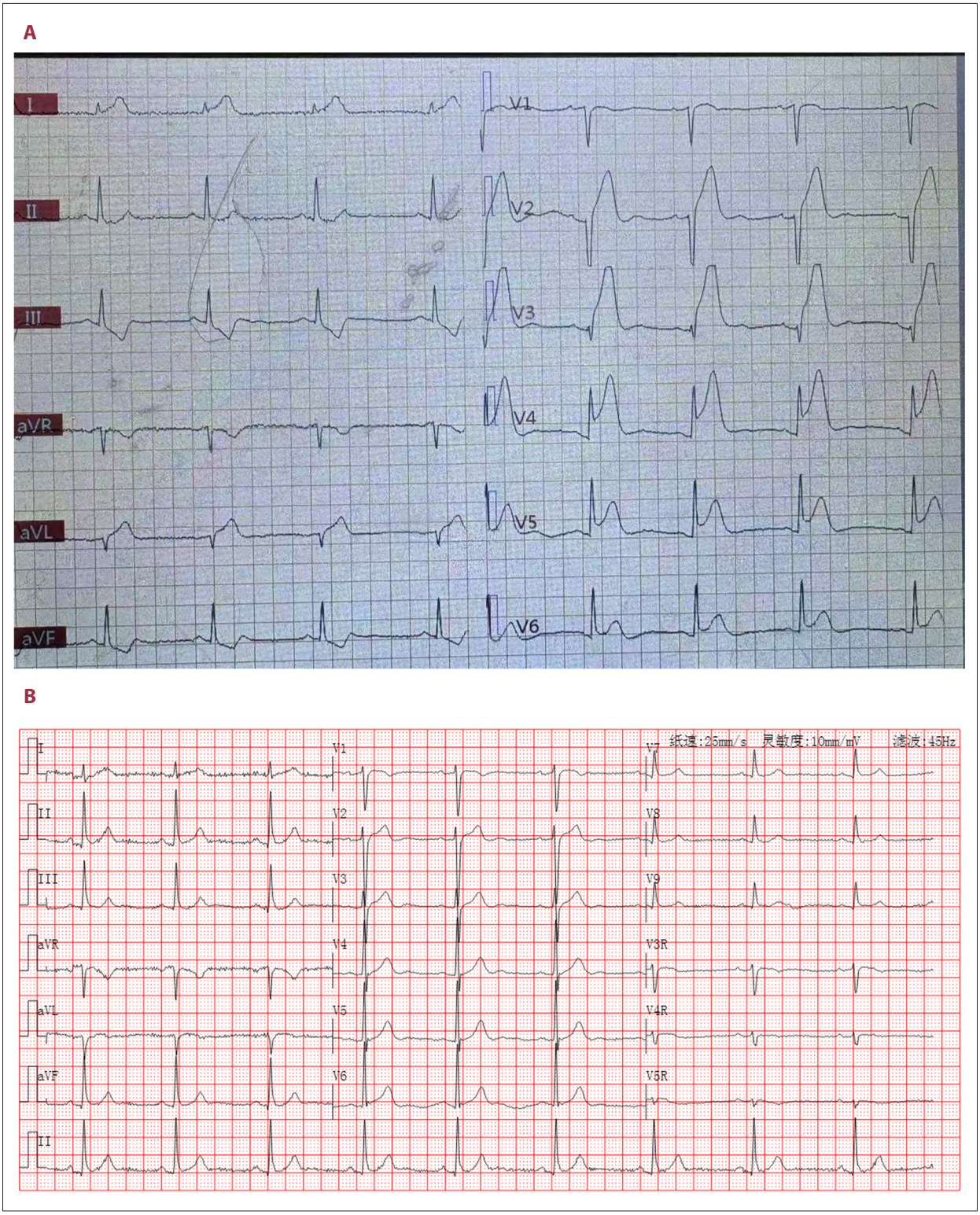

Figure 1. Electrocardiography (ECG) findings. (**A**) Admission ECG showing ST-segment elevation in leads V2-V6, consistent with anterior ST-segment elevation myocardial infarction (STEMI). (**B**) Post-percutaneous coronary intervention ECG demonstrating regression of ST-segment elevation. Both panels are original clinical images from this case and have not been previously published.

