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CORONARY EMBOLISM CAUSING MYOCARDIAL INFARCTION AFTER HEART VALVE SURGERY

INFARKT MIOKARDA IZAZVAN KORONARNOM EMBOLIJOM NAKON OPERACIJE SRČANIH ZALISTAKA

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Short title: Coronary embolism after heart surgery

Abstract

Introduction. Coronary embolism can rarely be a cause of myocardial infarction. It is usually associated with atrial fibrillation, dilated cardiomyopathy, bacterial endocarditis and underlying hypercoagulable state, as well as heart surgery. Case report. We report a case of a patient with severe mitral and tricuspid regurgitation, with no underlying coronary artery disease. The patient underwent heart valve surgery, and the immediate postoperative course was uneventful. Five days after the operation the patient sustained cardiac arrest, which was followed by a successful cardiopulmonary resuscitation. Electrocardiography showed atrial fibrillation with a significant ST segment elevation in the inferior leads. Urgent coronary angiography revealed a total occlusion of the right coronary artery, thus percutaneous coronary intervention was performed, after which flow restoration through the artery was achieved. The patient was discharged with triple antithrombotic therapy on the 20th postoperative day. Conclusion. Heart surgery could be followed by unexpected and potentially fatal complications, coronary embolism being one of them. The prompt and adequate reaction by the whole medical team is crucial for the patient's survival and recovery.

Key words: coronary embolism, myocardial infarction, atrial fibrillation, heart valve surgery.

Apstrakt


Ključne reči: koronarna embolija, infarkt miokarda, atrijalna fibrilacija, operacija srčanih zalistaka.

Introduction

Coronary embolism (CE) is a rare non-atherosclerotic cause of myocardial infarction (MI). In an autopsy study performed decades ago, CE was accountable for 13% of MI. A more recent clinical study showed even lower prevalence of CE in MI patients of only 2.9%.
The conditions associated with CE are atrial fibrillation (AF), dilated cardiomyopathy, bacterial endocarditis and underlying hypercoagulable state, as well as recent heart surgery.2,3

There is controversy regarding CE treatment. Different reperfusion strategies have all shown moderate success. Historically, drug therapy with anticoagulant and fibrinolytic agents was used. Nevertheless, percutaneous techniques such as percutaneous coronary intervention (PCI) thrombus aspiration, balloon angioplasty and stent implantation, have become preferred choice.2-6 Yet, consensus was not reached regarding recommendations for the optimal treatment.2

The significance of CE lies in the fact that, despite the low incidence, it represents an urgent, potentially fatal condition, with worse prognosis when comparing to MI caused by atherosclerosis.2

Case report

We report a case of a 61-year-old male who was admitted for elective surgery of heart valves. The patient complained of fatigue and shortness of breath. His heart rhythm was permanent AF, for which he was on oral anticoagulation therapy. The patient had a history of hypertension and hyperlipidemia, without other comorbidities. He was not a smoker and his family history was negative.

Echocardiography showed a severe mitral regurgitation (MR) with dilated mitral annulus (44 mm) and sclerotic lesions of mitral cusps and subvalvular apparatus. MR effective orifice area was 40 mm2 and regurgitant volume was 70 ml. Left atrium was extremely dilated with 57 mm in diameter and 142 ml in volume (indexed volume 69 ml/m2). Left ventricular ejection function was preserved (60%), however diastolic dysfunction type I (impaired relaxation) was present (E/E'=11). There was a moderate tricuspid regurgitation with a moderate pulmonary hypertension.

Coronary angiography prior to surgery excluded coronary artery disease (Figure 1-A). The patient was afterwards referred to cardiac surgeon for mitral and tricuspid valve surgery.

The patient underwent surgery in general anesthesia with the use of extracorporeal circulation. Mitral annuloplasty with the implantation of a rigid ring No. 30 and De Vega tricuspid annuloplasty were performed. The operation was completed successfully and the immediate postoperative course was uneventful.

