Syncope as initial symptom of ostial lesion of the left main coronary artery with cardiogenic shock

Sinkopa kao početni simptom ostijalne lezije glavnog stable leve koronarne arterije sa kardiogenim šokom

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Abstract

Introduction. Syncope represents a relatively atypical symptom of acute coronary syndrome. Syncope itself does not provide enough information to indicate an acute coronary event, especially a lesion of the left main coronary artery, without malignant rhythm and conduction disorders.

Case report. A male patient, aged 63, was admitted to the intensive cardiac care unit because of a short loss of consciousness, in sinus tachycardia, with signs of acute heart failure and being hypotensive. Electrocardiogram showed a possible acute anterior myocardial infarction, followed by cardiogenic shock and emergency coronary angiography (subocclusive ostial lesion of the left main coronary artery) and primary percutaneous coronary intervention with intraaortic balloon pump therapy was performed. A direct drug eluting stent was implanted with the optimal primary result.

Conclusion. The prompt diagnosis, especially in such relatively atypical clinical presentation, reperfusion therapy with primary percutaneous coronary intervention in acute myocardial infarction complicated by cardiogenic shock, contribute to the improvement in the survival rate and patient’s quality of life. This case report is clinically educative due to relatively atypical presentation and performed interventions.

Key words: myocardial infarction; syncope; shock, cardiogenic; coronary angiography; drug-eluting stents.

Syncope is a transient loss of consciousness precipitated by cerebral hypoperfusion, which is associated with the absence of postural tone and usually followed by a complete recovery within a few minutes. This clinical condition is a common medical problem with an estimated incidence of per 1,000 persons per year and accounts for 1% of emergency department visits and 6% of all hospital admissions.\(^1\)

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Syncope represents a relatively atypical symptom of acute coronary syndrome. Only 3.1% patients with syncope were diagnosed with acute myocardial infarction \(^2\). Syncope itself does not provide enough information to indicate an acute coronary event, especially a lesion of the left main coronary artery, without malignant rhythm and conduction disorders. Many of the missed diagnoses included syncope that resulted in medico-legal action involved. Therefore, establishing the diagnosis of syncope is important so that specific treatment can be administered to prevent future recurrences and eliminate the underlying predisposing disease.

Treatment of unprotected stenosis of the left main coronary artery (LMCA) still remains a challenge for interventional cardiologists. According to the current recommendations of the American Heart Association (AHA) and American College of Cardiology (ACC), unprotected LMCA stenosis represents an indication for cardio-surgical treatment, except in special situations where interventional cardiology has its place \(^3\). Some of them are ostial lesion of the left main stem, proximal lesion of the left main stem, acute myocardial infarction, and initial and developed cardiogenic shock \(^3\). According to current recommendations, the patients with acute myocardial infarction (AMI), complex mechanical complications or cardiogenic shock are supported using intra-aortic balloon pump (IABP) \(^4\). Nowadays it is recommended that even percutaneous coronary intervention (PCI) on LMCA is performed with the support of IABP or other circulatory support \(^5\).

For this reason, we presented a patient with syncope and acute myocardial infarction with incipient cardiogenic shock, diagnosed with unprotected ostial lesion of the left main coronary artery.

**Case report**

A male patient, aged 63, was admitted to the intensive cardiac care unit for a short loss of consciousness, without chest pain. There was no history of previous syncopal or presyncopal states or ischemic heart disease symptoms. Risk factors for ischemic heart disease were hypertension and smoking. At admission, the patient was conscious, hypotensive with blood pressure of 90/60 mmHg, with signs of acute heart failure and sinus tachycardia – heart rate of about 100/min, without malignant rhythm and conduction disorders. Electrocardiogram registered signs of possible acute anterior myocardial infarction, with incomplete left bundle branch block (LBBB) and first degree heart (AV) block (Figure 1). The initial laboratory tests showed increased values of myoglobin (261 ng/mL, reference values up to 107 ng/mL), whereas the levels of hs troponin and creatine kinase-MB (CK-MB) were within the reference range, due to patient’s early presentation. Initial echocardiographic examination showed ischemic cardiomyopathy with ejection fraction (EF) of 50%, and inferolateral basal and medial hypokinesia, apical anterior, basal inferior and medial anterior akinesia, with hyperkinetic movements of other segments. Soon after admission, the state of the patient worsened and the first signs of cardiogenic shock were registered (the mean arterial pressure – MAP of 60 mmHg, diuresis 30 mL/h). For this reason, the patient was initially administered midazolam, and the sedation was later maintained using propofol, and was supported with invasive mechanical ventilation (IMV) in the intermittent positive pressure ventilation (IPPV) mode with PEEP (positive end-expiratory pressure) 5 cm H\(_2\)O. The patient was treated with dual antiplatelet therapy (aspirin 300 mg, clopidogrel 600 mg), statin 20 mg through nasogastric tube, heparin (1,000 IU/h), and inotropic stimulation with dopamine 8 mcg/kg/min. Clinical status, electrocardiographic changes, increased level of myoglobin, and echocardiographic findings gave indications for emergency coronary angiography, which was performed within two hours after syncope. Coronary angiography of the left coronary artery indicated subocclusive ostial lesion of the left main coronary artery with TIMI (thrombolysis in myocardial infarction) grade 2 flow (Figure 2). The right coronary artery was without significant lesions (Figure 3). After consultation with the cardiac surgeon, because of the possibility for emergency surgical revascularization, primary percutaneous coronary...
intervention (pPCI) was performed. Periprocedural heparin (10,000 IU) was administrated intracoronary. Initial heparin infusion was stopped. As a result of the developing cardiogenic shock and lesion of the left main coronary artery, an intra-aortic balloon pump (IABP) with the ratio 1:1 was implanted before the intervention. The following were used in pPCI: 5F left Judkins guideing catheter 4.0, guidewire (Runthrough, Terumo Europe) and a drug-eluting stent (Nobori 3.5 × 14 mm, Terumo Europe).

