ST-ELEVATION MYOCARDIAL INFARCTION FOLLOWING THROMBOLYSIS FOR ACUTE STROKE: A CASE REPORT

Bo Wang  Hitesh Patel*  Tom Snow  Ed Leatham

ABSTRACT

Diseases of the coronary and cerebral vasculature share common aetiologies and hence are often linked. Whereas ischaemic stroke following an acute myocardial infarction is relatively common, the converse is much rarer. We present a case of a 69 year old patient with a ST-elevation myocardial infarction three days after systemic thrombolysis for acute ischaemic stroke. We discuss the possible underlying aetologies and the necessary investigations, and highlight the possibility of thrombosis as a paradoxical complication of thrombolysis.

Keywords: ST elevation myocardial infarction; Acute stroke; Thrombolysis; Prothrombotic state.

KEY POINTS

- ST elevation myocardial infarction following an acute stroke is not commonly seen and an underlying aetiology should be investigated for from several possible differentials.
- A prothrombotic state exists after systemic thrombolysis that can contribute to complications and impacts upon management.
- Glycoprotein IIb/IIIa inhibitor can be considered in patients with ischaemic cardiac chest pain despite recent thrombolysis for stroke.

CASE HISTORY

A 69 year old woman presented with a one hour history of right-sided weakness and dysphasia and had clinical findings suggestive of a stroke. Computed tomography (CT) scan of the head suggested a left middle cerebral artery territory ischaemic stroke. She was thrombolysed in accordance with the NINDS guidelines with 63mg of Alteplase, a recombinant tissue plasminogen activator.

* Hitesh Patel, Royal Surrey County Hospital, Guildford GU2 7XX, UK. E-mail: dochiteshpatel@hotmail.com
plasminogen activator, two hours after onset of symptoms. Admission electrocardiogram showed normal sinus rhythm with left axis deviation only (figure 1).[1]

Her past medical history included angina, for which she had a 3.5x12mm bare metal coronary stent inserted into a dominant left circumflex artery 13 months ago. She also underwent open biological aortic valve replacement with a 23mm Perimount valve for mixed aortic valve disease 2 months prior to this admission. Other medical problems included bronchiectasis, anaemia of unknown origin and depression. Her medications on admission consisted of aspirin 75mg once a day, bisoprolol 1.25mg once a day, amiodarone 200mg once a day (started after valve replacement to maintain sinus rhythm), simvastatin 20mg once a day, ferrous sulphate 200mg three times a day, omeprazole 40mg once a day.

The patient initially showed a good response with NIHSS (National Institute of Health stroke scale) score improving from 10 to 7. On the third day post stroke thrombolysis, she suddenly developed severe central chest pain. ECG showed significant ST elevation in anterior and inferior leads (figure 2). Whilst being assessed for chest pain, the patient arrested with an underlying rhythm of ventricular fibrillation. This was promptly defibrillated and the patient regained cardiac output within one cycle of cardio-pulmonary resuscitation by which stage there was resolution of the ST segment changes and chest pain.

The patient was admitted to the coronary care unit and was loaded with 300mg clopidogrel and was started on a Tirofiban (glycoprotein IIb/IIIa inhibitor) infusion. Aspirin was continued at 75mg once a day. A low-molecular-weight heparin was not given, despite the protocol for acute coronary syndrome, due to the increased risk of intracerebral haemorrhage with recent systemic thrombolysis. Troponin I at 12 hours was elevated at 18.41 and creatine kinase was 889. ECG four hours after the onset of chest pain showed Q waves in inferior leads and deep T wave inversion in the anterior leads (figure 3).

The patient was transferred to the local angioplasty referral hospital the following morning. A coronary angiogram showed unobstructed coronary arteries and a patent stent within a dominant left circumflex artery. Transoesophageal echocardiography (TOE) showed did not show any thrombus within the cardiac chambers. Saline contrast (bubble) echocardiography with Valsalva release did not reveal any intracardiac shunts.

The patient was transferred back to our hospital and eventually rehabilitated well from both events and was discharged to the community. Prior to discharge, she was anticoagulated with warfarin in addition to aspirin, a combination on which she still remains.
DISCUSSION

The incidence of acute ischaemic stroke after myocardial infarction (MI) has been shown to be around 2%.[2] Amongst other shared risk factors such as hypertension, diabetes mellitus and smoking, atrial fibrillation is the single most important risk factor and it occurs in up to 20% of patients after a ST-elevation myocardial infarction (STEMI)[3]. Poor ventricular function after a recent infarct with atrial fibrillation predispose to the formation of mural thrombi, which then becomes a source of emboli to the cerebral vasculature.

