CASE REPORT

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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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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 reported 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. In such case, the prompt and adequate reaction by the whole medical team is crucial for a patient's survival and recovery.

Key words:

coronary vessels; embolism; heart valves diseases; myocardial infarction; postoperative complications; resuscitation; treatment outcome.

Apstrakt

Uvod. Koronarna embolija je redak uzrok infarkta miokarda. Najčešće se povezuje sa atrijalnom fibrilacijom, dilatativnom kardiomiopatijom, bakterijskim endokarditisom i hiperkoagulabilnim stanjem, kao i sa operacijom srca. Prikaz bolesnika. Prikazan je slučaj bolesnika sa teškom mitralnom i trikuspidnom regurgitacijom, bez postojeće koronarne bolesti. Urađena je operacija srčanih zalistaka, a neposredni postoperativni tok je protekao uredno. Pet dana posle operacije došlo je srčanog zastoja koji je bio praćen uspešnim reanimacionim postupkom. Elektrokardiografski je registrovana atrijalna fibrilacija sa značajnom elevacijom ST segmenta u inferiornim odvodima. Urgentna koronarna angiografija je pokazala totalnu okluziju desne koronarne arterije, te se pristupilo perkutanoj koronarnoj intervenciji kojom je uspešno uspostavljen ponovni protok kroz arteriju. Bolesnik je otpušten 20. postoperativnog dana, sa trojnom antitrombotskom terapijom. Zaključak. Operacija srca može biti praćena neočekivanim i potencijalno fatalnim komplikacijama, među kojima je i koronarna embolija. Brza i adekvatna reakcija medicinskog tima ključna je za preživljavanje i oporavak bolesnika.

Ključne reči: koronarni krvni sudovi; embolija; zalisci srca, bolesti; infarkt miokarda; postoperative komplikacije; reanimacija; lečenje, ishod.

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

Introduction

Coronary embolism (CE) is a rare nonatherosclerotic cause of myocardial infarction (MI). In an autopsy study performed decades ago, CE was accountable for 13% of MI 1 . A more recent clinical study showed even lower prevalence of CE in MI patients of only 2.9% 2 .

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 ^{4–6}. Yet, consensus was not reached regarding recommendations for the optimal treatment.

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 reported 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. Mitral regurgitation (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 1A). The patient was afterwards referred to a 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 successfully completed and the immediate postoperative course was uneventful.

Postoperative electrocardiography (ECG) showed AF with a normal ventricular rate (Figure 2), there were no changes in comparison to the preoperative ECG.



Fig. 2 – Postoperative electrocardiographic finding (ECG): atrial fibrillation with normal ventricular response and without any ST segment abnormalities.





Fig. 3 – Electrocardiographic finding after the cardiopulmonary resuscitation: AF with elevation of ST segment in the inferior leads.

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 immediately initiated. 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 (CKMB)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, basae excess (BE) - 18.5 mmol/L, pO₂ 47 mm Hg, pCO₂ 31 mm Hg, SaO₂ 65% (nr > 94%)].

Dual antiplatelet therapy was immediately initiated, including aspirin and clopidogrel (loading doses 300 mg and 600 mg, respectively, followed by maintenance doses 100 mg/day and 75 mg/day, respectively). Urgent coronary angiography revealed a total occlusion of the distal right coronary artery (RCA) with thrombolysis in myocardial infarction (TIMI) 0 flow (Figure 1B). 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 RCA, so the whole thrombi could be aspirated. Thus, downstream abciximab was given, intravenous (iv.) bolus of 0.25 mg/kg followed by an infusion of 0.125 µg/kg/min for 12 h. 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 (represented normal epicardial reperfusion) (Figure 1C).

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 h monitoring of the ECG recorded AF with a good heart rate control. The patient was discharged on the 20th postoperative day in a good general condition with triple antithrombotic therapy including aspirin, clopidogrel and warfarin.

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 total occlusion of RCA seen by coronary angiography.

MI due to coronary artery injury during heart valve surgery is a rare but possible complication. Because of the

	Major criteria
٠	Angiographic evidence of coronary artery embolism and thrombosis without atherosclerotic components
٠	Concomitant coronary artery embolization at multiple sites
•	Concomitant systemic embolization without left ventricular thrombus due to acute myocardial infarction
	Minor criteria
٠	< 25% stenosis on coronary angiography, except for the culprit lesion
•	Evidence of an embolic source based on transthoracic echocardiography, transesophageal echocardiography, compute 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
٠	Two or more major criteria, or
•	One major criterion plus two or more minor criteria or

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

close proximity of the mitral annulus with the left circumflex coronary artery, this artery can be injured during mitral valve surgery ^{7, 8}. For the same reason, tricuspid valve surgery caries a risk of the right coronary artery injury ^{9, 10}. There are multiple mechanisms for these injuries, but the majority include mechanical obstructions caused by a surgical needle or a suture ^{8, 11, 12}.

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 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 was 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 was proposed as an underlying cause of 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 increased the suspicion for this mechanism. Clinical studies addressing this topic have shown that AF is the most common underlying cause of CE,^{2, 3} which supports this theory.

A similar case was reported by Wang et al. ¹³ in which MI occurred seven weeks after heart surgery in a 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 value at the moment of the incident was subtherapeutic, like in our case. Because this was a patient with permanent AF and acute MI, triple antithrombotic therapy was recommended for the first 6

months after discharge from the hospital. 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.

None of the eminent cardiovascular societies have published clinical guidelines concerning diagnosis and treatment of CE. Shibata et al. ² suggested the criteria useful for the CE diagnosis (Table 1). Our patient had one major criterion: angiographic evidence of coronary artery thrombosis. Minor criteria 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.

MI can be classified as the type 2 according to the universal classification of MI ¹⁴. This stands for MI secondary to an ischemic imbalance, which includes CE. MI cannot be interpreted as a surgical complication, considering that more than 72 hours passed between the operation and the event, and that during this time the patient was completely stable and had normal recovery.

Conclusion

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 in such case is crucial for a patient's survival and recovery.

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