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ORIGINAL ARTICLE

Intracranial aneurysms with perianeurysmal edema: Long-term outcomes post-endovascular treatment



Kenneth J. Sim^a, Bernard Yan^{a,b}, Richard J. Dowling^a,
Peter J. Mitchell^{a,*}

^a Department of Radiology, Neurointervention Service, The Royal Melbourne Hospital, Melbourne, Victoria, Australia

^b Department of Neurology, Melbourne Brain Centre, The Royal Melbourne Hospital, University of Melbourne, Parkville, Victoria, Australia

Available online 23 June 2014

KEYWORDS

Endovascular coiling;
Magnetic resonance
imaging;
Perianeurysmal
edema

Summary

Background and purpose: Perianeurysmal edema is rare and is associated with expansion of intracranial aneurysms despite adequate endovascular treatment. The natural history of this condition is poorly understood. We present a case series of perianeurysmal edema to investigate the incidence, natural history and management of these aneurysms.

Materials and methods: Retrospective analysis of endovascularly treated aneurysms from January 2001 to December 2012 was performed. Perianeurysmal edema either pre- or post-treatment as detected on MRI was used to identify cases.

Results: Ten of the 838 patients demonstrated perianeurysmal edema. The median age was 51.5 (interquartile range 48 to 59). Of the 10 subjects, 50% (5/10) were male. Perianeurysmal edema was detected after the initial presentation and treatment in 70% (7/10) of the patients, with one patient developing headache after endovascular coiling, while the remaining 6 were found on routine follow-up imaging. Only 4 of the patients were ever symptomatic in the setting of perianeurysmal edema, with 3 of these patients improved clinically following treatment of the aneurysm. Radiological follow-up showed expanding neck remnants leading to a second endovascular coiling procedure in 50% (5/10) of the patients and a third procedure in 20% (2/10) of the patients to attenuate aneurysm growth.

Abbreviations: MRI, magnetic resonance imaging; MRS, modified Rankin scale; DSA, digital subtraction angiography; CT, computed tomography; Acom, anterior communicating artery; ICA, internal carotid artery; FVL, Factor V Leiden; PED, pipeline embolization device; Pcom, posterior communicating artery; SAH, subarachnoid hemorrhage (SAH).

* Corresponding author. Department of Radiology, The Royal Melbourne Hospital, Grattan Street, Parkville, Victoria, Australia, 3050. Tel.: +613 9342 7000; fax: +613 9342 7482.

E-mail addresses: Peter.Mitchell@mh.org.au, mitchep@gmail.com (P.J. Mitchell).

<http://dx.doi.org/10.1016/j.neurad.2014.05.001>

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Conclusion: Ten of the aneurysms treated with endovascular therapy in our case series demonstrated perianeurysmal edema. They are mostly asymptomatic, with no cases of re-bleeding identified, however perianeurysmal edema is associated with aneurysm re-expansion despite initial endovascular treatment.

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Introduction

Perianeurysmal inflammation and expansion of intracranial aneurysms is a rare occurrence with only a few cases reported in the literature [1]. Inflammatory changes have been noted after spontaneous thrombosis of giant intracranial aneurysms [2–6] and following endovascular coiling [1,2,7–11] or flow-diversion treatment [12]. Imaging findings associated with perianeurysmal inflammation are not clearly defined in the literature, however high signal intensity on T2-weighted MRI in the perianeurysmal area consistent with vasogenic edema, aneurysm wall enhancement and aneurysm expansion with compression of the surrounding parenchyma are all potentially observed [1,7–11,13]. The underlying pathology remains uncertain, however intrasaccular thrombus and the chemical and cellular cascade associated with the clot formation, has been postulated as a potential mechanism [6,14–16]. Identifying which aneurysms are more likely to demonstrate perianeurysmal inflammation remains problematic due to the paucity of cases [1,11]. Furthermore, treatment of aneurysms with perianeurysmal inflammation is uncertain, with administration of steroids and further coiling having been reported in case reports [1,7–11,17]. This paper aims to focus on perianeurysmal edema and to the best of the authors' knowledge, we present the largest case series of aneurysms with perianeurysmal edema. We aim to better define the natural history and management approach to this condition.

Materials and methods

A prospectively collected database of all patients with intracranial aneurysms who underwent endovascular treatment between January 2001 and December 2012 at our institution was reviewed. Endovascular treatment included coiling, stent-assisted coiling, balloon-assisted remodeling or pipeline embolization device. There were a total of 838 patients reviewed. Patients presented either with SAH, symptoms related to mass effect from the aneurysm or as an incidental finding. Aneurysms with perianeurysmal edema were defined as demonstrating high signal intensity on T2-weighted MRI in the perianeurysmal area that was present either before or after endovascular treatment of the aneurysm. Acute ischemia was excluded by Diffusion Weighted Imaging. Word searches of imaging reports of patients who had endovascular aneurysm treatment and MRI with keyword 'edema' were performed to identify suspected cases, with two neuroradiologists (P.M. and R.D.) then independently reviewing the cases to confirm they

met the inclusion criteria. Follow-up of these patients was individualized depending on the clinical and angiographic outcomes, however MRI and/or DSA at 6 months with yearly MRI or CT angiogram thereafter was generally performed. The MRS was used to assess the overall independence and functioning of the patient and to grade the level of any potential patient morbidity.

Results

Ten patients out of the 838 included in the study demonstrated perianeurysmal edema either pre-procedure or on follow-up MRI (Figs. 1 and 2; Table 1). The median age was 51.5 (interquartile range 48 to 59). Fifty percent (5/10) were male. Three were smokers. Amongst the non-smokers, 2 had significant cardiovascular risk factors of which one also had Factor V Leiden disease. Another non-smoker had Protein S deficiency. Five patients initially presented with SAH, 3 with worsening headache with no evidence of SAH, one with a homonymous hemianopia and one with seizures. There was no apparent trend in aneurysm location with 3 basilar artery aneurysms, 3 anterior communicating artery aneurysms, 2 posterior communicating artery aneurysms and 2 internal carotid artery aneurysms. All 10 aneurysms had sacs that were embedded within the surrounding brain parenchyma. The initial endovascular procedure in 7 of the patients was endovascular coiling, with 3 undergoing stent-assisted coiling. Perianeurysmal edema was not detected after use of a Pipeline flow device.

In the patients presenting with SAH, only one patient had an MRI pre-endovascular treatment. Perianeurysmal edema was already evident on this MRI (Fig. 2) which may suggest rupture of an aneurysm with perianeurysmal edema. In the other 4 patients presenting with SAH, the onset of the perianeurysmal edema is uncertain with the edema in every one of the cases, first noted on their initial follow-up MRI scan. The timing of this MRI scan ranged from 4 to 60 months. Of those who did not have a SAH, the perianeurysmal edema was present prior to endovascular treatment in 2 of the 5 cases. Edema in the other 3 cases was first noticed on their initial follow-up MRI after coiling. The timing of this MRI scan ranged from 7 to 46 months. Out of the 4 patients who had a pre-endovascular treatment MRI, 3 of them demonstrated aneurysms that contained intra-aneurysmal thrombus (Figs. 1 and 2).

Only 4 of the patients were ever symptomatic in the setting of perianeurysmal edema, with one presenting with SAH, 2 with worsening headache with no SAH, and the other with a right homonymous hemianopia. Steroids achieved good clinical effect in the 2 with worsening headache with

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