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CASE REPORT

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Severe Inflammatory Reaction of the Optic System after Endovascular Treatment of a Supraophthalmic Aneurysm with Bioactive Coils

SUMMARY: We report on a patient who, after a symptom-free interval, developed severe vision impairment and whose MR imaging demonstrated extensive edema in the central nervous tissue neighboring the treated aneurysm. To our knowledge, this is an unreported complication of endovascular treatment of aneurysms.

The number of supraophthalmic internal carotid artery (ICA) aneurysms treated via an endovascular approach has significantly increased. Giant aneurysms over 25 mm seem to be more often associated with ophthalmic symptoms.¹ We report on a patient who, after a symptom-free interval, developed severe vision impairment and whose MR imaging demonstrated extensive edema in the central nervous tissue neighboring the treated aneurysm as a thus far not reported complication of endovascular treatment of aneurysms.

Case Report

A 46-year-old woman underwent MR imaging for unspecific headaches. No impairment of the visual system was present. In an otherwise normal MR imaging finding, an incidental supraophthalmic aneurysm of the right ICA was suspected. Digital subtraction angiography (DSA) confirmed 2 aneurysms of the right ICA (Fig 1A). The larger aneurysm measured 11 × 9 × 8 mm. A second medially located cavernous aneurysm measured 5 mm. Other aneurysms were not present.

In accordance with the International Study of Unruptured Intracranial Aneurysms,² elective coiling of both aneurysms was planned. After the first framing coil (Guglielmi detachable coil 18, 360°, 10 mm × 30 cm; Boston Scientific, Natick, Mass), 8 bioactive coils (Matrix detachable coil, Boston Scientific), measuring between 8 mm × 20 cm and 2 mm × 8 cm, were deployed into the aneurysm. The second aneurysm was coiled with 2 bare platinum coils (Protégé, ev3, Plymouth, Minn). No thromboembolic complications occurred during the intervention. Both aneurysms were successfully treated with coils. The ophthalmic artery remained patent.

Immediately after treatment, the patient did well and had no neurologic symptoms. During the following day, she had loss of the visual field in the right eye. Ophthalmologic examination on the fourth postinterventional day showed complete loss of the temporal visual field with residuals of the nasal and central visual field. No oculomotor disturbances were present.

MR imaging (Fig 1B, -C) on the fourth day following intervention demonstrated thickening of the optic chiasm with severe edema that extended into the right optic nerve and both optic tracts. The post-

contrast images showed enhancement in the chiasm close to the large aneurysm and in the adjacent basal brain parenchyma.

Steroid therapy was started with 12-mg dexamethasone per day. The patient recovered during the course of 7 days, and visual loss rapidly and completely resolved. MR imaging 9 days after the intervention showed a nearly complete regression of chiasmal swelling (Fig 1E, -F). Contrast enhancement decreased as well. Steroid medication was continued with 8-mg dexamethasone per day and was tapered to zero in the following 3 weeks.

Discussion

In the presented case, endovascular coiling of an aneurysm led to edema, swelling, and disturbances of the blood-brain barrier of central nervous tissue close to the treated aneurysm. It is not yet clear which pathophysiologic condition leads to this phenomenon, but the improvement following administration of steroids suggests an inflammatory reaction. The concept of endovascular treatment of an aneurysm is based on the occlusion of the aneurysm with thrombus and organization of the thrombus within the aneurysm during the course of the following weeks. Immediately after endovascular treatment with coils, thrombus formation begins within the aneurysm due to absent or turbulent flow or thrombogenicity of the surface of the coils. In the thrombus-formation phase, activated platelets release different cytokines.³ In the following days, immune cells migrate into the thrombus and induce an inflammatory response. Especially, macrophages stimulate fibroblasts, which promote the conversion of thrombus to fibrous tissue. This healing process is known to take weeks to months.^{4,5}

In our patient, we saw a subacute postinterventional inflammatory reaction, which started clinically 1 day after treatment and could be documented by MR imaging on the fourth day. Edema, contrast enhancement, and the immediate clinical and morphologic response toward the steroid treatment proved the inflammatory etiology of the observed reaction. We propose 3 potential mechanisms to explain this early inflammatory response during the thrombus-formation period.

First, coiling may lead to a slight increase of the aneurysm diameter and, therefore, lead to mechanical irritation of adjacent structures by increasing the mass effect. In MR imaging, however, there was no measurable difference in aneurysm size. In the literature, there are hints that the mass effect of an aneurysm can persist after coiling,⁶ but, to our knowledge, there are no reports of an acute new mass effect immediately after coiling.

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Fig 1. A, Conventional DSA of the right ICA. The finding is a large supraophthalmic aneurysm (arrowheads) and a second medially cavernous aneurysm (double arrow). D, DSA after coil occlusion of both aneurysms. B, C, E, and F, T2-weighted and T1-weighted contrast-enhanced images 4 days (B and C) and 9 days (E and F) after intervention. MR image at the fourth postinterventional day shows edema in both optic tracts and thickening and signal-intensity elevation in the right optic nerve and chiasm (double arrows in B). C, T1-weighted postcontrast image shows enhancement at the rim of the aneurysm and the chiasm. MR images 9 days postintervention (E and F) show that edema and swelling have regressed, as well as the enhancement.

Second, the phase of acute platelet aggregation and thrombus formation may lead to an inflammatory reaction due to the known cytokine release from activated platelets.³ This might suggest that the amount of thrombus within the aneurysm after treatment plays an important role. In our patient, we achieved a complete occlusion of the large aneurysm. One may presume that the fast occlusion of the aneurysm led to rapid thrombosis within the aneurysm with consecutive fast cytokine release. However, because our aneurysm was not exceedingly large or otherwise unusual, we cannot explain why the observed reaction does not happen more often.

A third possible stimulus could be the coil material itself. In our case, we used a combination of bare platinum and polyglycolic/polylactic acid (PGLA)-coated Matrix coils. The PGLA coating degrades and releases lactic and glycolic acid. The latter should increase proliferation of fibroblasts and collagen release.^{7,8} This degradation process of Matrix coils is supposed to start slowly after treatment and is reported to take approximately 12 weeks.⁹ If this description of the process was true, degradation products could not be the noxious stimulus for our observed acute inflammatory reaction. Another possibility could be the induction of a foreign-body reaction with inflammatory changes, which involve the adjacent tissue.

It is not clear which mechanism mediated the observed inflammatory reaction of the optic system. Potential causes include thrombus formation and cytokine release after aneu-

rysm occlusion, mass effect of the implanted coils, and a reaction to the PGLA coating of Matrix coils. The aim of this report was, however, to raise the awareness toward this yet unreported complication of coiling with bioactive coils.

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