Disappearance and reappearance of a cerebral aneurysm

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Letter to the Editor
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Sirs,

A 42-year-old male was hospitalized with acute-onset aphasia and right hemiparesis due to acute middle cerebral artery (MCA) superior division infarction. A computerized tomography angiography (CTA), obtained for evaluation of stroke etiology and vessel status, revealed a partially thrombosed unruptured saccular aneurysm on left MCA bifurcation (figure a, b). Following fractional neurological recovery, he developed sudden onset global aphasia and right hemiplegia on the sixth day of hospitalization. A second CTA, obtained for evaluating the possibility of an endovascular intervention to achieve acute recanalization, showed complete disappearance of the aneurysm along with thrombosis of the distal M1 segment of the left MCA (figure c,d). In addition, disseminated thrombosis of the great arteries including ascending and descending aorta, bilateral subclavian and iliac arteries were noted. Interventional treatment was then not attempted and intravenous heparin was initiated.

There was evidence for systemic hypotension, significant leukocytosis (36,500/mm3) and liver dysfunction that was considered to be secondary to systemic inflammatory response syndrome in the setting of severe hospital-acquired aspiration pneumonia. No acquired or hereditary thrombophilia could be demonstrated albeit an extensive work-up. While the patient remained clinically stable, a follow-up CTA obtained on nineteenth day showed reappearance of the aneurysm in addition to complete recanalization of MCA and other arteries (figure e,f).

Because of the clinical severity of infarction and denial of the patient for a surgical clipping of the aneurysm, he was transferred to a rehabilitation facility on subcutaneous low-molecular weight heparin treatment. Unfortunately, the patient was lost on follow-up after discharge from this facility, and we could not get in contact with him against our best effort.

Spontaneous disappearance and reappearance of a ruptured cerebral aneurysm is rarely documented, and is
generally considered a result of aneurysmal shrinkage or thrombosis [1]. In addition, severe cerebral vasospasm and the use of antifibrinolytics can also contribute to disappearance of aneurysms in cases with ruptured aneurysms [2]. On the other hand, the demonstration of such a dynamic phenomenon in unruptured cerebral aneurysms, as in the case presented herein, is much more uncommon. Although, we were unable to pinpoint a specific reason for the systemic thrombotic response in our patient, we believe that a transient hypercoagulability and hyperviscosity in the setting of systemic inflammation might have contributed to the process. Anticoagulation, together with the probable self-limiting nature of the inciting event, led to dissolution of thrombi both in the systemic circulation and within the aneurysm itself.

References