Kounis Syndrome Secondary to Use in a Middle-Aged Female

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DEAR EDITOR,

We present a thirty-six year old woman, referred herself into our hospital for a drug induced Kounis Syndrome, who suffered from twice severe cardiac shocks after taking 1.5g Cefuroximeaxetil by intravenous infusion. This patient showed allergic symptoms, changes those are relatively insensitive but potentially significant in echocardiographic images and electrocardiogram with high cardiac enzymes. Kounis syndrome caused by Cefuroximeaxetil intravenous infusion and intramuscular administration has been described in [1, 2]

INTRODUCTION

Kounis syndrome is characterized by cardiovascular symptoms and acute coronary events, accompanied with allergic reactions. Clinical symptoms include chest discomfort, dyspnea, faintness, nausea, pruritus and urticarial with signs such as hypotension, diaphoresis, pallor and bradycardia. Triggers such as drugs [3, 4], chemicals, foreign bodies and environmental exposures may stimulate inflammatory mediator releases. Drugs act as strong antigenic complex causing repetitive and persistent hypersensitivity stimulus to coronary arteries [5].

CASE PRESENTATION

This female patient was 36 years old, denied history of high blood pressure, drugs or food allergies, went into hospital for bilateral apocrine sweat gland excision in 2013-02-23. About 30 minutes before the operation, 250ml 0.9% NS and 1.5gcefuroximeaxetilwere given intravenously. During this operation, instant onset of symptoms occurred, as nausea, vomiting, dizziness, pallor, short of breath, heavily sweating and pink frothy sputum coughing. Considering this situation as she being allergic to cefuroxime axetil, intravenous infusion and operation were stopped immediately, 5 mg dexamethasone was injected 3 times, 40mg methylprednisolone was given intramuscularly along with dopamine intravenously.23:00 that night, her ECG showed ST elevation in leads V2,V3 and sinus tachycardia. She was transferred to respiratory intensive care unit. Physical examination showed mental depression, slightly lower sound of bilateral pulmonary breath with widespread rates, 125 beats per minute of heart rates with regular rhythm, no cardiac murmur, nor edema of lower limbs.

High doses of dopamine and norepinephrine were for maintaining blood pressure with continuous ECG monitoring and oxygen inhalation. Methylprednisolone was for resisting inflammation, metoprolam and granisetron was for preventing vomiting, loratadine and promethazine was for averting allergies, along with omeprazole sodium given as a protection of gastric mucosa. Current ECG indicated ST-stage ischemic changes in leads V5, V6 and sinus tachycardia with left atrial hypertrophy, (as shown in Figure 1b) meanwhile, echocardiogram showed systolic dysfunction in left ventricular, mild mitral regurgitation and pulmonary hypertension, with a EF of 0.26. (as shown in Figure 1c)

3:30 next day, a progressive decline of pulse oxygen, unstable blood pressure and rapid heart rate suddenly showed up. Noninvasive ventilator assisted ventilation in high oxygen flow was administered to her, succinylated gelatin was for enlarging blood volume, cedilanid was for cardiacinotropic action, lowering ventricular heart rate and diuresis. Dyspnea occurred and her breathing frequency was up to 40-47times per minute, meanwhile, her blood pressure maintained lower after given large doses of booster medication. Echocardiogram showed systolic and diastolic dysfunction in left ventricular and slight fluid in the pericardial cavity, with a higher right atrial pressure and a EF of 0.3 (as shown in Figure 1d).

An trachea was intubated and mechanic ventilation was for assisting her respiratory, along with a balloon counterpulsation performed to improve cardiac function, raise up blood pressure, increase coronary perfusion pressure and maintain stability of circulation system. Her renal function and urine volume switched back to normal the next day after treated with bedside hemodialysis for once 2013-02-25, her creatine kinase was 820.6UY/L, myocardial troponin was 5.26ng/ml, myocardial myoglobin was 97.7ng/ml, creatine kinase-MB was 89.52U/L, lactic dehydrogenase was 1046.4U/L and glutamic-oxalacetic transaminase was 169.1U/L During this course, cordarone was for reducing ventricular rate, midazolam was for sedation, morphine was for reducing pulmonary edema, hydrocortisone...
succinate was for anti-inflammation, mepem was for anti-infection, albumin and lehome was for nutritional support.

2013-03-25, her creatine kinase was 629.8UY/L, myocardial troponin was 0.17ng/ml, myocardial myoglobin was 303.4ng/ml, creatine kinase-MB was 17.17U/L, lactic dehydrogenase was 286.5U/L and glutamic-oxalacetic transaminease was 72.3U/L.

Meanwhile, her reexamine bedside heart ultrasonic showed slight pericardial effusion (as shown in Figure 1e). She was discharged with cardiology outpatient follow-up without obvious discomforts. She was in good health without any symptomatic disease occurred during the past 4 years. No significant abnormality was found in her recent heart ultrasonic (as shown in Figure 1f).

**DISCUSSION**

Pathogenic mechanism of Kounis Syndrome after an allergen invasion might work with heart tissue-resident mast cell mediators on the myocardium and the atherosclerotic plaque, or worsen cardiac preexisting disease by the hemodynamic changes [6] and trigger coronary artery spasm and plaque rupture which increase left ventricular pressure and decrease splanchnic, cerebral, and myocardial blood flow. Life-threatening cardiac arrests can be caused by the pathological accumulation of mast cells in organs [7]. Cefuroxime axetil, this drug, is essential for mast cell development, proliferation, survival, adhesion and homing.

Hemodynamic effect of Kounis Syndrome characterized by cardiogenic shock combining allergic shock with extensive peripheral vasodilation and myocardial suppression may put up severe arterial dilation and hypotension [8,9]. Treatment of Kounis syndrome is challenging because it needs management of both cardiac and allergic manifestations simultaneously. In this hemodynamically unstable patient, balloon pumping is a life-saving medical procedure. Medical use of dopamine, cordarone and norepinephrine successfully reversed coronary artery spasm, while dexamethasone and methylprednisolone was given to alleviate allergy symptoms by tempering her immune system response to the allergic mediators.

Treatment strategies of Kounis syndrome should mainly be symptomatic treatments with maintenance and support therapies. Determination of relative indexes and intensive care is of great significance for prognosis of those patients.

**CONCLUSION**

Physicians who run into clinical patients with heart problems accompany with chest pain or ST elevation should be aware of their allergies and medical histories [1, 8]. Kounis syndrome should be considered in the differential diagnosis of coronary syndrome and hypersensitivity myocarditis [10].

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**ETHICAL STANDARDS**

All human and animal studies have been approved by the appropriate ethics committee and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. All persons gave their informed consent prior to their inclusion in the study.

**REFERENCES**


