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Acute myocardial infarction following amoxicillin allergy: coronary angiography and intravascular ultrasound findings

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Amoxicillin is a widely prescribed antibiotic that might rarely cause severe allergic reactions and anaphylaxis in sensitive individuals. Acute cardiac emergencies are quite uncommon in the setting of drug allergies. Here we report the emergency management and IVUS study of a 40-year-old female patient presented with severe allergy resulting in myocardial infarction after oral amoxicillin intake.

Keywords: *thrombosis – hypercoagulable states – acute coronary care.*

Acute coronary syndromes following allergic reactions are rare but they may be life threatening. The underlying mechanism depends on various factors such as coronary vasospasm, increased coagulability, and epinephrine therapy¹⁻³. Decreased coronary perfusion resulting from hypotension associated with anaphylaxis can be another contributor for this scenario⁴.

Case report

A 40-year-old woman presented to our emergency room (ER) with retrosternal chest pain of 90 minutes duration following ingestion of her first dose of amoxicillin that had been started for acute tonsillitis. 20 minutes after the oral dose, she developed swollen extremities and mucous membranes and rash on her skin. Her symptoms were aggravated suddenly. She had started complaining of shortness of breath, tongue swelling and difficulty in swallowing without any fever before she had rushed into another ER facility. There, the patient's blood pressure was 110/60 mm Hg and she was initially treated with intravenous antihistamines and steroids. The symptoms had partially resolved and her blood pressure remained the same but she had started having retrosternal chest pain. Her ECG revealed acute inferior wall myocardial infarction and she was transferred to our ER for urgent catheterization. Her physical examination revealed

resolving skin rashes and red areas on her extremities. Her face and tongue were not swollen and she denied any difficulty in breathing. She had no coronary risk factors and denied oral contraceptive use. Urine toxicology was normal. Cardiac examination and vitals were in the normal range. There was minimal wheezing in the middle lung fields and oxygen saturation was 96%. Her ECG showed 1-2 mm ST elevation in leads II, III and avF, pathologic Q waves in III and avF and reciprocal 1 mm ST depression in avL (figure 1C). Troponin I level was 11.65 ng/ml. Coronary angiography revealed normal LAD and Cx arteries. There was a 40-50% stenosis and suspected accompanying thrombus in the right coronary artery (RCA) (figure 1A). There was TIMI III flow and grade 3 myocardial blush in the distal RCA region, therefore intravenous tirofiban (10 mcg/kg bolus followed by infusion of 0.15 mcg/kg/min 48 hours) therapy was started and the patient was transferred to the coronary care unit. Medical therapy included clopidogrel, metoprolol, ramipril and atorvastatin. The symptoms resolved and there was no complication in the follow-up. Biochemistry values were normal except elevated CK and CK-MB. Transthoracic echocardiography revealed segmental wall motion abnormality with inferior hypokinesis and global ejection fraction was 50%. On the third day, right coronary angiography did not show the pre-existing intracoronary thrombus (figure 1B). IVUS study revealed a non-critically stenosing smooth atherosclerotic plaque from proximal to distal RCA (figure 2). The infarct-related segment had an irregular fibrous cap and erosions over the plaque surface which was interpreted as plaque rupture with local thrombus formation resulting in myocardial infarction. The patient

