Case Report

Optimal treatment modality for coexisting acute myocardial infarction and ischemic stroke

Fatih Gungoren a, Feyzullah Besli a,⁎, Zulkif Tanriverdi a, Ozcan Kocaturk b

a Department of Cardiology, Harran University School of Medicine Hospital, Sanliurfa, Turkey
b Department of Neurology, Harran University School of Medicine Hospital, Sanliurfa, Turkey

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ABSTRACT

The simultaneous occurrence of acute ST-segment elevation myocardial infarction and acute ischemic stroke is an uncommon and complex clinical presentation. Although the medical treatment of both diseases is similar, data regarding optimal reperfusion therapy are limited. Nevertheless, use of tissue plasminogen activator may be a reasonable strategy for treatment of both diseases. We present a rare case of coexisting thrombosis of the coronary artery and mid cerebral artery that was managed successfully with tissue plasminogen activator thrombolytic therapy.

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1. Introduction

Acute myocardial infarction (MI) and ischemic stroke are the leading causes of mortality and morbidity worldwide [1]. Both diseases require prompt diagnosis and treatment since the myocardial cells and neurons rapidly undergo necrosis immediately after arterial thrombotic occlusion [1,2]. The main therapeutic strategy for both diseases is to reduce the infarct size by providing reperfusion to the occluded artery [3]. Timely reperfusion is mandatory for salvage of the ischemic area from irreversible damage. Although percutaneous interventions and tissue plasminogen activator (tPA) have been used as reperfusion strategies for ST-segment elevation MI (STEMI) for a long time, the first guidelines that recommended percutaneous thrombectomy and thrombolysis for acute stroke were published in 2003 [4]. The use of intravenous tPA within 3–4.5 h after the onset of acute ischemic stroke has demonstrated a significant treatment benefit [4]. However, data concerning the optimal reperfusion therapy for cases of coexisting acute STEMI and acute stroke are limited. We present the case of a 69-year-old man who presented to the emergency department with STEMI and ischemic stroke that was successfully treated with tPA.

2. Case presentation

A 69-year-old man with a history of smoking, hypertension, and ischemic stroke 5 years earlier presented to the emergency department with chest discomfort, right-sided hemiplegia and aphasia lasting for 30 min. His blood pressure was 125/70 mm Hg in the right arm and 120/70 mm Hg in the left arm. His pulse rate was 75 bpm, and the pulses were equal in all four extremities. Neurological examination revealed aphasia and right-sided hemiplegia with a muscle strength grade of 0/5 in both the right upper and lower limbs. The patient’s National Institutes of Health Stroke Scale (NIHSS) score was 13 at presentation.

The electrocardiogram revealed ST-segment elevation in the anterior leads (Fig. 1). The chest radiograph showed no widened mediastinum and was otherwise normal. Transthoracic echocardiography revealed anterior wall hypokinesis with a left ventricular ejection fraction (LVEF) of 35% and no intracardiac thrombus. Cerebral computed tomography revealed early signs of occlusion in the territory of the mid cerebral artery and no intracranial hemorrhage (Fig. 2).

The patient was diagnosed with coexisting STEMI and acute ischemic stroke. After a comprehensive evaluation (by a cardiologist and neurologist), intravenous tPA was administered at the dose recommended for acute STEMI (15 mg intravenous bolus, followed by 50 mg infusion over the next 30 min and then 35 mg infusion over the next 60 min) within the first hour of admission. Resolution of the ST-segment elevation was approximately 50% at 90 min after tPA administration on surface electrocardiography (Fig. 1). During the second hour of intravenous thrombolysis, his neurological findings recovered substantially. The patient’s motor deficits were almost completely relieved and his motor function improved to 80% by 24 h. Brain magnetic resonance imaging findings were consistent with subacute ischemic stroke at 48 h (Fig. 3). Coronary angiography revealed critical stenosis of the left anterior descending artery (LAD) (Fig. 4a) and a drug-eluting stent was successfully implanted without complications (Fig. 4b).

On echocardiography, the left ventricular ejection fraction had improved to 45% on day 8. Dual antiplatelet therapy (aspirin plus...
clopidogrel) and anti-ischemic medications (metoprolol and isosorbide-5-mononitrate) were given during hospitalization. The patient was discharged and had almost fully recovered from the hemiplegia and had partially recovered from the aphasia (NIHSS score: 4) by day 10.

