Short communication

A case of Kounis syndrome with anaphylactic shock secondary to penicillin G injection in a 32-year old woman

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A B S T R A C T

Our case report describes a 32-year old woman who had myocardial infarction with anaphylactic shock secondary to penicillin injection. She had hypotension and chest pain after intramuscular injection of 1200,000 U penicillin G injection for cricton tonsillitis. Her ECG showed ST elevation on D1 and aVL derivations and ST depression and T wave inversion on D2,D3,aVF, V3–6 derivations. Her ECG abnormalities regressed after the chest pain resolved. Her serum troponin level was elevated to 5.2 ng/ml. She had no pathology on echocardiographic examination. She was given anti-platelet and anti-thrombotic treatment, monitorized and followed in intensive coronary care unit. No cardiac complications were observed in her follow-up. Her coronary angiography was completely normal. The hyperventilation test to induce coronary spasm was negative during the coronary angiography. The myocardial injury seen in this case may be due to hypotension, allergic reaction itself, the potential vasospasm at the time of the anaphylactic shock or to intravenous adrenalin administered. Such cases of Kounis syndrome with anaphylactic shock are rarely observed. Emergency physicians should be aware of Kounis syndrome when there is a young patient without any risk factors having chest pain after an allergic insult.

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Introduction

Kounis syndrome is the coexistence of acute coronary syndromes with hypersensitivity reactions. There are many etiologies that have been reported including drugs, medical conditions, environmental exposure and stent implantation. There are 3 types of Kounis syndrome. Type 1 Kounis syndrome is the one seen in patients with normal coronary arteries. Type 2 is seen in patients with pre-existing atheromatous disease. Type 3 is stent thrombosis due to hypersensitivity reactions to the components of the stent. The exact mechanism of this syndrome is not clearly identified. There are some possible mechanisms. The most accepted mechanism is mast cell degranulation after the allergic insult. Chemical mediators released from mast cells are thought to induce coronary artery spasm or to promote platelet aggregation.1–4

Case report

We present a 32-year old woman who had no history of cardiovascular disease or any risk factor. She had migraine for 10 years. She was admitted to emergency department because of squeezing chest pain after intramuscular injection of 1200,000 U penicillin G for cricton tonsillitis. Her arterial blood pressure was 70/50 mm Hg. Her ECG showed prominent ST depression and T wave inversion on leads DII, DIII, AVF, V3–6, and 1 mm ST elevation on leads D1 and aVL (Fig. 1A). She was given 0.5 mg intravenous adrenalin. She chewed 300 mg of non-enteric acetylsalicylic acid. Her chest pain resolved in 5 min. And a second ECG afterwards showed only T wave inversion on leads D1 and aVL (Fig. 1B). Her blood biochemistry revealed an elevated serum troponin level of 5.2 ng/ml (reference range: 0–0.3). Echocardiography was normal. We admitted the patient to the intensive coronary care unit with a diagnosis of Kounis syndrome. She was started enoxaparine 6000 IU subcutaneously twice a day, clopidogrel 75 mg once a day orally. She had already taken acetylsalicylic acid. The next day she had coronary angiography. The coronary angiography showed completely normal coronary arteries (Fig. 2). A hyperventilation test was also performed during the procedure to provoke coronary spasm. But it was negative. The patient was discharged from the hospital with a prescription of acetylsalicylic acid 100 mg per day.

Discussion

Our case is a sample of Type 1 Kounis syndrome. There are not many cases of Kounis syndrome in the literature presenting after penicillin administration and accompanied by anaphylactic shock. One of these
cases was reported by Gikas et al., who presented a Kounis syndrome with anaphylactic shock in a young man induced by oral amoxycillin intake. Another case was a sample of Kounis syndrome with a previous history of significant coronary artery disease. The third case was presented by Viana-Tejedor et al. and was diagnosed to have type 2 Kounis syndrome. Tok et al. reported a case similar to ours. In this case a 52-year old man developed ST elevation on leads D1 and aVL after intramuscular penicillin injection due to criptic tonsillitis. But in this case the patient did not experience anaphylactic shock. A more recent case is reported by Bilgin et al. secondary to clarithromycin use in a 36 year-old woman. Kounis syndrome may also be seen in very young adolescent patients. Ilhan et al. presented a case in a 16 year-old child. Our patient differs from these cases. Because our patient did not have an allergy or cardiovascular disease history before. She experienced a very serious anaphylactic shock after penicillin injection which resulted in acute myocardial infarction.

There can be some possible mechanisms to explain the myocardial infarction in our case. The anaphylactic shock itself due to hypotension may be the cause. Because after saline infusion, when the blood pressure was normalized the chest pain regressed and ECG findings resolved. Although we could not demonstrate coronary spasm with hyperventilation method during the coronary angiography, at the time of anaphylactic shock acute coronary spasm might have contributed to the process. The allergic process itself may also be another explanation.

In conclusion, we should keep in mind the possibility of Kounis syndrome when there is a young patient without any risk factors having chest pain after an allergic insult.

Note: The patient and her husband gave informed consent for this case report. And this article fulfilled the principles of the ethical guidelines of the 1975 Declaration of Helsinki.

References