

Kounis Syndrome: Unveiling Allergic Triggers for Acute Coronary Events - A Case Study of Contrast Dye Induced Reaction during Percutaneous Coronary Intervention

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1. Introduction

Kounis syndrome (KS) is a condition characterized by an allergic reaction leading to an acute coronary syndrome. It was initially identified and reported by Kounis and Zavras in 1991 and is alternatively referred to as "allergic angina syndrome" or "allergic myocardial infarction" [1].

The initial case documenting an acute myocardial infarction along with urticaria, reported in 1950, involved a 49 - year - old man who was undergoing treatment with 300, 000 units per day of penicillin in oil [2]. KS can be provoked by a variety of factors, and ongoing research continues to reveal additional triggers. Among the most prevalent triggers of KS were antibiotics, accounting for 27.4% of cases, closely followed by insect bites at 23.4% [3], contrast media has been identified as the causative trigger in nearly 6% of the documented KS cases [4].

We are reporting a case of KS resulting from exposure to contrast dye during a percutaneous coronary intervention (PCI).

2. Case Description

36 - year - old Moroccan woman, with a medical history of uncontrolled type 1 diabetes, experienced classe III angina according to the Canadian Cardiovascular Society (CCS) scoring, for a month. She presented, in our facility, a week after a severe chest discomfort. It is to be noted that there are no known allergies or family background of medical conditions. At first medical contact, the patient was normotensive (100/60 mmHg), with normal heart rate (70 beats per minute), normal respiratory rate (15 cycles per minute) and pulse oximetry (97% on ambient air). The

electrocardiogram (ECG) showed inverted T waves on anterior leads (Figure 1). Laboratory investigation revealed elevated cardiac troponin (200 ng/l, normal range: 13 - 17 ng/l). Transthoracic echocardiography (TTE) showed an enlarged and dysfunctional left ventricle with akinesia of the anterior wall, the septal wall, and the apex (Figure 2).

A diagnostic coronary angiography has been indicated to assess the coronary vasculature.

The procedure, which was carried out 24 hours after admission, begun with the injection of iodinated contrast, Iopromide, into the left coronary arteries in a caudal view revealing a potential occlusion of the LAD, which was planned to be reassessed on an apical view for refined categorization. Additionally, there was a concurrent vasospasm in the circumflex artery (Figure 3).

However, shortly after, the patient began experiencing respiratory distress and upper limbs numbness. Her vital signs were: heart rate; 130 beats per minute, blood pressure; 70/ 40 mmHg, oxygen saturation; 85%. We also noted a generalized cutaneous rash (Figure 4).

We immediately adjudged these events to be caused by anaphylactic shock to the iodinated contrast, we interrupted the procedure to avoid extra use of Iopromide and, accordingly, administered an intramuscular injection of 0.3 milligrams (mg) adrenaline and 100 mg of Hydrocortisone hemi - succinate. The patient also received antihistamines and oxygen therapy. Her vital signs, namely blood pressure and oxygenation status gradually improved. The patient was then transferred to the intensive care unit and was closely monitored electrically, and echocardiographically, and by laboratory tests.

