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International Journal of Cardiology 139 (2010) e1 – e4

International Journal of Cardiology

www.elsevier.com/locate/ijcard

Letter to the Editor

Acute myocardial infarction and transient ischemic attack in a patient with lone atrial fibrillation and normal coronary arteries

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Received 1 April 2008; accepted 28 June 2008
Available online 19 August 2008

Abstract

We present the case of a 50-year old female with a history of paroxysmal atrial fibrillation and without any antithrombotic therapy, who was admitted to the neurologic department of our hospital with symptoms of cerebral ischemia. Two hours after the release of the neurological syndrome, she experienced an acute ST-segment elevation myocardial infarction (STEMI) of the inferior wall, which was not thrombolysed due to active menstruation. The coronary angiography was performed nine days later and it showed normal coronary arteries. This is the first case report of a TIA and an acute myocardial infarction due to atrial emboli, in a middle aged woman without any coronary lesions. In our opinion it points out the strength and the effectiveness of the internal fibrinolytic mechanisms.

Keywords: Lone atrial fibrillation; Acute myocardial infarction; Transient ischemic attack; Spontaneous fibrinolysis; Internal fibrinolysis

1. Case report

A 50-year old woman with a medical history of lone paroxysmal atrial fibrillation for the last 4 years and without any anticoagulation therapy, was admitted to the outpatient department of our hospital with diplopia, headache and paresthesias of the left side of her body. The physical examination revealed a female patient with a normal body mass index, who was afebrile and had stable vital signs. Her lungs were clear. There was an irregular rhythm (90 bpm) with clear first and second sound, without any murmurs, gallops, or rubs. The systolic blood pressure was 110 mm Hg. Her ECG showed atrial fibrillation. The radiological laboratory provided us with a normal chest X-ray and a brain CT scan with no signs of cerebral or subarachnoid hemorrhage or other intracerebral abnormality.

The patient was admitted to the neurological department with the diagnosis of cerebral ischemia. Six hours later, the neurological deficits had disappeared and the patient was feeling a mild headache. In eight hours, after her admission, she developed an intense chest pain with nausea and sweating which lasted for 2 h. The ECG showed atrial fibrillation with ST elevation in the leads II, III and aVf. Six hours after the onset of the chest pain, her biochemical values were, CPK 328 U/L, CKMB 68 UI/lt and TnI 3.2 ng/ml. TnI continued to increase until it was doubled, 8 h later. The rest enzyme kinetics were typical for myocardial infarction. She was given a loading dose of 300 mg of clopidogrel po, 500 mg of acetyl-salicylic acid po, 450 mg amiodaron IV and 30 mg of enoxaparine IV. Her treatment comprised clopidogrel 75 mg × 1po, acetyl-salicylic 100 mg × 1po, enoxaparine 70 mg × 2 SC, and amiodarone 900 mg iv with slow infusion for 5 days and then sotalol 80 mg × po. Despite the antiarrythmic agents, she was still having paroxysm of atrial fibrillation throughout her hospitalization.

The transthoracic echocardiogram revealed an hypokinesia of the inferior wall of the left ventricle and of the middle part of the intraventricular septum. A mitral valve prolapse with a mild insufficiency without any significant valvulopathy also

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0167-5273/$ - see front matter © 2008 Elsevier Ireland Ltd. All rights reserved.
doi:10.1016/j.ijcard.2008.06.085

