Case Reports in Neurology

Case Rep Neurol 2020;12:127–136

DOI: 10.1159/000508589
Published online: December 14, 2020

Neurosonology in Critical Care

Asymmetric TCD Findings in Malignant MCA Infarction, Resolution after Decompressive Hemicraniectomy: A Case Report

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Keywords
Transcranial Doppler · Hemicraniectomy · Malignant infarct

Abstract
Transcranial Doppler (TCD) is a non-invasive method for assessing cerebral hemodynamics in the acute phase of stroke. We report a case of a 33-year-old man who presented with a massive left hemispheric infarct developing into "malignant" MCA infarction. TCD was utilized to monitor intracranial hemodynamics while the clinical and neuroimaging findings were used to help us in the decision to proceed with decompressive craniectomy (DC). Pre-operatively, there was reduced mean flow velocities (MFV) of the middle cerebral artery (MCA) with increasing pulsatility index (PI) ipsilateral to the infarct. The subsequent but smaller rise in the PI in the contralateral MCA was suggestive of very high intracranial pressure (ICP) from massive brain swelling. Serial TCD examinations post-operatively showed normalization of the PI, and subsequent rise in the left MCA MFV. Clinical improvement was also noted as the TCD findings improved. The asymmetry in TCD findings can be attributed to occlusion of the MCA with subsequent spontaneous recanalisation, occlusion of the MCA with subsequent recanalisation due to the DC, or initial occlusion and subsequent pressure effects on the arterioles of the MCA due to the "malignant" edema of that hemisphere that was relieved by DC. This case illustrates the value of TCD as a useful modality in monitoring intracranial hemodynamics in acute stroke.

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Introduction

Decompressive craniectomy (DC) is a surgical technique aimed at relieving intractable intracranial hypertension following "malignant" middle cerebral artery (MCA) infarctions, intracranial hemorrhage, and traumatic brain injuries (TBI) [1]. In a review and meta-analysis comparing hemicraniectomy versus medical treatment, DC resulted in large reductions in mortality due to space-occupying middle cerebral artery infarctions [2]. It has been shown that cerebral hemodynamics assessed by CT perfusion improve after DC, particularly in those patients with these "malignant" cerebral infarcts [3].

Transcranial Doppler sonography (TCD) is an ultrasound imaging modality which is utilized to monitor in real time the cerebral hemodynamics on patients with cerebrovascular disorders [4]. The following parameters can be obtained with TCD: peak systolic velocity (PSV), end-diastolic velocity (EDV), mean flow velocity (MFV), pulsatility index (PI), and cerebral resistance index (R_i) to describe cerebral hemodynamics [5]. Furthermore, additional information can also be obtained on evaluating the waveform morphology [5].

As the intracranial pressure (ICP) rises, the PI starts to increase due to the concomitant reduction of the EDV in relation to the PSV. The continuous build-up of ICP equalizes the systemic diastolic pressure until the end-diastolic flow in the TCD disappears. This progressive increase of the ICP also brings about the reduction of the PSV. The reduced PSV is then followed by a negative diastolic flow (oscillating flow) indicating the absence of adequate cerebral blood flow consistent with cerebral circulatory arrest [6]. In the setting of malignant cerebral infarcts, TCD can detect increases in the PI correlated with the midline shift measured on the third day [7]. Decompression craniectomy for severe head injury with raised ICP has been shown to normalise the raised PI and low EDV [8].

We report a case of a patient who underwent serial TCD monitoring to explore the hemodynamic changes before and after decompressive hemicraniectomy for a "malignant" MCA infarction.

Case Presentation

A 33-year-old right-handed male presented with a right sided weakness and was seen by our service 25 h after the ictus. Neurological examination showed a Glasgow Coma Score of 11, NIHSS score of 20, globally aphasic with isocoric pupils and roving eyeballs. There was also a right central facial palsy and right hemiplegia (power 0/5 in right upper and lower limbs) with a positive Babinski sign. No arrhythmia was detected on cardiac exam. The patient came in with a non-contrast CT (Fig. 1) done 5 h from the ictus which revealed hyperacute infarct on the left parieto-temporal area with mass effect on the ipsilateral lateral ventricle without midline shift, as well as hyperdense focus suggestive of hemorrhagic transformation. A dense MCA sign on the left was also evident. He was subsequently referred to Neurosurgery for possible DC. There were insufficient time slots to schedule a CT angiogram; thrombectomy is not available in our centre.