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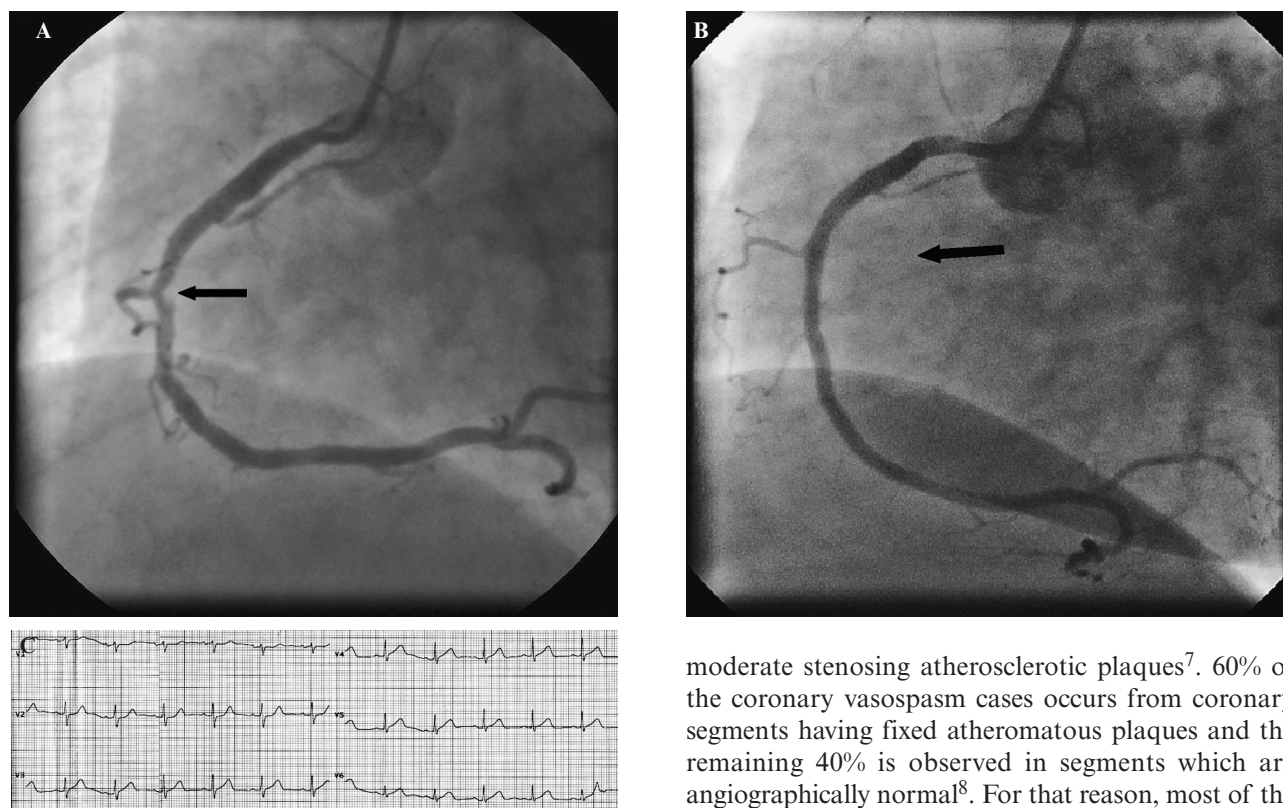


Fig. 1. – RCA angiography at pre- and post-tirofiban therapy (A and B). Infarct-related plaque was angiographically free from thrombus after 48 hours tirofiban therapy (arrows). 12-lead ECG is concordant with inferior myocardial infarction in ER (C).

was discharged on the fifth day without any complication, and she was referred to an allergy-immunology clinic.

Discussion

Data concerning myocardial infarction during allergic reactions are very limited but are present in the literature since 1982⁵. Possible aetiologic factors of myocardial ischaemia are vasospasm caused by anaphylactoid factors (histamine, prostaglandins, leukotrienes, cytokines, etc.), epinephrine therapy for anaphylaxis, increased coagulability from mast cells, and decreased myocardial perfusion following hypotension resulting from anaphylactic shock¹⁻⁴.

It has been demonstrated that coronary arteries possess stores of histamine which stimulates H1 and H2 receptors during the allergic process⁶. This is followed by coronary arterial vasospasm leading to what we call “allergic angina” and manifested as clinical, ECG and laboratory findings of classical angina pectoris and allergic reaction.

It is well known that many vulnerable atherosclerotic plaques are angiographically normal appearing because of compensatory vascular remodelling. Also, 75% of infarct-related thrombi originates from mild to

moderate stenosing atherosclerotic plaques⁷. 60% of the coronary vasospasm cases occurs from coronary segments having fixed atheromatous plaques and the remaining 40% is observed in segments which are angiographically normal⁸. For that reason, most of the atherosclerotic lesions resulting in myocardial infarction are thought to be haemodynamically silent. On the other hand, atheromatous plaques are rich in mast cells especially in their shoulder region which may render plaques unstable and contribute to atherothrombosis⁹. IVUS study has a major diagnostic role to study silent plaques and it can be speculated in our case that anaphylactoid factors and mast cells following an allergic reaction triggered coronary vasospasm, increased platelet aggregation with thrombus formation leading to atheromatous plaque rupture and resulting in myocardial ischaemia.

In our case, we have correlated, on temporal basis, the acute myocardial infarction with allergic phenomena due to amoxicillin administration. We interpreted myocardial ischaemia following an allergic reaction as an acute event that had triggered silent coronary artery disease with the vasoactive mediators. Although epinephrine therapy or prolonged hypotension could be a possible reason for myocardial ischaemia, our patient received only antihistaminic and steroid therapy and her blood pressure was normal during the entire course of the event.