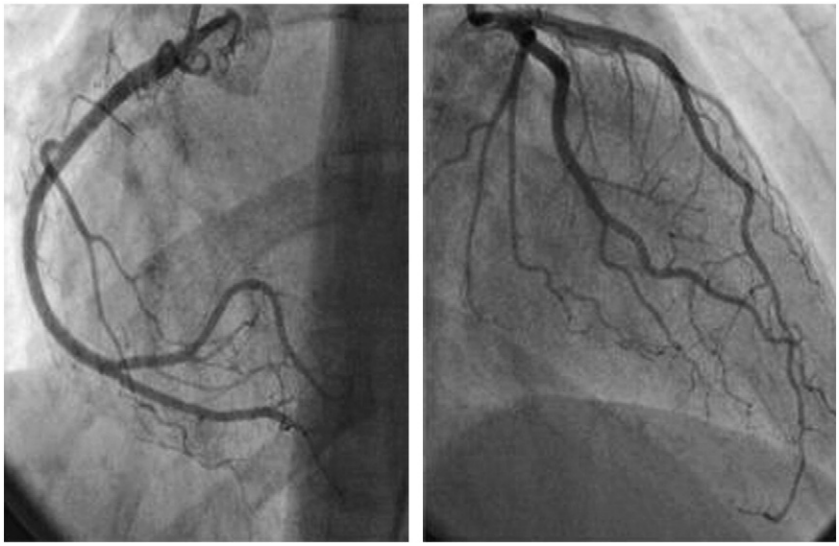

Fig. 1. The coronarography revealed normal coronary arteries.

found. The measurements of pericardium, aortic root and ascending aorta were found to be within normal values. The transeosophageal echo didn't show spontaneous echo contrast or any obvious thrombus in left atrial appendage. The intraatrial septum appears to be without lesions.

Nine days later the patient underwent a coronary angiography, which revealed normal coronary arteries with no atheromatic lesions (Fig. 1). Other examinations such as tripplex of carotic arteries showed no abnormal results. At her discharge, the patient had a small consistent hypokinesia of the inferior wall but she was in a good clinical condition. Six months later the transthorasic echo was still showing a mild hypokinesia of the basal inferior wall (Fig. 2).

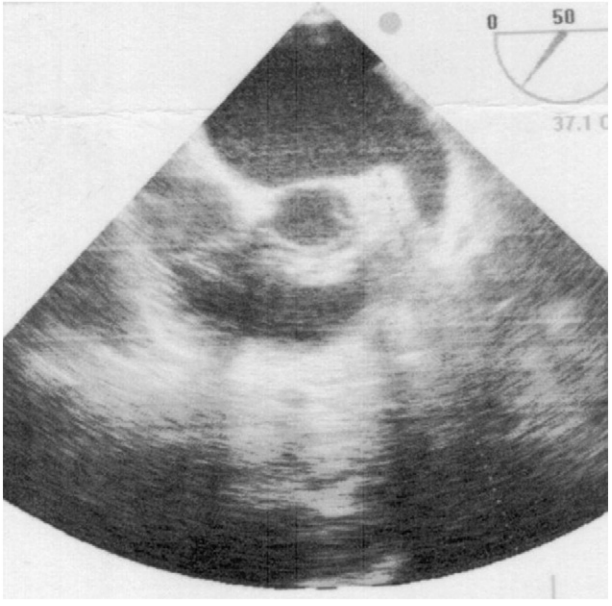

Fig. 2. The transesophageal echo displayed a normal left atrium.

2. Discussion

Atrial fibrillation (AF) is an arrhythmia associated with stroke [1] and systemic embolism [3]. It is also the most frequently encountered cardiac arrhythmia, with a prevalence of 2% to 4% in adults over age 60 years [4]. The term lone atrial fibrillation (LAF) is used for young patients (up to 65 years old) with no clinical or echocardiographic evidence of cardiac disease. LAF may be paroxysmal, persistent or permanent. It accounts for less than 15% of cases of chronic AF and up to 45% of cases of paroxysmal AF. It is significantly more prevalent among men than among women.

The correlation between atrial fibrillation and arterial embolisms is well established. The most common sites are, the brain, resulting, as in this case did, in cerebral ischemia (TIA or stroke), the eye, the kidneys, the intestine, the spine, and important arteries of the arms and legs. Obstruction of a coronary artery is rarely referred in the international bibliography [5,17].

The annualized rate of ischemic stroke is similar for those with intermittent [2,6] and sustained AF. According to the CHADS2 score [7], the most clinically useful model for the estimation of the risk of ischemic stroke in patients with AF, the annual risk is 0.49%, compared to the warfarin group which is 0.29%. This means that we must treat 500 patients with warfarin for one year to prevent one stroke. The most recent guidelines [8] for the management of atrial fibrillation in young patients with LAF suggest that aspirin (100–325 mg per day), offers a compensatory level of protection against thromboembolism.

