

A Case of Acute Myocardial Infarction 24 hours after Acute Ischaemic Stroke

Dear Editor,

It is well known that thrombolysis for acute myocardial infarction (AMI) may lead to disintegration of pre-existing intracardiac thrombi with subsequent cerebral embolisation. It appears plausible that the same mechanism could apply to patients with cardiac or arterial thrombi who are undergoing intravenous thrombolysis for acute ischaemic stroke. AMI immediately following tissue plasminogen activator (t-PA) treatment for stroke is a rare but serious complication. The disruption of intracardiac thrombus and subsequent embolisation to the coronary arteries may be an important mechanism in the development of AMI after t-PA treatment for acute ischaemic stroke.¹ However, the occurrence of AMI 24 hours after presentation with ischaemic stroke in patients with no apparent source of emboli might have a different mechanism other than thromboembolic mechanism. Concomitant plaque instability in coronary and carotid vascular beds may provide a possible mechanism.

We describe a case of AMI which occurred 24 hours after presentation with acute ischaemic stroke, for which intravenous t-PA was administered.

Case Report

A 57-year-old woman with a history of hypertension presented to our tertiary facilities with acute onset of left sided weakness and numbness. The patient initially experienced an attack of chest pain associated with shortness of breath and sweating which resolved spontaneously. She reported similar attacks of chest pain with less severity in the last few months prior to presentation. Ten minutes after resolution of the chest pain, the patient developed left sided weakness and numbness with no dysarthria. On admission, the patient was fully conscious with unsteady gait. Her NIH stroke scale was 8. Emergency computed tomographic (CT) scans of the brain was normal. Intravenous Alteplase was given and the patient had stationary course. However, 24 hours after the initial presentation, she developed severe chest pain and her electrocardiogram was compatible with acute inferior myocardial infarction (ST segment elevation in leads II, III, and aVF).

Emergency coronary angiography showed thrombotic subtotal occlusion of the right coronary artery (RCA) at its mid segment (Fig. 1A) with 2 significant lesions at the

proximal and mid thirds of the left anterior descending artery of 80% and 75% of stenosis, respectively, and a distal 70% stenosis at the distal left circumflex artery. Primary percutaneous coronary intervention (PCI) was successfully performed for the culprit lesion in the RCA with optimum results after balloon angioplasty and stenting (Fig. 1B) and the patient had subsequent uneventful course.

Laboratory data showed normal baseline troponin-I and creatine kinase MB with subsequent elevation during the second day of presentation, high homocysteine level, elevated high sensitivity C-reactive protein (hs-CRP) measuring 13.99 mg/L and high levels of total and LDL-cholesterols with high serum triglycerides.

Transthoracic 2D-echocardiography showed mild hypokinesia of the inferior wall. The left ventricular systolic function was normal with no intracardiac thrombi.

Magnetic resonance imaging of the brain revealed small recent infarction lesions over the left high frontal and parietal areas and right basal ganglia (Figs. 1C, 1D and 1E). The patient had an uneventful hospital course and was discharged in stable condition.

Discussion

In the current case, the possibility of development of acute coronary syndrome manifested by chest pain 10 minutes before the presentation of stroke does not reject the hypothesis of panvascular plaque activation. On the other hand it may support it. Moreover, the levels of troponin I and creatine kinase MB were normal during the first day of presentation, and this can exclude the occurrence of AMI before the onset of stroke.

It has been reported that disruption of intracardiac thrombus with subsequent embolisation to the coronary arteries may represent another possible mechanism in the development of immediate AMI after t-PA treatment for acute ischaemic stroke.¹

In this case, the possibility of development of ischaemic stroke secondary to slowly evolving AMI with formation of cardiac thrombi and subsequent cerebral embolisation is unlikely due to the inferior location of the infarction with normal left ventricular systolic function.²

