LETTER TO THE EDITOR

Before the Stump Flow on the TCD in SAH can be Attributed to an SAH Relapse, All Other Possible Causes must be Ruled Out

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Dear Editor,

We read with interest the article by Salhotra et al. about a 44-year-old woman with a World Federatoin of Neurological Sciences (WFNS) grade IV subarachnoid hemorrhage (SAH) due to spontaneous rupture of an internal carotid artery aneurysm with an initial Glasgow coma scale (GCS) score of 7.[1] After two days, the patient experienced a sudden drop in GCS with dilated pupils unresponsive to light, an increase in blood pressure, and a stump flow on transcranial Doppler (TCD), indicating circulatory arrest attributed to an increase in intracerebral pressure (ICP).[1] A control cerebral computed tomography (CCT) scan after administration of mannitol showed increased SAH with ventricular intrusion.[1] The study is impressive, but some points should be discussed.

The first point is that no cerebral imaging had been performed immediately after the drop in GCS.[1] To assess the cause of the stump flow in TCD, it would have been imperative to obtain parenchymal and vascular information about the cerebral status at the time of this deterioration. The stump flow may not only be due to an ICP increase, but also to embolism, spasm, reversible cerebral vasoconstriction syndrome (RCVS) or dissection. Therefore, magnetic resonance angiography (MRA) or computed tomography angiography (CTA) would have been mandatory. It is also conceivable that the increased ICP was caused by cerebral edema. It should also have been ruled out that the patient had suffered a second SAH due to a rupture of a second aneurysm. Parenchymal information would have been mandatory, also to rule out the possibility that the deterioration was due to an ischemic stroke caused by spasms.

The second point is that the results of the CTA or MRA were not reported. In order to recognize the location, type and morphology of the aneurysm, it would have been imperative to either present an image of it or describe it in detail. The side of the internal carotid aneurysm was also not reported. A CTA/MRA is also important because the deterioration of the GCS could be attributed to arterial spasm with consecutive ischemic strokes and elevated blood pressure.

The third point is that the coagulation parameters and platelet count were not reported.[1] It would have been imperative to report these results in order to rule out the possibility that re-bleeding was due to coagulopathy or excessive anticoagulation. We should also know the coagulation management after the first hemorrhage. It is also conceivable that the index patient had a pre-existing hereditary coagulopathy, so this should have been ruled out.

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The fourth point is that the further therapeutic treatment was not described. Did the patient receive ventricular drainage? Was the aneurysm coiled? What was the outcome, what modified Rankin scale did she achieve?

In summary, it can be said that the index study has limitations that relativize the results and their interpretation. Addressing these limitations could strengthen the conclusions and support the results of the study. Before attributing stump flow on the TCD to a second SAB in a patient with SAB, all other possible causes of stump flow on the TCD should be excluded.

Declarations

Availability of Data and Material

All data are available from the corresponding author.

Author Contribution

JF: Design and conception, discussed available data with coauthors, wrote the first draft, and gave final approval. SM: Contributed to literature search, discussion, correction, and final approval.

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Reference

1. Salhotra R. Transient cerebral circulation arrest in SAH. Indian J Crit Care Med 2024;28(6):620–621. DOI: 10.5005/jp-journals-10071-24719.