Does thrombolytic therapy harm or help in ST elevation myocardial infarction (STEMI) caused by the spontaneous coronary dissection?

Da li trombolitička terapija utiče negativno ili pozitivno na infarkt miokarda sa ST-elevacijom (STEMI) nastao spontanom disekcijom koronarne arterije?

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Abstract

Introduction. Spontaneous coronary artery dissection (SCAD) is a very rare disease with poor prognosis. It mainly affects young women free of risk factors for coronary artery disease (CAD) and women during the peripartum period. The prognosis for myocardial infarction caused by SCAD is poor, management is often difficult and guidelines still missing. Case report. We presented a woman with acute myocardial infarction of anterior wall of the left ventricle, caused by spontaneous dissection of medial segment of the left anterior descending coronary artery. We treated the patient with thrombolytic therapy and performed coronary angiography after that. Finally we decided to do nothing more. Two years later we performed coronary angiography again and founded the coronary artery normal. We also analyzed 19 cases published from 1996 to 2012 when coronary artery dissection had been treated with thrombolytic agent. Analysis revealed only one case of 19, with complication after treating SCAD with thrombolysis. Conclusion. Sometimes, regarding myocardial infarction in young women with no risk factors for CAD, especially in young women in peripartum, we should think about SCAD. The presented case, like eight others, demonstrates that good clinical outcomes can be achieved with thrombolysis. In spite of all this, we still need more data to verify that thrombolysis does not have to harm the therapy for SCAD. For the time being thrombolytic therapy could be an option.

Key words:
acute coronary syndrome; aneurysm dissecting; myocardial infarction; fibrinolytic agents; treatment outcome.

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Ključne reči:
akutni koronarni sindrom; aneurizma, disekanta; infarkt miokarda; fibrinolitici; lečenje, ishod.
Among reported case series ranging from 3 to 47 cases, there is the approximate 2:1 female predominance. About one third of the cases in women occur in the peripartum period. The clinical presentation of SCAD depends on the extent and the flow limiting severity of the coronary dissection, and ranges from asymptomatic to unstable angina, acute myocardial infarction, and ventricular arrhythmias to sudden cardiac death, and may be responsible for as many as 1 of 10 episodes of acute coronary syndrome in women younger than 50 years. Currently, clinical recognition of SCAD has increased as coronary angiography is utilised frequently in the clinical evaluation of patients with acute coronary syndromes. Moreover, intracoronary imaging techniques such as intravascular ultrasound (IVUS) and optical coherence tomography (OCT) have enabled a more detailed clinical assessment of SCAD.

The etiology of SCAD is unclear. Approximately one third of women with SCAD are pregnant or peripartum. Hormonal effects in the vessel wall such as reduced collagen synthesis, smooth muscle cell proliferation, and abnormalities in the proteoglycan matrix are implicated and may also explain cases of SCAD seen with oral contraceptive pill use. Dissection is caused when there is bleeding into the media of the artery, separating the vessel layers with subadventitial hematoma in the false lumen, compressing the true lumen to varying degrees.

The left anterior descending (LAD) coronary artery is the most frequently involved vessel in autopsy and angiographic series of the LAD artery accounts on average for 60% of cases. Patients have been treated successfully with medical therapy, coronary stenting, and coronary artery bypass grafting, depending on the extent and location of disease. In patients who have completed infarctions without residual ischemic symptoms medical therapy has been associated with good long-term outcomes. The role of thrombolysis in patients with ST elevation myocardial infarction (STEMI) is controversial.

In this article, we reported the case of STEMI caused by spontaneous coronary artery dissection, showing regression after conservative medical treatment. The management options and complications were discussed.

**Case report**

A 48-year-old female was admitted to the Emergency Department because of intense retrosternal pain of one hour duration and an electrocardiographic pattern of acute ST elevation anterior myocardial infarction (Figure 1). The patient was admitted to the intensive care unit and treated with intravenous thrombolysis (t-PA). We also administrated glycoprotein IIb/IIIa inhibitor (tirofiban), aspirin, clopidogrel, heparin, nitrates, and beta-blocker, and the patient’s clinical status progressively improved.

After the given therapy there was more than 50% resolution of ST segment elevation in leads V3, V4 and V5. Serial measurement of biochemical markers was consistent with myocardial necrosis. One day after admission we performed coronary angiography and found SCAD of the medial part of the LAD artery (Figure 2) with TIMI I-II flow in distal part of LAD. Neither were there atherosclerotic lesions in the affected vessel nor in the other coronary arteries and we decided to do nothing except medical therapy. After 5 days...
brain (B-type) natriuretic peptide (BNP) was highly elevated. Echocardiography performed two weeks post-admission revealed the akinetic apex and apical segments of the anterior and the inferior wall of the left ventricle. The global systolic performance of the left ventricle was satisfactory with the estimated ejection fraction of 45–50%. The patient was a smoker without any other conventional cardiovascular risk factor for coronary artery disease. In the thirteenth year of life she had infective endocarditis. She suffered from obsessive compulsive disorder and regularly visited the psychiatrist. Her past medical history was unremarkable and she denied any use of vasoconstricting or recreational drugs. She took no oral contraceptives (estrogen plus progestin) and had no a history of thromboembolic disease. All laboratory investigations performed in order to assess the inflammatory risk, coagulation abnormalities, as well as autoimmune disorders were found to be within normal limits. The patient had an uneventful clinical course and was discharged on a beta-blocker, aspirin, clopidogrel, statin and an ACE inhibitor.

