Case based learning points

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Allergic Acute Coronary Syndrome; A Fatal Presentation in the Emergency Department

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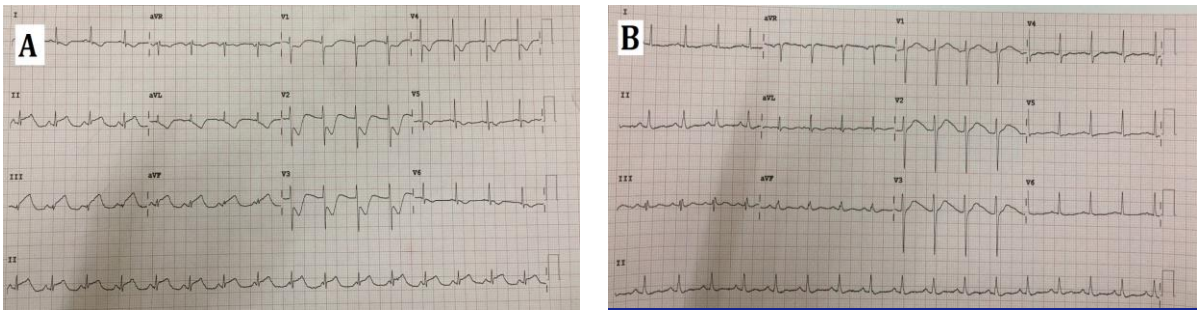

Figure 1: On arrival ECG showed inferior ST-segment elevation (A); The ECG obtained after treatment (B)

CASE PRESENTATION

Case 1

A 29-year-old man came to our emergency department (ED) with altered sensorium. According to his ten-year-old son, his father was eating food, and suddenly he felt severe chest pain and was then about to faint. Later, when we probed into the patient's history, we realized that an insect bit him on the left shoulder, following which he had developed chest pain. On arrival, patient's vital signs were as follows: heart rate (HR) 112 beats per minute, blood pressure (BP) 65/40 mmHg, respiratory rate (RR) 20 per minute, and pulse oximeter oxygen saturation (SpO2) 92% in room air. The patient was drowsy, responding to painful stimuli. Cardiac monitoring showed ST elevation in lead 2. Head to toe examination revealed flushing, diaphoresis, a red hyperemic rash extending over the left upper chest, shoulder, and back. Diffuse wheeze was noticed on auscultation of the chest. An electrocardiogram (ECG) was obtained, which showed inferior ST-segment elevation (Figure 1-A).

High-flow oxygen was initiated through a non-rebreather mask. The patient was treated with intramuscular adrenaline (0.5 mg, three doses, five-minute intervals), intravenous corticosteroid (hydrocortisone 200 mg once), intravenous antihistamine (diphenhydramine 50 mg once), intravenous 0.9% saline solution (0.9% normal saline (NS) 500 ml as a bolus, total of 3 boluses) and salbutamol nebulization (5 mg three times continuously, a total dose of 15 mg). The patient clinically improved, BP and ECG (Figure 1-B) rapidly normalized after prompt treatment in the ED. A focused echo was performed by the ED physician and showed good left ventricular contractility without noticeable regional wall motion abnormalities. Initial Troponin T was 0.17 ng/ml (our clinical laboratory cutoff was less than 0.13 ng/ml). The second set of Troponin T was 0.24.

The patient was admitted to the cardiac critical care unit. As a part of the cardiac workup, a treadmill test (TMT) was performed, which showed a result within the acceptable range. An official transthoracic echo was performed the next day, which showed normal findings with left ventricular ejection fraction of 67%. He was discharged in stable condition after two days of in-hospital observation. The patient underwent coronary angiography (CAG) after one week, which revealed mild coronary sclerosis without any stenosis.

Case 2

A 42-year-old female came to the ED with abdominal pain, abdominal computed tomography with contrast agent revealed acute appendicitis. But 10 minutes after contrast, the patient developed severe itchiness, chest discomfort, and diffuse rash throughout the body. Patient's vital signs were as follows: BP 70/54 mmHg, HR 118 beats per minute, RR 18 per minute, SpO2 94%. The patient was drowsy but responding to verbal

