

LETTER TO THE EDITOR

Kounis syndrome: Anaphylaxis causing coronary occlusion

Dear Editor,

A 53-year-old man was transferred by ambulance to our centre with retrosternal chest pain immediately after treatment for anaphylaxis. The patient initially sought care after experiencing shortness of breath, diaphoresis, nausea and dizziness following ingestion of amoxicillin/clavulanic acid. On arrival paramedics found the patient collapsed on his premises with cyanosed lips and generalised urticaria. The patient was treated with two injections of 0.5 mg adrenaline intramuscularly, and additionally given 700 mL i.v. isotonic fluid and 200 mg hydrocortisone intravenously. Approximately 2 min after the second dose of i.m. adrenaline and resolution of anaphylaxis symptoms, the patient began to experience central chest pain. ECG performed on route to our centre displayed sinus tachycardia and ST-elevation in leads II, III and aVF. Aspirin, fentanyl, heparin and ticagrelor were administered and the cardiac catheter laboratory was activated pre-hospitally. On arrival in the ED, the patient had minimal pain and vitals were BP 140/70 mmHg, heart rate 140/min and respiratory rate 20/min; peripheries were warm and well perfused. A subsequent ECG showed AF with presence of ST changes. The patient was transferred to the cardiac catheter laboratory where coronary angiogram revealed 50% stenosis of the LAD, minor disease of the circumflex, 60% stenosis mid-right coronary artery and 99% stenosis distal right coronary artery (Fig. 1). Drug eluting stents were placed in the right coronary artery (Fig. 2), and the patient had an uneventful recovery.

The concurrence of acute coronary syndrome with hypersensitivity

reactions is known as Kounis syndrome.¹ There are three variants of the syndrome in decreasing prevalence: type I: allergy-related angina due to coronary spasm; type II: allergy-related MI due to plaque rupture and thrombus formation; and type III: drug eluting stent thrombosis secondary to hypersensitivity reaction. Type II patients are believed to have inactive but quiescent pre-existing atheromatous plaques. An allergic insult then leads to plaque erosion or rupture and myocardial ischaemia.² Type I Kounis syndrome is most frequently reported (72.6% of cases), with type II representing 22.3% of cases and type III 5.1%.³

The incidence of Kounis syndrome is difficult to estimate, and it is likely that the condition is underreported,³ with limited Australian cases reported in the literature. A prospective study⁴ estimated the incidence of Kounis syndrome to be 19.4 per 100 000 admissions.⁴

MI in these circumstances can also be attributed to the therapeutic use of adrenaline for anaphylaxis, although i.m. adrenaline-induced MI is very uncommonly reported.² In this case, it is likely that the patient suffered Kounis syndrome type II, as there was evidence of coronary artery disease and had pre-existing atheromatous plaques as well as plaque rupture during catheterisation.

Emergency physicians are sometimes faced with patients who have two or more conditions that may require opposing treatments. In Kounis syndrome, anaphylaxis has the potential to alter the traditional initial management of acute coronary syndrome, with Table 1 summarising management considerations for the three variants of Kounis syndrome. These considerations outline the



Figure 1. Percutaneous coronary intervention, prior and post stent insertion in right coronary artery. Arrow indicates occlusion prior to stenting.



Figure 2. Angiogram post stent.

relative merits and risks and will inform the treating clinician to make the most appropriate decision for each individual situation.

Nitrates may not be appropriate in the setting of anaphylaxis as they may worsen hypotension. Additionally,

TABLE 1. Management considerations for Kounis syndrome variants†

Type I	Type II	Type III
May benefit from treatment of allergic reaction, circumventing further complications ⁵	Acute coronary event protocol ⁵	Acute coronary event protocol ⁵
H1 and H2 antihistamines (e.g. diphenhydramine 1–2 mg/kg) for non-severe symptoms ⁵	Caution with nitrates in hypotension ¹	Caution with nitrates in hypotension ¹
Must be given slowly as they can precipitate hypotension and compromise coronary blood flow ⁵	Beta-blockers contraindicated, ^{1,5} patients may benefit from glucagon ⁵	Urgent aspiration of intrastent thrombosis ⁵
There are no drugs of choice in initial severe allergic reaction ⁵	Opioids administered with caution, ^{1,5} consider fentanyl ¹	Mast cell stabilisers along with corticosteroids and anti-histamines to potentially alleviate allergic symptoms from stent implantation ⁵ Opioids administered with caution, ^{1,5} consider fentanyl ¹

†Type I: allergy-related angina due to coronary spasm; type II: allergy-related MI due to plaque rupture and thrombus formation; type III: drug eluting stent thrombosis secondary to hypersensitivity reaction.

opioids such as morphine may induce mast cell degeneration, aggravating the anaphylaxis. Beta-blockers counteract the actions of adrenaline in anaphylaxis and some literature suggests not using them at all when acute coronary syndrome is suspected in the setting of anaphylaxis.¹ Even aspirin is potentially detrimental, producing more anaphylaxis mediators through cyclo-oxygenase inhibition.¹

These medicines are commonly used in the early hospital management of acute coronary syndrome, and it is unclear what the optimum management for patients with concurrent anaphylaxis and MI is. This case highlights the need to outweigh the relative urgency and risk of anaphylaxis and its treatment against the risks of acute coronary syndrome and its treatment.

Competing interests

GK is a section editor for *Emergency Medicine Australasia*.

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