Case Report

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Acute ST-segment elevation myocardial infarction after intravenous thrombolysis in acute cerebral infarction: A case report and Literature review

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Abstract

Background: Acute ischemic stroke is a common emergency in neurology. At present, there are two common treatment methods, intravenous thrombolysis and transcatheter mechanical thrombectomy. However, there are few reports of acute myocardial infarction after intravenous thrombolysis with Alteplase(rt-PA). However, the pathogenesis of acute myocardial infarction after thrombolysis and its relevance to thrombolytic therapy are still unclear. This article combines case reports to illustrate its possible pathogenesis and review of related literature.

Case presentation: This article reports a very rare case of a 48-year-old patient who developed obvious chest tightness and chest pain after thrombolytic therapy. Blood test showed significant increase in troponin-I (cTnI) and creatine kinase-MB isoenzyme (CK-MB). The patient's ECG also showed a junctional escape, with significant elevation of ST-segment in leads II,III,and aVF. Subsequently, coronary angiography confirmed acute total occlusion of the right coronary middle segment, and primary PCI treatment was performed. In the end, the patient was discharged successfully.

Conclusion: The incidence of acute myocardial infarction (AMI) after acute ischemic stroke (AIS) thrombolysis is low, but the mortality rate is very high. Primary PCI is the key to saving patients' lives.

Keywords: acute ischemic stroke; acute myocardial infarction, thrombolytic therapy, aspirin, clopidogrel, bivalirudin

Background

With the establishment of the stroke center, more and more patients with acute ischemic stroke (AIS) are being treated. Ultra-early intravenous thrombolysis is one of the internationally recognized effective treatments. However, there are few reports of acute myocardial infarction after intravenous thrombolysis with Alteplase(rt-PA). However, the pathogenesis of acute myocardial infarction after thrombolysis and its relevance to thrombolytic therapy are still unclear. This article combines case reports to illustrate its possible pathogenesis and review of related literature.

Case presentation

A 48-year-old right-handed man with a history of hypertension and type 2 diabetes mellitus presented to the emergency department with sudden weakness of the left limb for 3 hours. A cranial computed tomography showed no obvious cerebral hemorrhage and lacunar cerebral infarction (Figure 1). The first electrocardiogram was accidentally lost, but the emergency cardiac injury markers, such as troponin-I (cTnI) and creatine kinase-MB isoenzyme (CK-MB) were not abnormal. The patient was diagnosed with AIS (National
Institute of Health Stroke Scale, NIHSS: 8) and received thrombolytic therapy with recombinant human tissue plasminogen activator(rt-PA) within 1 hour after emergency treatment. The process went smoothly, and the NIHSS score was 2 immediately after the thrombolysis. After another hour, the patient developed chest tightness and chest pain. At that moment, the blood pressure of the patient was 134/76 mmHg, with a heart rate of 49
beats per minute. Reexamination of ECG showed sinus bradycardia, ventricular escape, junctional escape, and inferior ST-segment elevation (Figure 2). Repeated biochemical test results showed troponin-I was 2.14 ng/ml and CK-MB rose to 37 U/L. A diagnosis of inferior wall acute myocardial infarction (AMI) was made. A bedside transthoracic echocardiography (TTE) showed no thrombus in the heart cavity. Loading dose of dual antiplatelet drugs with aspirin (300 mg) and clopidogrel (300 mg) was chewed. Considering the hemorrhagic transformation after AIS, we use bivalirudin as an intraoperative anticoagulant. The coronary
angiography in the middle part of the right coronary was com-

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pletely occluded, and a Firebird 3.0*23mm stent was implanted (Figure 3,4). After the operation, the patient's hemodynamics stabilized and were transferred to the intensive care unit for monitoring. Bivalirudin was administered intravenously until 4 hours after procedure, and the dose was adjusted to maintain ACT between 250 and 300 seconds. The patient was discharged with a stable condition on the fifteenth day after admission.

