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KOUNIS SYNDROME – AN ALLERGIC HEART ATTACK

Anaesthesiology

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ABSTRACT

Kounis syndrome is an unexpected emergency secondary to an allergic reaction to commonly used drugs.It is commonly reported with the use of anaesthetic drugs & antibiotics.But its incidence is rare with analgesics.Herein we report a case where an unexpected catastrophe had occurred post operatively with the use of Tramadol.

KEYWORDS

Tramadol, Myocardial infarction, Allergy, Kounis syndrome

INTRODUCTION

The first case of myocardial infarction during an allergic reaction in humans was reported in 1950 [1]. In 1991, Kounis and Zavras described a 'syndrome of allergic angina' as the simultaneous occurrence of chest pain and allergic reactions, accompanied by clinical and laboratory findings of angina pectoris, caused by inflammatory mediators released during the allergic process [2].It is hypothesized that vasospastic angina could be induced by functional and metabolic changes in the heart caused by the release of inflammatory mediators such as histamine and leukotrienes acting upon the smooth muscle of the coronary arteries following a serious allergic insult [1, 2].Several drugs, conditions, environmental exposures, latex, foods, venom and toxins have been reported to cause this rare-life threatening syndrome. Perioperative anaphylaxis occurs in 1:3 500–1:20 000 cases, with a mortality of 9% [3] It accounts for 9–19% of anaesthetic associated complications and 5–7% of deaths during anaesthesia [3,4].The most common agents implicated in perioperative anaphylaxis are neuromuscular blocking agents (58%), latex (19.6%) and antibiotics (12.8%) [5].Kounis syndrome is probably under-reported, with anaesthesiologists and intensivists generally unaware of the condition. Herein, we report the case of a patient developed coronary vasospastic angina (type 1 variant of Kounis syndrome) immediately after the administration of tramadol.

CASE REPORT

A 23-year-old man was admitted to our ICU postoperatively following incision & drainage of a Parotid abcess which is of two weeks duration under general anaesthesia.Any adverse event was not observed in the operating room.During clinical follow-up in the ICU,50 mg tramadol was givens by IV injection for pain relief. Just immediately after the administration of the tramadol IVinjection,he began to present feeling of instability, excessive sweating, palpitations & retrosternal chest pain.His past medical history was unremarkable with no cardiovascular risk factors, systemic illness or atopic disease. However,he was consistently having high blood sugars since the time of admission.HbA1c revealed to be 7.8 % suggesting undiagnosed diabetes mellitus.Following the event, the electrocardiogram (ECG) showed 2.5 mm ST-segment elevation in leads II,III,aVF along with V4 – V6 and marked reciprocal ST-segment depression in leads V1 – V3 (Fig. 1). Then,Cardiology consultation was given. Physical examination revealed a blood pressure of 80/48 mmHg with a regular pulse of 122 beats/min, respectively & required vasopressor support.Cardiovascular and respiratory auscultation findings were unremarkable. Troponin T level (990 ng/L) and CK-MB level (65 IU/L) were high. A transthoracic echocardiography examination revealed global hypokinesia with akinetic septum and moderate left ventricular dysfunction with an ejection fraction of 35%. Standard treatment for acute coronary syndrome with subcutaneous injection of 1mg/Kg enoxaparin, 80mg Atorvastatin,a loading dose of 300 mg Aspirin and 300 mg Clopidogrel were administered. During the follow-up,the patient's chest pain was relieved and ST segment elevation was resolved . A coronary angiography was performed immediately and revealed normal right and left coronary arteries . Thus, his medical therapy was continued with analgesic, antibiotic, heparin, statin ,75 mg/ day clopidogrel & Aspirin. No recurrent angina was observed during hospitalization, and he was extubated on the fourth day once he was off the vasopressor support. Follow-up ECG was unremarkable. Transthoracic echocardiography examination was repeated before the discharge and revealed that regional wall motion abnormality was vastly improved with improved ejection fraction.

We explored the other possible allergic medications prior to the tramadol, which might have gone unnoticed. He had received 4.5 grams IV Piperacillin & Tazobactum & respective general anaesthetic drugs, but any symptom of a possible allergic reaction was not observed during the administration of these drugs. We had hypothesized that the hypersensitivity reaction following IV administration of tramadol probably caused the anginal attack and ECG changes and, thereby, the diagnosis of Kounis syndrome was made. The patient was subsequently found to be growing Burkholderia psuedomallaei in the blood & a diagnosis of Melioidosis was made. Treatment was started accordingly.

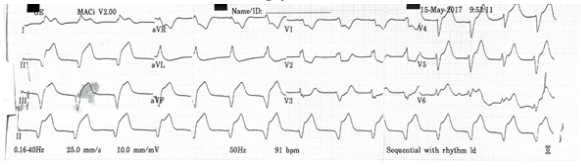

Figure 1: Electrocardiogram (ECG) showing 2.5 mm ST-segment elevation in leads II, III, aVF along with V4 – V6 and marked reciprocal ST-segment depression in leads V1 – V3

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