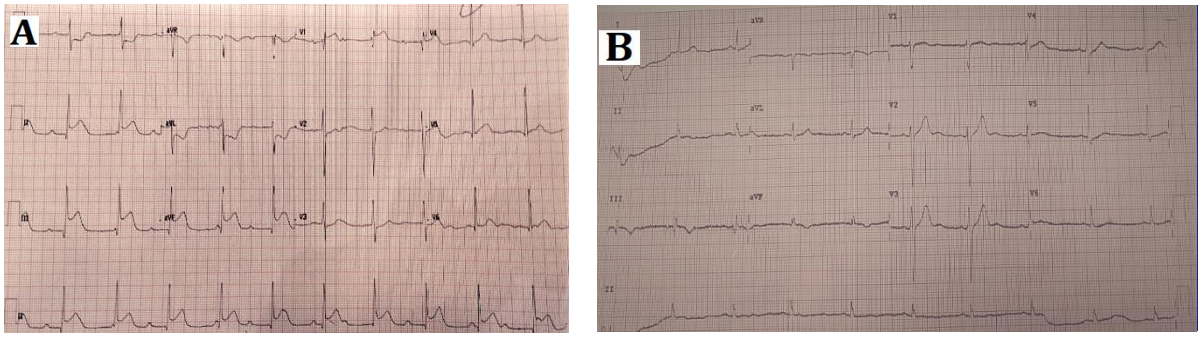

Figure 2: The ECG obtained after intravenous contrast administration showed inferior ST-segment elevation (A); The ECG obtained after treatment (B)

commands. Head to toe examination revealed total body flushing, diaphoresis, and a widespread urticarial rash. Diffuse wheeze was noticed on auscultation of the chest. An ECG was obtained, which showed inferior ST-segment elevation (Figure 2-A).

High-flow oxygenation was initiated. The patient was treated using intramuscular adrenalin administration (2 doses of 0.5 mg, five-minute interval), hydrocortisone 200 mg IV bolus, diphenhydramine 50 mg IV bolus, intravenous 0.9% saline solution (500 ml as a bolus, total of 4 boluses). Blood pressure and ECG (Figure 2-B) became normal and the patient clinically improved within one hour of anaphylaxis presentation after rapid assessment and treatment using IV medication. A focused echo was performed by the ED physician, which showed obvious good left ventricular contractility without noticeable regional wall motion abnormalities. Troponin T value raised to 0.37ng/ml, subsequent serial troponin was within the normal range. Cardiology consultation was sought. A transthoracic echocardiogram was obtained, which had absolutely normal findings. After cardiology clearance, she underwent appendectomy surgery. The post-surgery period was uneventful.

She was discharged after six days of in-hospital stay. As a part of the cardiac workup, a treadmill test was performed after 15 days, which showed a result within the acceptable range. Since the patient was allergic to contrast agent and was completely asymptomatic, she did not opt for coronary angiography.

LEARNING POINTS

Both cases described above were dealing with a typical presentation of allergic acute coronary syndrome (AACS) described as Kounis Syndrome (KS) in medical literature (1). Nicholas Kounis described the first case of AACS/KS in 1991 in medical literature and revealed that AACS incidence rate is around 9/100000 cases per year (2). AACS/KS is not a rare clinical presentation, but its diagnosis can be missed. It is a complex coronary disease, which needs urgent medical intervention, so every emergency physician should be aware of it.

AACS/KS may present in the form of angina (acute coronary spasm), acute myocardial ischemia, or in-stent thrombosis in those who already have a stent in coronary blood vessels. The pathogenesis of AACS is due to the quick release of inflammatory markers such as histamine, leukotriene, thromboxane, and mast cells, during acute allergic insult in coronary vessels (3).

The most common complaint in AACS/KS is chest pain followed by diaphoresis, headache, and altered sensorium. The most common sign is hypotension, followed by ECG changes. The most common ECG findings are ST elevation in inferior and lateral leads.

Troponin was elevated in both cases, but serial troponins were negative once they were treated for anaphylaxis. A study conducted by Lippi et al., suggested that Troponin would be elevated even in those with normal anaphylaxis reaction compared with healthy individuals and stated that coronary vessels could be the target vessels in anaphylaxis (4).

The diagnosis and management of AACS/KS are somewhat tricky. The treatment of anaphylaxis may worsen the disease process (for example, intramuscular Adrenalin may cause myocardial hypoperfusion, ultimately leading to ACS). Mortality is unavoidable if the diagnosis is delayed. Once anaphylaxis is treated in AACS/KS, a full cardiac workup should be done to rule out the other causes of acute myocardial infarction. An allergy workup is also required for assessing allergies such as environmental factors, multiple drugs, and food.

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