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Can Convexity Subarachnoid Hemorrhage be Caused by Rupture of a Saccular Aneurysm?

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Sir,

A 51-year-old female came to emergency service with the complaint of a sudden-onset, one-hour-duration long, witnessed episode of aphasia. No headache was declared. When she arrived to the hospital three hours later, she had no symptoms. Her neurological examination was normal. Medical history elicited alcoholism, heavy cigarette smoking, and depression. She used irregularly quetiapine, diazepam, paroxetine, and mirtazapine for a long time. An emergent head CT, obtained four hours after symptom onset, showed a subarachnoid convexity hemorrhage (cSAH) located in the precentral sulcus of the frontal lobe. There was no subarachnoid hyperdensity within the Sylvian and basal cisterns (Figure 1).

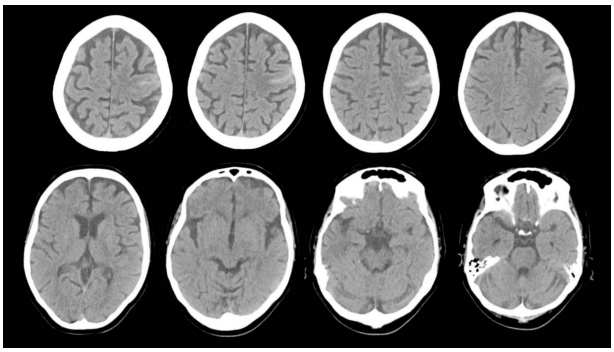

Figure 1. Plain Ct: upper bank: hyperderdensity filling prefronal sulcus on the left compatible with csah. Lower bank: no hyperdensity visible within Sylvian's sistern, pentagone and subdural spaces.

Given the presence of strokelike episode and a plenty of vasoactive drugs were used, a CT angiography was ordered with the provisional diagnosis of cerebrovascular arteriopathy. Of very much surprise to us, a seven-millimeter left middle cerebral artery (MCA) bifurcation saccular aneurysm was seen (Figure 2). Of note, no vasospasm or venous thrombosis was present.

At this point, the aneurysm was presumed to be coincidental given absence of spatial relationship between the locations of the aneurysm and subarachnoid blood. However, MR imaging obtained 12 hours after symptom onset, clearly documented that SAH was indeed present in the stem of the Sylvian fissure, next to the aneurysmal dome, pushing arachnoidal blood upward to the cortical sulci (Figure 3).

A complete obliteration of the aneurysm was achieved uneventfully with a technique utilizing Y-stenting plus coiling on the first morning after the event (Figure 4).

Convexity SAH, also known as cortical or convexal SAH, is not a rare entity. Based on our own analysis of nine published series [1–10] enrolling minimum 10 cases (n = 272), two most prevalent causes are cerebral amyloid angiopathy (CAA, in 24%) and reversible cerebral vasoconstriction syndrome (RCVS, in 20%). Etiology remained undetermined in 21%. Other prevalent causes are carotid artery steno-occlusive lesions in 7.4%, posterior reversible encephalopathy syndrome (PRES) in 7%, cerebral venous thrombosis in 6.6%, endocarditis in 3.7% and vasculitis in 2.4%. Cerebrovascular malformations such as cavernoma, thrombosed developmental venous anomaly, and dural fistula, were detected in 3%. Hemostatic abnormalities such as overanticoagulation and thrombocytopenia were found in 2.2%. The remaining causes, extracted from a diversity of case reports, include Moya Moya disease [11], hyperperfusion syndrome [10], cerebral hypotension [3], malignant hyper-

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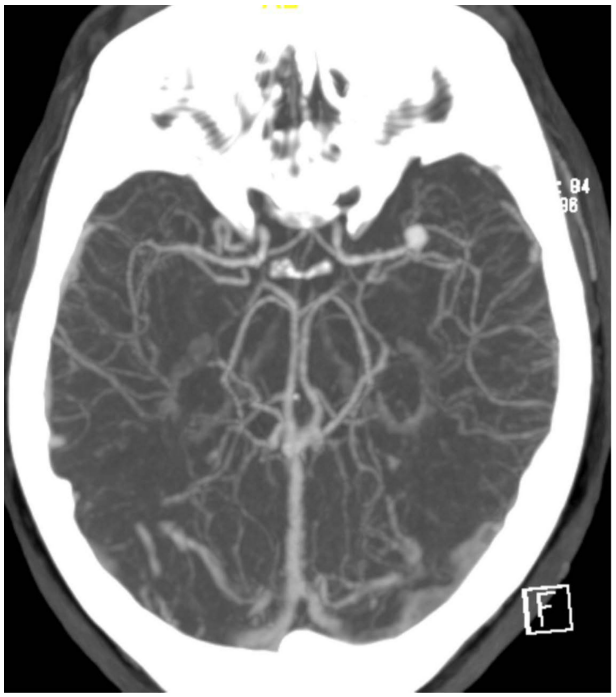

Figure 2. A seven-millimeter saccular aneurysm in the left MCA bifurcation (CTA).

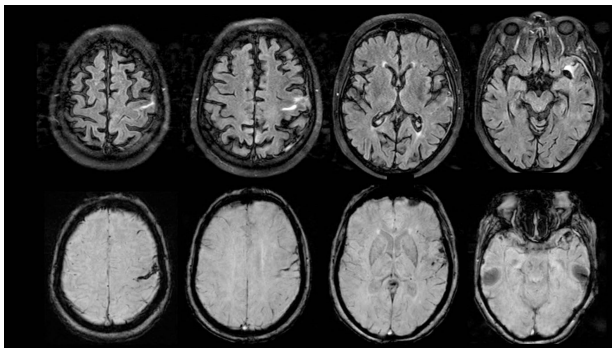

Figure 3. FLAIR (upper bank): hyperintensities located in lateral portion of the presentral sulcus, in the stem of the Sylvian fisue with a minute subdural hematoma over convexity; SWI (lower bank): hypointesities located in same regions compatible with blood signal. Hyponintense signal within lateral Slyvian sistern is well visible in SWI (third from the right, lower bank.

tension [2], cerebral abscess [1], and cortical infarct [4]. No cSAH caused by ruptured saccular aneurysm was reported in the germane literature. Aneurysmal rupture can cause pure acute subdural hemorrhage [12] or intracerebral hemorrhage but never cause an isolated cSAH. Saccular aneurysms found in RCVS patients with cSAH

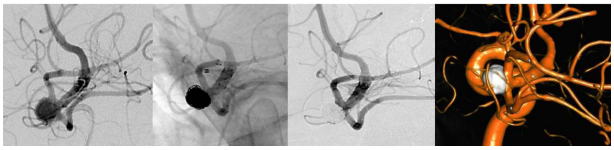

Figure 4. Procedural images of treatment of saccular aneurysm. Complete obliteration was documented.

are considered as coincidental, if not being a predisposing factor of RCVS [13].

If we rely on head CT scan alone, the case presented herein could be diagnosed as aneurysm-related cSAH. However, this is a misdiagnosis because brain MRI demonstrates certainly the presence of acute blood in the cisterns immediately adjacent to the culprit aneurysm. This case clearly reminds us the necessity to always perform brain MRI in every case of cSAH. This is critical not only for elucidation of frequent etiologies, such as CAA and PRES, but also to achieve accurate diagnosis and classification of SAH as nicely documented in this case vignette.

Acknowledgements

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