Neurosonology in Critical Care

Transcranial Doppler in the Detection and Management of Arterial Vasospasm after Aneurysmal Subarachnoid Haemorrhage

Narayanaswamy Venketasubramanian  Manish Taneja  David Choy
Raffles Neuroscience Centre, Raffles Hospital, Singapore, Singapore

Keywords
Transcranial Doppler · Vasospasm · Subarachnoid haemorrhage

Abstract
Delayed cerebral ischaemia (DCI) and cerebral infarction is a much-feared complication of aneurysmal subarachnoid haemorrhage (aSAH). It has been largely attributed to focal hypoperfusion from reversible cerebral arterial narrowing, "vasospasm," from the effects of prolonged exposure of the arteries to perivascular blood and oxy-haemoglobin. Transcranial Doppler (TCD) provides a non-invasive method for detecting and monitoring vasospasm. We report a 38-year-old lady who developed sudden dizziness and catastrophic generalised headache with neck pain (Pain Score 10/10) while voiding her bowels. She subsequently became drowsy and was brought to hospital. On examination, she was already alert and orientated. Blood pressure was 175/109 mm Hg. Her neurological examination was normal but for severe neck stiffness to passive flexion. Computed tomography of the brain showed extensive SAH. Cerebral angiography revealed a 6 × 3 mm aneurysm along the posteromedial aspect of the supraclinoid left internal carotid artery. She underwent aneurysm coiling that night. She was given intravenous and then oral nimodipine. TCD monitoring of the circle of Willis on day 14 detected very
high velocities in the right and left middle cerebral arteries, mean velocity 187 and 141 cm/s, middle cerebral artery/internal carotid artery ratio 6.03 and 4.15, suggestive of severe and moderate vasospasm, respectively. She did not develop any related neurological symptoms or deficits. She was maintained in a euvoletic state and given high volumes of intravenous saline (2.4 L/day). Repeat TCD 7 days later was normal. The intravenous saline was gradually tailed off and she was subsequently discharged. TCD has an important role in the non-invasive detection and monitoring of vasospasm after aSAH.

Introduction

Aneurysmal subarachnoid haemorrhage (aSAH) has an incidence of 2–16 per 100,000 [1], a mortality that ranges between 8 and 67%, and significant dependence of 8 and 20% among survivors [2]. Apart from the effects of the initial and recurrent haemorrhage, a much-feared complication is delayed cerebral ischaemia (DCI) and cerebral infarction. It occurs in approximately 30% of those surviving the initial haemorrhage, usually between days 4 and 10 after SAH, and resolves by 21 days [3, 4]. DCI has been largely attributed to focal hypoperfusion from reversible cerebral arterial narrowing from "vasospasm" from the effects of prolonged exposure of the arteries to perivascular blood and oxy-haemoglobin [5], though other mechanisms are likely to also have a role, including early brain injury and cell death, blood-brain barrier disruption and initiation of an inflammatory cascade, microvascular spasm, microthrombosis, cortical spreading depolarisations and failure of cerebral autoregulation [6]. There are a number of techniques to detect vasospasm: transcranial Doppler (TCD) is a rapid, non-invasive, safe, portable, repeatable accurate bedside test for the assessment of the intracranial arteries of the circle of Willis [7]. This is a case report of the use of TCD for the detection and monitoring of vasospasm after aSAH.

Case Report

The patient is a 38-year-old lady who developed sudden dizziness and catastrophic generalised headache with neck pain (Pain Score 10/10) while voiding her bowels. She subsequently became drowsy and was brought to hospital. She had not had headaches before. She had a past history of hypertension in pregnancy. There was no family history of neurological illnesses or intracranial aneurysms.