An initial Transcranial Doppler (TCD) examination (Fig. 2) was done 25 h post ictus which showed asymmetric findings, with decreased mean flow velocities (MFV) on all segments of the left MCA starting at a depth of 50 mm. The homologous MCA on the right showed normal findings. Additionally, an increased mean flow velocity was seen on the left anterior cerebral artery (ACA). Doppler signal was unobtainable at the distal left MCA at a depth of 45 mm. The

PI at the proximal segment of both MCAs were normal at 60 mm but increased more distally (60 mm: RMCA PI – 1.0; LMCA PI – 0.91; 50 mm LMCA PI – 1.28; RMCA PI – 0.87). "Medical decompression" with mannitol was given. No follow-up TCD was done immediately after institution of mannitol.

Follow-up TCD study (Fig. 3) was done 35 h after the ictus following the increase of NIHSS 20 to NIHSS 22. There was further asymmetry with decreased MFV on all segments of the left MCA starting at a depth of 50 mm with minimal flow at the distal MCA (45 mm depth). An increased PI on the proximal segment of both MCAs at 60 mm (RMCA PI – 1.15; LMCA PI – 1.48) was also observed.

Repeat neuroimaging (Fig. 4) showed a massive infarction at the left MCA territory with midline shift. DC was then immediately performed that day, the second day of stroke.

TCD follow-up was carried out on day 1 post-operatively (Fig. 5). Persistent asymmetry with minimal flow was seen on the distal MCA segment of the left, with increasing MFV noted on the right MCA, which may be indicative of increase compensatory flow to the pial collaterals. Increase in the MFV of the left ACA was still apparent. The PI of both proximal MCAs at 60 mm depth returned to the normal range.

A significant improvement of the patient's neurological status was observed post-operatively. He was able to maintain wakefulness and to follow some commands. By his 7th post-operative day, there was further improvement of his neurological status. He became more awake, with an NIHSS score of 15 (muscle strength: right arm – 2/5; right leg – 3/5). Follow-up TCD showed an increase of the MFV of the left MCA (Fig. 6). The increased MFV of the left ACA noted in the previous TCDs was no longer present. The patient was eventually discharged after a few more days.

Discussion

This is a case of a 33-year-old male, who developed left "malignant" MCA infarction characterized by right sided weakness, aphasia and progressive unresponsiveness. Subsequent workup showed large left hemispheric infarct in the left parieto-temporal area with mass effect. Aside from the clinical and neuroimaging presentation TCD examination findings – reduced MFV and increasing PI consistent with progressive ICP from brain edema and swelling, in spite of "medical decompression" – gave us more confidence to proceed with DC.

Insonation of basal cerebral blood vessels using TCD can assess cerebral hemodynamics non-invasively even in the acute phase of ischemic stroke. Monitoring of the cerebral blood flow velocity (FV), PI and characteristic waveform patterns allows accurate and rapid interpretation of the hemodynamic changes occurring intracranially [9]. Some studies have shown that the mean cerebral blood flow within the ischemic core is lower in malignant MCA infarctions as compared with smaller MCA infarcts [3]. It should be noted that significant midline shift or large focal lesions may cause some displacement of the basal cerebral arteries, affecting the detection and measurement of arterial flow. Still, TCD offers an inexpensive and continuous means of monitoring vessel patency in real-time [6]. In addition, while TCD may not be the most accurate way of measuring ICP, it may have a role in monitoring changes in ICP, especially shifts in the PI [10].

In our patient, the TCD findings are suggestive of initial left MCA distal severe stenosis/occlusion manifested by reduced flow velocity, and flow diversion or compensatory flow to the ipsilateral ACA. The subsequent TCD study showed further reduction in left MCA flow and

increased PI possibly from worsening of the left MCA distal severe stenosis/occlusion, and raised ICP. The post-operative TCD suggests that the raised ICP had reduced significantly as the PI bilaterally became normal. The normalization of left MCA PI may also be due to reduction in the initial left MCA distal severe stenosis/occlusion. The high left MCA velocities and low PI on post-operative day 7 may be due to hyperemia from persisting vasodilation with loss of vasomotor reactivity from neurovascular decoupling due to severe ischemia.

