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RESEARCH ARTICLE

A RARE CASE OF KOUNIS SYNDROME PRESENTING TO THE EMERGENCY DEPARTMENT

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ABSTRACT

Anaphylaxis is a serious life- threatening allergic reaction and requires emergent diagnosis and treatment. The first – line pharmacological treatment in anaphylaxis is intramuscular adrenaline. Concurrent occurrence of acute coronary syndromes with hypersensitivity reactions is a rare but possible occurrence known as 'Kounis Syndrome'. We report a case of a young healthy male with no significant risk factors for coronary artery disease developed ventricular tachycardia and myocardial infarction following therapeutic dose of intramuscular administration of adrenaline for an anaphylactic reaction to the ED.

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INTRODUCTION

Anaphylaxis is a serious life- threatening allergic reactionand requires emergent diagnosis and treatment. The first – line pharmacological treatment in anaphylaxis is intramuscular adrenaline. Kounis syndrome is the concurrent occurrence of acute coronary syndromes with hypersensitivity reactions (Kounis, 1991 and Petrus Fourie, 2016). It has been established as a hypersensitivity coronary disorder induced by various conditions, drugs, environmental exposures, foods and coronary stents. Allergic, hypersensitivity, anaphylactic and anaphylactoid reactions are associated with this syndrome. Vasospastic allergic angina, allergic myocardial infarction and stent thrombosis with occluding thrombus infiltrated by eosinophils and/or mast cells. There are three principal variants of the syndrome: type 1 is allergy – related angina due to coronary spasm, whereas, type 2 is allergy related myocardial infarction due to plaque rupture or erosion and type 3 is allergy – related stent thrombosis with re – infraction. Adrenaline induced allergic myocardial infarction during management of anaphylaxis has been reported only on a few occasions.

Case History: A 31-year- old previously healthy male came to the ED with an urticarial rash all over the body, one hour after ingestion of Augmentin – 625 duo Tablet, which he was prescribed earlier that day for Sinusitis. On arrival to the ED his airway was patent, saturation was 96% on room air, respiratory rate was 20/minute, pulse rate was 114/minute, blood pressure was 132/80 mmHg. Cardiac monitor showed sinus tachycardia. On examination, he was having erythema all over his body, and bilateral wheeze. He was treated initially with Intravenous H1- receptor blocker, Corticosteroid and H2 receptor blocker. As the patient continued to worsen and developed shortness of breath, monitor showed vitals; HR - 124/min, BP- 140/84 mmHg, saturation – 96% on room air (RA). He was given 0.5 ml of intramuscular adrenaline (1:1000), administered into the antero -lateral side of the thigh, 2 minutes following which, patient became more restless, and developed palpitations, chest tightness and retrosternal chest pain. Cardiac monitor showed Monomorphic Ventricular Tachycardia, with HR - 184/min, BP – 154/96 mmHg, saturation – 95% on RA. Immediately patient was administered intravenous Amiodarone, as per the ACLS protocol. Following which, the rhythm, reverted to Sinus Rhythm. Electrocardiogram showed ST segment depressions in leads II, III, aVF and V3 to V5 and ST segment elevation in leads I and aVL. Patient's saturation was 95% on room air, HR – 98/min, blood pressure fell to 80/50mmHg, for which he was given a bolus of 1litre NS, but as it did not help, patient was subsequently started on Inotropes, in view of hypotension. Cardiology team was alerted about the changes noted in the Electrocardiogram (ECG). On advice of the cardiologist, patient was administered antiplatelet, statins and anticoagulants and the Electrocardiogram repeated after 2 hours showed a normal sinus rhythm.

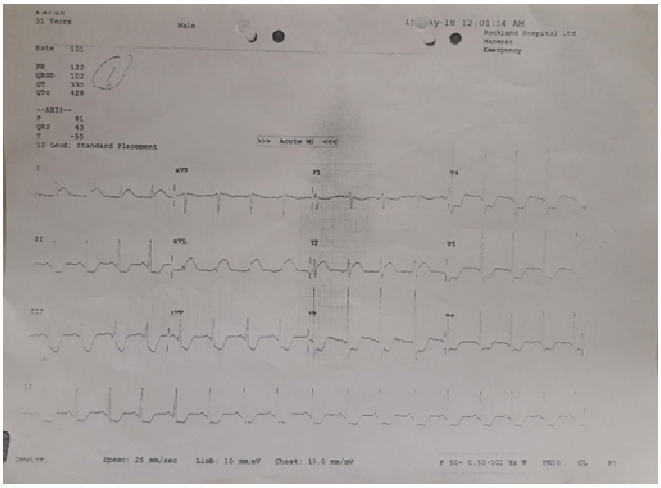

Figure 1. Electrocardiogram after administration of Intramuscular Adrenaline

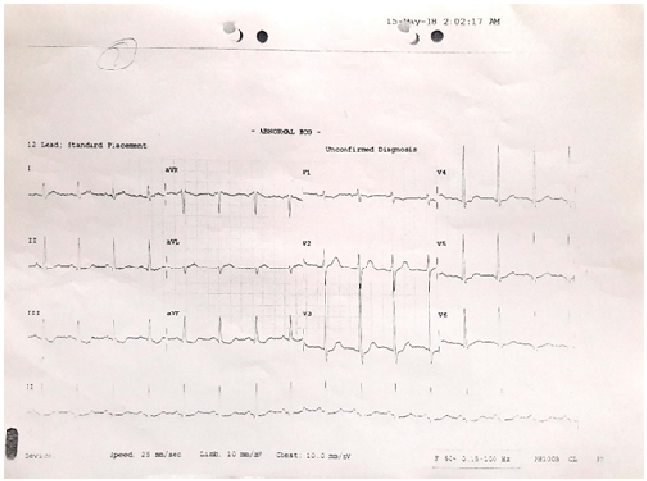

Figure 2. Two hours after the initial Electrocardiogram

Troponin T was positive with a titre of 0.994 ng/ml (Normal Range: < 0.01ng/ml). CPK-MB was positive with a value of 66.3 IU/L (Normal Range: 5 – 25 IU/L). The tests were repeated on the second day and were still positive with a Trop T tire of 0.974 ng/ml and CPK-MB OF 54.7IU/L (< 24 IU/L). Further investigations included, 2D echocardiogram which revealed an ejection fraction of 60% with no regional wall motion abnormalities. Coronary Angiogram, was done which revealed slow flow in LAD, but no atherosclerotic plaques. Complete blood count showed Leucocytosis. Renal functions were normal.

