Perianeurysmal edema as a predictive sign of aneurysmal rupture

Report of 2 cases

**FELIX HENDRIK PAHL, PH.D.,** 1,2 **MATHEUS FERNANDES DE OLIVEIRA, M.D.,** 2 **NELSON PAES FORTES DINIZ FERREIRA, M.D.,** 3 **LEONARDO LOPES DE Macedo, M.D.,** 4 **ROGER SCHMIDT BROCK, M.D.,** 2 AND **VALÉRIA CARDOSO DE SOUZA, M.D.** 5

1Department of Neurosurgery, Hospital do Servidor Público Estadual de São Paulo; 2Department of Neurosurgery, Hospital Sirio Libanês, São Paulo; 3Radiology Service, Hospital do Coração, São Paulo; 4Radiology Service, Cedimensional, Juiz de Fora, Minas Gerais; and 5Endovascular Service, Hospital Alemão Oswaldo Cruz, São Paulo, Brazil

Subarachnoid hemorrhage following intracranial aneurysmal rupture is a major cause of morbidity and mortality. Several factors may affect the probability of rupture, such as tobacco and alcohol use; size, shape, and location of the aneurysm; presence of intraluminal thrombus; and even the sex of the patient. However, few data correlate such findings with the timing of aneurysmal rupture. The authors report 2 cases of middle-age women with headache and MRI findings of incidental aneurysms. Magnetic resonance imaging showed evidence of surrounding parenchymal edema, and in one case there was a clear increase in edema during follow-up, suggesting a progressive inflammatory process that culminated with rupture. These findings raise the possibility that bleb formation and an enlargement of a cerebral aneurysm might be associated with an inflammatory reaction of the aneurysm wall, resulting in perianeurysmal edema and subsequent aneurysmal rupture. There may be a temporal link between higher degree of edema and higher risk for rupture, including risk for immediate rupture. (http://thejns.org/doi/abs/10.3171/2014.6.JNS132558)

**Key Words** • subarachnoid hemorrhage • aneurysm • treatment • vascular disorders

Subarachnoid hemorrhage (SAH) following intracranial aneurysmal rupture is a major cause of morbidity and mortality. Several factors may affect the probability of rupture, such as use of tobacco or alcohol; size, shape, and location of the aneurysm; presence of intraluminal thrombus; and even the sex of the patient. However, few data correlate such findings with the timing of aneurysmal rupture. Hiu et al. highlighted the role of surrounding brain edema as an early manifestation in the course to aneurysmal rupture. We report 2 similar cases, suggesting that perianeurysmal parenchymal edema is a marker preceding acute aneurysmal rupture, and discuss pertinent hypotheses.

**Case Report**

**Case 1**

A 37-year-old woman presented for neuroradiological investigation of a 3-month history of progressively worsening pulsatile headache. Her medical history was negative for smoking, alcohol intake, and medication use. She denied any family history of intracranial aneurysms. Her clinical and neurological examinations were unre- markable. Brain MRI revealed a saccular aneurysm of approximately 5 mm at the anterior communicating artery complex (Fig. 1A). It also showed vasogenic edema surrounding the aneurysm, characterized by hypointensity on T1-weighted and hyperintensity on T2-weighted and FLAIR sequences (Fig. 1A).

The patient was referred for neurological follow-up. At 2-month follow-up, her headache persisted and was progressively worsening, and another MRI study was obtained. Imaging now revealed increased edema and an aneurysm bleb (Fig. 1B). Six days after the second MRI, the patient presented with “the worst headache of her life” followed by impairment of consciousness. Urgent head CT revealed a Fisher Grade 4 SAH with an intraparenchymal hemorrhage (IPH) in the right gyrus rectus, adjacent to the aneurysm (Fig. 1C).

The patient was evaluated by the neurosurgical team and underwent aneurysm clipping (Fig. 1D). She recovered well and was discharged with a Glasgow Outcome Scale score of 5.

