



Correspondence

Intraoperative anaphylaxis, cardiac arrest and hypertrophic cardiomyopathy: Implications for Kounis syndrome


Anaphylactic reactions occurring during general anesthesia constitute the most severe and frequent forms of drug-induced hypersensitivity reactions. Although bronchospasm, cutaneous and mucus signs and symptoms are easily recognized, signs and symptoms that mainly involve the cardiovascular system constitute diagnostic and therapeutic problems. In the very interesting report published recently [1], a 75-year-old woman, hypertensive, smoker and allergic to penicillin and cephalosporin with vancomycin spacer placement, developed twice hypotensive cardiac arrest with pulseless electrical activity during preoperative general anesthesia for left total hip re-implantation. Bupivacaine, fentanyl, lidocaine, propofol, succinylcholine and vancomycin were given induction for induction. She was resuscitated successfully with chest compressions, epinephrine and diphenhydramine administration and vancomycin withdrawal. Serum tryptase and troponin were raised suggesting myocardial ischemia. Transthoracic echocardiography revealed hypertrophic cardiomyopathy with asymmetrical septal hypertrophy and severe mitral regurgitation.

This report raises important questions:

1. Vancomycin was the most likely cause for the patient's intraoperative anaphylaxis since the patient had vancomycin spacer placement. This patient was allergic to penicillin and cephalosporin and such patients have more symptoms than do mono-sensitized individuals [2]. IgE antibodies with different specificities have additive effects and even small amounts of corresponding antigens can trigger mediator release when the patient is simultaneously exposed to them [3]. The patient had received pre-operatively apart from vancomycin and 5 anesthetic agents including bupivacaine, fentanyl, lidocaine, propofol, succinylcholine that could have acted as antigens with additive effects. The authors wisely did not perform vancomycin skin testing due a high rate of false positivity but searching for specific IgE antibodies for each of the given drugs could have identified the culprit agent.
2. Post-event electrocardiogram was unchanged from baseline, but serum troponin and tryptase were raised. These findings are compatible with the diagnosis of allergic myocardial infarction or type II Kounis syndrome [4]. This variant includes patients with culprit but quiescent pre-existing atheromatous disease in whom the acute release of inflammatory mediators can induce coronary artery spasm progressing to acute myocardial infarction with increased troponin and cardiac enzymes. Coronary spasm might be transient, lasting shortly, and is unpredictable. The electrocardiogram might be normal and only ambulatory electrocardiographic detect the spasm. The patient was smoker and hypertensive, therefore quiescent pre-existing atheromatous disease could not be excluded.
3. The patient was found to have a previously undiagnosed hypertrophic cardiomyopathy with asymmetrical septal hypertrophy and

severe mitral regurgitation. Coexisting of hypertrophic cardiomyopathy with coronary spasm is not frequent. In a study of 36 patients with hypertrophic cardiomyopathy, challenged with acetylcholine, coronary vasospasm was induced in 10 (28%). It was concluded that coronary vasospasm appears to play significant role in etiology of myocardial ischemia in patients with hypertrophic cardiomyopathy. Smoking might be a major risk factor for coexistence of coronary vasospasm. The patient was nicotine dependent and this supports our view that this patient had suffered a Kounis type II coronary spasm.

4. Anaphylaxis created acute decrease in preload, producing dynamic left ventricular outflow obstruction and subsequently pulseless electrical activity arrest. The heart and coronary arteries are primary targets of anaphylactic reactions and the acute decrease in preload was due to reduced cardiac output from coronary vasoconstriction rather than due to fluid extravasation [5]. The reduced preload is accompanied by increased troponin levels due to myocardial damage as has been occurred in the described patient.

All cases of perioperative coronary anaphylaxis require additional work-up to identify the offending agent and to avoid future reactions. This workup should include, apart from detailed history of drug allergy and drug adverse reactions, troponin and tryptase measurements, estimation of IgE and especially specific IgEs for the drugs involved, patch testing, skin prick, and intradermal skin testing when and where these are appropriate.

References

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