We offer a few possible explanations for the sequential clinical, imaging and asymmetric TCD findings in our patient. The occurrence of acute ischemic stroke with initial left MCA occlusion have caused reduced left MCA velocities and flow diversion to the left ACA. This was followed by spontaneous recanalization and return of flow along the left MCA. Reperfusion of the MCA can occur at any level of the middle cerebral artery, although most frequently, in the distal segments, with majority of cases occurring during the first 48 h [11]. This re-establishment of circulation may contribute to the development of a focal "malignant" cerebral edema after stroke and further neurological deterioration [12]. The raised ICP can lead to the reduced consciousness and worsening of the neurological status. The increasing trend of PI pre- and reduction post-operatively is highly suggestive of the increased ICP expected to occur in large hemispheric malignant infarcts. The high PI values returned to normal few days post operatively indicating the normalisation of the intracranial pressure after surgical decompression. The post-operative increase of the left MCA velocities is probably due to post-reperfusion hyperemia. The only impact on the right MCA was a slightly increased PI at the same time the PI increased significantly in the left MCA, possibly from increased ICP. The problem with this explanation is that the proximal left MCA was patent based on the initial TCD study. The possible hyperdense left MCA sign may have been a false positive – there may however have been left MCA severe stenosis/occlusion distally due to distal embolization after a proximal clot that had spontaneously dissolved between the time of the CT scan and the TCD.

Another possible explanation is similar to the above; except the recanalisation was not spontaneous but rather due to the decompression surgery. This phenomenon has been reported before [13]. A 43-year-old patient with "malignant" edema after embolic occlusion of the right MCA underwent DC. Ultrasound had detected a sub-totally occluding thrombus in the right internal carotid artery and a partial occlusion of the ipsilateral MCA. After the surgery, complete recanalization of the affected vessels was observed. As with the first possible explanation above, the problem with this explanation is that the proximal left MCA was patent based on the initial TCD study.

A third possible explanation is related to the first, but that the rise in PI is due to the "malignant" MCA infarct itself. Asymmetric short systolic spikes have been found in the MCA ipsilateral to a "malignant" MCA infarct [14]. These spikes are indicative of very high resistance distally. It is possible that the focal edema was extremely severe and compressed the arterioles that were coursing on and through the left cerebral hemisphere, causing these spikes to be seen in the feeding more proximal MCA. In a case series of 30 patients with supratentorial intracerebral hemorrhage, ipsilateral low flow velocities and raised ipsilateral PI was found [15]. The elevated ratios of ipsilateral-to-contralateral PI correlated primarily with hemispheric lesion volume. This asymmetry may be reflecting compartmentalised intracranial pressure gradients. It can be expected, therefore, that after decompression craniectomy, the very high pressures would reduce, and the PI would return to normal.

Conclusion

This case report has shown the possible value and utility of TCD in the decision to perform DC. We have utilized TCD in addition to the clinical and neuroimaging parameters in monitoring intracranial hemodynamics. The role of DC on MCA hemodynamics after large hemispheric infarctions needs further study.

Acknowledgement

This research was supported by the Department of Neurology of Jose R. Reyes Memorial Medical Center especially the Stroke and Neurovascular Service and all the residents-in-training who have been directly involved in the management of this patient. We thank the TCD technician Ms. Marissa Teves for her help in doing the series of TCD studies.

Statement of Ethics

The subject gave his written informed consent to publish this case report, (including publication of images). The patient confidentiality and anonymity of data were ensured by the authors.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Funding Sources

There was no funding received for the publication of this paper.

Author Contributions

J.C.N. conceived the idea of writing this case report. JC.N., N.V. and M.A.D.G. analyzed and interpreted data for the work. HGB drafted the manuscript. J.C.N. and N.V. edited it and made the necessary revisions. Final approval was given by all authors.