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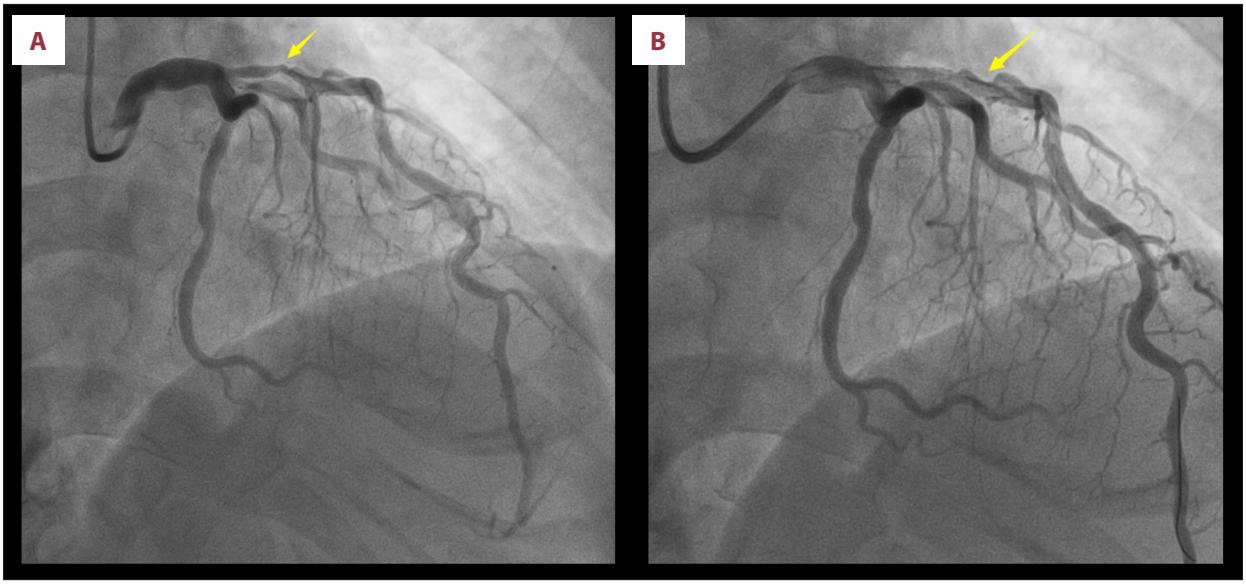

Figure 2. Coronary angiography. (**A**) Severe (approximately 90%) proximal left anterior descending (LAD) artery stenosis with Thrombolysis in Myocardial Infarction (TIMI) grade II flow before intervention. (**B**) Final angiogram after balloon predilation, drug-eluting stent implantation, and postdilation demonstrating TIMI grade III flow without clinically significant residual stenosis. Yellow arrows indicate the culprit proximal LAD lesion before percutaneous coronary intervention and the treated proximal LAD segment after percutaneous coronary intervention. Both panels are original clinical images from this case and have not been previously published.

Therefore, the present case illustrates the diagnostic tension between 2 possible explanations. Specifically, cephalosporin-associated KS has been reported; Fang et al described variable cephalosporin-related presentations, including STEMI and shock [4]. Adachi et al reported that cephalosporin-associated KS can occur without cutaneous manifestations [5]. Pharmacovigilance data also identify antibiotics as important triggers of reported KS cases [6]. However, the patient had 90% fixed proximal LAD stenosis with impaired TIMI grade II flow, which alone provides a strong conventional explanation for anterior STEMI. Consequently, a coincidental plaque-related STEMI temporally associated with cefaclor intake remains as plausible as a hypersensitivity-triggered event.

