Assigning developmental venous anomaly thrombosis to SARS-CoV-2 only when the causal connection has been proven

TO THE EDITOR: I read with interest the article by Ironside et al.1 on a 28-year-old male with right-sided parieto-occipital intracerebral bleeding due to thrombosis of a right occipital developmental venous anomaly (DVA) (Ironside N, Petrosian D, Abbas S, et al. Developmental venous anomaly thrombosis in a patient with coronavirus disease 2019-associated hypercoagulability: illustrative case. J Neurosurg Case Lessons. 2023;5(6):CASE22487). The patient underwent emergent decompressive hemicraniectomy with external ventricular drain placement and received intravenous heparin.1 His modified Rankin scale score at the 2-month follow-up was 2.1 The study is excellent but has limitations that are cause for concern and should be discussed.

The assumed causal relationship between severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and DVA thrombosis with secondary lobar bleeding has not been proven.1 The index patient had a mild respiratory infection 4 weeks before the cerebral hemorrhage and tested positive for SARS-CoV-2 using a home antigen test.1 In a study of 316 paired samples, only 33 samples were positive for antigen testing and reverse transcriptase–polymerase chain reaction (RT-PCR) by both methods.2 The sensitivity of the antigen test was calculated to be only 84.6%.2 A second argument against a possible causal relationship is the long latency period of 4 weeks between the mild respiratory infection and presentation because of the cerebral bleeding. A caveat in this regard is that there is no mention of whether the patient was tested for SARS-CoV-2 on admission or whether the RT-PCR result was not reported. Moreover, tests for hypercoagulability were noninformative. Therefore, other pathophysiological mechanisms should be considered to explain DVA thrombosis. Because the patient also had arterial hypertension, we should know if his blood pressure was elevated at presentation. We should also know if he had regularly consumed nonalcoholic liquids and if he had a leisurely or active lifestyle.

Surprisingly, the workup for hypercoagulability was noninformative.1 Because venous thrombosis is usually accompanied by increased D-dimer, we should know if this parameter was actually normal or if it was not determined before craniotomy.

It would also be interesting to know whether the patient was tested for any infectious or immunological parameters, particularly C-reactive protein, leukocyte count, lymphocyte typing, cytokines, and chemokines. Was his platelet function checked, and was it normal?

Overall, the interesting study has limitations that call the results and their interpretation into question. Addressing these issues would strengthen the conclusions and could improve the status of the study. As long as the causal connection between SARS-CoV-2 and thrombosis of a DVA has not been clearly proven, a causal connection cannot be established.

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References

Disclosures
The author reports no conflict of interest.

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RESPONSE
No response was received from the authors of the original article.

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