There are few cases of antibiotic-related anaphylaxis complicated with ischaemic ECG changes and myocardial infarction in the literature^{10,11}. The novelty of this case report consists of the angiographic and IVUS findings which show thrombosis in coronary arteries responsible for the ischaemic episode. The thrombus-containing segment was visualized as free of thrombus after tirofiban therapy. IVUS examination revealed non-critical atherosclerotic plaque formations

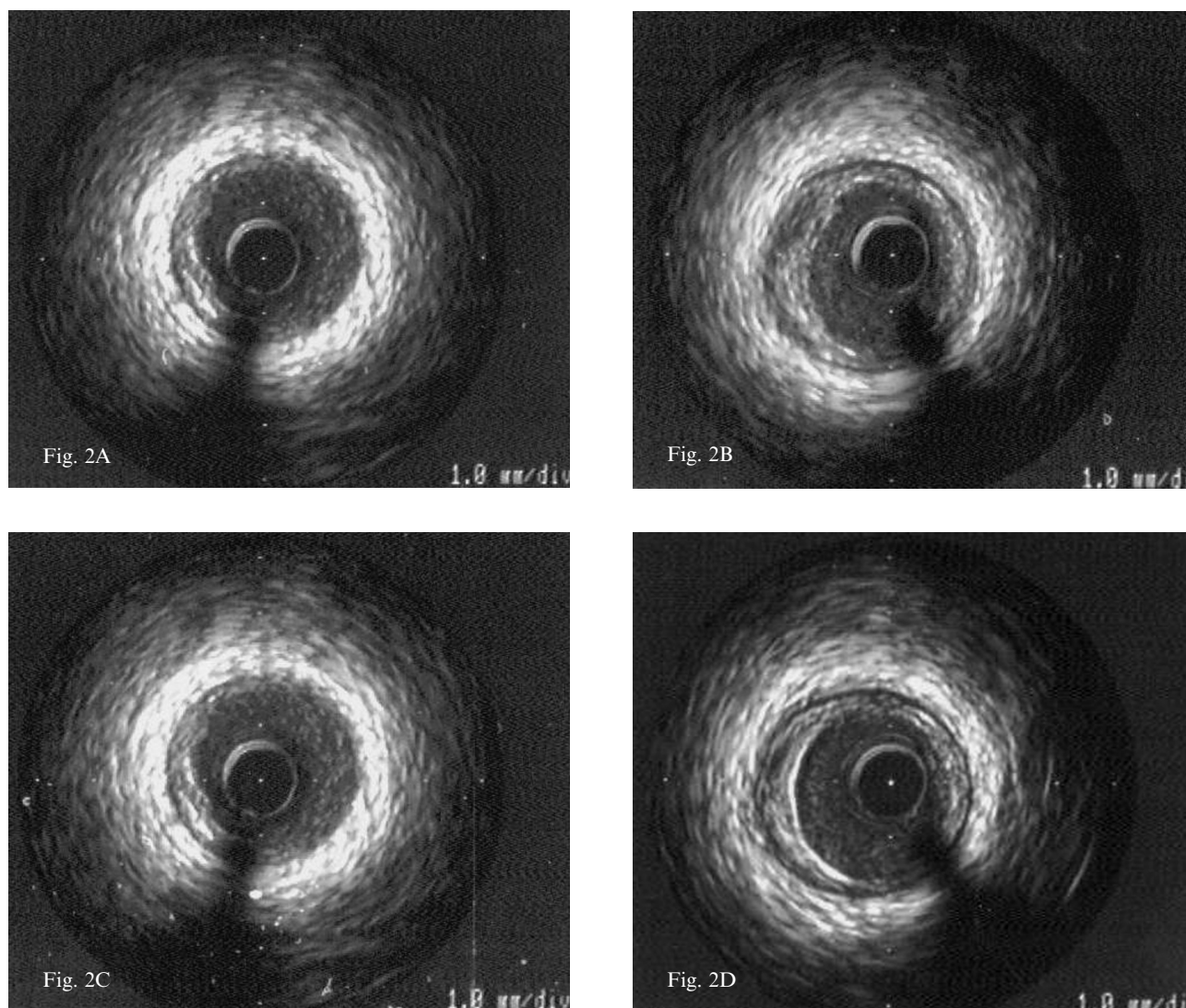


Fig. 2. – IVUS examination of the infarct-related segment revealed significant irregularity over the plaque surface where the thrombus had settled (A, B and C). Distal to the infarct-related segment a smooth, regular fibrous cap (D) is present.

from proximal to distal RCA and plaque rupture in the infarct-related segment. For patients presenting with any allergic reaction or anaphylaxis complicated with myocardial infarction, early coronary angiography with IVUS is important for choosing the optimal management strategy.

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