The patient remained asymptomatic and 2 years later was subjected to second coronary angiography, which showed complete healing of the previous LAD dissection (Figure 3). There was normal sinus rhythm on ECG, with micro R wave in V2 and V3 with slightly negative T wave in D2, D3, aVF and from V4 to V6 (Figure 4).

We also retrieved literature (source PubMed) dealing with trombolytic therapy of SCAD. The retrieving process is presented in Figure 5. We identified 18 case reports in which...
trombolytics had been used in treatment of SCAD. Analysis of these cases including own one (this case) revealed only one case with complications as a consequence of thrombolytic use (Table 1).

**Discussion**

SCAD is a very rare disease with poor prognosis occurring more commonly in women, particularly in the peripartum period. The majority of affected individuals (approximately 75%) are young women without risk factors for atherosclerosis, of whom approximately 30% are in the peripartum period. SCAD usually involves a single vessel (ie, the LAD artery in women and the right coronary artery in men). There is no consensus on the treatment of SCAD. All three, medical, percutaneous coronary interventions and surgical approaches have been employed, but no randomized control trials have compared the three approaches. However, coronary dissection may regress spontaneously.

We analyzed 19 cases reports published from 1996 until 2012 with coronary artery dissection treated with thrombolytic agent. Successful use of thrombolytic agents have been published in eight cases including our. In 11 cases, thrombolytic agents did not help to re-establish coronary flow, but only in one case complications were described that may be associated with thrombolytic therapy. Two patients died, not from the effects of thrombolytic therapy, but from large myocardial infarction.

The presented case was conservatively managed, including thrombolysis, glycoprotein IIb/IIIa inhibitor, and clopidogrel, and the patient had no recurrence of chest pain in a long-term follow-up of two years. Also, control coronary angiography after two years was normal.

**Conclusion**

SCAD dissection is a rare and uncommon cause of acute coronary syndrome that should be considered in young patients, particularly women, which is presented with myocardial infarction. It is known that thrombolytic therapy can

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**Table 1**

<table>
<thead>
<tr>
<th>Case report</th>
<th>Year</th>
<th>Journal</th>
<th>Thrombolytic</th>
<th>Successful</th>
<th>Complication</th>
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<tr>
<td>Leclercq F, et al.</td>
<td>1996</td>
<td>Eur Heart J.</td>
<td>rt-PA</td>
<td>yes</td>
<td>no</td>
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<tr>
<td>S. Narasimhan, et al.</td>
<td>2004</td>
<td>IJTCVS</td>
<td>Streptokinase + rt-PA</td>
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<td>Maeder M, et al.</td>
<td>2005</td>
<td>Intern Journal of Cardiology</td>
<td>rt-PA</td>
<td>no</td>
<td>no</td>
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<tr>
<td>Evangelou D, et al.</td>
<td>2006</td>
<td>Intern Journal of Cardiology</td>
<td>Tenecteplase</td>
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<td>Cano O, et al.</td>
<td>2009</td>
<td>Intern Journal of Cardiology</td>
<td>Tenecteplase</td>
<td>no</td>
<td>?</td>
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<tr>
<td>T. Karaahmet T, et al.</td>
<td>2009</td>
<td>Anadolu Kardiyl Derg</td>
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<td>Saadat H, et al.</td>
<td>2009</td>
<td>Int J Angiol</td>
<td></td>
<td>no</td>
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<td>Andreou AY, et al.</td>
<td>2009</td>
<td>Exp Clin Cardiol</td>
<td></td>
<td>no</td>
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<td>Andreou AY, et al.</td>
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<td>Motreff P, et al.</td>
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<td>Almafragi A, et al.</td>
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<td>Ito H, et al.</td>
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<td>Am J Cardiol</td>
<td></td>
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<td>Hidalgo-Urbano RJ, et al.</td>
<td>2011</td>
<td>Rev Esp Cardiol</td>
<td></td>
<td>no</td>
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<td>Jović Z, et al.</td>
<td>2015</td>
<td>Vojnosanit Pregl (this issue)</td>
<td>rt-PA</td>
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**rt-PA** – recombinant tissue plasminogen activator
lead to complications when applying for SCAD, but there is still no clear evidence of this. In the analysis that we conducted, there was only one case of 18 previously published complications after treating SCAD with thrombolysis. The presented case, like eight others, confirm that good clinical outcomes can be achieved with thrombolysis, glycoprotein IIb/IIIa inhibitors and long-term dual antiplatelet therapy. In spite of all this, we still need more data to verify that thrombolysis does not harm the therapy for SCAD.

REFERENCES


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