

Concurrent stroke and ST-elevation myocardial infarction: Is it a contraindication for intravenous tenecteplase?

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SUMMARY

Acute ischemic stroke (AIS) and acute ST-elevation myocardial infarction (STEMI) are leading causes of mortality worldwide. Concurrent AIS presentation with STEMI is rare and potentially fatal. Most importantly to date many centres in Malaysia are still not aware on how to treat this condition. We report a case of AIS, which was treated with intravenous tenecteplase (TNK) according to ischemic stroke dosage and lead to improvement of neurological deficit.

INTRODUCTION

Stroke as a ramification of myocardial infarction is common potentially leading to a high mortality rate. However, concurrent presentation of ischemic stroke and acute myocardial infarction that is equally critical is rare and therapeutically challenging. There are several contraindications for thrombolytic treatment in acute ischemic stroke (AIS) and recent myocardial infarction is included as a contraindication due to its' potential risk of myocardial rupture and haemopericardium; a rare complication of a recent ST-elevation myocardial infarction (STEMI).¹ According to previous reports we know that thrombolytic therapy is the fastest way to provide reperfusion of blood supply to the brain and myocardium.² In this case report, we demonstrate the successful treatment with intravenous TNK 0.4mg/kg (ischemic stroke dosage).

CASE REPORT

A previously healthy 33-year-old man was newly diagnosed with diabetes mellitus presented with a history of sudden onset of right-sided body weakness and slurred speech consistent with a stroke. The initial assessment revealed a moderate-severe stroke with National Institute of Health Stroke Scale (NIHSS) of 11. While assessing for intravenous thrombolysis for ischemic stroke, he started to complain of chest pain and difficulty in breathing. Further examination showed blood pressure of 140/95mmHg, regular pulse rate of 88 beats per minute and capillary blood glucose was 12mmol/l. He began to have global aphasia and visual inattention with obvious right-sided hemiplegia. Cardiovascular and respiratory examinations did not show any signs of cardiac failure. The non-contrasted computed tomography (CT) brain revealed early changes of left middle

cerebral artery (MCA) territory and the Alberta Stroke Program Early CT Score (ASPECTS) of 7 (Figure 1). An ECG showed extensive anterior and inferior ST-elevation (Figure 2). He was thrombolysed with tenecteplase 0.4mg/kg at 180 minutes from the onset of stroke symptoms and planned for emergency percutaneous coronary intervention which he later was unable to afford. Follow-up troponin I was elevated from 51.4 to 219 056pg/ml but his NIHSS improved to 9 at 24 hours and he had no recurrent chest pain with ECG showing the resolution of ST-elevation. The follow-up NCCT at 24 hours later showed no haemorrhagic transformation and echocardiogram revealed reduced ejection fraction of 45% with apical septal and lateral wall hypokinesia with no evidence of left ventricular clot. He was admitted to the cardiology care unit for a week to observe for arrhythmia but there was no atrial fibrillation recorded. He also received intravenous infusion of unfractionated heparin for three days and dual anti-platelet (clopidogrel and aspirin) with close monitoring for 24 hours after intravenous thrombolysis. He was discharged with NIHSS of 9 after three weeks of admission with improving neurological deficit of mild dysphasia but was able to take food orally.

DISCUSSION

Treatment Decision for AIS Associated with STEMI

The inclusion of recent myocardial infarction as a contraindication for IV r-tPA was not based on any randomized clinical trial. Furthermore, the reported risk of myocardial rupture or hemopericardium is very low and was associated with previous extensive anterior STEMI. In 2016 the American Stroke Association has updated and therefore suggested for cases of acute myocardial infarction with concurrent stroke, patients should be thrombolysed with stroke treatment dose followed by emergency percutaneous coronary intervention (PCI) and AIS patient may receive intravenous r-tPA 8 weeks after an episode of STEMI.³ Although endovascular clot-retrieval (ECR) could be considered in this new era of stroke thrombectomy, this procedure is not widely available including at our centre at the time presentation. To date, we have not encountered any report regarding the combination of PCI-ECR.