On examination, she was already alert and orientated. Blood pressure was 175/109 mm Hg on the right arm when sitting, pulse rate was 79/min, respiratory rate was 14/min, Pain Score was 5/10, and Glasgow coma scale was 15. There was no aphasia, anopia nor neglect. Pupils were equally sized and briskly reactive to light. Fundoscopy was normal. There were full eye, face, jaw, palate and tongue movements. The neck was extremely stiff to passive flexion. Limb tone, power, reflexes and coordination were normal. Pain sensation was intact.
Computed tomography (CT) of the brain showed extensive subarachnoid haemorrhage (Fig. 1). Urgent cerebral angiography revealed a 6 × 3 mm aneurysm along the posteromedial aspect of the supraclinoid left internal carotid artery, just proximal to the bifurcation (Fig. 2). The neck measured 2 mm, the aneurysm had a complex shape and a wide base with a clot at the apex. Three Target coils (Stryker, MI, USA) were used to embolize the aneurysm. An extra-ventricular drain was inserted the next day after a CT brain scan showed early hydrocephalus and was converted to a ventriculoperitoneal shunt 10 days later. She was given intravenous nimodipine 10 mg/h for 2 days, then oral nimodipine 60 mg every 4 h for 21 days. Levetiracetam 500 mg b.d. was given prophylactically to prevent seizures. Intravenous labetalol was also needed for the first 2 days for the severe hypertension and to keep her systolic blood pressure below 160 mm Hg.

She underwent regular TCD of the circle of Willis arteries. On the 14th day, very high velocities were detected in the right and left middle cerebral arteries (mean velocity 187 and 141 cm/s, middle cerebral artery [MCA]/internal carotid artery ratio 6.03 and 4.15, respectively), suggestive of severe and moderate vasospasm, respectively (Table 1). She did not develop any related neurological symptoms or deficits. She was maintained in a euvolemic state and given high volumes of intravenous saline (2.4 L/day). A repeat TCD 7 days later was normal (Table 2). Blood pressure, pulse rate, temperature, haematocrit and arterial CO$_2$ were not significantly different between the recordings. The intravenous saline was gradually tailed off and she was discharged from hospital 2 days later.

She remained well on clinical follow-up with no further events. She subsequently underwent a flow diverter insertion as there was still a residual but asymptomatic aneurysmal neck. She was put on anti-platelets and commenced on anti-hypertensive medication for persistently elevated blood pressure.

**Discussion**

There are a number of available tools for the detection of vasospasm; these include CT angiography (CTA), MR angiography (MRA), digital subtraction angiography (DSA), as well as inferences from CT perfusion (CTP) [8]. These techniques suffer from the issues of cost, portability and 24-h availability (all), radiation and reactions to contrast (CTA, DSA and CTP), invasiveness and stroke risk (DSA), and the need for specialised software (CTP). The advent of TCD has revolutionized the detection of vasospasm [9]. It has a Class IIa Level of Evidence B recommendation to monitor for the development of arterial vasospasm [10]. It is presently widely used in neurosurgical intensive care units for the detection and monitoring for vasospasm after aSAH. TCD evidence of vasospasm is highly predictive of DCI, with a sensitivity of 90% (95% CI 77–96%), specificity of 71% (95% CI 51–84%), positive predictive value of 57% (95% CI 38–71%), and negative predictive value of 92% (95% CI 83–96%) [11].

Diagnostic criteria have been proposed for the TCD diagnosis of vasospasm. For the MCA, flow velocities of less than 120 cm/s or greater than 200 cm/s, a rapid rise in flow velocities or a higher Lindegaard ($V_{mca}/V_{eoa}$) ratio (6 ± 0.3) may predict the absence or presence of clinically significant angiographic MCA vasospasm [12, 13]. For the basilar artery (BA), a
BA/extracranial vertebral artery ratio >2 was associated with 73% sensitivity and 80% specificity for BA vasospasm [14]. The diagnosis of vasospasm was made using the above-mentioned criteria. The patient had repeated studies, and the velocities became normal again.

The patient’s initial blood flow velocities were normal. They subsequently rose, which may be due to many factors, including fever, rising heart rate and mean arterial blood pressure, falling hematocrit, rising arterial CO₂ content and therapeutic interventions’ influence on flow velocities [15]. Other factors that may influence velocities include technical factors, vascular anatomy and patient age. Thus, her rising velocities were attributed to rapidly progressive arterial stenosis that subsequently quickly reversed: “vasospasm.” The use of the Lindegaard ratio (intracranial MCA velocity to extracranial internal carotid artery velocity) also removed the impact of extracranial, physiological and therapeutic factors [13]. Nimodipine has no effect on vasospasm; it is used as a prophylactic drug for its calcium channel blocking and neuroprotectant properties against the effects of cerebral ischaemia.

