LETTER TO THE EDITOR

Transient Cerebral Circulation Arrest in SAH

Ripenmeet Salhotra

Keywords: Cerebral circulatory arrest, Neurocritical care, Subarachnoid haemorrhage, Transcranial Doppler, Transcranial ultrasound.
Indian Journal of Critical Care Medicine (2024): 10.5005/jp-journals-10071-24719

Dear Editor,

Transcranial color doppler ultrasonography (TCCD), performed using point-of-care ultrasound machines, has been increasingly employed by intensivists at the bedside, to monitor changes in cerebral hemodynamics in patients with acute neurological conditions, including stroke, subarachnoid hemorrhage (SAH), raised intracranial pressures (ICP) and mid-line shift.[1,2] It has been instrumental in detection of cerebral circulation arrest to aid in brain death determination.[3] Subarachnoid hemorrhage is a life-threatening condition that has a high median case fatality of 27–44%.[4] One of the major complications of SAH is rebleeding, which occurs in 10–20% of patients and is associated with poor outcomes.[5] The occurrence of a transient cerebral circulatory arrest during rebleeding is rarely reported. In this letter, we report a case of a 44-year-old woman with SAH who experienced a transient cerebral circulatory arrest recorded on TCCD during a rebleed.

A 44-year-old woman was admitted to the intensive care unit with a diagnosis of World federation of Neurological Society grade IV (WFNS grade IV) SAH due to a rupture of internal carotid artery (ICA) aneurysm made at another hospital 2 days ago. The initial CT scan was suggestive of diffuse thin subarachnoid bleed (Fisher grade-II). She was on invasive mechanical ventilation and had a Glasgow coma scale (GCS) rating of 7. A definitive procedure (coiling of aneurysm) was planned. A few hours after admission, she suddenly had a fall in her GCS associated with dilated pupils-unreactive to light, and a surge in her blood pressure. A bedside TCCD was performed by the attending intensivist (Fig. 1). On insonation of the right middle cerebral artery (MCA), small, short systolic flow peaks (velocity less than 50 cm/sec)

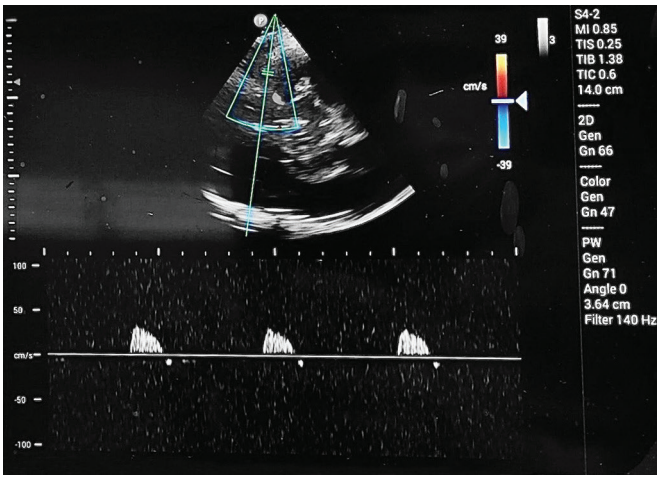

Fig. 1: Short systolic spikes pattern seen on TCCD

Department of Anaesthesiology and Critical Care, School of Medicine, Amrita Vishwa Vidyapeetham, Faridabad, Haryana, India

Corresponding Author: Ripenmeet Salhotra, Department of Anaesthesiology and Critical Care, School of Medicine, Amrita Vishwa Vidyapeetham, Faridabad, Haryana, India, Phone: +91 9810390927, e-mail: rippan.salhotra@gmail.com

How to cite this article: Salhotra R. Transient Cerebral Circulation Arrest in SAH. Indian J Crit Care Med 2024;28(6):620–621.