**Case 2**

A 45-year-old woman presented with dizziness lasting 2 weeks. Her medical history was negative for smoking, alcohol intake, and medication use, and she denied any family history of intracranial aneurysms. Her clinical
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and neurological examinations were also unremarkable. Brain MRI showed a saccular aneurysm of approximately 16 mm in size at the left carotid artery in contact with the left frontobasal parenchyma (Fig. 2A). Once again, there was vasogenic edema surrounding the aneurysm, as shown by hypointensity on T1-weighted and hyperintensity on T2-weighted and FLAIR sequences (Fig. 2A).

The patient was referred to undergo elective diagnostic angiography and aneurysm management. Shortly after leaving the physician’s office and arriving at home, she developed a thunderclap headache followed by somnolence and was taken to hospital. Head CT revealed a Fisher Grade 4 SAH and a voluminous IPH in the left frontal lobe, adjacent to the aneurysm (Fig. 2A, lower right). She was evaluated by the local neurosurgical team and recovered well after aneurysm coiling and craniotomy for evacuation of the hematoma (Fig. 2B and C). She was discharged with a Glasgow Outcome Scale score of 4.

Discussion

Brain edema may develop around partially thrombosed, large, or giant aneurysms. It is also well documented after coiling and stenting procedures. The underlying pathophysiology probably involves regional blood flow disturbances and the presence of an inflammatory process. Such findings are usually absent in the setting of unruptured aneurysms.

However, there is mounting evidence for a role of inflammation in the pathogenesis and growth of intracranial aneurysms. Mediators including tumor necrosis factor, macrophages, and reactive oxygen species might be present and play a role in aneurysm wall remodeling and risk for rupture.

Hiu et al. reported the first case of progressive perianeurysmal edema preceding the rupture of a small saccular aneurysm in a 71-year-old woman. They described a T2-elongated area around the dome of the aneurysm buried in the brainstem, suggesting perianeurysmal edema formation. The edema progressed with the formation of a bleb and an increase in aneurysm size over the following 3 years. The aneurysm ruptured as a brainstem hemorrhage without any subarachnoid clots 3 days after the final control MRI, which showed a significant increase in edema and aneurysm size and marked expansion of the bleb.

Both of our cases were very similar, involving previously healthy, middle-aged women with no risk factors or family history of intracranial aneurysms, who presented with headache and MRI findings of incidental aneurysms. In both cases, MRI revealed surrounding parenchymal edema. In the patient in Case 1, a clear increase in edema was shown during follow-up, suggesting a progressive inflammatory process that culminated with rupture. In the case reported by Hiu et al., the interval between symptom onset and rupture was 3 years. In both of our cases, this interval was considerably shorter, on the order of months. The patient in Case 2 presented with a dramatic rupture shortly after initial medical evaluation. Additionally, in all 3 cases, the aneurysm was intraparenchymal, and IPH was observed after rupture. Such features can impair clinical status and course and may suggest that perianeurysmal edema is an outcome factor.

Fig. 1. Case 1. A: The first MR images disclosed a saccular aneurysm of approximately 5 mm in the anterior communicating artery complex invading the right frontobasal parenchyma. B: Follow-up MR images 2 months later revealed evidence of a bleb formation and increase in edema sign. C: The first brain CT scans after sudden headache, disclosing Fisher Grade 4 SAH and right frontal intraparenchymal hemorrhage. D: Brain CT scans obtained after definite management of the aneurysm.
These findings raise the possibility that bleb formation and enlargement of a cerebral aneurysm might be associated with perianeurysmal edema and subsequent aneurysmal rupture. Such edema may be due to a pinpoint leak in the aneurysm; aneurysmal thrombosis, leading to inflammation and consequent weakening of the aneurysmal wall; or exposure of the intraaneurysmal blood flow to subendothelial tissue, which is a potential cause of wall destabilization. There may be a temporal link between greater severity of edema and higher risk of rupture, including risk of immediate rupture. However, not all types of edema have the same clinical significance. For example, coiling and stenting are sometimes followed by edema but seldom by vessel rupture, which suggests involvement of different etiologies and inflammatory cascades.

Although our experience is still limited and based on the cases reported herein, we strongly encourage that MRI be performed in patients with a diagnosed or suspected cerebral aneurysm so as to properly evaluate for perianeurysmal edema. If this finding is present, we recommend immediate hospitalization and discussion of definitive management of the aneurysm.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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