The incidence of acute MI following a stroke is not known but there have been other case reports[4,5,6]. The pathogenesis in all these cases was thought to be disruption of a cardiac thrombus, which gave rise to an embolic stroke and an embolic MI. 26% of patients presenting with acute stroke or transient ischaemic attacks have intra-cardiac thrombus[7,8]. Embolic myocardial infarction, although rare, has been described elsewhere in the context of atrial myxoma, aortic valve endocarditis, dilated cardiomyopathy and atrial septal defects (ASD)[4,6]. In this case however, no definitive evidence for such an embolic aetiology was found. Therefore the authors had to consider several possible differentials (Table I). In managing this patient, each differential had to be considered and investigated for, accordingly.

De novo thrombus in this patient is raised in the context of recent systemic thrombolysis. A paradoxical prothrombotic state exists after thrombolysis due to the selective action of tissue-plasminogen activators on fibrin degradation, leaving thrombin exposed and thus resulting in increased platelet aggregation.[10] Studies have also shown a transient but significant increase in total thrombin levels after thrombolysis, possibly via direct stimulation of the coagulation cascade by plasminogen activators.[11] This may explain the good evidence for early use of heparin and antiplatelets such as aspirin and clopidogrel after thrombolysis for STEMI for preventing vessel reocclusion.[12,13,14]

Management of patients after stroke thrombolysis is different from patients after STEMI thrombolysis. Early anticoagulant and antiplatelet therapy is not recommended after stroke thrombolysis due to the associated risk of intracerebral haemorrhage. However, in stroke as in MI, there is a significant reocclusion rate, estimated at 13%.[1] Recently, work has been directed into evaluating any possible benefit of early antiplatelet therapy. Some early trials have shown that patients taking aspirin long-term show better outcomes when thrombolysed for acute stroke.[15] Further to this, a large randomised controlled trial is currently being carried out looking at early use of antiplatelet agents with stroke thrombolysis.[16]

We thought that out of the differential mechanisms discussed in table 1, the most likely for our patient were: 1. Embolisation of existing thrombus following thrombolysis ; 2. De novo thrombus formation in the pro-coagulant state following thrombolysis. Neither of these theories could be proven despite extensive investigations. On balance of probability we started
anticoagulation with warfarin to reduce the risk of further potential thromboembolic events.

CONCLUSION

Acute STEMI after a recently thrombolysed ischaemic stroke is not a frequent event, but one that requires decisive treatment balancing the risk - benefit of relieving a coronary obstruction to that of intracerebral haemorrhage. We have reported a successful outcome following early use of a glycoprotein IIb/IIIa inhibitor whilst awaiting transfer for urgent coronary angiography. It is important to consider aetiological investigations in the subsequent management of these patients including TOE and saline contrast echocardiography as this would alter management.

Figure 1. ECG on admission
Figure 2. ECG at onset of chest pain

Figure 3. ECG at resolution of chest pain and after GpIIb/IIIa inhibitor
Table I. Possible pathogenic mechanisms

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>For</th>
<th>Against</th>
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<tr>
<td>Mural thrombus leading to embolic stroke then MI</td>
<td>Recent cardiac surgery; post-operative atrial fibrillation</td>
<td>No thrombus seen on TOE (95% sensitive)</td>
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<tr>
<td>De novo thrombus in coronary artery following thrombolysis</td>
<td>Pre-existing ischaemic heart disease; prothrombotic state after systemic thrombolysis</td>
<td>Normal coronary angiogram</td>
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<td>Stent thrombosis following thrombolysis</td>
<td>Bare metal stent thrombosis likely; prothrombotic state after systemic thrombolysis</td>
<td>Normal coronary angiogram; ST elevation distribution not in keeping with stent position</td>
</tr>
<tr>
<td>Paradoxical emboli through an ASD causing stroke then MI</td>
<td>Unlikely</td>
<td>No ASD seen on TOE/Bubble echo</td>
</tr>
<tr>
<td>Coronary artery spasm</td>
<td>Spontaneous resolution; normal coronary angiogram</td>
<td>Persistent ischaemic changes on ECG; cardiac-specific markers elevated</td>
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REFERENCES


