

Kounis syndrome presenting with cardiogenic shock

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To the Editor

Multiple factors may cause coronary artery spasm.^{1–4}

Kounis syndrome has been found to be associated with coronary artery spasm and acute coronary syndrome developing due to mast cell degranulation. It is relatively newly described and is being reported more and more frequently. The drugs, endovascular devices, environmental agents and several allergic diseases likely to cause Kounis syndrome have been described.⁵ All patients presented with acute coronary syndrome and anaphylactic shock in the absence of hemodynamic failure. No patient was reported with cardiogenic shock.

Methods

A 21-year-old female patient without any history of cardiac complaints was hospitalized for a nasal septum deviation operation. There were no abnormal findings in the preoperative physical examination, laboratory examination or on chest radiography (Figs 1 and 2). Prior to induction of general anesthesia an arterial line was inserted, pulse oximeter and central catheter were placed. The septum was anesthetized with 1% lidocaine with 1/100 000 epinephrine. General anesthesia was induced with intravenous propofol and atracurium. Approximately 5 min later the patient became hypotensive and marked ST elevation was seen on the monitor. The operation was cancelled and the patient was transported to the coronary care unit. Marked ST elevation in D1 and aVL leads and reciprocal changes in inferior leads were noted on an electrocardiogram, compatible with acute high lateral wall infarction (Fig. 3). Transthoracic echocardiogram showed severe hypokinesia of the left ventricle, moderate mitral regurgitation with normal mitral valves and the estimated ejection fraction was noted as 30%. Although the patient was being prepared for coronary angiography, blood pressure worsened and

ventricular fibrillation (VF) occurred. Defibrillation with a biphasic defibrillator (200 J) was performed six times. Magnesium sulfate 2 g and amiodarone 300 mg were administered intravenously for refractory VF. Approximately 5 min after cardiopulmonary resuscitation, vasopressor and inotropic support and placement of an intra-aortic balloon pump (IABP), the patient's hemodynamic status was stabilized and she was taken to the cardiac catheterization room for emergency catheterization.

Results

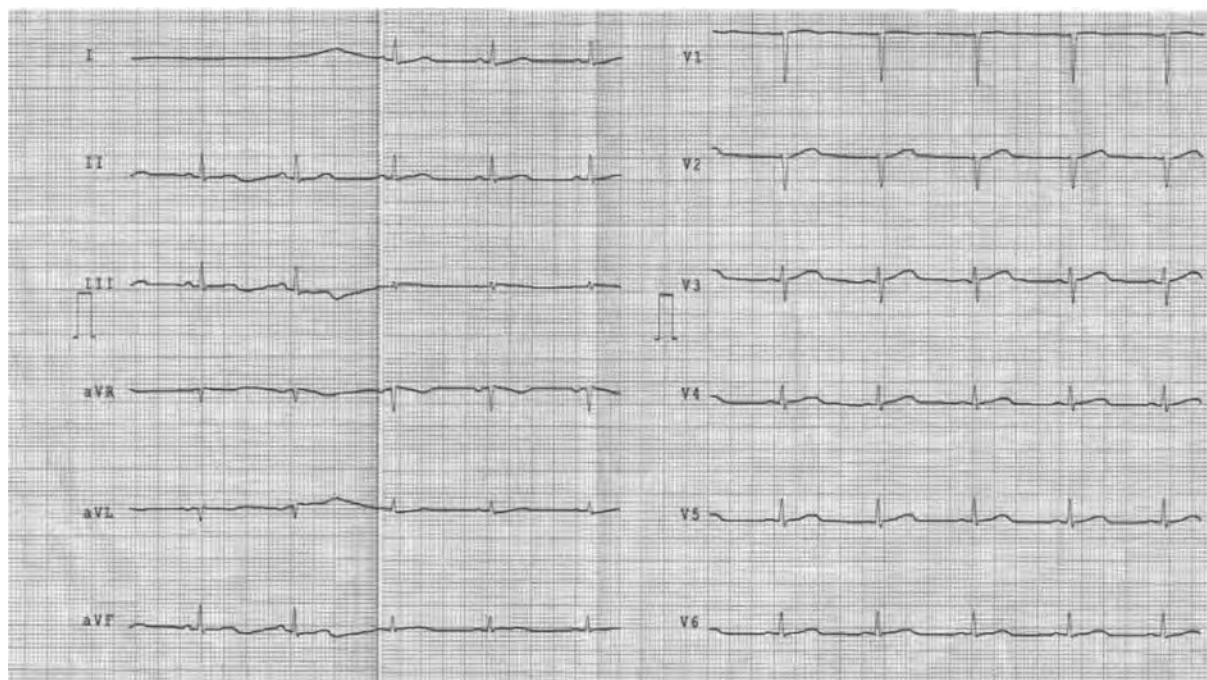
The coronary arteries were observed to be normal in coronary angiography (Fig. 4a and b) and left ventricular end-diastolic pressure (LVEDP) of 28 mmHg was found. Intravenous vasopressor treatment, due to the patient's being hypotensive, and inotropic treatment for failure in her systolic functions were initiated and the patient was again taken to the coronary ICU. In addition, intravenous steroid and intravenous antihistamine treatment were administered because of the likelihood of development of allergic myocarditis. Her chest radiography was consistent with pulmonary edema, her cardiothoracic index (CTI) was less than 0.5 because of acute myocardial dysfunction (Fig. 5). The laboratory findings of the patient taken on admission (approximately 4 h after induction of anesthesia) were as follows: WBC $26 \times 10^9/l$, eosinophil count 0, troponin 6.2 ng/ml, creatine kinase (CK) 865 U/l, CK-MB: 42 U/l and immunoglobulin E 76 IU/ml (normal <150 IU/ml).