Utilization of TNK for AIS

From other reports few authors believe (recombinant tissue plasminogen activator) r-tPA should be considered if the

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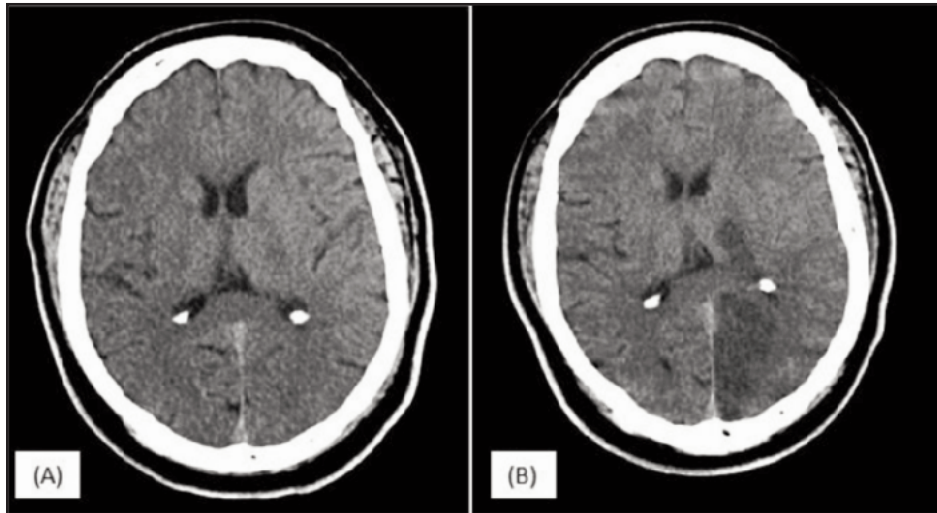


Fig. 1: (A) Pre-thrombolysis: Left insular ribbon sign and internal capsule subtle hypodensity, (B) 24-hour after thrombolysis: Left hemisphere effacement of sulci and PCA territory established stroke which possibly contributed by cardioembolic event.

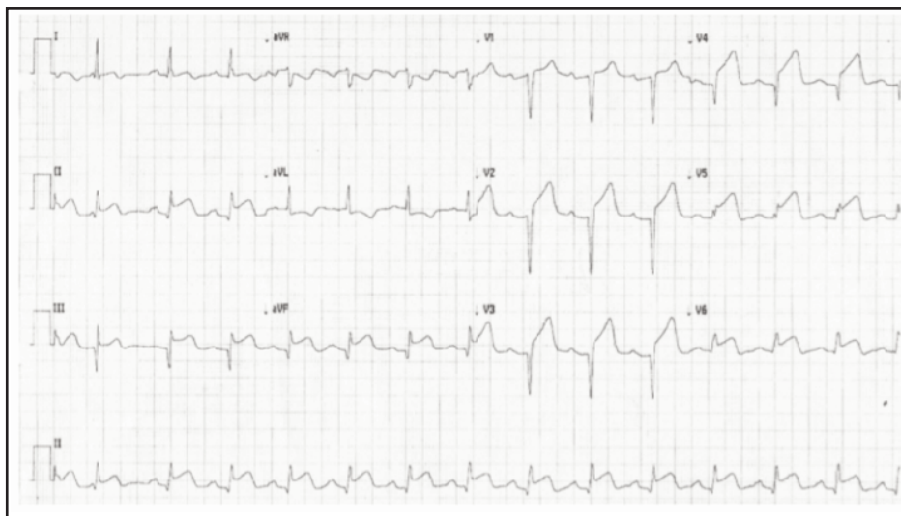


Fig. 2: Electrocardiogram on presentation showed extensive anterior and inferior ST-elevation.

brain and myocardium are affected simultaneously.² In our case we utilized intravenous TNK, which is known to be effective for the treatment of STEMI and commonly used as an agent in our STEMI treatment protocol. TNK is found to be fibrin more specific in comparison to r-tPA and can be given as a single bolus. Several clinical trials with intravenous TNK have shown to result in good clinical outcome in mild acute ischemic stroke with a similar safety profile with alteplase (r-tPA) and more recently TNK prior to endovascular thrombectomy with emergence of large vessel occlusion may provide better reperfusion with better functional outcome than alteplase.^{4,5}

CONCLUSION

Treatment decision in cases of concurrent AIS and STEMI should be individualized. The benefit and the risk of reperfusion treatment should be explained before excluding the patient from further treatment. As in our case, an immediate multidisciplinary decision to initiate treatment

has resulted in clinical improvement with early intravenous TNK treatment.

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