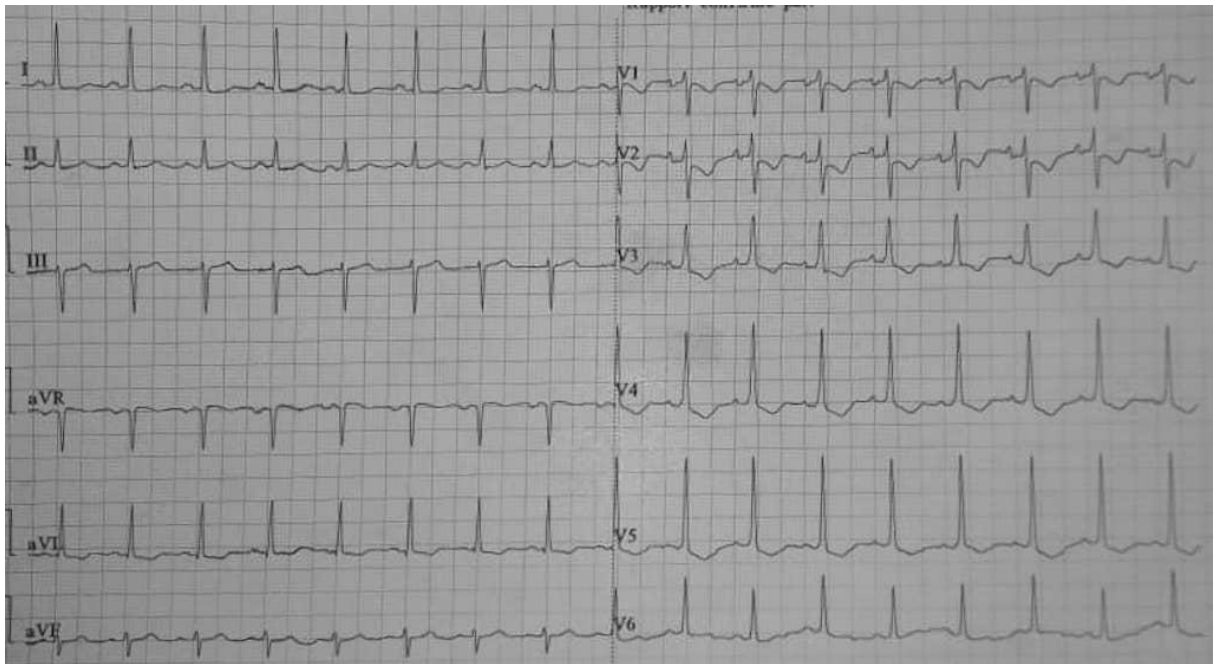


Figure 1: ECG (12 leads) showing negative T waves on the anterior leads

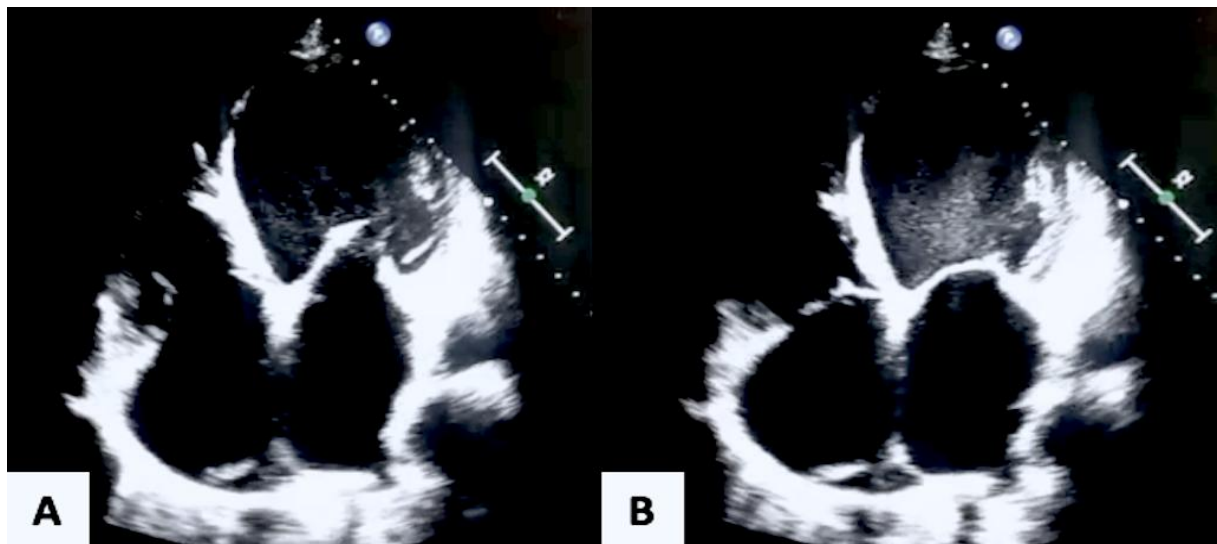


Figure 2: Apical Four - chamber view in end - diastole (A) and end - systole (B) showing a dilated and dysfunctional left ventricle