Moreover, transthoracic 2D-echocardiography ruled

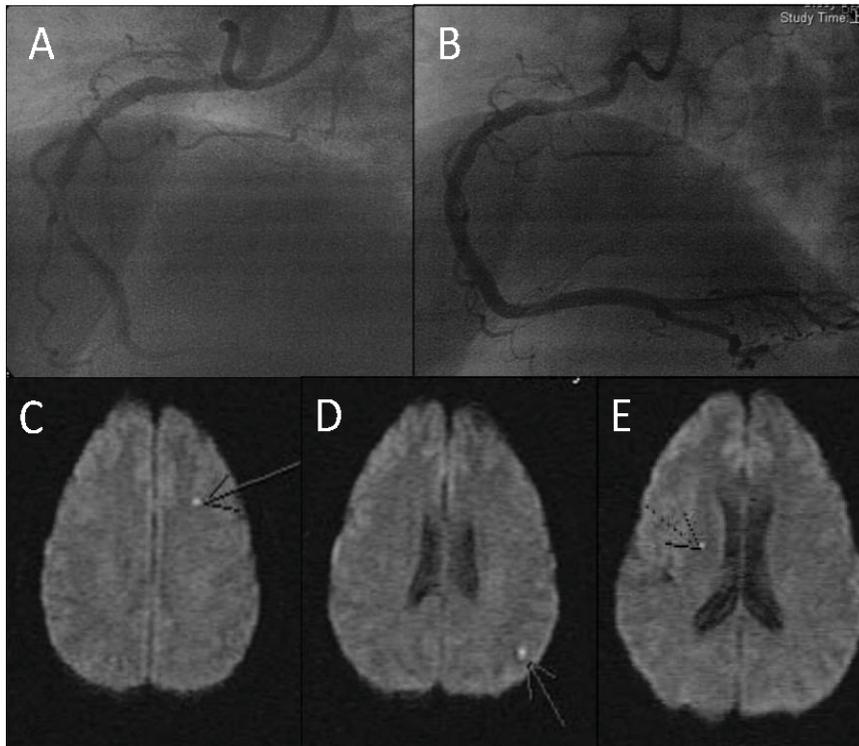


Fig. 1 Coronary angiogram of the RCA at the left anterior oblique view with thrombotic subtotal occlusion at its mid segment (Panel A). Final angiogram showing full recanalization of RCA by primary percutaneous coronary intervention (Panel B). Brain MRI showing small recent infarction lesions over the left high frontal (Panel C), parietal areas (Panel D) and right basal ganglia (Panel E).

out large intracardiac thrombi and excluded the possibility of cardioembolic mechanism for the development of the ischaemic stroke. However, transthoracic echocardiography has a limited ability to exclude the presence of patent foramen ovale and interatrial septal aneurysm which can cause cryptogenic stroke as a diagnosis of exclusion that is impossible to verify.

This supports the hypothesis that panvascular plaque activation in the carotid and coronary vascular beds may provide an explanation for the development of AMI in patients with acute stroke.

The same mechanism was presumed in patients with AMI with subsequent cerebral ischaemic events, based on the finding that the risk of stroke during the acute phase of AMI was similar in patients with evidence of ventricular thrombus compared to those without it.³

In the European Carotid Surgery Trial,⁴ among patients with cerebrovascular accidents, those with angiographically irregular carotid plaques were more likely to have subsequent acute coronary events compared with patients having smooth carotid plaques.

It was hypothesised that plaque instability is influenced by systemic factors such as inflammation, infection, autoimmunity or genes that are present in certain individuals. This puts the atherosclerotic plaques of the entire circulation

at an increased risk of this phenomenon.⁴

It was not surprising that the level of hs-CRP was markedly elevated in the present case, providing evidence of systemic inflammation and a possible link between carotid and coronary plaque instability. This inflammatory link may have relevant implications for understanding the mechanisms of acute widespread atherothrombotic plaque inflammation.⁵

Conclusion

AMI following acute ischaemic stroke can be explained by a different mechanism other than disintegration of intracardiac or intra-arterial thrombi by thrombolytic therapy with subsequent coronary embolisation. The sequence of events in our case supports the hypothesis of panvascular plaque activation in the carotid and coronary arteries as an alternative mechanism.

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