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POSTER

Looking for the culprit in stroke: what to do when the plot thickens?

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Abstract

Background: Stroke etiological investigation is usually a standardized process, where clinical and test findings are taken together. In a significant proportion of cases, no definite etiology is found, either because of negative findings or multiple causes. Here, diagnostic and therapeutic decisions were not straight-forward.

Case report: 70-year-old man complained of acute left hemiparesis. He had left homonymous hemianopia, sensory extinction and mild hemiparesis. CT showed a right temporo-parietal infarction. Echocardiogram, cervical and transcranial Doppler and 24h Holter monitoring had no significant findings. After 4 months, he was enrolled in an embolic stroke of undetermined source clinical trial. After 1 month, he had self-limited episodes of left hand tingling and later was admitted for left leg paresis. His aPTT was prolonged. CT showed a new right caudate-capsular infarction. MRI showed recent right subcortical temporo-parietal ischemia; MRA revealed irregular caliber and stenosis of the internal carotid artery (ICA), compatible with dissection or other vasculopathy, which improved in follow-up MRA, 7 days later. CSF analysis, including VZV, was normal. Trial oral anticoagulant (OAC) was stopped. After 1 month, he noticed a throbbing headache and red right eye. Angiography and ultrasonography revealed right thrombotic proximal ICA occlusion. OAC was started. After 1 month, readmission due to multiple episodes of left-side weakness. Doppler monitoring showed microembolization with right ICA origin. Hypocoagulation was stopped. Aspirin was started with no recurrent events within a 2-month follow-up.

Conclusion: Although a clear-cut etiology was elusive, the consensus was ICA dissection. Discussing safety/efficacy of novel OAC in ICA dissection is warranted.

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