Source of support: Nil

Conflict of interest: None

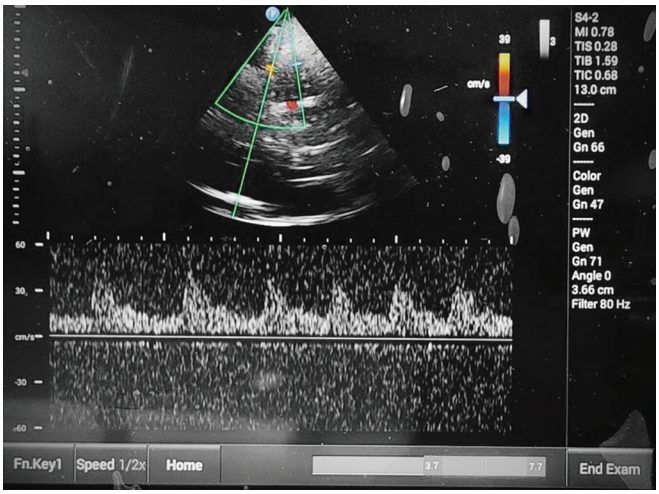

Fig. 2: Normal TCCD flow waveform with restoration of positive diastolic flow after 30 minutes

and absent diastolic flow were seen. These flow patterns on TCCD are referred to as: "systolic spikes".[1,3] The systolic spike pattern is highly characteristic for cerebral circulatory arrest.[3] It is produced by a severe increase in ICP causing cessation of effective cerebrovascular blood flow. As a response to this TCCD finding, cerebral decongestants, namely mannitol and 3%-saline, were rapidly administered to reduce the ICP and improve cerebral perfusion. A repeat TCCD after 30 minutes showed a 'normal' looking right MCA flow waveform with the positive diastolic flow (Fig. 2) this was accompanied by the return of pupillary reactivity to light. A repeat CT scan of the brain performed subsequently confirmed the presence of a diffuse thick subarachnoid space bleed with intraventricular extension (Fisher grade-IV). Aneurysmal rebleeding was assumed to be the cause of catastrophic raised ICP.

Transcranial color doppler ultrasonography has been validated as a bedside tool to detect cerebral circulatory arrest and as an ancillary test to diagnose brain death.[1,3] The presence of a systolic spike pattern, as seen in our patient, is highly suggestive of cerebral circulatory arrest. Systolic spikes are sharp unidirectional velocity signals in early systole of less than 200 milliseconds duration, less than 50 cm/s peak systolic velocity, and without a flow signal during the remaining cardiac cycle. These, along with the absence of previously recorded flow in intracerebral arteries, qualify as sonographic criteria of cerebral circulatory arrest.[3] We postulate that transient a catastrophic increase in ICP caused by a rebleed in subarachnoid space caused this pattern which was reversed over time by a decrease in ICP. This phenomenon could have implications for the management of SAH patients, as it could indicate a high risk of brain injury and death. Transcranial color doppler ultrasonography could be a useful tool to detect and monitor this condition and guide therapeutic interventions. We hope that this case report will stimulate further research and discussion on this topic.

Orcid

Ripenmeet Salhotra https://orcid.org/0000-0001-8987-6102

References

1. Robba C, Goffi A, Geeraerts T, Cardim D, Via G, Czosnyka M, et al. Brain ultrasonography: Methodology, basic and advanced principles and clinical applications. A narrative review. Intensive Care Med 2019;45(7):913–927. DOI: 10.1007/s00134-019-05610-4.
2. Kapoor I, Pandit S, Prabhakar H, Mahajan C. Comparison between transcranial sonography and computerized tomography scans to assess the midline shift in patients with traumatic brain injury. Indian J Crit Care Med 2023;27(1):64–66. DOI: 10.5005/jp-journals-10071-24376.
3. Ducrocq X, Hassler W, Moritake K, Newell DW, von Reutern GM, Shiogai T, et al. Consensus opinion on diagnosis of cerebral circulatory arrest using Doppler-sonography: Task Force Group on cerebral death of the Neurosonology Research Group of the World Federation of Neurology. J Neurol Sci 1998;159(2):145–150. DOI: 10.1016/s0022-510x(98)00158-0.
4. Marcolini E, Hine J. Approach to the diagnosis and management of subarachnoid hemorrhage. West J Emerg Med 2019;20(2):203–211. DOI: 10.5811/westjem.2019.1.37352.
5. Naidech AM, Janjua N, Kreiter KT, Ostapkovich ND, Fitzsimmons BF, Parra A, et al. Predictors and impact of aneurysm rebleeding after subarachnoid hemorrhage. Arch Neurol 2005;62(3):410–416. DOI: 10.1001/archneur.62.3.410.