The patient was managed according to guidelines by a multidisciplinary team, with early CT scan, DSA, aneurysm coiling, blood pressure control, nimodipine, external ventricular drainage insertion for early hydrocephalus and then conversion to ventriculoperitoneal shunt [11]. Her vasospasm was also managed by maintaining euvolemia as per the guidelines. Treatment with haemodynamic augmentation by triple-H (hypertension, hypervolaemic haemodilution) therapy is unproven. If the patient does not respond, induced hypertension is recommended, failing which intra-arterial vasodilators and balloon angioplasty are options.

Conclusions

Close monitoring for vasospasm is needed after aSAH to reduce the risk of DCI. TCD is a useful non-invasive inexpensive technique for the detection and monitoring of vasospasm in aSAH.

Statement of Ethics

This research complies with the guidelines for human studies and was conducted ethically in accordance with the World Medical Association Declaration of Helsinki. The subject has given her informed consent to publish her case (including publication of images).

Conflict of Interest Statement

The authors have no conflicts of interest to declare.
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Author Contributions

Narayanaswamy Venketasubramanian: neurologist, conceived the project, performed the transcranial colour-coded sonography, wrote the paper.

Manish Taneja: interventional radiologist, discussed the project, provided and interpreted the neuroimages, performed the cerebral angiogram and aneurysm coiling, critically reviewed the paper.

David Choy: neurosurgeon, discussed the project, was in overall charge of the patient’s care, critically reviewed the paper.

References

1 Feigin VL, Lawes CM, Bennett DA, Barker-Collo SL, Parag V. Worldwide stroke incidence and early case fatality reported in 56 population-based studies: a systematic review. Lancet Neurol. 2009 Apr;8(4):355–69.
2 Nieuwkamp DJ, Setz LE, Algra A, Linn FH, de Rooij NK, Rinkel GJ. Changes in case fatality of aneurysmal subarachnoid haemorrhage over time, according to age, sex, and region: a meta-analysis. Lancet Neurol. 2009 Jul;8(7):635–42.
3 Roos YB, de Haan RJ, Beenen LF, Groen RJ, Albrecht KW, Vermeulen M. Complications and outcome in patients with aneurysmal subarachnoid haemorrhage: a prospective hospital based cohort study in the Netherlands. J Neurol Neurosurg Psychiatry. 2000 Mar;68(3):337–41.
4 Hjдра A, Van Gijn J, Stefanko S, Van Dongen KJ, Vermeulen M, Van Crevel H. Delayed cerebral ischemia after aneurysmal subarachnoid hemorrhage: clinicopathologic correlations. Neuroradiology. 1986 Mar;36(3):329–33.
5 Mayberg MR. Cerebral vasospasm. Neurosurg Clin N Am. 1998 Jul;9(3):615–27.
6 Boddamski KP, Guilfoyle M, Helmy A, Huuskonen T, Czonyka M, Kirolos R, et al. The pathophysiology and treatment of delayed cerebral ischaemia following subarachnoid haemorrhage. J Neurol Neurosurg Psychiatry. 2014 Dec;85(12):1343–53.
7 Venketasubramanian N, Mohr JP. CT vs MRI in Coma or other Neurologic Emergencies. J Crit Illn. 1991;(Sept):850.
8 Washington CW, Zipfel GJ; Participants in the International Multi-disciplinary Consensus Conference on the Critical Care Management of Subarachnoid Hemorrhage. Detection and monitoring of vasospasm and delayed cerebral ischemia: a review and assessment of the literature. Neurocrit Care. 2011 Sep;15(2):312–7.
9 Aaslid R, Marlowalder TM, Nornes H. Noninvasive transcranial Doppler ultrasound recording of flow velocity in basal cerebral arteries. J Neurosurg. 1982 Dec;57(6):769–74.
10 Connolly ES Jr, Rabinstein AA, Carhuapoma JR, Derdeyn CP, Dion J, Higashida RT, et al.; American Heart Association Stroke Council; Council on Cardiovascular Radiology and Intervention; Council on Cardiovascular Nursing; Council on Cardiovascular Surgery and Anesthesia; Council on Clinical Cardiology. Guidelines for the management of aneurysmal subarachnoid hemorrhage: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2012 Jun;43(6):1711–37.
11 Kumar G, Shahripour RB, Harrigan MR. Vasospasm on transcranial Doppler is predictive of delayed cerebral ischemia in aneurysmal subarachnoid hemorrhage: a systematic review and meta-analysis. J Neurosurg. 2016 May;124(5):1257–64.
12 Sloan MA, Alexandrov AV, Tegeler CH, Spencer MP, Caplan LR, Feldmann E, et al.; Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. Assessment: transcranial Doppler imaging in the detection and management of arterial vasospasm after subarachnoid haemorrhage.
Doppler ultrasonography: report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. *Neurology*. 2004 May;62(9):1468–81.