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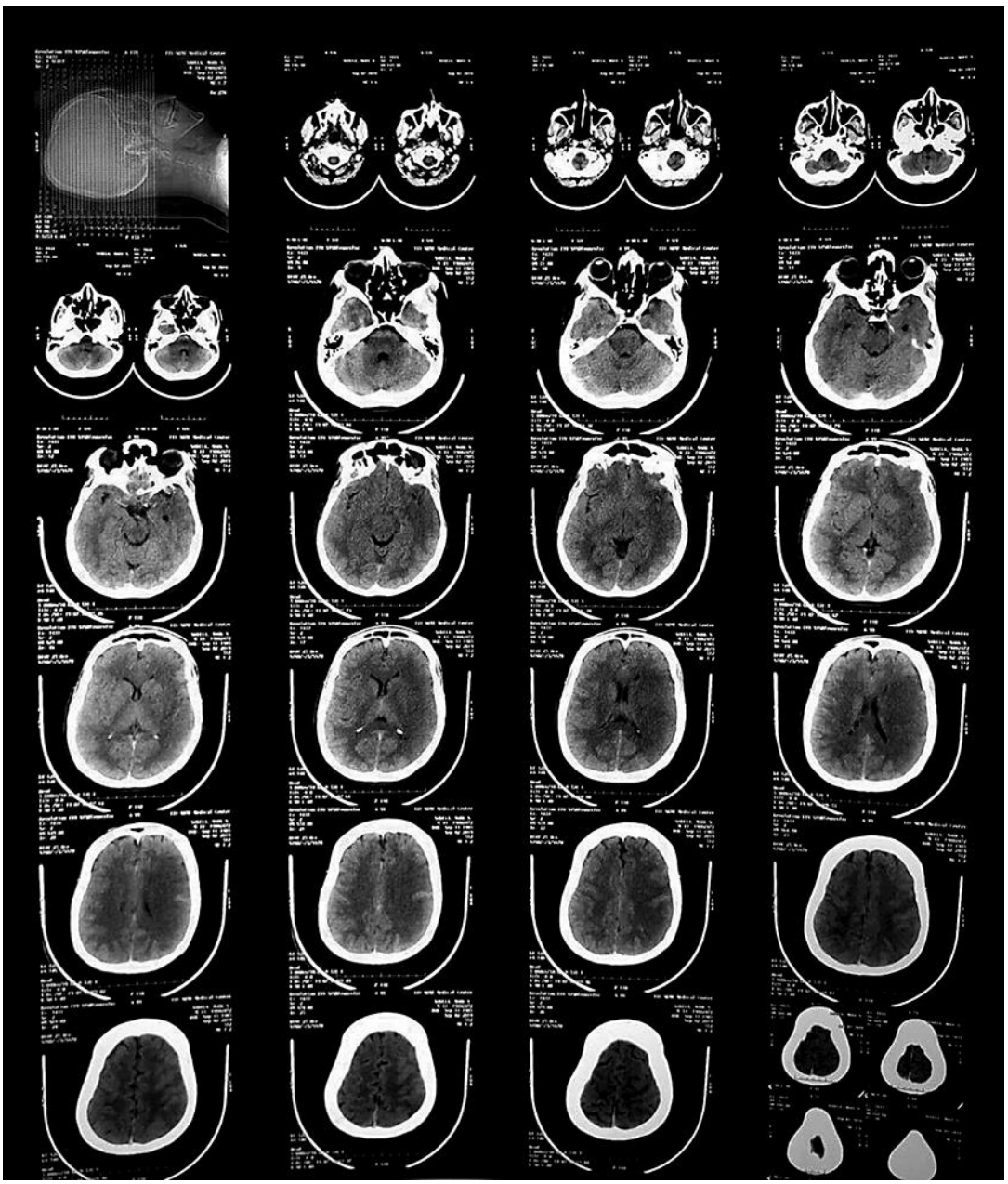

Fig. 1. Non-contrast CT done 5 h from the stroke onset.

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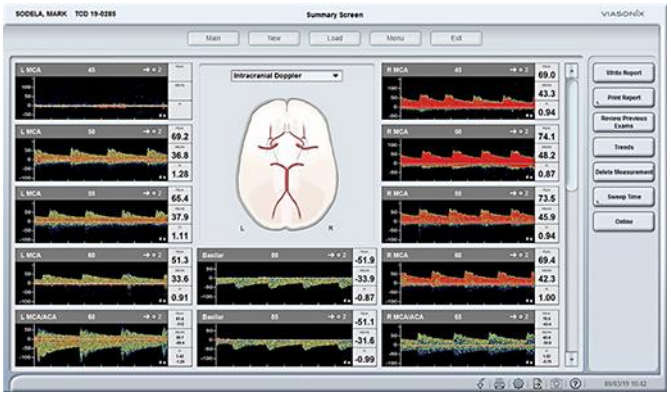

Fig. 2. Initial TCD done 25 h post ictus.

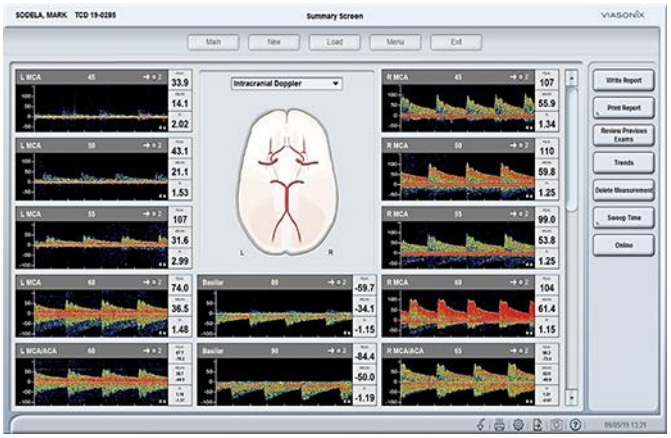

Fig. 3. Repeat TCD done 35 h post ictus.

