

## Real-time Monitoring of Blood Flow Changes during Intravenous Thrombolysis for Acute Middle Cerebral Artery Occlusion

Dear Editor,

We report a 62-year-old Chinese man who presented 90 minutes after right-sided weakness and inability to speak of sudden onset. He had been taking medications for hypertension and hypercholesterolaemia for 5 years. On examination, he was conscious with an irregularly irregular pulse and blood pressure of 168/95 mmHg. Neurological examination was remarkable for a forced left gaze, global aphasia and profound right hemiplegia (Power, Medical Research Council – MRC grade 0) with the National Institute of Health Stroke Scale (NIHSS) score of 23 points. While no early ischaemic changes were noted in the non-contrast-enhanced brain computed tomography (CT), CT angiography (Fig. 1A) revealed an occlusion of left proximal middle cerebral artery (MCA).

Intravenous tissue plasminogen activator (IV-TPA) was initiated at 135 minutes after the symptom-onset (0.9 mg/kg body weight with 10% given as IV bolus and the rest infused over 60 minutes). We performed continuous monitoring of the left MCA flow with 2-MHz Transcranial Doppler (TCD) and the spectral flow signals were graded according to the Thrombolysis In Brain Ischaemia (TIBI)

grading system.<sup>1</sup> Pre-TPA bolus TCD showed high resistance flow signals (TIBI grade 1) with flow diversion into the ipsilateral anterior cerebral artery (Fig. 1B). At 17 minutes after the TPA bolus, rapid recanalisation (within 15 seconds) of the MCA (to TIBI 5) was achieved (Fig. 1C). This was associated with immediate and significant clinical recovery, as evidenced by the return of motor power in the extremities and the speaking of a few words (NIHSS 15 points). We observed frequent spontaneous embolic signals (Fig. 1D) for the next 15 minutes followed by re-occlusion of the MCA (Fig. 1E) and clinical deterioration (NIHSS 23 points). However, recanalisation with normal flow signals was achieved again at 40 minutes after the TPA bolus and clinical recovery (Fig. 1F). TIBI grade 5 signals persisted during the rest of TCD monitoring and no further embolic signals were observed. NIHSS at the completion of TPA infusion was 11 points.

The repeat brain CT scan, performed 22 hours of symptom-onset, showed a left striato-capsular infarction (Fig. 1G) with no evidence of haemorrhagic transformation. Further clinical improvement was noted during the rest of his hospital stay (Power in the right upper and lower limbs – MRC grade