13 Lindegaard KF. The role of transcranial Doppler in the management of patients with subarachnoid haemorrhage—a review. *Acta Neurochir Suppl (Wien)*. 1999;72:59–71.

14 Sviri GE, Ghodke B, Britz GW, Douville CM, Haynor DR, Mesiwala AH, et al. Transcranial Doppler grading criteria for basilar artery vasospasm. *Neurosurgery*. 2006 Aug;59(2):360–6.

15 D’Andrea A, Conte M, Cavallaro M, Scarafile R, Riegler L, Cocchia R, et al. Transcranial Doppler ultrasonography: from methodology to major clinical applications. *World J Cardiol*. 2016 Jul;8(7):383–400.

**Fig. 1.** CT brain scan showing massive subarachnoid haemorrhage.
Fig. 2. Cerebral angiogram showing the aneurysm.
### Table 1. Transcranial Doppler of the circle of Willis at day 14

| Artery | Right | | | | | | Left | | | | | |
|--------|-------|---|---|---|---|---|---|---|---|---|---|
|        | depth, mm | PSV, cm/s | EDV, cm/s | MV, cm/s | PI | depth, mm | PSV, cm/s | EDV, cm/s | MV, cm/s | PI |
| MCA    | 40     | 265 | 148 | 187 | 0.63 | 45     | 217 | 103 | 141 | 0.80 |
|        | 45     | 210 | 113 | 146 | 0.66 | 50     | 184 | 96  | 127 | 0.68 |
|        | 50     | 241 | 119 | 159 | 0.77 | 55     | 176 | 91  | 119 | 0.72 |
|        | 55     | 135 | 166 | 80  | 0.78 | 60     | 141 | 71  | 94  | 0.75 |
|        | 60     | 150 | 86  | 107 | 0.60 | 65     | 84  | 37  | 53  | 0.89 |
| ACA    | 65     | –94 | –47 | –62 | 0.75 | 65     | –103 | –61 | –75 | 0.57 |
| PCA    | 55     | 129 | 54  | 79  | 0.95 | 55     | 55  | 40  | 39  | 0.64 |
|        | 60     | 180 | 98  | 125 | 0.65 | 60     | 96  | 43  | 61  | 0.87 |
|        | 65     | 76  | 34  | 48  | 0.86 | 65     | 69  | 32  | 44  | 0.85 |
| tICA   | 67     | 81  | 45  | 57  | 0.62 | 66     | 94  | 39  | 57  | 0.97 |
| VA     | 50     | –32 | –17 | –22 | 0.66 | 55     | –37 | –21 | –26 | 0.63 |
|        | 60     | –34 | –16 | –22 | 0.82 | 60     | –35 | –14 | –21 | 1.04 |
|        | 65     | –42 | –21 | –28 | 0.77 | 65     | –42 | –17 | –25 | 1.02 |
|        | 70     | –54 | –24 | –34 | 0.89 | 70     | –45 | –19 | –28 | 0.93 |
|        | 75     | –59 | –28 | –38 | 0.83 | 75     | –71 | –36 | –47 | 0.73 |
|        | 80     | –61 | –23 | –35 | 1.07 | 80     | –74 | –29 | –44 | 1.03 |
|        | 85     | –48 | –23 | –32 | 0.78 | 85     | –58 | –23 | –35 | 1.00 |
| BA     | 90     | –74 | –34 | –48 | 0.84 | 95     | –82 | –39 | –53 | 0.82 |
| eICA   | 55     | –50 | –25 | –34 | 0.73 | 50     | –81 | –39 | –53 | 0.79 |
| MCA/ICA ratio | 6.03 | | | | | 4.15 | | | | |

