Kounis syndrome induced by administration of iron sucrose

Case Report

Zhou Hao¹, Chen linzhi², Huang weijian¹, Gao zhan¹

1. the cardiology department of the first affiliated hospital of Wenzhou medical university
2. the laboratory department of Wenzhou central hospital

Corresponding Author:
Gao Zhan.
the cardiology department of the first affiliated hospital of Wenzhou medical university, Wenzhou, Zhejiang 325000, P.R. China.
E-mail address: lyonx@126.com.
Fax number: (+86)0577-55578999

Abstract: Anaphylactic reaction accompany with acute myocardial infarction was referred as “Kounis syndrome”. Although underlying mechanism was still unclear, coronary artery spasm and peripheral vasodilation induced by inflammatory mediators were most incriminated. Here we report a Kounis syndrome case, in which coronary artery spasm and peripheral vasodilation were confirmed by coronary angiography and Ganz-swan catheter respectively, which was not definitely proven by previous case reports. And also Iron products induced Kounis syndrome has not been reported yet.

Keywords: Kounis syndrome, coronary artery spasm, iron sucrose

1. Introduction
Anaphylactic shock is a life-threatening emergency characterized by systemic vasodilation and fluid redistribution. Acute myocardial infarction has been reported to accompany and complicate such allergic reactions, which is known as Kounis syndrome. Here, we report a case of 57-year-old man who presented with anaphylactic shock and subsequent acute ST-segment elevation.
myocardial infarction (STEMI) induced by the intravenous administration of iron sucrose, which diagnosed base on coronary artery spasm and peripheric vasodilatation.

2. Case report

A 57-year-old man, with no previous history of hypertension, was prescribed iron sucrose intravenous infusion for iron-deficiency anemia in an outpatient setting. During the first iron sucrose injection, the patient complained of dyspnea, malaise, and sweating. The symptoms reoccurred on the second day when the patient received the second dose of iron sucrose; the dyspnea became more severe, and the patient's condition quickly deteriorated with continuous coughing and pink frothy sputum. The blood pressure rapidly decreased to 60/40 mmHg, and the heart rate increased to 140 bpm. The patient was transferred to our hospital immediately.

On admission, following the administration of dopamine and norepinephrine, the patient's clinical condition was as follows: pulse rate, approximately 96 bpm; BP, 60/40 mmHg; and SaO2, 70–75%. Blood gas analysis suggested severe hypoxemia, and cardiac troponin I levels were higher than 50 µg/L (normal, 0–0.15 µg/L) with the electrocardiogram (ECG) showing ST-segment elevation in leads I and aVL, which indicated acute lateral wall STEMI (Fig. 1). The echocardiogram demonstrated slightly impaired left lateral ventricular contraction and no mechanical complications, and computed tomography (CT) revealed acute pulmonary edema and effusion (Fig. 2).

Figure 1. Electrocardiogram showing ST-segment elevation in leads I, aVL, and V6 on admission and regression on day 2 of hospital admission.
The patient was diagnosed with acute STEMI and cardiogenic shock, and coronary angiography was performed immediately. This revealed a 70–80% diffuse lesion in the mid-distal end of the left circumflex artery (LCx) with TIMI grade 3 flow (Fig. 3), which corresponded to the lateral wall infarction indicated by ECG and echocardiogram; no lesions were found in the left main coronary artery (LMCA), right coronary artery, or left anterior descending (LAD) artery. However, since merely lateral wall infarction rarely results in hemodynamic collapse, we also performed ventriculography, which showed essentially normal findings apart from slightly impaired interior wall contraction.

Swan-Ganz catheterization was performed just after the angiography, revealed normal systemic vascular resistance index (2345 dyne•s-1•cm-5•m-2) with relatively normal heart function (cardiac output, 4.5 L/min; cardiac index, 2.4 L•min-1•m-2) under the administration of dopamine at 5 µg•kg-1•min-1 and norepinephrine at 0.7 µg•kg-1•min-1.
Figure 3: Coronary angiography showing a transient diffuse lesion in the mid-distal end of the left circumflex artery.

Since this patient suffered severe hypoxemia and hypotension and had no severe stenotic lesion in the LMCA or LAD, and also no clues for mechanical complications or pericardial tamponade. We considered it was unlikely that the occurrence of STEMI had triggered shock. Given the specific symptoms after the intravenous injection of iron sucrose, i.e., hypovolemia and vasodilation (decreased systemic vascular resistance in the absence of norepinephrine), fluid redistribution (bilateral pulmonary exudation), relatively reserved cardiac output (confirmed by echocardiogram, ventriculography, and Swan-Ganz catheterization), it was reasonable to consider that the patient suffered STEMI secondary to anaphylactic shock.

After anaphylactic shock was suspected, the circulating IgE levels on day 8 of hospital admission were assessed and were observed to be 56.42 IU/mL (normal, 1.27–241.3 IU/mL). After rehydration therapy and treatment with a vasoactive agent, the patient’s condition steadily improved and stabilized, and the ECG revealed ST-segment regression on day 2 of hospital admission (Fig. 1). Chest CT showed decreased pulmonary exudation on day 14 of hospital admission (Fig. 2). In repeat coronary angiography on day 24 of hospital admission, no lesions were found; in fact, even the diffuse lesion noted in the previous angiography in the LCx had disappeared (Fig. 3). The patient was
discharged at day 25 of hospital admission.

3. Discussion

Anaphylaxis accompanied by STEMI is known as Kounis syndrome, which was first defined by Kounis and Zavras in 1991 [1]. Several theories have been proposed to explain this association, including the involvement of mast cell degranulation, the release of chemical mediators, plaque rupture, and coronary artery spasm [2]. In our case, coronary artery spasm was demonstrated by the transient diffuse lesion in LCx, which was rarely observed in previous Kounis syndrome case reports. Swan-Ganz catheterization was also not frequently performed in previous Kounis case, in our case, swan-Ganz catheterization was critical by revealing decreased systemic vascular resistance. Which implicate anaphylactic shock. Since anaphylactic shock was not among our first differential diagnoses; therefore, we did not immediately test for IgE and iron-specific IgE. While these tests were performed on day 8 of hospital admission, the negative results neither supported nor excluded anaphylaxis since the half-life of IgE is only for 48 h [3]. Moreover, allergies with negative IgE response are not rare [4].

Iron products are a very common allergen, and multiple reports have described anaphylaxis induced by the administration of iron products[5], some of them were strongly suspect to be Kounis syndrome[6]. However, to the best of our knowledge, this is the first report to describe the occurrence of Kounis syndrome induced by the intravenous administration of iron sucrose. We expect our findings to be clinically useful in treating such patients.

4. References


