

Acute Inferior ST Elevation Myocardial Infarction (STEMI) After Thrombolytic Therapy for Acute Ischemic Stroke (A case report)

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Abstract

Intravenous thrombolysis is the mainstay of treatment for acute ischemic stroke, provided that treatment is initiated within 3 to 4.5 hours of clearly defined symptom onset. In literature there are many complications of recombinant tissue plasminogen activator (rtPA) as hemorrhagic events or allergic reactions. Acute myocardial infarction is a rare complication of thrombolytic agent with an unknown incidence. We present the case of a 61-year-old male developed inferior & posterior ST elevation myocardial infarction (STEMI) after successful thrombolysed acute ischemic stroke (AIS). He was treated by primary percutaneous coronary intervention (PCI) to totally occluded RCA that successfully recanalized. The case demonstrates incidence of STEMI after stroke thrombolysis. Although, mechanisms are still unclear but there have been a number of proposed mechanisms for post-rtPA thromboembolic phenomenon including disruption of unstable plaque of pre-existing mural or intracardiac clot. Our reported case shed light on the association between fibrinolytic therapy and ischemic stroke.

Keywords:

Acute ischemic stroke, Thrombolytic therapy, Acute inferior STEMI, Case report, Emboli, recombinant tissue plasminogen activator (rtPA)

Background:

Coronary artery disease (CAD) and cerebrovascular disease (CVD) are sharing same common risk factors as age, DM, HTN, dyslipidemia and cigarette smoking. Acute atherosclerotic cardiovascular events have the same management with possible subsequent complications. Intravenous thrombolysis with recombinant tissue plasminogen activator (rtPA) is the first line treatment in patients with acute ischemic stroke within 3 to 4.5 hours from onset in selected patients. The main target of thrombolysis is the recanalization which has been associated with improved outcomes and reduced mortality. The risks and benefits of treatment outcomes are related to patient and clot-specific factors that are assessed in many established prognostic scores, the most famous one is National Institutes of Health Stroke Scale (NIHSS). Clot features such as size, location, source and composition are considered to influence outcomes. Patient factors such as age, diabetes, pre-existing intracranial pathology, infarct volume on CT, surgery or trauma as well as degree of neurological deficit can suspect outcome. All of these prognostic scores do not assess the suspected acute ischemic cardiovascular event risk.

In literature there are many complications of rtPA as hemorrhagic events or allergic reactions. Only a few cases of acute myocardial infarction are reported as post thrombolytic

complication, and the mechanisms are still unclear but There have been a number of proposed mechanisms including atherosclerotic plaque disruption and thromboembolism.

Case presentation:

A 61-year-old man presented to the emergency department with facial drooping, left-sided weakness, and numbness that occurred 1 hour before admission. He had medical history of hypertension and dyslipidemia, but no other neurological symptoms.

On admission, his vital signs were stable, and his electrocardiogram (ECG) revealed a normal sinus rhythm with a heart rate of 75 bpm (Figure 1A). Initial blood testing was unremarkable with normal complete blood count, renal function, and coagulation. His cardiac troponin level was 0.01 ng/mL (0.00–0.4 ng/L).

Two hours after the occurrence of symptoms, he was diagnosed with stroke and his National Institutes of Health Stroke Scale score was 9. After excluding hemorrhage on CT, we administered thrombolytic therapy with tissue plasminogen activator intravenously at 0.9 mg/kg body weight according to standard protocol (total dose 90 mg infused over 60 minutes, with 10% of the total dose administered as an initial bolus over 1 minute).

Two hours after the completion of the recombinant tissue plasminogen activator (rtPA) infusion, the patient developed central chest pain with acute inferio-posterior right ventricular persistent ST elevation myocardial infarction on ECG (Figure 1B). Five minute later, patient develop VT which successfully cardioverted by 1 DC shock (200 J). At this time, his cardiac troponin levels were 17 ng/mL and 20 ng/mL two and six hours later.