Discussion and conclusions

AMI is a rare complication of intravenous thrombolytic therapy for AIS, with extremely high mortality. In the past, only 12 cases were reported all over the world on the past 15 years (table 1) [1-10]. Previous case reports analyzed the causes of the disease and put forward two possible mechanisms: one is that intravenous thrombolysis may lead to the lysis of the original thrombus in the heart and further embolism of the coronary artery [11]. Previously, systemic embolism secondary to microthrombosis after thrombolysis has been reported. In addition, research has shown that the incidence of coronary embolism is 2.9%, and atrial fibrillation is the most common cause [12]. There is one previously reported case of mural thrombus detected by cardiac ultrasound [1]. The low detection rate may be related to the low sensitivity of cardiac ultrasound. In some cases, cardiac ultrasound was performed after thrombolysis, when thrombus disintegration may have occurred and cannot be detected. Secondly, the secondary procoagulant state after systemic thrombolysis can further aggravate coronary thrombosis [4]. The secondary procoagulant state induced by drugs further causes thrombosis in the originally narrow coronary artery, leading to AMI. According to the anatomical and hemodynamic characteristics of the coronary artery, the left anterior descending artery is the most prone to embolism [1], while the blood flow velocity of the right coronary artery is relatively slow, which is more prone to thrombosis. Moreover, the occluded arteries of 12 patients reported previously were located in the right coronary artery or left anterior descending artery. Mostly, the right coronary artery is the main source of blood supply to the inferior myocardium, and inferior myocardial infarction is more prone to insufficient blood volume, mainly manifested as hypotension and bradycardia, with higher mortality. Among the patients with thrombolysis-related myocardial infarction reported at present, cerebral infarction also occurred in the middle cerebral artery region. The middle cerebral artery supplies the insular cortex, which plays an important role in autonomic nervous regulation, and its involvement is easy to lead to arrhythmia [3]. Arrhythmia may further deteriorate the coronary blood supply. However, it should also be pointed out that cardiogenic cerebral embolism caused by atrial fibrillation cannot be excluded from the causes of cerebral infarction. The simultaneous presence of cardiocerebral embolism may promote the deterioration of the disease. Moreover, the shedding of cardiac thrombus will lead to coronary embolism, which is also one of the possible causes of AMI. Unfortunately, patients with AMI have a sudden onset and fail to bedside echocardiography, so it is difficult to identify mural thrombosis and the possible location of the thrombus. This disease still needs to be differentiated from Takotsubo cardiomyopathy, which is a disease of myocardial damage caused by sympathetic activation and excessive catecholamine release [13]. This disease is easy to occur in postmenopausal women, manifested as chest pain, dyspnea, hypotension and other discomforts. Additionally, ST-T changes can be found in electrocardiogram (ECG), and troponin and brain natriuretic peptides (BNP) may increase, resulting in misdiagnosis as ischemic cardiomyopathy. Echocardiography presents characteristic and transient left ventricular hypokinesia or dyskinesia at the base or apex. Coronary angiography can differentiate it from ischemic cardiomyopathy [14]. About 44% of patients with Takotsubo cardiomyopathy have ST segment elevation without corresponding ST segment depression. In this case, the ECG of the patient showed ST segment elevation in leads II, III and aVF, which was a typical inferior myocardial infarction supplied by the right coronary artery. This patient failed echocardiography or coronary angiography in time, but ECG suggested that it is unlikely to be Takotsubo cardiomyopathy.

The 2018 Chinese Guidelines for AIS proposed that standard intravenous thrombolysis should be carried out in acute cerebral infarction complicated with AMI, and bridging with coronary angiography and emergency stent implantation should be performed according to the condition [15]. However, according to the previous literature, 7 of the 12 patients underwent primary PCI immediately after the detection of myocardial infarction, and 3 patients survived after PCI. Although the survival rate of primary PCI was only 42.9%, all patients who failed to surgery died. Therefore, primary PCI may be the best treatment to save such patients. Nevertheless, hemorrhagic transformation after thrombolysis in AIS is another problem that we have to face. Usually, coronary intervention needs unfractionated heparin for anticoagulation, which increases the risk of hemorrhagic transformation. Therefore, during the procedure, we chose bivalirudin for anticoagulation and monitored activated clotting time (ACT) to maintain ACT between 250 s and 300 s. After the PCI, we also proved that bivalirudin was safe and effective in coronary intervention after thrombolysis in AIS.

After intravenous thrombolysis in acute stroke, AMI is a very rare complication with extremely high mortality of ultra-early intravenous thrombolysis. However, it should be pointed out that the correlation between intravenous thrombolysis and myocardial infarction still needs further study and data analysis with large sample size. Because of its low incidence and high mortality, it is difficult for clinical prevention in advance. However, the onset of AMI after thrombolysis is very rapid (13 relevant cases were statistically analyzed in this study, all of those presented onset within 4 h after thrombolysis). Based on this case and literature reports, we also suggest that patients with previous coronary artery diseases or stent implantation should receive a cardiac ultrasound and dynamic ob-

Clinical Imaging and Case Reports (ISSN 2770-9205)

servation of myocardial damage markers as soon as possible while thrombolysis, which may help to early detect the changes in the condition. Active emergency coronary intervention after myocardial infarction may be the best choice to save the lives of the patients