Reviewing international medical journals, we came across enough articles investigating the relationship between cerebral ischemic attacks and elevation of cardiac troponin or CKMB isoenzyme. It seems that elevated troponin I in stroke patients, has to do with a minor myocardial injury due to

sympathoadrenal activation [9–12]. It is found that damage of specific brain regions like right insula is related with higher values of TnI but this is a matter that is still under investigation [13]. The magnitude of troponin elevation in these reports is less than that seen with acute myocardial infarction due to coronary artery occlusion [9] and it is associated with the severity of stroke rather than the underlying heart disease [14]. Additionally, transient ischemic attacks do not usually increase cardiac troponin. CKMB, a less specific biochemical marker for cardiac damage, is often elevated even if TnI is normal, and is likely to be noncardiac in origin [15]. Some patients, approximately 9% may develop acute myocardial infarction during the first few days of their cerebral ischemia but as coronary angiography shows later, this is a consequence of the underlying coronary heart disease that they already suffer from [16]. The pathophysiology of these events is explained by the sympathetic nervous stimulation that increases the oxygen consumption of a heart with a defective coronary reticulum.

In our case, a woman with a transient ischemic attack had typical electrocardiographic and laboratory findings of an acute myocardial infarction. The possibility of myocardial damage due to adrenal hyperactivity is doubtful due to the high values of TnI in association with CKMB and SGOT curves. Also the localization of the malfunctioning area of the myocardium using the ECG was comparable with the hypokinetic area as it was displayed by the ultrasound examination. So there is a strong piece of evidence that there had been a necrosis of a part of the inferior wall of the left ventricle. The hypothesis of an arterial vasospasm that lasted long enough to cause a necrosis cannot explain fully our case because the onset of the episode happened over 2 h after the release from the neurological syndrome and the patient didn't respond to the intravenous infusion of nitroglycerine. The only hypothesis that can offer a satisfying explanation for the two entities (myocardial infarction and TIA) at the same time is that of an arterial (coronary and cerebral respectively) embolism.

Coronary artery embolism, is a situation extremely rare in clinical practice. The topography of the coronary ostiums inside the sinuses of valsava, protects them against the aortic jet and the small particles that it may carry. Though it is occasionally referred in literature, it has been described as a complication of various cardiac diseases such as atrial fibrillation [17], dilated cardiomyopathy [18,19], infective endocarditis [20], prosthetic valve [21] or even deep venous thrombosis [22] (paradoxical embolism). The embolisms were attributed to poor anticoagulation, high coagulability or defective C or S protein. The further investigation of these events with coronary angiography, indicated total or subtotal occlusions of coronary arteries which were treated with intracoronary thrombolysis, ballooning, stenting or thrombectomy. In respect of our patient, we believe that both obstructed arteries (cerebral and coronary) were spontaneously recanalised, as it was disclosed by the disappearance of neurological defects as well as by the coronary angiography.

Spontaneous thrombolysis is a process initiated by the tPA or by pro-urokinase (pro-UK) released from endothelial cells. These agents act on plasminogen, converting it to the active molecule of plasmin. The whole process may take hours to several days for completion. Spontaneous resolution of occlusions in coronary arteries [23], veins or even synthetic grafts [24,25] have has been reported in the past. Although these mechanisms appear to be rare or ineffective, recent surveys revealed that inadequate fibrinolytic activity, intensifies the severity and the frequency of coronary thromboembolic events [26–28]. Further study of this process and the incoming of a global test for it's measurement [29], will enable us a) to detect agents, among common used drugs, such as antidiabetics or antihypertensives that may hinder spontaneous fibrinolysis and b) to separate the patients, who suffer from coronary heart disease, into a high risk group that needs a more aggressive antiplatelet therapy [29].

Acknowledgement

The authors of this manuscript have certified that they comply with the Principles of Ethical Publishing in the International Journal of Cardiology [30].

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