The Journal of Emergency Medicine, Vol. 49, No. 2, pp. e39–e43, 2015

0736-4679/$ - see front matter

ELSEVIER

http://dx.doi.org/10.1016/j.jemermed.2015.02.044

Clinical Communications: Adults

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MYOCARDIAL INFARCTION IN THE SETTING OF ANAPHYLAXIS TO CELECOXIB: A CASE OF KOUNIS SYNDROME

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☐ **Abstract—Background: Acute coronary syndromes in the setting of an allergic or hypersensitivity reaction are known as Kounis syndrome. The syndrome involves release of inflammatory mediators after an allergen exposure that leads to coronary artery vasospasm or platelet activation. A variety of foods, drugs, and environmental exposures have been implicated in this condition. Case Report: The case involves a 62-year-old woman with dyspnea, chest pain, and transient ST-segment elevation after ingesting celecoxib. Her symptoms resolved with treatment for a suspected allergic reaction. Although she did have mild elevation of serum cardiac biomarkers, subsequent cardiac catheterization demonstrated normal coronary arteries. Why Should an Emergency Physician Be Aware of This?: This is the first reported case of ST-segment elevation myocardial infarction after allergy to celecoxib. Knowledge of Kounis syndrome will better prepare physicians in both its identification and clinical management.** © 2015 Elsevier Inc.

☐ **Keywords—Kounis syndrome; celecoxib; ST-segment elevation; anaphylaxis; hypersensitivity reaction**

INTRODUCTION

Kounis syndrome is the occurrence of acute coronary syndrome secondary to an allergic or hypersensitivity reaction (1). The syndrome is caused by inflammatory mediators released during mast cell activation that induce coronary artery vasospasm and platelet activation (1). A variety of foods, drugs, and environmental exposures have been implicated in this condition (2). Despite numerous case reports, Kounis syndrome remains a poorly recognized clinical entity. We report a case of Kounis syndrome in an adult female shortly after ingesting celecoxib. Awareness of this phenomenon and its proposed treatments will better prepare physicians in both identification of Kounis syndrome and its clinical management.

CASE REPORT

A 62-year-old female with a medical history of hypertension, hypothyroidism, osteoarthritis, and Samter's syndrome (i.e., bronchial asthma, nasal polyposis, and aspirin sensitivity) sought medical attention after experiencing sudden shortness of breath 15 min after taking her prescribed celecoxib. There were no other recent ingestions or known exposures that may have precipitated an allergic response. The patient further noted persistent chest tightness, emesis, skin flushing, and pruritus. She had been taking celecoxib on an intermittent basis for arthritis over the previous 6 months. She denied any previous reaction to this medication. Her only known allergy was an anaphylactic response to aspirin 20 years earlier, which she noted was similar to her current symptoms. The patient was otherwise healthy, does not smoke, and has no significant family history of medical illnesses. She was a seasonal resident on an island off the coast of Maine.

On initial assessment by island emergency medical services, the patient appeared to be in significant respiratory distress with flushed and diaphoretic skin, but no visible urticaria or angioedema. Initial vital signs included a heart

Received: 17 April 2013; Final submission received: 9 January 2015;
Accepted: 27 February 2015