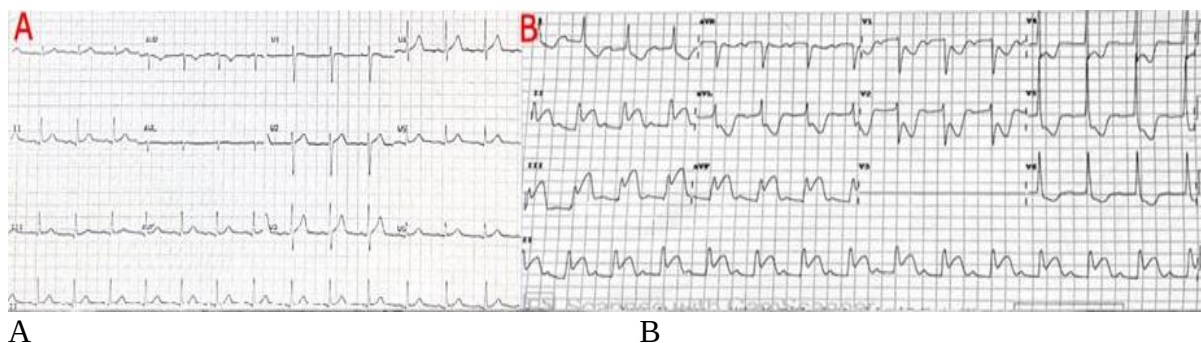


Figure 1A: 12 lead ECG at admission with no ischemic changes. **1B:** 12 lead ECG show ST elevation in lead II, III, aVF with ST depression & T wave inversion in V1-V6, I, and aVL.

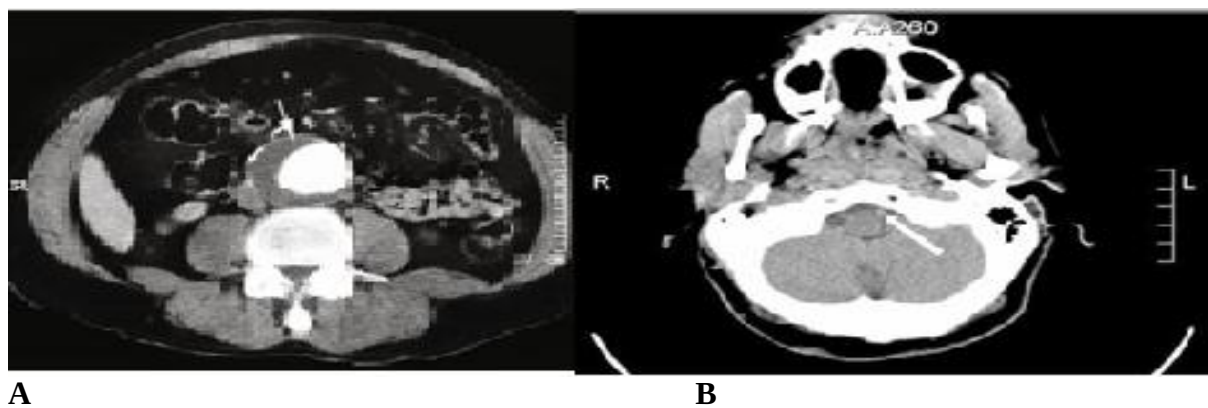


Fig. 2; CT angiogram shows atheromatous plaque of abdominal aorta (A), CT brain

shows right vertebral artery occlusion with no haemorrhage(B).

In the meantime, an urgent computed tomography (CT) brain (Figure 2B) and CT aortogram (Figure 2A) were performed to exclude vascular or pericardial hemorrhage, prior shifting for primary coronary intervention(PCI).

Pre-angiogram transthoracic echocardiography (Figure 4A) demonstrated normal left ventricular ejection fraction of 60% with hypokinetic inferior and posterior walls and mild eccentric mitral incompetence, no mural thrombus was seen.

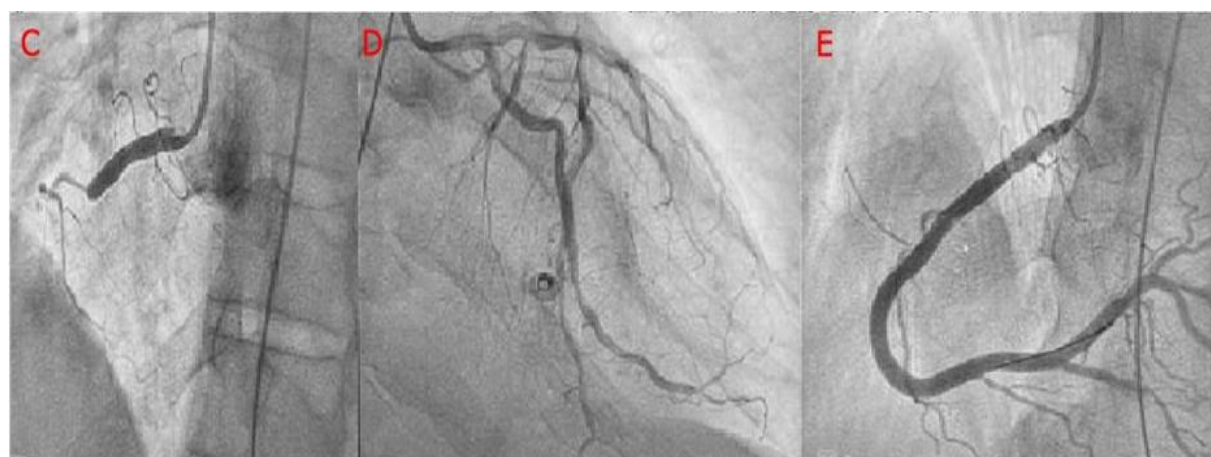


A

B

Fig.4; TTE shows normal LV with no intracardiac thrombi or shunts in apical four chamber view (A) TEE shows no mural, left atrial appendage thrombi or intracardiac shunts (B).