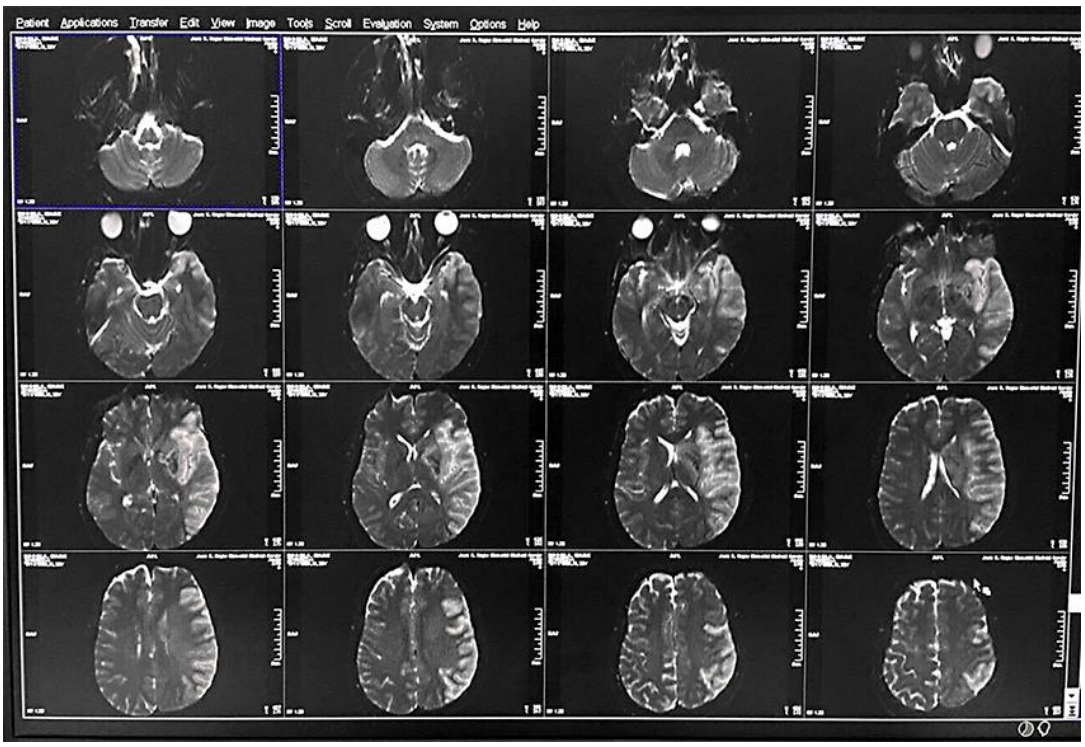

Fig. 4. Repeat neuroimaging (MRI) done preoperatively.

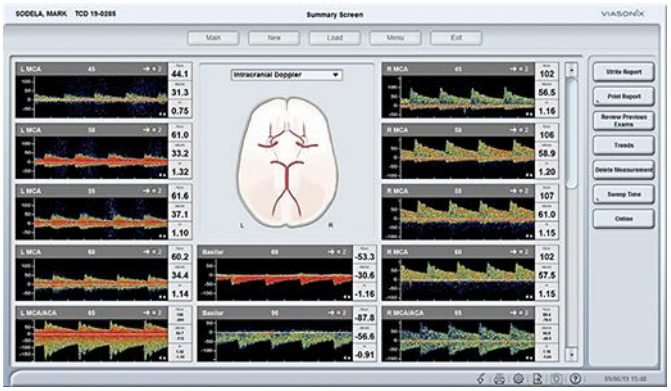

Fig. 5. TCD on 1st post op day.

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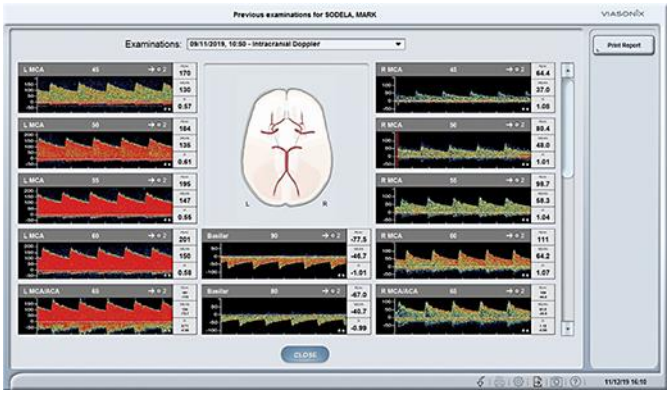

Fig. 6. TCD done 7 days post op.