The patient was extubated on the second day and the IABP was removed on the third day. One week later all cardiac biomarkers and electrocardiography, echocardiography and chest radiography findings were found to be normal. Subsequently, the patient was discharged on the tenth day. The patient received steroid and antihistamine treatment during her stay in hospital. The patient had been administered an angiotensin-converting enzyme inhibitor and beta blockers because of having left ventricular systolic dysfunction during her hospital stay, and after discharge these drugs were stopped. On prick skin testing for anesthetic drugs, a strongly positive reaction to lidocaine was noted.

Comment

In 1991, Kounis and Zavras⁶ described the 'syndrome of allergic angina' as the coincidental occurrence of chest

Fig. 1



ECG showed normal electrocardiogram.

pain and allergic reactions caused by inflammatory mediators released during the allergic insult. Many conditions that may result in Kounis syndrome were defined. Hymenoptera stings and drug reactions are responsible for a great majority of these conditions.⁵ The list of the drugs that can cause Kounis syndrome was issued by

Fig. 2

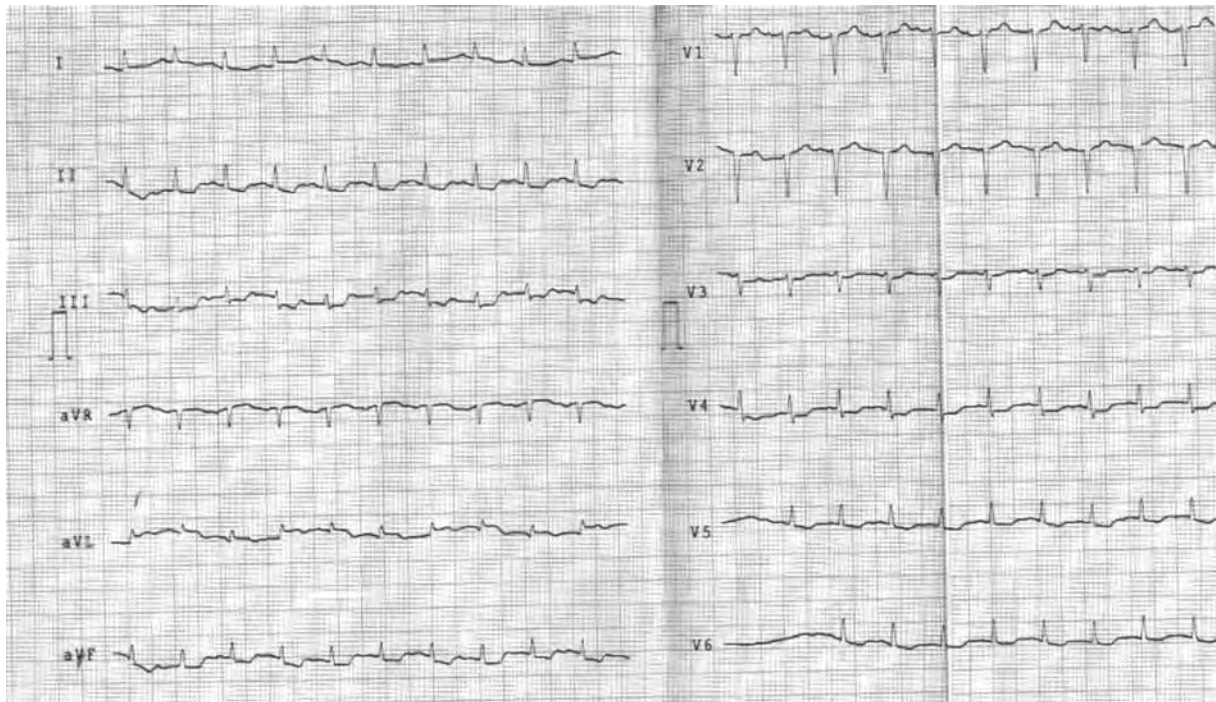


Preoperative evaluation; normal chest radiography.

