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Case Report Open Access

Acute ST-Segment Elevation Induced by Intravenous Contrast Medium

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Introduction

Acute ST elevation myocardial infarction following contrast medium use is rare. It falls under the spectrum of "cardiac anaphylaxis". Exact pathophysiological mechanism is unclear, but it appears to involve coronary vasospasm mediated by the vasoactive substances released during anaphylaxis.

Case Report

We present a case of a 37 year old Caucasian male with a history of HTN, active tobacco use and hepatitis C untreated due to poor compliance with previously known history of contrast allergy who presented to the emergency room with chest pain atypical for angina, palpitation and cough. Initial evaluation showed patient to be hemodynamically stable with blood pressure 114/76 mmHg, pulse rate 78 bpm, respiratory rate 16 per min and Oxygen saturation (SpO_2) of 98%. His first ECG in the ER revealed no evidence of active ischemia, and Chest X-ray revealed no acute cardiopulmonary process (Figure 1).

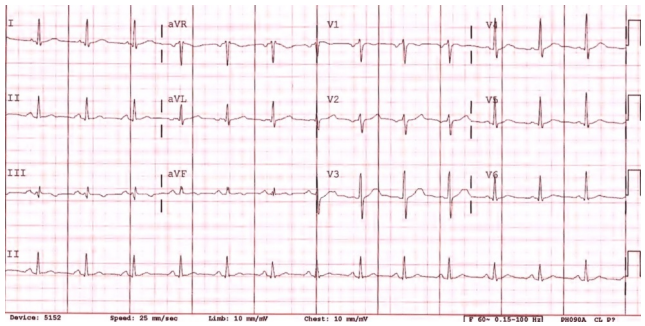

Figure 1: Initial ECG.

CBC	CMP	Others
Hb 15.6 gm/dl	Na 136 mmol/L	APTT 31
Hct 46.7 %	K 3.1 mmol/L	PT 12.3
WBC 5,700	BUN 17 mg/dL	PT 1.1
Plat 204,000	Crea 1.1 mg/dL	D-dimers 1,015
MCV 87.2 fL	ALT 65 U/L	Troponin I <0.045 ng/ml
	AST 37 U/L	

Table 1: Pertinent lab indices.

Laboratory indices, however, showed a positive D-dimer (a value of 1,015 ng/ml) while his initial troponin I was negative (Table 1). He was pre-medicated with IV Methylprednisone 125 mg, IV Benadryl 25 mg and IV Pepcid 20 mg prior to CAT scan of the chest with IV contrast which excluded a pulmonary embolus.

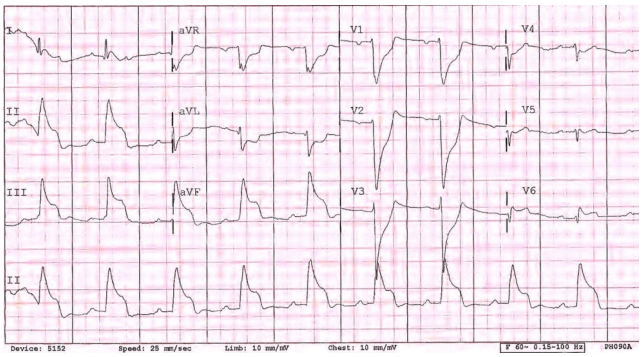

Figure 2: ECG post contrast.

A few minutes after this procedure, he developed typical angina, urticarial rash on his torso and arms with mild swelling of his lips and ECG done showed ST-segment elevation involving the inferior leads II, III and a VF with reciprocal changes in the anterolateral leads (Figure 2).

His systolic blood pressure dropped to about 90 mmHg and with mild tachycardia. He subsequently received IV Decadron 20 mg, IV Benadryl 25 mg, IV Pepcid 20 mg and IV heparin 5000 units as well as a bolus of a litre of Normal Saline. Patient had already received a full dose Aspirin 325 mg on route to the hospital before presentation.

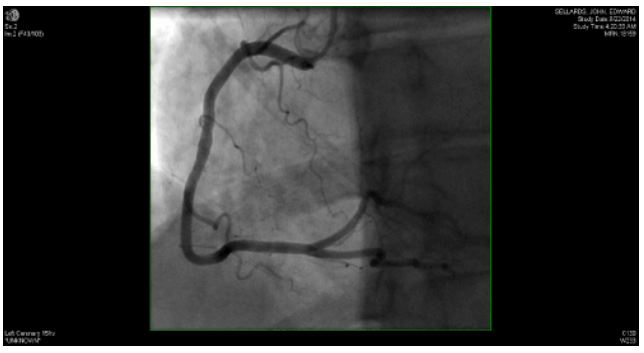

Figure 3: Right Coronary system.

No epinephrine was given as his hemodynamic status remained relatively stable and a suspected ischemic process was on going. Subsequent repeat ECG showed near resolution of ST-segment elevation. The catheterization lab was immediately activated and coronary arteriography done showed no obstructive lesion or thrombus in the right or left coronary system (Figures 3 and 4). He was observed in the coronary care units for over 24 hours prior to being discharged in a stable condition.

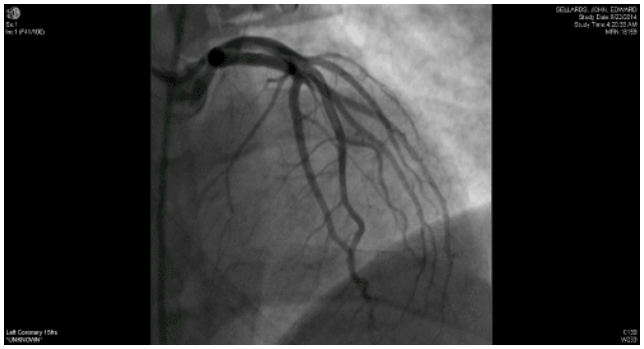

Figure 4: Left Coronary system.

Discussion

Anaphylactic reaction may trigger vascular insult in various ways largely by release of inflammatory mediators and a known but rare potential complication is acute coronary syndrome either in patients with existing coronary artery disease or rarely in those with normal coronaries [1]. Coronary vasospasm is the proposed underlying mechanism of allergy-induced coronary syndromes. Mast cells have been demonstrated within the human heart to be located perivascular in proximity to myocytes [2] and activation of these cells through immunological stimuli lead to release of potent vasoactive and pro-inflammatory mediators (histamines, prostaglandins, leukotriene, cytokines etc.) which can directly or indirectly induce coronary spasm.

The mast cells also through some of these pro-inflammatory agents can be pro-coagulant inducing platelet aggregation and thrombus formation [2]. In our patient, because of the sequence of his angina following administration of contrast medium, cardiac anaphylaxis was strongly considered as an etiological process. Also given the transient nature of the ST elevation with absence of obstructive disease or thrombus on the coronary arteriography, vasospasm is the proposed causative mechanism. Ours could well be considered a type of Kounis syndrome which is the occurrence of acute coronary syndromes with conditions associated with mast cell activation, including allergic or hypersensitivity and anaphylactic or anaphylactoid insults [3]. In case of coronary vasospasm in the setting of anaphylaxis or allergic insult, supportive therapy with a vasodilator if patient is hemodynamically stable and steroid may be effective in management [4].

References

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