CASE REPORT

Stroke thrombolysis complicated by ST elevation myocardial infarction (STEMI)

Mark Flower, James Harvey, Jonathan Hunter

Department of Medicine,
Redcliffe Hospital, Redcliffe,
Queensland, Australia

Correspondence to

Dr Mark Flower,
mark.flower@uqconnect.edu.au

Accepted 9 February 2018

SUMMARY

An 84-year-old male Jehovah's Witness presented to the emergency department 1 hour after onset of left facial droop and left upper limb weakness. Thrombolytic stroke treatment was commenced as per local thrombolytic protocol with intravenous recombinant tissue plasminogen activator (rtPA) at 2 hours and 25 min following onset of symptoms. Almost immediately after rtPA infusion the patient reported chest pain and had ECG changes consistent with a diagnosis of anterior ST elevation myocardial infarction. At angiogram, a graft study showed severe native coronary artery disease. The left internal mammary artery graft was patent to the left anterior descending artery (LAD); however, the apical LAD was occluded, with the appearance suggestive of embolic occlusion.

BACKGROUND

Current evidence supports the use of recombinant tissue plasminogen activator (rtPA) as the standard of care for acute stroke in selected patients. The goal of thrombolysis is recanalisation 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. Trial and registry data have demonstrated that administration of treatment is

time critical. Delay to initiation of treatment is associated with both reductions in benefit and increased risk of complications. Clot features such as size, location, source and composition are also considered to influence outcomes. Patient factors which have been associated with poorer outcome include increasing age, diabetes, pre-existing intracranial pathology, infarct volume on CT, recent myocardial infarction, surgery or trauma as well as degree of neurological deficit as calculated using the National Institutes of Health Stroke Scale (NIHSS).¹

The most well-documented risks pertain to bleeding complications, the most feared of which is intracranial haemorrhage.² With increasing clinical experience of rtPA, rarer complications have also been identified. Notable among these are anaphylactic reactions and embolic phenomenon.³ There have been a number of proposed mechanisms for post-rtPA thromboembolic phenomenon including hypercoagulable state after lysis, disruption of unstable plaque and dissemination due to breakup of pre-existing mural or atrial appendage clot.^{4,5}

CASE PRESENTATION

This case presents a thrombolysis for acute stroke complicated by ST elevation myocardial infarction (STEMI) (figure 1). The patient was an 84-year-old male Jehovah's Witness who experienced central

Figure 1 ECG at onset of chest pain. aVF, augmented vector foot; aVL, augmented vector left; aVR, augmented vector right.

To cite: Flower M, Harvey J, Hunter J. *BMJ Case Rep* Published Online First: [please include Day Month Year]. doi:10.1136/bcr-2017-223580

Figure 2 Angiographic distal LAD occlusion. LAD, left anterior descending artery; LIMA, left internal mammary artery; RPD, right posterior descending.

chest pain almost immediately after completion of rtPA infusion. Medical history was significant for transient ischaemic attack, ischaemic heart disease, hypertension, dyslipidaemia, insulin-dependent type 2 diabetes, early Parkinson's disease, chronic obstructive pulmonary disease not requiring oxygen therapy, stage I chronic kidney disease, benign prostatic hypertrophy requiring permanent indwelling urethral catheter, osteoporosis,

Figure 3 Graft anatomy.

obstructive sleep apnoea and hypothyroidism. Functional status prior to presentation was consistent with New York Heart Association class I. A four-vessel coronary artery bypass graft had been performed 11 years prior ([figure 2](#)).

Medications included clopidogrel (75 mg in the morning), carvedilol (25 mg twice daily), spironolactone (12.5 mg every night), atorvastatin (20 mg every night), isosorbide mononitrate (60 mg in the morning), thyroxine (75 mg every night), indacaterol (300 mcg in the morning), levodopa/benserazide (200/50 mg four times a day), risidronate (150 mg monthly), dutasteride/tamsulosin (500/400 mcg in the morning), cholecalciferol (1000 units in the morning) and aspart/insulin protamine suspension 30/70 (20 units in the morning, 29 units every night).

The patient presented to the emergency department 1 hour after onset of left facial droop and left upper limb weakness. He had been recently well after completing treatment for an uncomplicated urinary tract infection 1 month prior.

On examination, his blood pressure was 114/60 mmHg, heart rate 65 bpm, SpO₂ 95% on room air, respiratory rate was 16/min, and afebrile. Neurological deficit included left-sided upper and lower limb drift with partial sensory deficit and neglect. There was partial left lower facial palsy and he was able to obey only one-stage commands (NIHSS 8). Examination of the chest revealed scant bibasilar crepitations and an ejection systolic murmur loudest at the aortic area that did not radiate. Abdominal examination was unremarkable.

Thrombolytic protocol with rtPA at a dose of 73.8 mg (0.9 mg/kg) was started at 2 hours 25 min after onset of symptoms. A 10% bolus was given over 1 min as per local protocol with the remaining 90% over 1 hour. Within 10 min after the completion of the rtPA infusion, the patient developed central chest pain and anterior ST elevation on ECG. An urgent chest CT angiogram was performed which excluded vascular dissection or pericardial haemorrhage prior to transfer to a tertiary hospital for urgent angiogram.