PSV, peak systolic velocity; EDV, end diastolic velocity; MV, mean velocity; PI, pulsatility index; MCA, middle cerebral artery; ACA, anterior cerebral artery; PCA, posterior cerebral artery; tICA, terminal internal carotid artery; VA, vertebral artery; BA, basilar artery; eICA, extracranial internal carotid artery.
Table 2. Transcranial Doppler of the circle of Willis at day 21

| Artery | Right | Left |
|--------|-------|------|
|        | depth, mm | PSV, cm/s | EDV, cm/s | MV, cm/s | PI  | depth, mm | PSV, cm/s | EDV, cm/s | MV, cm/s | PI  |
| MCA    | 40  | 100 | 48 | 65 | 0.79 | 40  | 125 | 57 | 80 | 0.86 |
|        | 45  | 101 | 45 | 64 | 0.89 | 45  | 101 | 47 | 65 | 0.83 |
|        | 50  | 91  | 40 | 57 | 0.91 | 50  | 97  | 45 | 63 | 0.82 |
|        | 55  | 111 | 44 | 66 | 1.01 | 55  | 124 | 57 | 79 | 0.85 |
|        | 60  | 131 | 55 | 80 | 0.94 | 60  | 107 | 58 | 74 | 0.66 |
|        | 65  | 95  | 48 | 64 | 0.74 | 65  | 101 | 52 | 68 | 0.73 |
| ACA    | 67  | -43 | -23 | -30 | 0.66 | 76  | -68 | -36 | -47 | 0.68 |
| PCA    | 55  | 88  | 41 | 57 | 0.82 | 55  | 58  | 23 | 35 | 0.79 |
|        | 60  | 101 | 54 | 70 | 0.67 | 60  | 95  | 49 | 64 | 0.72 |
|        | 65  | 103 | 50 | 68 | 0.79 | 65  | 118 | 65 | 82 | 0.65 |
| tICA   | 65  | 87  | 43 | 58 | 0.76 | 69  | 79  | 40 | 53 | 0.73 |
| VA     | 40  | -28 | -15 | -19 | 0.67 | 50  | -33 | -15 | -21 | 0.86 |
|        | 45  | -33 | -16 | -22 | 0.77 | 55  | -36 | -16 | -23 | 0.87 |
|        | 50  | -44 | -21 | -28 | 0.79 | 60  | -43 | -20 | -28 | 0.85 |
|        | 55  | -41 | -19 | -26 | 0.81 | 65  | -47 | -22 | -30 | 0.83 |
|        | 60  | -38 | -19 | -25 | 0.75 | 70  | -49 | -23 | -31 | 0.84 |
|        | 65  | -48 | -22 | -31 | 0.83 | 75  | -40 | -17 | -25 | 0.89 |
| BA     | 80  | -39 | -18 | -25 | 0.81 | 90  | -65 | -31 | -42 | 0.81 |
|        | 85  | -54 | -26 | -36 | 0.78 | 95  | -44 | -18 | -26 | 1.02 |
|        | 90  | -65 | -31 | -42 | 0.81 | 100 | -60 | -32 | -42 | 0.67 |
|        | 95  | -44 | -18 | -26 | 1.02 | 105 | -81 | -39 | -53 | 0.79 |
|        | 100 | -60 | -32 | -42 | 0.67 | 110 | -65 | -33 | -45 | 0.80 |
|        | 105 | -81 | -39 | -53 | 0.79 | 115 | -62 | -31 | -41 | 0.76 |
| eICA   | 45  | -49 | -22 | -31 | 0.85 | 45  | -49 | -27 | -34 | 0.66 |

MCA/ICA ratio

| Right | 2.58 |
|-------|------|
| Left  | 2.36 |

PSV, peak systolic velocity; EDV, end diastolic velocity; MV, mean velocity; PI, pulsatility index; MCA, middle cerebral artery; ACA, anterior cerebral artery; PCA, posterior cerebral artery; tICA, terminal internal carotid artery; VA, vertebral artery; BA, basilar artery; eICA, extracranial internal carotid artery.