Postoperative electrocardiography (ECG) showed AF with a normal ventricular rate (Figure 2), there were no changes in compare to the preoperative ECG. Anticoagulation therapy with low-molecular weight heparin was initiated, followed by a graduate introduction of warfarin after the removal of chest tubes on the 2nd postoperative day, with a starting dose of 2.5 mg/day and a gradual increase to 5 mg/day.

On the 5th postoperative day the patient complained of a severe chest pain with a sudden onset, which was followed by a cardiac arrest due to ventricular fibrillation. The cardiopulmonary resuscitation (CPR) was initiated immediately. The patient was intubated and mechanically ventilated, and after ten attempts of defibrillation, along with medical support, return of spontaneous circulation was achieved.

The laboratory analyses at the moment of the incident showed INR 1.2, despite the target values being set to 2.0-3.0, and a significant increase in cardiac enzymes levels (creatine kinase 1,471 U/l, creatine kinase MB 154 U/l, high-sensitive troponin T >40,000 ng/l) and D-dimer (>10,000 mg/ml).
ECG showed AF with a significant ST segment elevation in the inferior leads (Figure 3). The patient was in cardiogenic shock with mean arterial pressure of 50 mmHg and heart rate of 130 bpm, thus noradrenaline infusion was administered. Arterial blood gases showed metabolic acidosis (pH 7.11, lactate 10 mmol/l, BE -18.5 mmol/l, pO$_2$ 47 mmHg, pCO$_2$ 31 mmHg, SaO$_2$ 65%).

Dual antiplatelet therapy was initiated immediately, including aspirin and clopidogrel (loading doses 300 mg and 600 mg, respectively, followed by maintenance doses 100 mg/day and 75 mg/day). Urgent coronary angiography revealed a total occlusion of the distal right coronary artery with Thrombolysis in Myocardial Infarction (TIMI) 0 flow (Figure 1-B). Primary PCI was indicated. After an initial balloon dilatation which did not result in restoration of TIMI flow, multiple thrombi aspirations were performed. However, due to distal embolization, aspiration catheter could not be placed deep into the postero-lateral branch of the right coronary artery, so the whole thrombi could be aspirated. Thus, downstream abciximab was given, i.v. bolus of 0.25 mg/kg followed by an infusion of 0.125 µg/kg/min for 12 hours. Furthermore, due to the large thrombus burden, two drug-eluting stents were implanted resulting in optimal outcome and TIMI 3 flow restoration at the end of the procedure (Figure 1-C).

After the successful PCI the patient was returned to the intensive care unit. Repeated echocardiogram showed normal function of the mitral and tricuspid valves, but akinesia of the inferior wall was registered. Despite the timely performed PCI, mechanical ventilation and noradrenalin infusion, the patient was still hypotensive and hypoxic, which, along with extremely elevated D-dimer level, raised a suspicion for pulmonary embolism. However, it was excluded by computerized tomography pulmonary angiography.

The ECG changes gradually resigned. The intensive care treatment resulted in hemodynamic stabilization and oxygen level restoration, so after five days the patient was extubated and measures of early rehabilitation were initiated.

Further course was uneventful. The warfarin dose was increased to the maximal 10 mg/day in order to reach the therapeutic INR value. A 24-hour monitoring of the ECG recorded AF with a good heart rate control. The patient was discharged on the 20-postoperative day in a good general condition with triple antithrombotic therapy including aspirin, clopidogrel and warfarin. Since this is a patient with permanent AF and acute MI, triple antithrombotic therapy is recommended for the first 6 months. After that period aspirin or clopidogrel could be excluded, and after 12 months, if no major adverse cardiac events occur, single therapy with warfarin could be considered.

Three months following discharge, the patient was asymptomatic with no signs of heart failure, New York Heart Association (NYHA) class I and Canadian Cardiology Society (CCS) grade I. ECG showed AF with a good rate control and no signs of myocardial ischemia and lesion. Echocardiography revealed normal function of the mitral and tricuspid valves with normal systolic function of the left ventricle and no wall motion abnormalities.