INVESTIGATIONS

Laboratory investigations showed creatinine 101 µmol/L and haemoglobin 11.5 g/dL. The remainder of the full blood count, coagulation studies and electrolyte and liver function tests were otherwise normal. CT brain and cerebral angiogram demonstrated complete occlusion of the right intracranial vertebral artery. Radiological appearance was suspicious for thromboembolism.

At angiography, graft study showed severe native coronary artery disease. The left internal mammary artery (LIMA) graft was patent to the left anterior descending artery (LAD); however, the apical LAD was occluded, with the appearance suggestive of acute subtotal embolic occlusion ([figure 3](#)). Angiographic appearance suggested that this thrombus had likely migrated distally in the LAD since onset.

After angiogram, echocardiogram demonstrated a left ventricular ejection fraction of 31% with dyskinetic apex consistent with new-onset heart failure with reduced ejection fraction. No mural thrombus was seen. Peak serum troponin was 81 µg/L (<0.04 µg/L).

CT imaging performed 24 hours following thrombolysis excluded haemorrhagic transformation. The patient was noted to have paroxysmal atrial fibrillation during admission prior to referral to rehabilitation. He was commenced on therapeutic dabigatran, which was selected due to the availability of a reversal agent in the context of being a Jehovah's Witness.

OUTCOME AND FOLLOW-UP

At discharge 1 month after presentation, there was limited remaining neurological deficit. The patient was able to mobilise and transfer independently and returned home with a plan for titration of heart failure medications and repeat echocardiogram.

DISCUSSION

STEMI as a complication of thrombolysis is a rare but recognised complication.^{4 6 7} We feel this is an important entity for all medical officers involved in the treatment of acute stroke to be aware of. The authors suggest that both stroke and STEMI were of thromboembolic origin, and angiographic findings supported this. Mural thrombus was not seen at angiogram or echocardiogram, but it was considered that mural thrombus may have been disrupted by thrombolytic treatment. Paroxysmal atrial fibrillation was demonstrated after angiogram which may have been pre-existing and have predisposed to thrombus. Given the coronary anatomy, it was considered likely that thrombus that was caught in LIMA graft flow would tend to migrate to the LAD and then distally.

The described phenomenon has been reported previously in case reports but we believe this is the first case where STEMI

onset has been seen almost immediately following thrombolysis.^{6 7}

Contributors MF: drawing of figure 2; attainment of angiogram imaging; attainment of ECG imaging; prepared the first draft, edited subsequent drafts, submission of article. JHu: significant contribution to draft writing; contacting patient for consent; draft reviewing; case finding of similar cases. JHa: review of draft article; technical input to imaging; analysis of angiogram report. The three authors were involved in the care of the patient and planning case submission.

Funding This research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

© BMJ Publishing Group Ltd (unless otherwise stated in the text of the article) 2018. All rights reserved. No commercial use is permitted unless otherwise expressly granted.

REFERENCES

- Embersson J, Lees KR, Lyden P, *et al.* Effect of treatment delay, age, and stroke severity on the effects of intravenous thrombolysis with alteplase for acute ischaemic stroke: a meta-analysis of individual patient data from randomised trials. *Lancet* 2014;384:1929–35.
- Dereck L, Nighoghossian N. Intracerebral haemorrhage after thrombolysis for acute ischaemic stroke: an update. *J Neurol Neurosurg Psychiatry* 2008;79:1093–9.
- Miller DJ, Simpson JR, Silver B. Safety of thrombolysis in acute ischemic stroke: a review of complications, risk factors, and newer technologies. *Neurohospitalist* 2011;1:138–47.
- Wang B, Patel H, Snow T, *et al.* ST-Elevation myocardial infarction following thrombolysis for acute stroke: a case report. *West Lond Med J* 2011;3:7–13.
- Mehdiratta M, Murphy C, Al-Harathi A, *et al.* Myocardial infarction following t-PA for acute stroke. *Can J Neurol Sci* 2007;34:417–20.
- 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;13:64–6.
- Wallace EL, Smyth SS. Spontaneous coronary thrombosis following thrombolytic therapy for acute cardiovascular accident and stroke: a case study. *J Thromb Thrombolysis* 2012;34:548–51.
- Joo H, Wang G, George MG. A literature review of cost-effectiveness of intravenous recombinant tissue plasminogen activator for treating acute ischemic stroke. *Stroke Vasc Neurol* 2017;2:73–83.

Learning points

- While current evidence supports the use of recombinant tissue plasminogen activator as the standard of care for acute stroke in selected patients, clinical experience with this intervention is still accumulating.
- This case demonstrates the importance of remaining vigilant for thrombotic complications of thrombolysis as well as the historically well-documented complications.
- A detailed history and examination is vital in early diagnosis with prioritisation of investigations and urgent initialisation of treatment shown to improved outcome.

Copyright 2018 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- Submit as many cases as you like
- Enjoy fast sympathetic peer review and rapid publication of accepted articles
- Access all the published articles
- Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow