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Embolic Stroke Complicating Thrombolysis in Acute Myocardial Infarction: A Case Report

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ABSTRACT

Stroke remains a rare but catastrophic complication of thrombolysis. Intracranial hemorrhages are the primary neurological complication of thrombolytic therapy, but systemic embolisation shortly after thrombolytic therapy has also been reported, presumably caused by the dislodging of a pre-existing cardiac thrombus. In this report we have described a 59 year old male, case of acute Inferior and extensive Anterior wall STEMI developed slurring of speech with jargon with gait ataxia with swaying to the left and right hemiparesis, 6 Hours post thrombolysis with streptokinase and anti coagulation with low molecular heparin. These findings were suggestive of embolic infarctions in multiple vascular territories of left MCA, left PCA and right PICA.

KEYWORDS: *WSN Streptokinase (STK), ST Elevated Myocardial Infraction (STEMI), Embolic Infarctions, tissue Plasminogen Activator (t-PA)*

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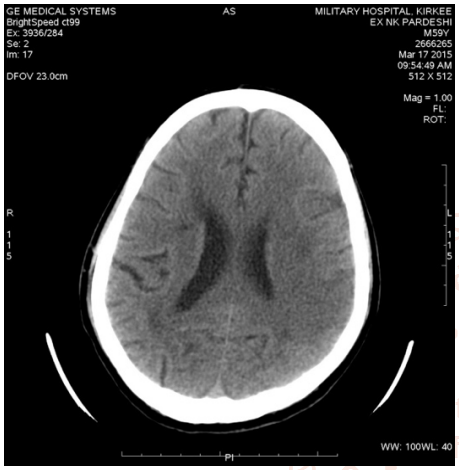
INTRODUCTION

Stroke remains a rare but catastrophic complication of thrombolysis. The overall risk of stroke from thrombolytic therapy for acute myocardial infarction in properly selected patients is low compared with the impressive reduction in mortality associated with the use of thrombolytic agents. While intracranial haemorrhages are the primary neurological complication of thrombolytic therapy, systemic embolisation shortly after thrombolytic therapy has been reported, presumably caused by the dislodging of a pre-existing cardiac thrombus.

Case: 59 year old male, chronic smoker an old case of primary hypertension, presented to our centre with anginal chest pain of four hours duration. The chest pain was of sudden onset, continuous, retrosternal, burning type, rated as ten on ten on visual analogue scale, associated with diaphoresis and two episodes of vomiting with no radiation. He denied any history of dyspnoea, syncope or palpitations. On examination he was hemodynamically stable. Jugular venous pulse was not raised and pedal edema was absent. Cardiovascular and chest examination was normal. ECG revealed features consistent with Inferior wall and extensive Anterior wall ST elevated myocardial infraction (STEMI). Troponin-T was positive. He was given Aspirin 325 mg stat, Clopidogrel 300 mg stat and Atorvastatin 80 mg stat. being a non PCI centre he was immediately thrombolysed with Streptokinase (STK) 1.5 million units administered over 45 minutes. ST elevation in inferior and lateral leads had shown a 50 % decline in amplitude of ST segments. Post STK he was started on Low molecular weight heparin 60 mg subcutaneous 12 hourly in view of extensive anterior wall MI.

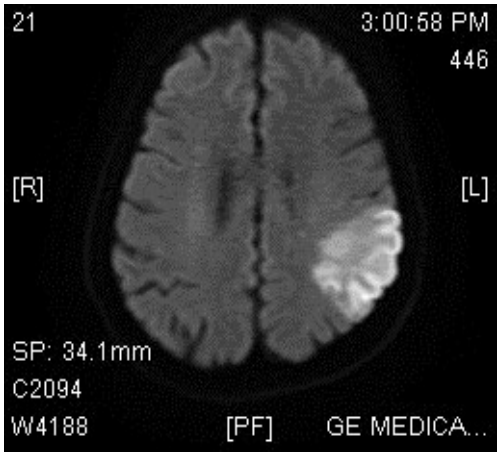
Six hours post thrombolysis he developed slurring of speech with jargon with gait ataxia with swaying to the left. He also complained of inability to hold onto a glass of water with his right hand. His pulse was

regular with no carotid or subclavian bruit. Neurological examination revealed Wernicke's aphasia, Right upper motor neuron facial palsy, Right sided spastic hemiparesis. Power was 4/5 right upper and lower limbs with hyperreflexia and extensor plantar response on the right side with National Institute Of Health Stroke Scale (NIHSS) score of nine. Heel to shin test and finger nose test was positive on the left side suggesting ipsilateral cerebellar hemispheric lesion. ECG monitoring revealed sinus rhythm. NCCT brain did not reveal any areas of haemorrhage in the brain. However, loss of grey white matter junction was present in left parietal lobe (Picture 1).

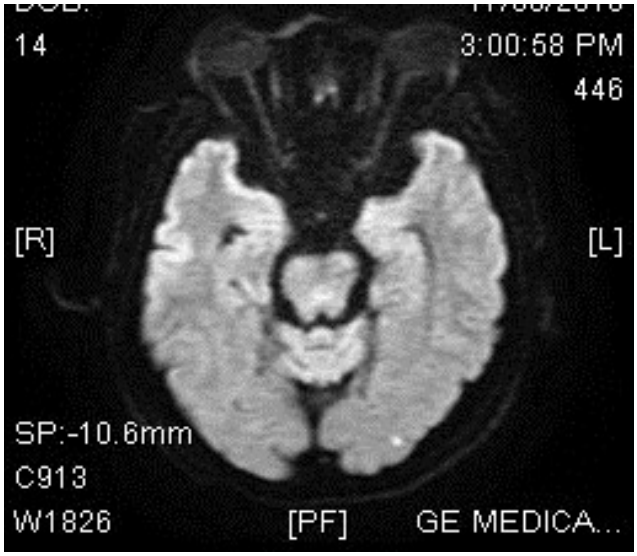


Picture 1: NCCT Brain reveals loss of grey white matter junction at left parietal lobe.

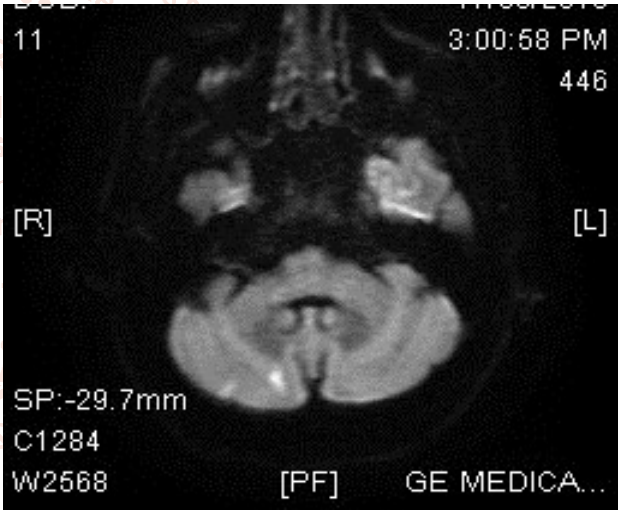
MRI brain revealed an area of acute restriction of diffusion in left parietal lobe (Picture 2). Smaller similar foci of restriction of diffusion were noted in left occipital lobe (Picture 3a) and right cerebellar hemisphere (Picture 3b). These areas are seen to be mildly hypointense on T1W1 and hyperintense on T2W1 and FLAIR. These areas were seen to be hypointense on ADC maps. These findings were suggestive of embolic infarctions in multiple vascular territories of left MCA, left PCA and right PICA.



Picture 2: MRI Brain DWI image with b value of 1000 shows an area of acute restriction of diffusion involving the cortex and subcortical white matter of left parietal lobe posterior to central sulcus.

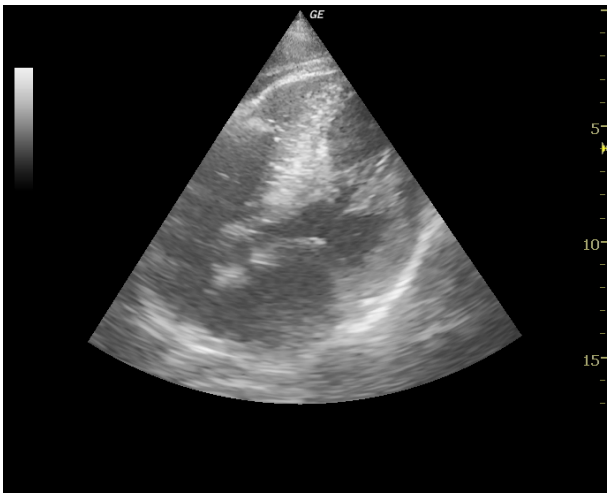


Picture 3a: MRI Brain DWI image with b value of 1000 shows an area of acute restriction of diffusion in left occipital lobe



Picture 3b: MRI Brain DWI image with b value of 1000 shows an area of acute restriction of diffusion in right cerebellar hemisphere

Carotid Doppler was normal and bedside echocardiography showed LV dysfunction with an EF of 35%, akinetic basal mid apical anterior septal and infundibular LV wall with no LA/LV clot (Picture 4). His antiplatelet medications, anticoagulants, ACE inhibitors and beta blockers were continued. Follow up after 7 days revealed that patient was ambulant, angina free at rest; NYHA Class II with partial recovery of neurological deficits. His speech remained slurred and there were no residual motor deficits and modified Rankin scale (mRS) was 1.



Picture 4: 2D Echo showing LV dysfunction with an EF of 35%, akinetic basal mid apical anterior septal and infundibular LV wall

Case Discussion: Strokes post thrombolysis in acute myocardial infraction (AMI) has been a plausible and well recognised complication in medical literature of yesteryears. Out of 20768 patients thrombolysed as part of GISSI 2 trials, a total of 236 (1.14%) had stroke in the hospital, 0.36% had haemorrhagic stroke, 0.48% had ischemic strokes and 0.30% had strokes due to undefined cause [1]. It is surprising and indeed pertinent to note that ischemic strokes were more common than the hemorrhagic stroke in the study. The concomitant presence of diffuse atherosclerotic vascular disease, atrial fibrillations, mural thrombus, pump failure, cholesterol embolisation probably explains the predominance of ischemic and embolic strokes in AMI post thrombolysis, but in our case n such conditions was there.

GISSI 2 study also documented a small but significant excess of stroke with tissue Plasminogen Activator (t-PA) as compared with those who had received streptokinase (1.33% vs 0.94%, adjusted OR1.42 95% CI :1.08 -1.94) [1]. This translates into four excess events with t-PA for every thousand patients treated. The cost effectiveness of Streptokinase over t-PA (US$50 for 1.5 MU STK vs US$ 600 for 50mg t-PA) and comparable in hospital mortality and major cardiac complications further substantiates the safety, efficacy and feasibility of use of streptokinase in resource limited health care settings[2].

Large studies have shown that the incidence of ischemic strokes is higher amongst elderly, individuals with anterior wall myocardial infarction and poor Killip class conditions which by themselves are associated with an increased risk of ventricular thrombus and embolic strokes[1]. Our patient had extensive anterior wall MI with an LV ejection fraction of 35 %, and Killips Class I, though the conventional risk factors for emboli like atrial fibrillation, mural thrombus or significant carotid plaque burden were absent.

The 2013 guidelines of AHA recommends that patients with STEMI undergoing reperfusion with fibrinolytic therapy should receive anticoagulant therapy for a minimum of 48 hours, and preferably for the duration of the index hospitalization, up to eight days or until revascularization is performed[3]. Although use of streptokinase forbids the use of heparin routinely it was recommended that it may be given as an intravenous bolus and infusion for patients receiving streptokinase if they are at high risk for systemic embolization, as highlighted in our case. It is noteworthy that the trials on anticoagulant use post thrombolysis did not show any significant increase in the incidence of stroke (risk with heparin 1.14% vs 1.13% risk without heparin) [3].

Conclusion: Stroke in the setting of thrombolysis in AMI portends a poor prognosis but our patient had minimal residual disability probably due to embolic nature of the event.

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