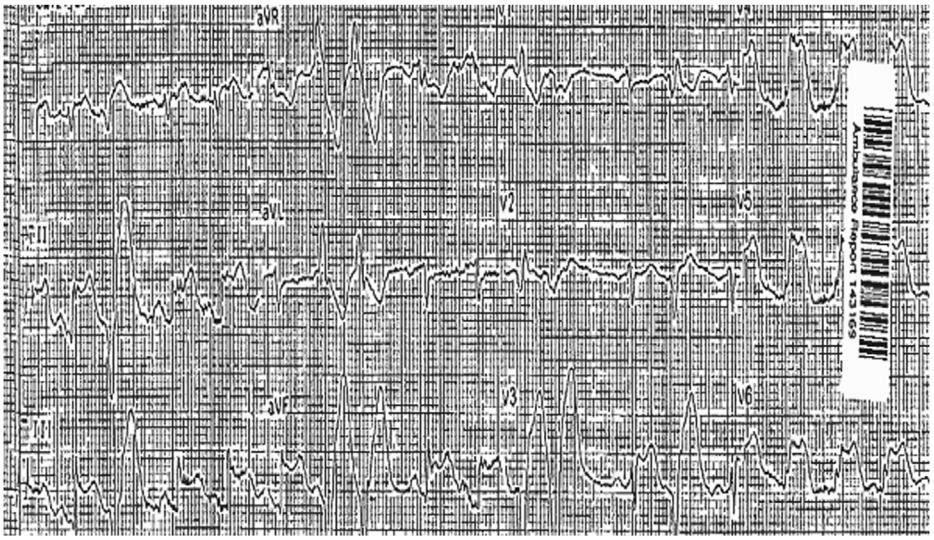

Figure 1. Prehospital 12-lead electrocardiogram showing >4 mm of ST-segment elevation in inferior (II, III, aVF) and anterolateral (V3, V4, V5, V6) leads and ST-segment depression in aVL.

rate of 170 beats/min, blood pressure 82/62 mm Hg, respiratory rate of 20 breaths/min, and oxygen saturation of 86% on ambient air. Cardiac and pulmonary auscultation were unremarkable. Transport time from the scene to the nearest hospital was prolonged due to the need for both rescue boat and ambulance transfers. En route to the hospital, the patient received supplemental oxygen, 1 liter of normal saline, 50mg of intravenous diphenhydramine, and a 2.5 mg albuterol sulfate nebulization. Her hemodynamics and oxygenation improved during transport. Upon arrival to the mainland, a prehospital 12-lead electrocardiogram (ECG) revealed sinus rhythm with 4-mm ST-segment elevations in the inferior and anterolateral leads accompanied by premature ventricular contractions. Reciprocal changes were evident in aVL (Figure 1).

The patient arrived at a nearby tertiary care center 1 h and 15 min after the onset of her symptoms. At that time, her dyspnea and chest discomfort had markedly improved although ST-segment elevations and depression persisted on ECG (Figure 2). Chest x-rays and routine laboratory studies were without abnormalities. Cardiac biomarkers returned mildly elevated, with a troponin T of 0.41 ng/mL, peak total creatinine kinase 218 U/L, and creatinine kinase MB equaling 10.5 ng/mL. While awaiting cardiac consultation, she was administered i.v. doses of methylprednisolone (125mg), unfractionated heparin (60U/kg bolus then 12U/kg/hr drip), and eptifibatide (180mcg/kg bolus followed by 2mcg/kg/min drip) as well as clopidogrel (600mg) taken orally. Aspirin was never given due to reported allergy. Serial ECGs revealed normalization of

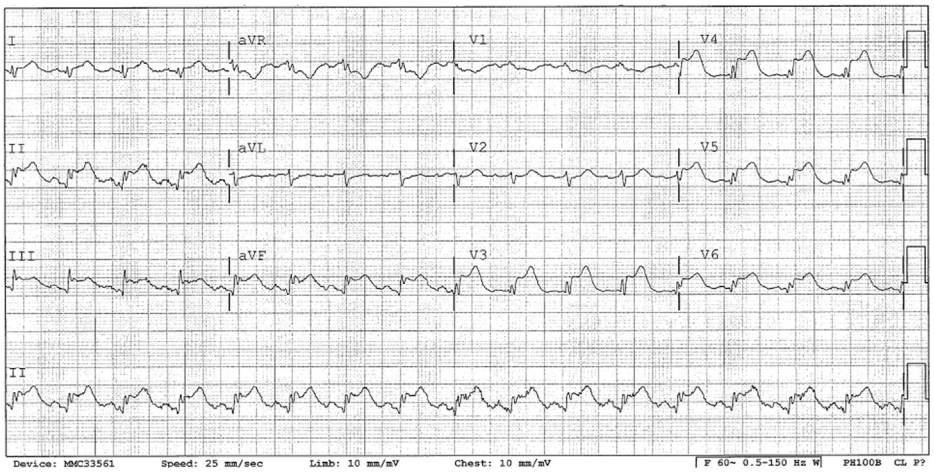

Figure 2. Initial electrocardiogram obtained on arrival to the emergency department and over an hour after symptom onset reveals persistent ST-segment elevation in leads II, III, aVF as well as leads V3–V6. Reciprocal ST-segment depression in aVL continues.