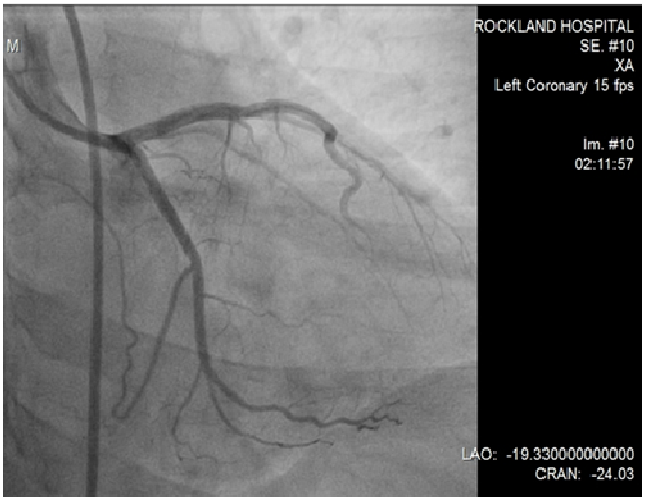

Figure 3.

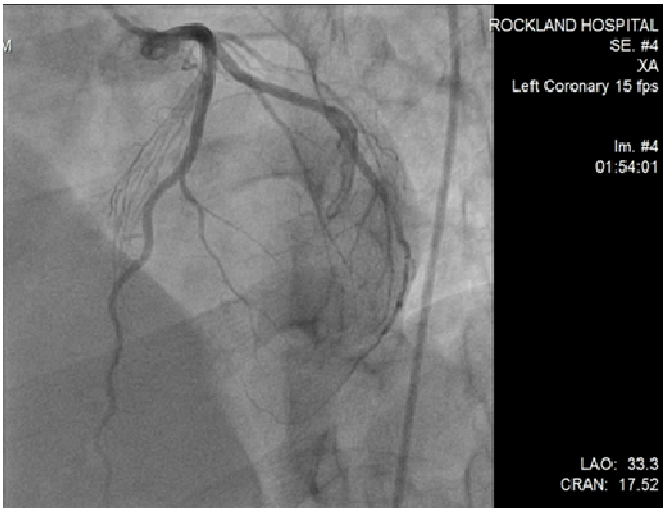

Figure 4. Coronary Angiogram – Normal Coronaries

He was previously healthy, has no history of any predisposing factors, nor did he have a history of any co-morbidities like asthma, diabetes mellitus, hypertension or coronary artery diseases. He has had no history of any known allergies. Patient was asymptomatic during the hospital stay and was discharged after 2 days. At subsequent reviews after 1 week and 15 days respectively he remained asymptomatic.

DISCUSSION

Anaphylaxis is a serious life- threatening allergic reaction, with a rapid onset; it may cause death and requires emergent diagnosis and treatment. The first – line pharmacological treatment in any patient suspected to have anaphylaxis is 1:1000 dilutions of 0.3 - 0.5 ml of intramuscular adrenaline (Tintinalli, 2016). Adrenaline acts on alpha and beta adrenergic receptors. Through the alpha adrenergic receptors adrenaline cause vasoconstriction, increase peripheral vascular resistance whereas, through the beta adrenergic receptors it causes increased inotropic and chronotropic effects. In addition to these, adrenaline has a number of undesirable side effects, like palpitations, tremors, dizziness, and also more severe side effects like ventricular arrhythmias, pulmonary oedema, hypertensive crisis, myocardial ischaemia or infarction (Tintinalli, 2016). Adrenaline induced myocardial infarction in the setting of anaphylaxis could be due to the anaphylaxis itself known as Kounis syndrome, or due to the effect of adrenaline (Jayamali, 2017). Adrenaline induced myocardial infarction during management of anaphylaxis has been reported only on a few occasions. A similar case as ours, was reported by Jayamali and colleagues were a previously healthy male developed myocardial infarction after use of adrenaline, and coronary arteries were found to be normal (Jayamali, 2017). In another case reported by Bawaskar and colleagues, a middle aged female developed severe allergic reaction and myocardial infarction after consumption of pantoprazole (Himmatrao, 2017). However, the benefits of early administration of adrenaline in anaphylaxis outweigh its side effects.

Conclusion

It was therefore, thought that this patient might have had a coronary vasospasm to Epinephrine, or vasospastic allergic myocardial infarction – 'Kounis syndrome', which might have lead the course of events. Although development of allergic vasospasm to Epinephrine is a rare occurrence, its

administration through intramuscular route is considered to be the first line of treatment for anaphylaxis which is a life – threatening condition. During an allergic reaction, histamine dilates the coronary arteries (via H_1 – receptors on vascular smooth muscle cells) and causes arrhythmias and atrio – ventricular conduction blocks in healthy individuals. Systemic allergic signs and symptoms accompanied by clinical, electrocardiographic or laboratory findings of myocardial ischaemia constitutes Kounis Syndrome.

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