Trans-femoral coronary angiogram revealed total occluded right coronary artery and atherosclerotic left system with non-significant lesion. After wiring of RCA and gentle low pressure balloon pre dilatation, mid segment subtotal RCA lesion appeared and one DES 4.00x38 mm was deployed with final TIMI 3 flow and no distal embolization (Figure 3C-E).



C

D

E

Fig.3; Right & left coronary angiogram show totally occluded RCA(C), Atherosclerotic

left system angiography with non-significant lesion (D), Post successful PCI to RCA (E).

Thromboembolic events were excluded by Transesophageal echocardiogram (TEE) with contrast (Figure 4B), carotid artery duplex and CT aortogram(2A).

The patient was discharged after 5 days with no further adverse events. One month later, the patient fully independent with no neurological deficit.

Discussion:

We present a case of acute inferior ST elevation myocardial infarction (STEMI) after thrombolytic therapy for acute ischemic stroke. The onset of STEMI as a complication of thrombolysis is rare but noted before.

The normal ECG and troponin values at admission were strong indicators that the STEMI did not precede stroke onset. The earliest onset (2h) of myocardial infarction after fibrinolytic treatment for AIS was reported in a few published relevant cases is 2 hours [5-9]. Manea et al. [4] suggested in their clinical case, two possible pathogenic mechanisms for the development of STEMI immediately after thrombolytic therapy for acute ischemic stroke [4].

One is cardioembolic STEMI due to fragmentation of a pre-existent intracardiac thrombus induced by thrombolytic treatment [4]. In a large study, [10] the prevalence of coronary artery embolism was 2.9%, with atrial fibrillation (AF) as the most common cause (73% of cases) and multivessel embolism occurring in 15% of the patients. AF was associated with an ≈ 2 -fold increased risk of MI in other studies, partly because of the embolic risk, and also because of an inflammation-associated prothrombotic state [11]. In our case, we exclude embolic event by Transesophageal echocardiogram (TEE) with contrast, carotid artery duplex and CT aortogram.

The second possible mechanism of STEMI is local thrombosis on an unstable atheroma plaque in a stenotic coronary artery [4]. An unexpected prothrombotic status has been recently posited to arise immediately after rtPA therapy, and this could further promote coronary thrombosis. The free plasmin formed as a result of the systemic lytic state can activate contact factors, factor V, and possibly prothrombin; the thrombin bound to fibrin is progressively exposed as the clot undergoes lysis and has the potential to locally activate platelets and accelerate coagulation.[13] This prothrombotic status supposedly induced by rtPA administration could partly explain the myocardial reinfarction that occurs in 3% to 6% of patients, despite successful coronary thrombolysis[12] or arterial reocclusion with early neurological deterioration in 14% to 34% of thrombolysed stroke patients.[12,14] .

In the ARTIS trial, [15] early administration of aspirin in patients with AIS treated with alteplase not only failed to improve the outcome at 3 months but also increased the risk of SICH. Hence, it has been argued that a potentially threatening therapy, such as thrombolysis, should not be used unless a complete evaluation, including vascular imaging, has been obtained. However, in the acute setting, the neurologist must balance the pressure of time against the need for additional diagnostic testing.

Conclusion:

Intravenous thrombolysis is the mainstay of treatment for acute ischemic stroke but The STEMI could be a rare complication of stroke thrombolysis with an early onset after i.v. rtPA

administration (<2hour in our patient) and if confirmed, it should trigger rapid initiation of coronary revascularization procedures.

Some recent small-scale trials investigated the association of administration of some antithrombotics in the first 24hours after thrombolysis as adjuncts to rtPA treatment and the prevention of some early lethal or disabling events, such as reocclusion of the coronary or cerebral arteries after successful recanalization.so, we need to highlight the importance of further research on the early initiation of antiplatelet with rtPA to prevent their pro thrombotic effect.

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The authors do not acknowledgment any further contributions to this manuscript.

Compliance with ethical standards

Conflict of interest

The authors declare that there is no conflict of interest regarding this article.

Research involving human and/or animal right

There was no research involved animals performed by any author. All procedures performed in human participants were approved by the ethical standards of the institutional committee and in accordance with the 1964 Helsinki Declaration and its later amendments.

Informed consent

Informed consent was obtained from the patient included in the study.

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