A structured adverse drug reaction assessment was performed using the Naranjo probability scale [7]. The score was 2: +1 for previous reports of similar reactions, +2 for symptom onset after cefaclor administration, and -1 for the presence of a plausible alternative cause (severe fixed proximal LAD disease). No points were assigned for dechallenge, rechallenge, objective drug levels, placebo response, dose-response relationship, previous similar reactions, or confirmatory objective evidence. This score corresponds to a possible adverse drug reaction and supports only cautious consideration of cefaclor as a potential trigger.

The available data do not demonstrate a systemic allergic reaction. The patient had no urticaria, rash, wheezing, dyspnea, angioedema, oropharyngeal edema, swelling, or hypotension at presentation. The eosinophil count was 0.5×10^9/L, which is at or near the upper limit of normal in many clinical laboratories and does not provide clear evidence of eosinophilia. Serum tryptase, histamine, total IgE, and specific IgE were not measured during the acute phase; no outpatient allergy testing was performed. Therefore, mast cell activation and cefaclor hypersensitivity remain unproven in the present case.

Diagnostic evaluation in similar cases should be pragmatic and should not delay STEMI treatment. Current anaphylaxis guidelines recommend obtaining serum tryptase as early as possible after symptom onset, ideally within 1 to 2 hours and no later than 4 hours, followed by measurement of a baseline level (after recovery) for comparison [8,9]. Total and specific IgE testing and allergy/immunology consultation may help guide future drug avoidance. When available, optical coherence tomography or intravascular ultrasound can help distinguish plaque rupture or erosion from dynamic coronary spasm superimposed on fixed stenosis. In our patient, optical coherence tomography and intravascular ultrasound were not performed; thus, plaque morphology and the presence or absence of superimposed spasm could not be determined.

Patient management in the present case was consistent with the immediate priority in STEMI: urgent reperfusion. Guideline-directed STEMI care, including primary percutaneous coronary intervention, dual antiplatelet therapy, and secondary prevention, remains essential even when KS is suspected [10]. If

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systemic hypersensitivity or anaphylaxis is clinically evident, antihistamines, corticosteroids, and epinephrine may be required; however, epinephrine should be administered with careful monitoring in patients with concurrent myocardial ischemia because it can increase myocardial oxygen demand and may exacerbate vasoconstriction [8,9]. In the present case, no antiallergic therapy was administered because there were no systemic allergic manifestations.

Alternative explanations should also be considered. Antecedent respiratory symptoms indicate that infection-related inflammation may have contributed to plaque instability, given that respiratory infections have been associated with an increased short-term risk of ACS [11]. However, inflammatory markers such as C-reactive protein and procalcitonin were not available. Takotsubo syndrome and myocarditis were considered less likely because coronary angiography identified an obstructive culprit lesion; the ECG changes and symptoms promptly improved after revascularization.

This case is clinically informative not because it proves KS, but because it demonstrates how a potential allergic coronary event should be evaluated when emergency reperfusion takes priority. The report underscores the importance of obtaining a careful drug exposure history, systematically documenting allergic manifestations, collecting early allergy biomarkers when feasible, and avoiding overstating causality when definitive investigations are unavailable.

Conclusions

This case describes anterior STEMI occurring approximately 1 hour after cefaclor exposure. The temporal association prompted consideration of KS, but severe fixed proximal LAD stenosis provides a strong conventional explanation; the absence of serum tryptase, histamine, and IgE testing, allergy follow-up, and intracoronary imaging precluded confirmation of a hypersensitivity-mediated mechanism. The main clinical message is that suspected allergic coronary events should prompt careful documentation and early biomarker sampling when feasible, but urgent STEMI reperfusion should not be delayed.

Institution Where the Work Was Performed

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Patient Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. All images were anonymized and obtained as part of routine clinical care.

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Artificial Intelligence Disclosure

OpenAI ChatGPT was used for language editing, grammar correction, and improvement of readability during manuscript preparation. It was not used for data collection, data analysis, statistical analysis, image generation, image manipulation, or interpretation of clinical findings. All clinical content, diagnostic reasoning, and final manuscript decisions were reviewed and approved by the authors, who take full responsibility for the accuracy and integrity of the work.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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