Table 1

Age/sex	Previous coronary Artery stent implant	criminal vascular	Stroke territory	MI territory	Presentation	Onset time when t-PA	Thrombus	primary PCI	Outcome	Author
62/F	No	LAD	Right MCA	Anterior wall	Hypotension	3h	Yes	Yes	Dead	Meissner W[1]
88/F	No	Unknown	Possible Right MCA	Unknown	Chest pain Hypotension	70min	Unknown	No	Dead	Fitzek S[2]
65/F	No	RCA	Right MCA	Inferior wall	Hypotension bradycardia	75min	No	No	Dead	Mehdiratta M[3]
81/F	No	RCA	Right MCA	Inferior wall	Hypotension bradycardia	70min	No	Yes	Dead	Mehdiratta M[3]
78/M	Unknown	Unknown	Right MCA	Inferolateral wall	Chest pain Hypotension	3h	Unknown	No	Dead	Sweta A[4]
58/F	Unknown	Unknown	Left MCA	Anterior and lateral wall	Hypotension bradycardia	3h	No	No	Dead	Sweta A[4]
70/M	Yes	RCA	Left ICA	Inferior wall	Hypotension bradycardia	4h	No	Yes	Survived	Wallace EL[5]
78/M	Unknown	RCA	Right MCA	Inferior wall	Chest pain Palpitation	4h	No	Yes	Survived	Almasi M[6]
79/M	No	RCA	Left MCA	Inferior wall	Chest pain hypotension bradycardia	15min	No	Yes	Survived	Yang CJ[7]
75/F	No	LAD	Right MCA	Anterior wall	Chest pain	15min	No	Yes	Dead	Mannino M[8]
87/F	Unknown	RCA	Right MCA	Anterior wall	Chest pain	100min	No	Yes	Dead	Manea MM[9]
71/F	Yes	RCA	Left MCA	Inferior wall	Chest pain sweat	3h	No	No	Dead	Hang J[10]
47/M	No	RCA	Right MCA	Inferior wall	Chest pain Palpitation	1h	No	Yes	Survived	Present case

Clinical Imaging and Case Reports (ISSN 2770-9205)

Figure 1 Emergency cranial CT showed no obvious abnormality, suspected right middle cerebral artery high density sign

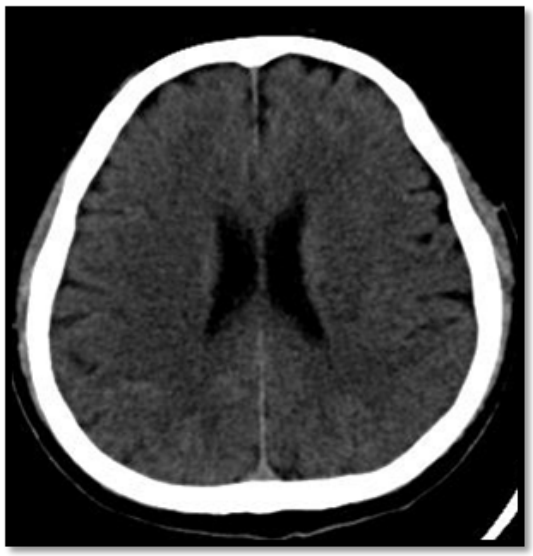


Figure 2 The ECG showed sinus bradycardia, ventricular escape, junctional escape, and ST-segment elevation in lead II, III and aVF

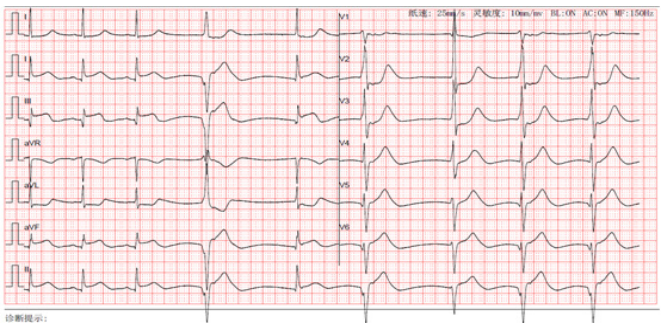


Figure 3 The middle segment of RCA was completely occluded (red arrow)

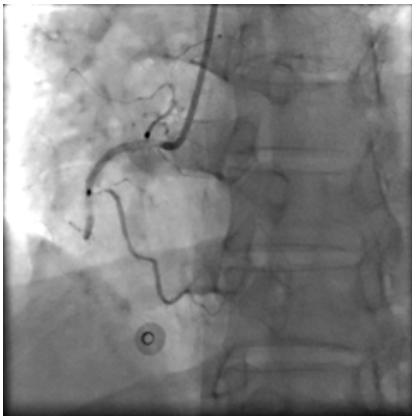
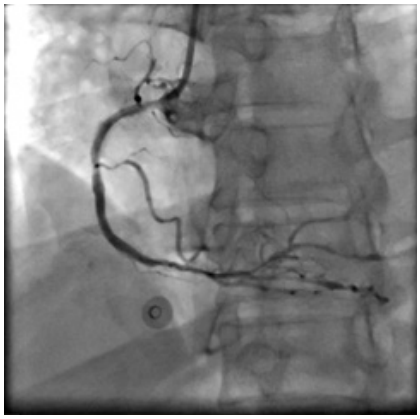


Figure 4 The results of one stent was implanted in RCA.