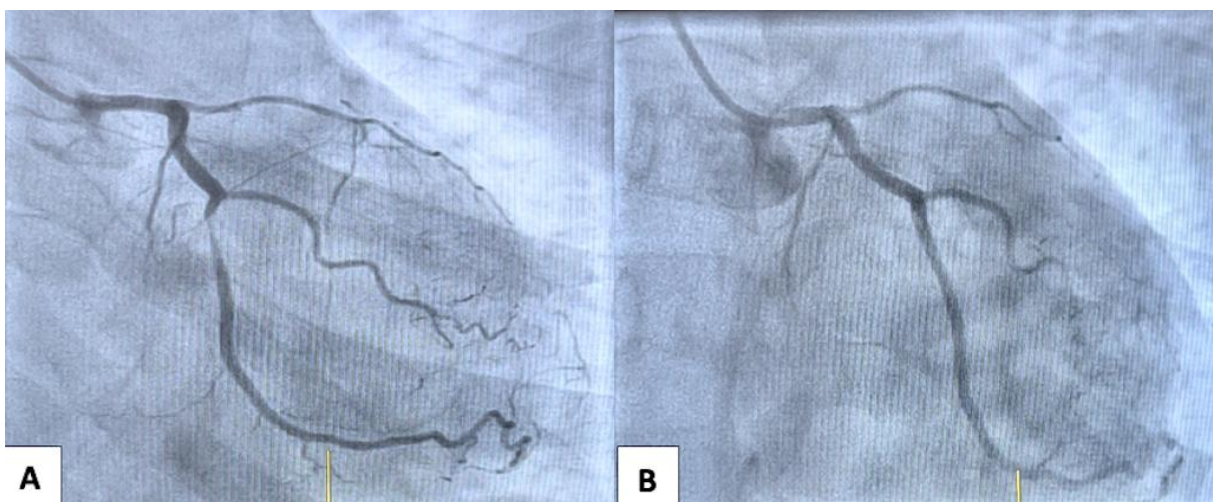


Figure 3: A - Caudal view of coronary angiography showing severe circumflex artery vasospasm, B - Caudal angulation showing the spontaneous resolution of the vasospasm



Figure 4: Emergence of skin rash shortly after iodine contrast injection

3. Discussion

KS is characterized as the simultaneous occurrence of acute coronary syndromes linked to the activation of mast cells and platelets in the presence of allergic or anaphylactic reactions, its pathophysiology entails coronary artery spasm and/or the erosion or rupture of atherosclerotic plaques in response to an allergic reaction [3].

KS can be categorized into three types: type 1, characterized by acute coronary syndrome (ACS) resulting from coronary spasms without any evidence of coronary artery disease; type 2, involving ACS from coronary spasms or plaque rupture in the setting of coronary artery disease; and type 3, characterized by coronary thrombosis containing mast cells and eosinophils [5].

The identification of KS is based on clinical manifestations, along with laboratory testings, elevated levels of several inflammatory biomarkers, such as tryptase, eosinophils, and IgE, have been identified [4]. Electrocardiographic, echocardiographic, and coronary angiographic presentations are also crucial for both diagnosing and managing KS [6].

In most cases, the electrocardiogram (ECG) typically displays ST - T changes indicative of ischemia, and ST elevation myocardial infarction (MI) is the most frequent manifestation. The echocardiogram can reveal regional wall motion abnormalities in the area supplied by the affected artery. Furthermore, Coronary angiography can provide a conclusive diagnosis, by revealing evidence of coronary vasospasm or stenosis [3].

KS triggered by contrast agents primarily consisted of type I and II cases, frequently observed in elderly male patients. The majority of contemporary iodinated contrast media fall into the categories of low - osmolality contrast media (LOCM) and isoosmolar contrast media (IOCM). The majority of KS cases were associated with LOCM, namely iopromide [6].

As in our case, the allergic reaction, manifested clinically and angiographically, was secondary to Iopromide injection.

Managing KS is complex due to the need for addressing both allergic reactions and cardiac symptoms simultaneously. There is a lack of specific guidelines for managing KS, and the evidence regarding treatment efficacy primarily relies on individual case reports or case series concerning the management of ACS [4].

For patients with the type I variant, similarly in our case, the aim of the management is to address the allergic reaction that can subsequently, lead to a resolution of the cardiac manifestations. Corticosteroids and supportive therapy with H1 and H2 antihistamines, can be used to reduce inflammation and alleviate allergic symptoms respectively. Fluid resuscitation would be appropriate if patients experience distributive shock. However, the use of epinephrine requires careful consideration due to its potential to exacerbate myocardial ischemia, prolong the QTc interval, and trigger coronary vasospasm and arrhythmias. Finally, the use of vasodilators like calcium channel blockers and nitrates can effectively alleviate coronary vasospasm caused by hypersensitivity [3 - 7].

Although, Contrast - induced KS can lead to severe complications, with a cardiac arrest occurrence of almost 23% and a mortality rate standing at 7.7% [6]; in general, type 1 KS has a favorable prognosis, and the majority of patients can anticipate a full recovery with suitable treatment [3].

4. Conclusion

KS appears to be infrequent, but its frequency could be underestimated because of inadequate awareness and insufficient recognition. Contrast - induced KS is an uncommon manifestation that can be overlooked by interventional cardiologists.

Therefore, during coronary angiography, it is of utmost importance to be highly suspicious of KS resulting from the use of contrast dye for optimized management.

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