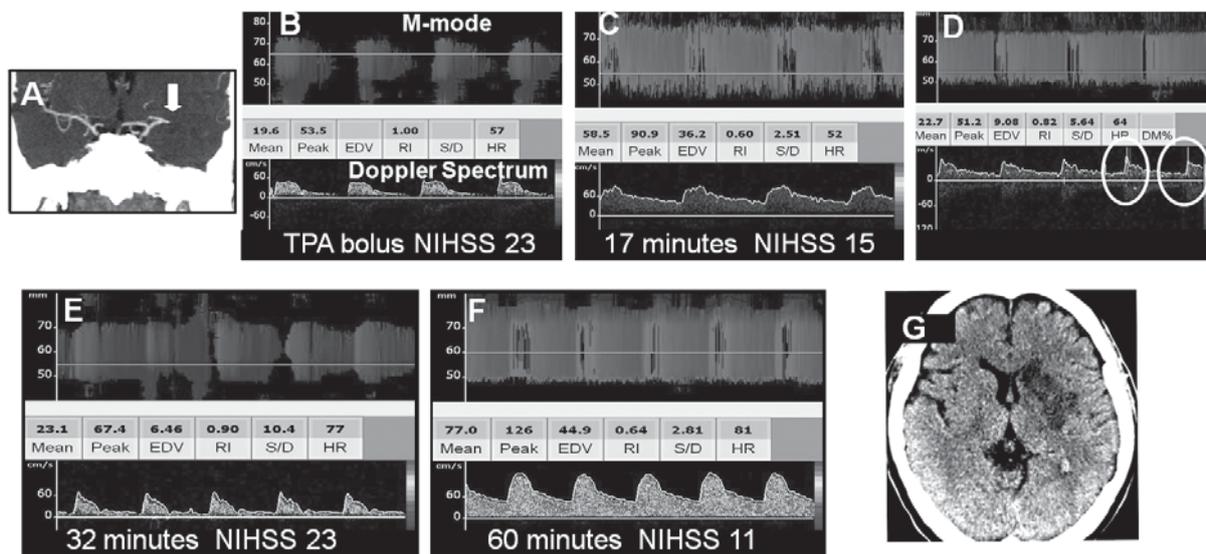


Fig. 1. Dynamic real-time transcranial Doppler (TCD) findings during intravenous thrombolysis for acute left middle cerebral artery (MCA) occlusion. Baseline contrast computed tomography (CT) angiography shows MCA occlusion on the pre-tissue plasminogen activator (TPA) (A). TCD at the time of TPA bolus (B) shows high-resistance spectral flow pattern (TIBI grade 1) and intermittent signals only during systole on M-mode. Rapid recanalisation (C) was noted at 17 minutes into the TPA infusion. Frequent spontaneous microembolic signals were noted (in panel D within the circles on the flow spectra) for the next 15 minutes followed by re-occlusion (E). Continued monitoring revealed recanalisation (F) again at 40 minutes that persisted until the end of TPA infusion. These dynamic changes in the MCA flow were accompanied by appropriate and significant clinical recovery. Final infarction is seen on the brain CT performed on day 2 (G).

4 and 5, respectively, with NIHSS score of 7 points at the time of discharge on day 5). His carotid duplex study was unremarkable. Transthoracic echocardiography showed a dilated left atrium. However, no intracardiac thrombus was noted. He continued to make good recovery with further rehabilitative physiotherapy and when seen after 3 months in the outpatient clinic, he could communicate normally and walked unaided (NIHSS 2 points, contributed by partial right haemianopia and mild facial weakness).

Our case describes the real-time dynamic changes in the flow pattern during continuous TCD monitoring in a patient presenting with acute middle cerebral artery occlusion and being treated with IV-TPA. The changes in arterial patency were closely associated with changes in the neurological status.

IV-TPA induces thrombolysis and remains the only FDA (Food and Drug Administration)-approved therapy for ischaemic stroke within 3 hours from symptom onset. TCD is a non-invasive examination that provides a reliable evaluation of intracranial blood flow patterns in real-time, adding physiological information to the anatomical information obtained from other neuroimaging modalities.<sup>2</sup> TCD is relatively cheap, can be performed at the bedside, allows monitoring in acute emergency settings and may help in selecting patients for additional interventional measures. Furthermore, continuous exposure of clot to 2-MHz pulsed-wave TCD ultrasound during IV-TPA infusion is known to enhance thrombolysis.<sup>3,4</sup>

Our case signifies the value of TCD monitoring during IV-TPA therapy. TCD provides extremely useful real-time information about the dynamic changes in the arterial patency during intravenous thrombolysis. A close temporal relationship between the changes in neurological

status and the arterial patency provides further evidence that arterial recanalisation is important for clinical recovery.<sup>5</sup> Demonstration of arterial recanalisation during thrombolysis can help in predicting a good prognosis in acute stroke patients.

#### REFERENCES

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Aftab Ahmad,<sup>1</sup>*MRCP*, Kewin TH Siah,<sup>1</sup>*MBBS*,  
Sze E Tan,<sup>1</sup>*MBBS*, Hock L Teoh,<sup>1</sup>*MRCP*, Bernard PL Chan,<sup>1</sup>*MD*,  
Benjamin KC Ong,<sup>1</sup>*MD*, Vijay K Sharma,<sup>1</sup>*MD, RVT*

<sup>1</sup> Division of Neurology, National University Hospital, Singapore  
Address for Correspondence: Dr Vijay K Sharma, Division of Neurology,  
National University Hospital, Singapore 119074.  
Email: drvijay@singnet.com.sg