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Coronary Vasospasm after Giving Adenosine for Supraventricular Tachycardia

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Abstract

Palpitations due to supra-ventricular tachycardia are a common mode of presentation to the emergency department. A 12 lead electrocardiography usually leads to immediate diagnosis and prompt management of this condition. Adenosine injection is the treatment of choice for rapid termination of supra-ventricular tachycardia which is widely used. It is generally considered safe and serious side effects are rare. We presented a rare case of a post-menopausal female admitted with supra-ventricular tachycardia and after injection of Adenosine, chest pain with a transient elevation of ST segment occurred. The ST segment changes reverted back to normal after a few minutes spontaneously without any therapy. This phenomenon could be explained due to coronary vasospasm produced by Adenosine. A review of literature revealed anecdotal cases of adenosine induced possible coronary vasospasm.

INTRODUCTION

Palpitation due to supra-ventricular tachycardia is a common mode of presentation to the emergency department. A 12 lead electrocardiography usually leads to immediate diagnosis and prompt management of this condition. Adenosine injection is the treatment of choice for rapid termination of supra-ventricular tachycardia and is widely used for the purpose. It is generally considered safe and serious side effects are rare. We presented a case of a post-menopausal female admitted with supra-ventricular tachycardia and after injection of Adenosine, chest pain with a transient elevation of ST segment happened. ST segment changes reverted back to normal after a few minutes spontaneously without any therapy. This phenomenon could be explained due to coronary vasospasm produced by Adenosine. A review of literature revealed anecdotal cases of adenosine induced possible coronary vasospasm.

CASE PRESENTATION

A 53-year-old post-menopausal female presented to the emergency department with complaints of palpitations since last night associated with vomiting. There was no complaint of chest pain, dyspnea, abdominal pain or diaphoresis. She had no history of cardiovascular disorders. On examination she was conscious, well-oriented with a pulse rate of 180/min and regular. The blood pressure recorded in the right arm in supine position was 110/80 mm of Hg.

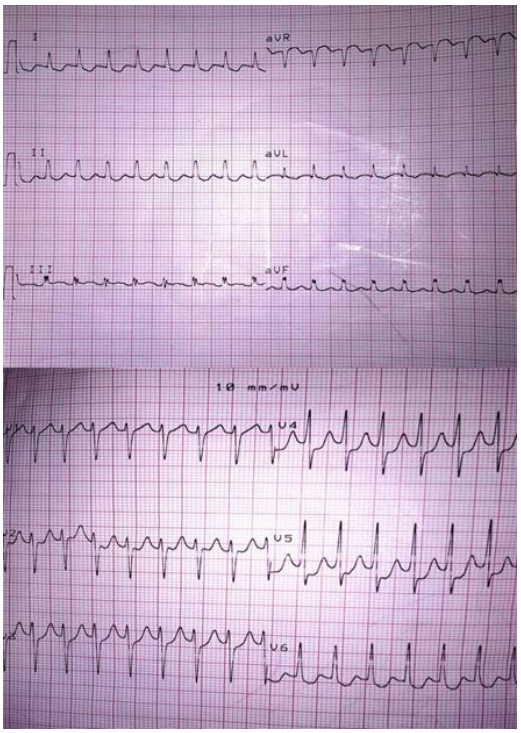

Figure 1. The Arrhythmia Electrocardiography showed a Regular Narrow Complex Tachycardia with a Rate of 200 Beat/Minute

She was tachypneic with a rate of 28/min with arterial saturation of 98% breathing ambient air. Jugular venous pulsations were prominent. The rest of examinations were unremarkable. All routine hematological and biochemical investigations were within the normal limits. Electrocardiography revealed a narrow complex tachycardia with regular RR rate of about 200/minute. No obvious p waves were visible (Fig 1).

The electrocardiography was suggestive of atrioventricular (AV) nodal reentrant tachycardia. As the patient was hemodynamically stable and there was no response to vagal maneuvers, the patient received adenosine intravenously in the antecubital vein in a total dose of 6 mg interrupting the tachycardia. Soon the patient developed chest pain. The electrocardiography at this time showed a new ST elevation in inferior leads and ST depressions in leads I and aVL (Fig 2)

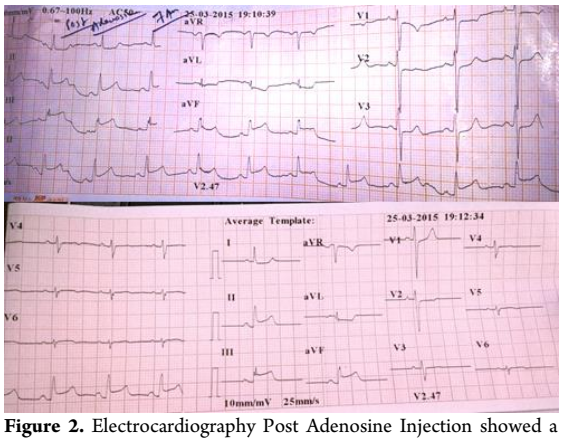

Figure 2. Electrocardiography Post Adenosine Injection showed a New ST Elevation in Inferior Leads and ST Depressions in Leads I and aVL.