Mazarakis *et al.*⁷ As was defined by Dr Kounis, there are two variants of the syndrome. Coronary artery spasm (biomarkers may be normal or increased) is present in type I variant, triggered by an allergic insult in individuals having normal coronary arteries in the absence of any predisposing factors of coronary artery disease. Due to allergic insult, erosion or rupture of atheromatous plaque that has been present and has remained silent appears in type II variant.⁵ The allergic reaction was triggered by lidocaine in our case. As the coronary angiography of the patient was observed to be normal and there were no accompanying atherosclerotic risk factors it was thought to be type I variant.⁸

In recent comments, Kounis syndrome was described as associated with stent thrombosis, hypersensitivity myocarditis and nickel allergy secondary to an atrial septal occluder device. A hypersensitivity suppressive agent might be used to prevent stent thrombosis and nickel allergy in the future. Furthermore, less allergenic struts can be used in these devices.^{9–11}

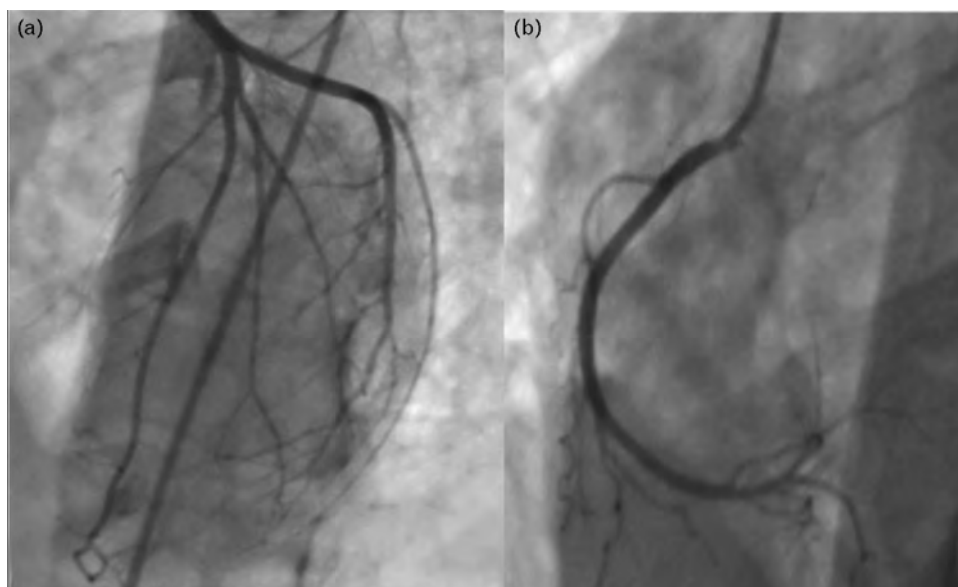
Anaphylactoid reactions owing to lidocaine have been reported^{12–14} but Kounis syndrome has yet not been reported in the literature. The presentation of our case with cardiogenic shock was interesting. There were several findings proving the diagnosis of cardiogenic shock, including severe systolic dysfunction (ejection fraction 30%) on admission echocardiographic examination, hypotension, clinical and radiographical pulmonary edema, increased LVEDP (reflected pulmonary capillary wedge

Fig. 3

ST elevation in D1 and aVL leads and reciprocal changes in inferior leads.

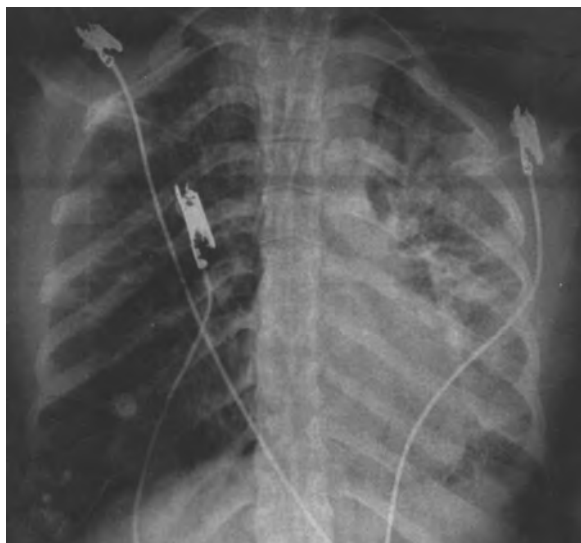
pressure as there was no mitral stenosis). The cardiogenic shock was treated by IABP, intravenous inotropic vaso-pressor and by controlled diuretics. Steroid and antihistamine therapy administered for the likely allergic cause provided complete clinical and hemodynamic recovery.

Cardiogenic shock has not yet been reported in the literature; however, anaphylactic reaction due to cardiovascular collapse has been very frequently reported.^{15–18} In conclusion, our case may provide new insight into Kounis syndrome. In the reports published so far, it has

Fig. 4

(a): Coronary angiography showing normal left coronary artery and circumflex artery. (b) Coronary angiography showing normal right coronary artery.

Fig. 5



Chest radiography was consistent with pulmonary edema; CTI was <0.5.

been shown that the syndrome presents with acute coronary syndrome and/or anaphylactic shock without hemodynamic failure. It should be kept in mind that in cardiogenic shock of unknown origin Kounis syndrome might be present.

Acknowledgement

There are no conflicts of interest.

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