Case Report

Acute Myocardial Infarction in a Patient with Normal Coronary Arteries After an Allergic Reaction

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The case is described of a man aged 51 years who, one hour after a coronary angiographic examination that showed coronary arteries without lesions, suffered an acute inferior myocardial infarction as the result of an acute allergic reaction, probably to the iodinated contrast agent that was used. Acute myocardial infarction following an allergy is an extremely rare clinical entity that, according to existing data, is due to the local release of vasoconstrictive substances as a result of the allergic reaction.

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A man aged 51 years, a smoker with hyperlipidaemia, was admitted to hospital for a coronary angiographic examination that had been programmed because of episodes of retrosternal pain at rest and signs of myocardial ischaemia on exercise testing. The patient's history was uneventful, apart from an allergy to aspirin and agricultural chemicals (nettle rash). On clinical examination he had normal appearance and nutrition, his heart sounds were distinct with no additional sounds, and there were no pathological findings related to other systems. His pulse was regular at 70 beats/min and his blood pressure was 130/70 mmHg. Blood and standard biochemical tests showed no abnormal findings. The 12-lead ECG was normal and the chest X-ray and cardiac ultrasound examination were free of abnormal findings. A few hours after his admission, the patient underwent coronary angiography, which showed coronary arteries free of lesions, and was then returned to his ward.

About one hour later, the patient exhibited a generalised maculopapular rash on his trunk and limbs, which was accompanied by intense itching. Corticosteroids and antihistamines were given and the patient's blood pressure remained at 130/70 mmHg. A half hour later, when the allergic rash had still not completely receded, the patient experienced acute, constrictive precordial pain with reflection to the back and accompanied by nausea and sweating. The ECG showed ST-segment elevation 2 mm in leads II, III, avF (Figure 1).

Intravenous nitrates and heparin were given and subsequently, since the ST-segment elevation and pain persisted, the patient was sent for a new angiogram, which showed complete occlusion of the right coronary artery at its origin (Figure 3). The patient was immediately given intracoronary nitrates and the filling of the right coronary artery was completely restored (Figure 4), while the pain receded. The ECG immediately afterwards also showed complete recovery with disappearance of the ST-segment elevation (Figure 2). The patient stayed in hospital for a total of seven days with no further complications, under treatment with corticosteroids and antihistamines. During his hospitalisation he showed fluctuations in

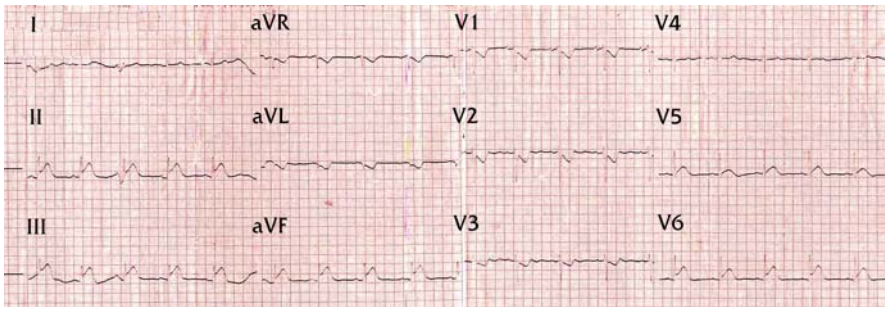

Figure 1. Patient's ECG showing ST-segment elevation in the inferior wall leads.

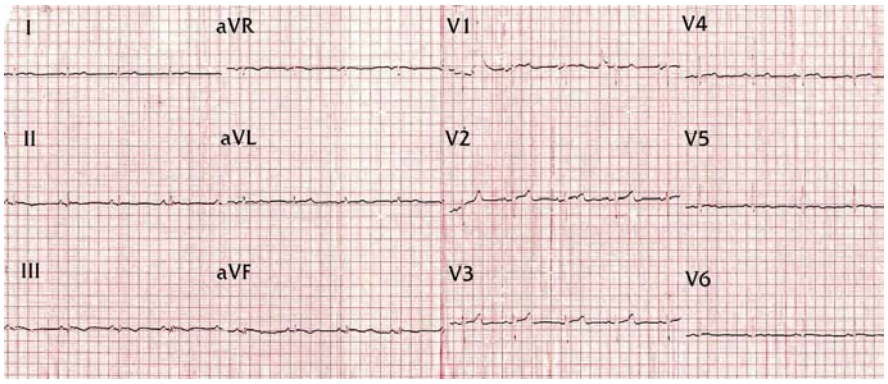

Figure 2. ECG after the administration of intracoronary nitrates and complete relief of the patient's symptoms, where the ST-segment elevation has disappeared.

enzyme levels consistent with a small degree of myocardial necrosis (CK-MB up to 50 IU/l, troponine up to 80 ng/dl) but without any ECG changes. He was discharged in very good general condition, under treatment with nitrates, aspirin, diltiazem, as well as methylprednizolone and setirizine for a further seven days.