Abbreviations

AIS: Acute ischemic stroke ECG: Electrocardiogram; CT: Computed tomography; AMI: Acute myocardial infarction; PCI: Percutaneous coronary intervention; CK-MB: Creatine kinase-MB isoenzyme; LAD: Left-anterior descending; RCA: Right coronary artery; ACT: Activated clotting time; MCA: Middle cerebral artery; ICA: Internal carotid artery

Ethics approval and consent to participate

Not applicable.

Consent for publication

Informed consent for the publication of the case report was obtained from the patient in written form.

Availability of data and material

The data analyzed in the case report are not publicly available due to the privacy policy of the hospital, but are available from the corresponding author upon reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

CY and FLZ, the co-first authors, performed the diagnostic coronary angiography for the patient and wrote the manuscript. CY was also the corresponding author. YSK performed the percutaneous coronary intervention. HGW, performed the percutaneous coronary intervention for the patient. CF wrote the manuscript, and made the illustrations. All authors

read and approved the final manuscript. All authors agreed to their contribution.

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References

1. Meissner W, Lempert T, Saeuberlich-Knigge S, et al. Fatal embolic myocardial infarction after systemic thrombolysis for stroke. Cerebrovasc Dis. 2006;22(2-3):213-214.

2. Fitzek S, Fitzek C. A Myocardial Infarction During Intravenous Recombinant Tissue Plasminogen Activator Infusion for Evolving Ischemic Stroke. Neurologist. 2015;20(3):46-47.

3. Mehdiratta M, Murphy C, Al-Harthi A, Teal PA. Myocardial infarction following t-PA for acute stroke. Can J Neurol Sci. 2007;34(4):417-420.

4. Sweta A, Sejal S, Prakash S, et al. Acute myocardial infarction following intravenous tissue plasminogen activator for acute ischemic stroke: An unknown danger. Ann Indian Acad Neurol. 2010;13(1):64-66.

5. Wallace EL, Smyth SS. Spontaneous coronary thrombosis following thrombolytic therapy for acute cardiovascular accident and stroke: a case study. J Thromb Thrombolysis. 2012;34(4):548-551.

6. Almasi M, Razmeh S, Habibi AH, Rezaee AH. Does Intravenous Administration of Recombinant Tissue Plasminogen Activator for Ischemic Stroke can Cause Inferior Myocardial Infarction?. Neurol Int. 2016;8(2):6617. Published 2016 Jun 29.

7. Yang CJ, Chen PC, Lin CS, et al. Thrombolytic therapy-associated acute myocardial infarction in patients with acute ischemic stroke: A treatment dilemma. Am J Emerg Med. 2017;35(5):804.e1-804.e3.

8. Mannino M, Asciutto S, Terruso V, et al. Myocardial Infarction Following Intravenous Thrombolysis for Acute Ischemic Stroke: Case Report and Literature Review. J Stroke Cerebrovasc Dis, 2017, 26(6): e105-e107.

9. Manea MM, Dragoş D, Stoica E, et al. Early ST-segment elevation acute myocardial infarction after thrombolytic therapy for acute ischemic stroke: A case report. Medicine, 2018, 97(50): e13347.

10. Jin H, Chen BY, Sun X, et al.Acute myocardial infarction after intravenous thrombolysis: a case report and literature review. Chin J Cerebrovasc Dis,2020,14(2):e117-e120.

11. Tanaka K, Ohara T, Ishigami A, et al. Fatal Multiple Systemic Emboli after Intravenous Thrombolysis for Cardioembolic Stroke. J Stroke Cerebrovasc Dis, 2014, 23(2): 395-397.

12. Shibata T, Kawakami S, Noguchi T, et al. Prevalence, clinical features, and prognosis of acute myocardial infarction attributable to coronary artery embolism [J]. Circulation, 2015, 132(4): 241-250.

13. Hajsadeghi S, Rahbar MH, Iranpour A, et al. Dobutamine-in-duced takotsubo cardiomyopathy: a systematic review of the Literature and case report. Anatol J Cardiol, 2018, 19(6): 412-416.

14. Ali A, Ahmad MQ, Malik MB, et al. Neurogenic stunned myocardium: a literature review. Cureus, 2018, 10(8): e3129.

15. Chinese Society of Neurology, Chinese Stroke Society. Chinese guidelines for diagnosis and treatment of acute ischemic stroke 2018.J Neurol, 2018, 51(9): 666-682.