3. Discussion

Simultaneous thrombosis of two vascular regions distant from one another, which is called cardiocerebral infarction, is a rare clinical situation and can be synchronous (thrombosis of two vessels simultaneously) or metachronous (thrombosis of one vessel precedes that of the other) [5]. In 2010, Omar et al. [6] described cardiocerebral infarction in a patient with concomitant acute ischemic stroke and acute MI.

The incidence of simultaneous cardiocerebral infarction (0.009%) has rarely been reported in literature [7]. The diagnosis of simultaneous cardiocerebral infarction requires the combined acute onset of a focal neurological deficit and chest pain or evidence of a MI, including electrocardiogram changes or elevated cardiac enzymes [7].

The mechanisms underlying cardiocerebral infarction can be explained as follows: (i) conditions resulting in concurrent cerebral – coronary infarction; (ii) cardiac diseases leading to cerebral infarction; and (iii) brain–heart axis dysregulation or cerebral infarction causing MI [7,8]. Several conditions can result in cardiocerebral infarction, including atrial fibrillation, aortic dissection, and pre-existing intracardiac thrombus. In our patient, there was no atrial fibrillation, aortic dissection, or intracardiac thrombus.

Although the role of primary percutaneous interventions (e.g., endovascular thrombectomy) in acute stroke has become increasingly popular, it is not clear whether the physician should perform percutaneous intervention first in concomitance of STEMI and acute ischemic stroke (primary percutaneous coronary interventions or endovascular thrombectomy) [9]. If percutaneous intervention for either stroke or STEMI only is performed, the remaining untreated event can worsen. Moreover, a history of recent ischemic stroke is considered a contraindication for intravenous tPA in acute STEMI [10].

No clinical study has addressed the decision-making process in such cases; however, thrombolytic therapy may be preferable because it is the standard treatment for acute ischemic stroke in patients who arrive within 4.5 h after onset and it is an alternative treatment to primary percutaneous coronary interventions for STEMI within 12 h after onset [3,11]. However, there is no information on the recommended type, total dose, or duration of thrombolytic therapy required for these cases. Among the variety of thrombolytic agents available, including alteplase (tPA), streptokinase, reteplase, and tenecteplase, tPA is likely the best option as it is the only drug approved for stroke [4]. The total dose and duration of tPA administration differ between STEMI and stroke; 100 mg over 1.5 h during the first 12 h is generally recommended for STEMI, whereas 0.9 mg/kg over 1 h (maximum dose of 90 mg, with 10% of the dose given as an intravenous bolus) is recommended for stroke in first 4.5 h [4,11]. The American Heart Association/American Stroke Association (AHA/ASA) recommends intravenous tPA administration for simultaneous cardiocerebral infarction and followed by percutaneous coronary angioplasty [12]. However, the AHA/ASA did not mention the specific management or dose of tPA according to acute MI subtypes.

A few cases of treatment success have been reported previously. Maciel et al. administered intravenous tPA using a stroke protocol (0.9 mg/kg over 1 h, total dose 80 mg) to a 44-year-old man with concomitant acute inferior MI and stroke [10]. After tPA therapy, their patient’s NIHSS score had improved from 11 to 4. However, there is no information about the cardiac outcomes in this case. Cai et al. [13] also administered intravenous tPA (0.9 mg/kg over 1 h; total dose, 60 mg) to a 59-year-old man presenting with stroke following acute non-STEMI. After tPA therapy, their patient’s neurological symptoms and signs improved substantially; however, this case was not STEMI. They-electively performed coronary angiography 3 months later and implanted a stent in the LAD and two stents in the left circumflex artery. However, since our patient had anterior STEMI and remarkably reduced LVEF, we preferred to administer intravenous 100 mg tPA as

![Fig. 1. Electrocardiography on admission to the hospital showing ST-segment elevation in the anterior leads and ST-segment resolution after intravenous thrombolytic therapy.](image1)

![Fig. 2. Brain computed tomography showing no hemorrhage before intravenous thrombolytic therapy.](image2)
recommended by the STEMI protocol and achieved satisfactory results. The ST segment elevation in the electrocardiogram was resolved and the neurological symptoms improved after tPA therapy in our patient. Therefore, we think that tPA may be given according to the STEMI protocol in patients with simultaneous cardiocerebral infarction especially in anterior STEMI with reduced LVEF.

4. Conclusion

Considering the insufficient data in the literature, treatment decisions for simultaneous STEMI and acute ischemic stroke should be individualized. We believe that in cases of coexisting two-vessel occlusion, thrombolytic therapy in the MI protocol may be a reasonable option. Further study is needed to determine the ideal management modality that provides the best outcome for this rare and devastating clinical condition.

References