Discussion

The fact that an allergic reaction can cause angina or myocardial infarction, though rarely, has been known for a number of years[1-7] and is believed to occur via two different mechanisms: either by causing coronary artery spasm or through rupture of an atheromatous plaque and the creation of thrombus.[8]

Figure 3. Left anterior oblique angiographic projection of the right coronary artery, showing full occlusion of the artery at its origin.

In the case of spasm, the prevailing view is that mediators of the allergy that cause vasospasm are released from mast cells in the adventitia of coronary vessels and the perivascular region. Such mediators include serotonin, catecholamines, prostaglandins, leukotrienes, thromboxane and histamine, whose role has been studied the most.[9-12] More specifically, coronary arteries are known to have two kinds of histamine receptor, H_1 and H_2.[13] The role of the H_2 receptors is not considered to be particularly important. However, stimulation of the H_1 receptors with small doses of histamine in patients with healthy coronary arteries and with no history of ischaemic heart disease causes vasodilation in both the epicardial coronary arteries and the smaller resistance vessels, via the release of NO (endothelium-dependent vasodilation).[14] It has also been observed that stimulation of those receptors in some patients who have a history of angina causes spasm of the epicardial coronary arteries.[15] This is a particularly interesting finding that could be due to an increased concentration of mast cells in the adventitia of the patients' coronary arteries[16,17] and hence to the release of histamine in relatively high concentrations, and/or to the coexistence of endothelial damage in those coronary arteries, something that would disturb their tone and lead to spasm rather than

Figure 4. Left anterior oblique angiographic projection of the right coronary artery after the administration of intracoronary nitrates, showing full restoration of the blood flow.

vasodilation after the local release of histamine. The fact that experiments have shown large concentrations of histamine to cause vasoconstriction rather than vasodilation supports the former view.[18]

As regards the rupture of atheromatous plaque as the result of an allergic reaction, this would obviously require the presence of atheromatous disease. Here again, a fundamental role is played by the mast cells, which apart from vasoconstrictory mediators also secrete enzymes with proteolytic properties, such as chymase and tryptase. These enzymes degrade ingredients of the fibrous cap of the atheromatous plaque via the activation of metalloproteinase, rendering it vulnerable to rupture and thrombosis.

It should be noted finally that every serious allergic reaction that causes hypotension, tachycardia and sometimes severe hypoxygenaemia (anaphylactic shock), can cause myocardial ischaemia in patients with subclinical coronary artery disease, and that the epinephrine that is administered externally for the treatment of such conditions could contribute to this.

In the case of the specific patient, the cause of the acute infarction was spasm in the right coronary artery, as proved by the second coronary angiogram. We can hypothesise that the mechanism behind the spasm was the local release of vasoconstrictory mediators of the allergic reaction, since the patient was not taking any other drugs that might have caused the spasm. Also, in view of the fact that the patient had taken no other medication and had not been exposed to any likely allergen before the angiography, we can surmise that the acute allergic reaction was probably due to the iodinated contrast agent. Although we know from earlier reports that iodinated contrast agents can cause coronary artery spasm following an allergic reaction, this is the first case where the spasm was also documented angiographically after the contrast agent was administered.

As regards the therapeutic approach to patients with coronary spasm following an allergic reaction, this should include vasodilators, such as nitrates, and calcium channel inhibitors, which are in any case the treatment of choice in every case of coronary spasm. In contrast, the role of corticosteroids and antihistamines, apart from their clear usefulness in the treatment of systemic manifestations of the allergy, has not been fully determined. In other words, it is not known to what extent these, and other pharmaceutical agents that have a stabilising action on the membrane of mast cells or restrict the action of mediators of the allergy, have a role in the treatment of acute coronary events that are caused by allergic reactions. In the future, the better study and understanding of the mechanisms through which an allergy causes acute coronary syndromes should lead to more specialised and effective therapeutic interventions.

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