During cannulation of the left coronary artery, its occlusion occurred as a result of ostial lesion, so immediately before the intervention, the guidewire was placed with the stent in the catheter, together cannulating the left coronary artery. A direct stent was deployed at 14 atm and the primary result of the intervention was optimal (Figures 4, 5). In order to prevent acute stent thrombosis, after the stent deployment, glycoprotein IIb/IIIa inhibitor abciximab was administered intracoronary, and continued intravenously during following 12 hours. After the intervention the patient still had signs of cardiogenic shock and was supported with the intra-aortic balloon pump. Right cardiac microcatheterisation was performed through the right interior jugular vein with a pulmonary artery catheter (Swan-Ganz). The value of pulmonary capillary wedge pressure (PCW) was 8 mmHg. These hemodynamic parameters can be explained with the sedation of the patient on IMV with PEEP 5 cm H2O, which caused systemic and pulmonary vasodilatation and lower PCW. Expansion of the volume was initiated using glucose and electrolyte solutions, along with inotropic stimulation with dopamine 8 mcg/kg/min. After this treatment, hemodynamic stabilisation was achieved (MAP 75 mmHg), PCW increased to 18 mmHg and diuresis was 150 mL/h. As a result of intermittent hemodynamic and respiratory instability, in the following four days the patient was on IMV in IPPV mode of ventilation, supported with IABP. After that, the patient was hemodynamically and respiratory sufficient, so he was extubated and circulatory support was stopped. The intra-aortic balloon pump removed without haemorrhagic complications. Carotid arteries ultrasound showed no significant lesions of carotid arteries. Follow-up echocardiographic examination showed EF of 45%, anteroseptal hypokinesia with mediopial septal akinesia. On the 15th day after admission, the patient was discharged with the diagnosis of acute anterior myocardial infarction (on the basis of electrocardiographic changes, and later increased levels of hs troponin and CK-MB, echocardiographic findings and the results of coronary angiography) (Figure 6), in cardiologically stable condition with double antiplatelet therapy (aspirin 300 mg, clopidogrel...
150 mg), beta-blocker (metoprolol 100 mg), ACE inhibitor (ramipril 2.5 mg), statin (atorvastatin 20 mg), diuretic (furosemid 40 mg) and aldosterone antagonist (spironolactone 25 mg). In the following 12 months the patient was without subjective complaints. Follow-up echocardiographic examination showed increased EF of 55%, hypokinesia apical-septal basal inferior. A stress exercise test using the Bruce protocol was performed and no signs of reduced coronary flow were registered. Ambulatory Holter-electrocardiography monitoring showed sinus rhythm, with the average heart rate 69/min, rare ventricular and supraventricular premature beats. No conduction disorders were detected.

**Discussion**

Cardiovascular causes are the most common life-threatening conditions associated with syncope, and these can be divided into arrhythmogenic, structural, and ischemic. Syncope from a sudden disruption in cardiac output is the deadliest form of syncope. Arrhythmogenic causes of syncope can include ventricular tachycardia, bradycardia (e.g. Mobitz type II or 3rd degree heart (AV) block), and significant sinus pauses (more than 3 seconds). Ischemia includes acute coronary syndromes, acute myocardial infarction. Among structural abnormalities are: valvular heart disease, such as aortic or mitral stenosis, cardiomyopathy (e.g., ischemic, dilated, hypertrophic), aortic dissection, atrial myxoma, and cardiac tamponade. Cardiac syncope more often occurs in patients older than 45 years. Life-threatening causes of syncope beside cardiovascular causes, include hemorrhage, and subarachnoid hemorrhage. Approximately 15% of the following life-threatening conditions present with syncope: subarachnoid hemorrhage, acute coronary syndrome, aortic dissection, leaking aortic aneurysm, and ruptured ectopic pregnancy. Many of the missed diagnoses of these five conditions that resulted in medico-legal action involved presentations that included syncope.