This suggested evolving ST elevation myocardial infarction in the inferior wall region. However, these changes were short lived and within a few minutes all ST segment changes reverted back to normal without any specific treatment (Fig 3).

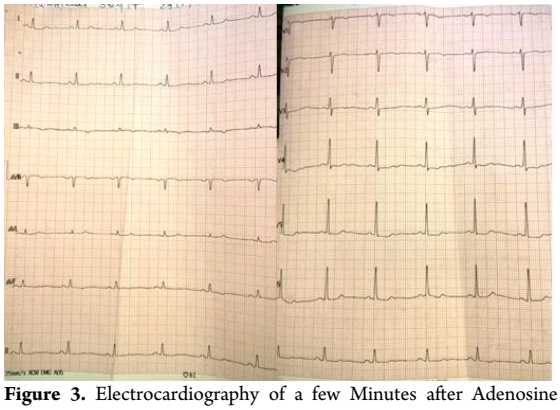

Figure 3. Electrocardiography of a few Minutes after Adenosine Injection, all ST Segment Changes reverted Back to Normal.

Transthoracic echocardiography revealed normal left ventricle with no wall motion abnormalities. Hence, these changes were interpreted as Adenosine induced coronary vasospasm. We did not obtain troponin.

DISCUSSION

The narrow complex tachycardia is defined as tachycardia with the presence of QRS duration of less than 120 ms duration which are typically of supra-ventricular origin [1]. The supra-ventricular tachycardia may be classified on the basis of origin into sinoatrial tachycardia, atrial tachycardia, junctional tachycardia, AV node reentrant tachycardia and AV reciprocating tachycardia using an accessory AV pathway.

The use of vagal maneuvers including the Valsalva maneuver and carotid sinus massage is a proper treatment option for these patients but with a success rate of less than 50% in most studies [2, 3].

Adenosine is used as drug of choice for prompt conversion of supra-ventricular tachycardia to sinus rhythm because of its high efficacy (90–95%) and very short duration of action. For this purpose, it is given in a bolus dose of 6 mg followed, if necessary by another dose of 12 mg. Its mechanism of action involves activation of an inward rectifier K+ current and inhibition of calcium dependent action potentials. When given as a bolus dose, adenosine directly inhibits AV nodal conduction. Given as an infusion, it is also used to study coronary circulation whereby it leads to coronary hyperaemia and steal [4].

Its common side effects are flushing, bronchospasm and high-grade AV block. Less common adverse effects are headache, hypotension, nausea and paresthesias, atrial fibrillation and ventricular tachycardia. There are reports of adenosine leading to myocardial infarction. The first case was reported by Polad JE and Wilson LM, where a 65-year-old woman with history of ischemic heart disease underwent standard adenosine stress test for myocardial perfusion imaging, but sustained inferior myocardial infarction during the final stages of the stress test [5]. Adenosine use leading to coronary vasospasm is extremely rare phenomenon and should be kept in mind [6].

Coronary vasospasm, or smooth muscle constriction of the coronary artery, is an important cause of chest pain which can lead to myocardial infarction (MI). Severe chest pain, usually without physical effort and with a concurrent electrocardiography showing transient ST elevation, is the key for the diagnosis of coronary artery spasm [7].

In a review performed by Menyar AA, among 220 articles (>12000 cases) related with MI with normal coronary angiogram, 50 articles (~100 cases) reported the role of drug in MI secondary to coronary artery spasm. Coronary spasm was associated with 12 illicit substances in teenagers (i.e., cocaine, marijuana, alcohol, butane and amphetamine) and with 19 types of medications (i.e., over-the-counter, chemotherapy, antimigraine and antibiotics) without any relation to age of the patient [8]

CONCLUSIONS

Adenosine is one of the most commonly used anti arrhythmic drugs and is considered very safe. It is also used widely to study coronary circulation as it produces coronary hyperaemia. But it may not be safe always as it causes myocardial ischemia due to coronary steal phenomenon and can rarely lead to coronary vasospasm as in our case. Its side effects should be borne in mind and it should be used cautiously in high risk groups which are yet not defined.

Disclosures

None

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