**Discussion**

The unexpected life-threatening event in the postoperative course of our patient was evidentially caused by MI. This was confirmed by the ECG which showed ST segment elevation in the inferior leads, as well as the elevated levels of cardiac enzymes in the blood and the coronary angiography finding.
MI due to coronary artery injury during heart valve surgery is a rare but possible complication. Because of the close proximity of the mitral annulus with the left circumflex coronary artery, this artery can be injured during mitral valve surgery. For the same reason, tricuspid valve surgery carries a risk of the right coronary artery injury. There are multiple mechanisms for these injuries, but the majority include mechanical obstructions caused by a surgical needle or a suture.

In all the published cases MI occurred during the operation or in the following few hours. Considering that in our case more than 96 hours have passed since the end of the operation and that the patient was completely stable during this time, without any ECG or laboratory abnormalities, this mechanism is excluded as the cause of the MI.

The patient had AF and the oral anticoagulant therapy was administered after the operation. The INR values, however, did not yet reach the optimal therapeutic range. For this reason, CE is proposed as an underlying cause of the MI. AF could have led to a thrombus formation in the extremely dilated left atrium, which then embolized to the coronary artery through the normal anatomic path. The thrombi aspirations during the PCI increase the suspicion for this mechanism. Clinical studies addressing this topic showed that AF is the most common underlying cause of CE, which supports this theory.

A similar case was reported by Wang et al. in which MI occurred seven weeks after heart surgery in patient with prior history of AF. In this case, a definitive diagnosis of CE was established after echocardiographic finding of a thrombus in the left atrium. The INR at the moment of the incident was, like in our case – subtherapeutic.

None of the eminent cardiovascular societies have published clinical guidelines concerning diagnosis and treatment of CE. Shibata et al. suggested the criteria useful for the CE diagnosis (Table 1). Our patient had one major criterion: angiographic evidence of coronary artery thrombosis. Minor criteria that were present were: <25% luminal stenosis on other coronary arteries except for the culprit lesion; and presence of the risk factors for thromboembolism, such as AF, cardiomyopathy and recent cardiac surgery. Presence of one major and two minor criteria are sufficient for the diagnosis of CE.

The MI can be classified as the type 2 according to the Universal classification of myocardial infarction. This stands for MI secondary to an ischemic imbalance, which includes CE. The MI cannot be interpreted as a surgical complication, considering that more than 72 hours have passed between the operation and the MI, and that during this time the patient was completely stable and had normal recovery.

In conclusion, it should be noted that heart surgery could be followed by a wide range of complications and although a large number of them are well known and predictable, there are some that are rare and unexpected but potentially fatal, coronary embolism being one of them. The prompt and adequate reaction by the whole medical team was in this case crucial for the patient's survival and recovery.

References

Table 1

Criteria for the diagnosis of coronary embolism

<table>
<thead>
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<th>Major criteria</th>
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<tr>
<td>- Angiographic evidence of coronary artery embolism and thrombosis without atherosclerotic components</td>
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<tr>
<td>- Concomitant coronary artery embolization at multiple sites</td>
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<td>- Concomitant systemic embolization without left ventricular thrombus due to acute myocardial infarction</td>
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### Minor criteria

- <25% stenosis on coronary angiography, except for the culprit lesion
- Evidence of an embolic source based on transthoracic echocardiography, transesophageal echocardiography, computed tomography, or magnetic resonance imaging
- Presence of embolic risk factors: atrial fibrillation, cardiomyopathy, rheumatic valve disease, prosthetic heart valve, patent foramen ovale, atrial septal defect, history of cardiac surgery, infective endocarditis, or hypercoagulable state

### Definite diagnosis of coronary embolism

Two or more major criteria, or
One major criterion plus two or more minor criteria, or
Three minor criteria

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**Fig. 1** - Coronary angiography: A) prior to the surgery: normal right coronary artery; B) after the CPR following cardiac arrest: occlusion of the right coronary artery (arrow); C) after the PCI: recanalized right coronary artery

**Fig. 2** - Postoperative ECG: AF without any ST segment abnormalities

**Fig. 3** - ECG after the CPR: AF with elevation of ST segment in the inferior leads