Syncope without chest pain is not the most typical symptom of acute coronary syndrome. In acute coronary syndrome, syncope is mostly caused by malignant rhythm and conduction disorders, and in fewer cases is caused by large myocardial damage. There is no much literature data on syncope after acute coronary event as neuromediated reaction. Sympathetic withdrawal seems to be the most likely mechanism of syncope. In the presented case, the most probable cause of syncope was cerebral hypoperfusion, which occurred due to incipient cardiogenic shock, since unstable atherosclerotic plaque in the ostium of the left main stem caused transient myocardial ischemia with lower cardiac output. Strategies in diagnostic syncope include the patient’s history, special tests like carotid sinus massage, tilt-table tests, echocardiography (ECHO), exercise stress test, cardiac monitoring, Holter monitor, external loop recorder, implantable loop recorder and electrophysiologic study. In emergency cases, like the presented one, for the diagnosis etiology of syncope were ECG, cardiac monitoring, ECHO and coronary angiography were important facts. Exercise stress tests, ambulatory ECG Holter monitor, loop recorders are important facts for the patient’s follow up.

Emergency echocardiography is an important and useful tool for establishing the diagnosis. That provides diagnostic and prognostic information on heart diseases that predispose patients to syncope, including the assessment of cardiac size, left-ventricular function, wall motion, valvular heart disease, pulmonary pressure or right ventricular strain and pericardial effusions. It has also become an established tool for diagnosing coronary artery disease.

Following the algorithm for emergency treatment of cardiovascular patients (ECG, biomarkers, echocardiography, and monitoring hemodynamic parameters), the precise diagnosis of acute myocardial infarction was quickly made and adequate treatment was provided. Primary percutaneous coronary intervention is nowadays an option for treatment of acute myocardial infarction, and it should be performed by the center with a large number of patients, which is able to perform the interventions 24 hours, 7 days a week, 365 days a year. Concerning treatment of lesions of the left main coronary artery, surgical myocardial revascularization...
Culprit lesions, when anterograde or retrograde flow is compromised, and when the patient is hemodynamically unstable; it is believed that in these circumstances PCI may enable a faster outcome than CABG. Taking all considerations of ACC/AHA from 2009 in treatment of lesions on the left main stem PCI does not require follow-up coronary angiography. Emergency PCI is recommended for unprotected lesion of the left main stem in the treatment of acute myocardial infarction, with "culprit" lesions, when anterograde flow is compromised, and when the patient is hemodynamically unstable; it is believed that in these circumstances PCI may enable a faster outcome than CABG. Taking all these facts into account, the treatment using PCI for the said patient was chosen, because the patient was in the state of initial cardiogenic shock, the ostial lesion of the left main coronary artery was suitable for PCI and it was quicker to treat it in this manner than by using surgical myocardial revascularization.

A drug-eluting stent (DES) was implanted, in the presented patient because the results of randomised studies showed that there was a significantly lower rate of major adverse cardiac events (MACE), mortality, and repeated revascularisation after 6 month, 12 months, 2 years and 3 years, in comparison with bare metal stents (BMS).

Cardiogenic shock is a cause of a high mortality rate in patients with acute myocardial infarction. Treatment of cardiogenic shock as a complication of acute myocardial infarction includes hemodynamic stability achieved by medication therapy or circulatory support and emergency revascularization using PCI or CABG. Medication therapy includes antiplatelet and antithrombotic drugs, vasopressors and inotropic agents. Antiplatelet and antithrombotic therapy should be done automatically in acute myocardial infarction. Volume application is often used, although it has not been analyzed in randomised studies. Vasopressors and inotropic agents are used because of their positive hemodynamic effect, but neither of them leads to permanent symptomatic improvement, and many even reduce survival rate, which may be associated with cell dysfunction caused by these drugs. A recent randomised study compared norepinephrine and dopamine in cardiogenic shock. Dopamine was associated with a higher mortality rate and more adverse effects, such as arrhythmia. In hypotension with other signs of cardiogenic shock, norepinephrine is recommended as the first choice. It should be initially administered in low doses and gradually titrated until systolic pressure reaches values of over 80 mmHg. After that, dobutamine can be administered together with norepinephrine for better contractility. In the presented case, hemodynamic stability was achieved using dopamine and volume application.

A SHOCK study revealed that patients with cardiogenic shock treated with pPCI or emergency surgical revascularization had better prospects of survival in comparison with patients with initial medication treatment.

Intra-aortic balloon pump is widely used in the treatment of cardiogenic shock and it represents the first line of circulatory support. A TACTICS study 5 has shown that IABP does not contribute to the reduction in intra-hospital mortality, but it brings about the improvement in a six-month mortality rate. A IABP-SHOCK II study has shown that the use of intra-aortic balloon counterpulsation does not significantly reduce a 30-day mortality in patients with cardiogenic shock complicating acute myocardial infarction for whom an early revascularization strategy was planned. Recent meta-analysis evidenced it in relation to survival rate when using IABP in cardiogenic shock. IABP therapy is considered to be a class IIb indication (European Society of Cardiology guidelines) for the management of cardiogenic shock.

Conclusion

This case report shows that the fast diagnosis, especially in patients with relatively atypical clinical presentation, and application of reperfusion therapy using percutaneous coronary intervention in patients with acute myocardial infarction complicated by cardiogenic shock, as well as application of adequate medication therapy and circulatory and respiratory support, contribute to the improvement in the survival rate and the quality of life of such patients.

REFERENCES


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