Transformation of a Cranial Fusiform Aneurysm into a Pseudotumoral-Like Mass Prior to Spontaneous Occlusion and Regression

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Summary

This report documents the evolution of a non-ruptured cranial fusiform aneurysm that underwent both spontaneous occlusion and regression. During this process, unique images of the aneurysm as a pseudotumoral-like mass were obtained. The pseudotumoral-like mass most likely reflected inflammation and secondary neovascularization within the aneurysm, supporting the theory that spontaneous aneurysmal healing involves an inflammatory process.

Introduction

Non-ruptured intracranial aneurysms that spontaneously occlude and regress are rare occurrences^{1,2}. The processes involved in both spontaneous occlusion and regression are not well understood. In this report we present the iconography of a non-ruptured intracranial aneurysm that underwent spontaneous occlusion and regression. At one point during this transformation, angiography with hyperselective catheterization yielded an image of the aneurysm as a pseudotumoral-like mass. We suppose that this image reflected inflammation and secondary neovascularization within the

aneurysm, providing a possible mechanism for aneurysmal healing. To our knowledge, detailed imagery capturing this phase of spontaneous aneurysmal healing has not been reported in the literature to date.

Case Report

A 28-year-old woman with no medical history was admitted to the emergency department for intense headache, acute episodes of blurred vision and dysphasia. MRI demonstrated a single aneurysm arising from a branch of the left insular segment of the middle cerebral artery. The lesion measured 1.3 cm and was surrounded by adjacent edema (Figure 1A). Angiography confirmed what appeared to be a patent, partially thrombosed fusiform aneurysm at a branch of the middle cerebral artery at the level of the left operculum (Figure 2A). Three days later, angiography performed before scheduled embolization revealed that the aneurysm had already undergone complete spontaneous thrombosis (Figure 2B). Twenty-four hours later, complaining only of mild headache, the patient was discharged following a baseline head CT scan.

Fifteen days after discharge, the patient was admitted once again to the emergency department for intense acute headache, blurred vision lasting 15 minutes and varying dysphasia. CT scan showed that compared to 15 days prior, there was decreased density of the aneurysmal body as well as increased perilesional hypodensity. Two days later, an MRI reconfirmed the

Abbreviation Key:

MRI: Magnetic Resonance Imaging;

CT: Computed Tomography;

MCA: Middle Cerebral Artery;

M2: Sylvian Segment of the Middle Cerebral Artery;

M3: Cortical Segment of the Middle Cerebral Artery.

Figure 1 A) MRI (on day 1) demonstrating a 1.3 cm extra-axial lesion, with minimal surrounding edema, corresponding to a partially thrombosed aneurysm which remains patent at a branch of the left insular middle cerebral artery. B) MRI (on day 22) reconfirming the presence of the aneurysm with perilesional edema. C) MRI (on day 27) showing a decrease in aneurysm size and perilesional edema compared to five days prior. D) MRI (11 months after discharge) revealing near complete regression of the aneurysm.

Figure 2 A) Angiography (on day 1 of first admission) confirming a partially thrombosed fusiform aneurysm measuring 12×7 mm at a branch of the middle cerebral artery at the level of the left operculum. An arrow points to the parent artery feeding the aneurysm. B) Angiography (on day 4) before scheduled embolization revealing complete spontaneous thrombosis of the aneurysm. An arrow points to the parent artery that is now thrombosed at its distal end. C) Angiography (on day 25) with hyperselective catheterization of the left MCA parent artery revealing both generalized opacification and probable neovascular arborization within the fusiform aneurysm. The aneurysm does not appear to supply the normal distal circulation of the parent artery. An arrow points to the parent artery that now feeds the pseudotumoral-like mass.

presence of an aneurysm with perilesional edema (Figure 1B). The MRI was followed directly by an angiography with selective contrast injection at the left internal carotid artery. This examination depicted a “blush-like” parenchymography of the fusiform aneurysm which remained occluded. Seventy-two hours later, with the intent to coil both the aneurysm and parent artery, repeat angiography under general anaesthesia was performed. Hyperselective catheterization of the left MCA parent artery revealed that the aneurysm had taken on an atypical pseudotumoral-like form (Figure 2C). No embolization was performed. MRI 48 hours later showed that compared to the MRI taken five days prior, both the aneurysm and perilesional

edema had decreased (Figure 1C). Throughout her admission, the patient remained stable complaining only of tension type headache and was discharged after a total of seven days in hospital. Follow-up MRI was obtained 11 months later, which revealed near complete regression of the aneurysm (Figure 1D).

Discussion

The spontaneous complete occlusion of a cerebral aneurysm is an infrequent event, whose precise mechanisms remain uncertain¹⁻⁴. Many effects, such as endothelial damage within the aneurysm, increased coagulability within the an-

eurysm as well as the ratio between aneurysmal neck and body size have all been theorized as contributing factors⁴⁻⁷. Spontaneously occluding cerebral aneurysms can induce edema leading to mass effect. The accumulation of thrombotic material within a thrombosed aneurysm, neovascularization and secondary intramural hemorrhage, as well as the development of intrathrombotic capillary channels have all been hypothesized as causes of this mass effect⁸⁻¹². Interventions performed for symptomatic aneurysms include bypass procedures, proximal clip application or endovascular occlusion followed by aneurysmectomy^{10,13}. We however did not perform endovascular occlusion upon hyperselective catheterization because the image of the aneurysm was particularly unexpected and atypical (Figure 2C). Endovascular occlusion of the unanticipated mass was considered, however it was ultimately decided that the most sensible approach would be to follow the evolution of the lesion with control MRI. Because no biopsy and histological specimen were available for analysis, it is impossible to know with certainty the mechanisms underlying the pseudotumoral aspect seen in Figure 2C. It is unlikely that this image represented recanalization and reactivation of the aneurysm, for the flow within the aneurysm did not join the distal arterial circulation and the aneurysm eventually under-

went gradual regression (Figure 1). It is more probable that the aneurysm had undergone inflammation and secondary neovascularization within its wall and body. We assume the “blush-like” image obtained prior to hyperselective catheterization was created by the *vasa vasorum* supplying the wall of the inflamed aneurysm. Inflammation induced neovascularization is associated with intramural hemorrhage and the growth of giant cerebral aneurysms¹². Why in this case a presumably equivalent inflammatory mechanism led to healing, as opposed to hemorrhage and growth, remains unknown.

Conclusion

The spontaneous occlusion and regression of a cerebral aneurysm is not only an exceptional and unpredictable event, but is one whose mechanisms are not well understood. This report depicts unique images of what appears to be inflammation and secondary neovascularization in an aneurysm that spontaneously regressed. Despite the fact that no firm recommendations can be induced from a single patient, this case supports the theory of inflammation and secondary neovascularization as a mechanism of healing in spontaneously thrombosed intradural aneurysms.

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