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ACUTE ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION ASSOCIATED WITH DICLOFENAC-INDUCED ANAPHYLAXIS: CASE REPORT

By J. W. B. de Groot, MD, PhD, A. T. M. Gosselink, MD, PhD, and J. P. Ottervanger, MD, PhD

Abstract A patient in whom acute myocardial infarction developed during diclofenac-induced anaphylaxis is described. ST-segment elevation myocardial infarction is a rare complication of anaphylactic reactions, but can occur even in patients with angiographically normal coronary arteries. Physicians should be aware of such a complication in order to diagnose it early and treat it properly. In the patient described here, according to the temporal relationship with diclofenac intake and the exclusion of coronary stenosis, it is probable that diclofenac caused the symptoms. To our knowledge, this is the first reported case of Kounis syndrome due to diclofenac. The patient's recovery was uneventful. (*American Journal of Critical Care*. 2009;18:388,386-387)

The wife of a 48-year-old man called for an ambulance 15 minutes after her husband became unconscious. Paramedics found him incontinent of feces and urine. His medical history included surgery for a herniated disc. He had no history of allergies, eczema, or asthma; he used no medication; and he had eaten his usual breakfast. He was a smoker. His father and grandfather had died of cardiovascular events. The man's blood pressure when the ambulance arrived was 60/40 mm Hg and his heart rate was

100/min. Electrocardiography showed ST-segment elevations in leads II, III, and aVF (see Figure). An acute inferolateral myocardial infarction was diagnosed, and following intravenous fluid replacement the patient was transported to the hospital for coronary angiography.

On admission, clinical examination revealed diffuse erythema with urticaria but no angioedema. The patient reported at this point that his symptoms started after he took 50 mg diclofenac for back pain. He had used diclofenac several years earlier without any problems. Within minutes after ingesting the drug, he experienced pruritus of the lips and tongue, dizziness, nausea, and, eventually, collapse.

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Figure A, Electrocardiogram obtained by paramedics shows ST-segment elevations in leads II, III, and aVF. B, Coronary angiogram shows no significant coronary stenosis.

Treatment was started with 0.5 mg adrenaline and 2 mg clemastine intravenously, and intravenous administration of fluids was continued. He then underwent coronary angiography. The coronary

arteries showed no significant stenosis (see Figure). After coronary angiography, the patient's blood pressure was 90/60 mm Hg; the rash disappeared within hours and the electrocardiographic findings returned to normal. The level of troponin T became elevated (0.28 ng/mL) 3 hours after admission. Level of creatine kinase and creatine kinase-MB fraction remained normal. His full blood count and results of a coagulation screening were within normal limits. Total levels of immunoglobulin E and tryptase measured after 10 hours were normal. Immunoglobulin E specific to a wide range of common

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inhalant and food allergens was negative. By the next day, the patient had recovered uneventfully.

Anaphylaxis is a medical emergency that is manifested by a broad array of signs and symptoms that can be deceptively similar to those of other diseases. Drugs can cause immunoglobulin E-mediated anaphylaxis. Although we cannot entirely exclude significant atherosclerotic burden leading to plaque rupture, such was probably the case in our patient. Nonsteroidal anti-inflammatory drugs are the second most commonly implicated class of medications causing anaphylaxis,¹ and aspirin-induced myocardial infarction has been reported before.²

The concurrence of acute coronary syndromes with mast cell activation induced by allergic and anaphylactic reactions is sometimes referred to as Kounis syndrome.³ The main pathophysiological mechanism of Kounis syndrome is amplified degranulation of mast cells. During mast cell degranulation, several vasoactive compounds (histamine, leukotrienes, and serotonin) and collagen-degrading compounds (neutral proteases) are released locally and in the peripheral circulation, causing a powerful vasoconstriction in the coronary arteries. Additionally, histamine-mediated endothelial activation contributes to progression of acute coronary syndrome by provocation and initiation of coronary artery spasm and/or plaque rupture. Furthermore, histamine activates platelets, triggering thrombosis.³

Conclusion

ST-segment elevation myocardial infarction is a rare complication of anaphylactic reactions, but can occur even in patients with angiographically normal coronary arteries. Physicians should be aware of such a complication in order to diagnose it early and treat it properly. In our patient, according to the tempo-

ral relationship with diclofenac intake and the exclusion of coronary stenosis, it is probable that diclofenac caused the symptoms. To our knowledge, this is the first reported case of Kounis syndrome due to diclofenac. In ST-segment elevation myocardial infarction, angiography with, if necessary, angioplasty is the best approach to diagnose and treat coronary occlusions.⁴ Continuous or delayed allergen absorption could further aggravate anaphylaxis, and a late-phase reaction represents a potential risk in all anaphylactic reactions. Therefore, treatment with adrenaline, antihistamines, and corticosteroids⁵ should be started in addition to coronary intervention.

FINANCIAL DISCLOSURES

None reported.

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NSAIDs are the second most commonly implicated medications causing anaphylaxis.

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