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Neurovascular Compression Syndrome after Coiling Intracranial Aneurysm

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Abstract

Objectives: Endovascular treatment of intracranial aneurysms using detachable coils is an established method by interventional radiologists. Next to prevention of subarachnoidal hemorrhage, prospective and retrospective studies have shown relief of symptoms caused by the mass effect of the aneurysm following this treatment.

Patients and methods: We present cases of endovascular treated intracranial aneurysms in patients developing focal neurological symptoms due to a local perianeurysmal inflammation. Furthermore, we review the literature to increase awareness of this complication, its pathophysiology and therapeutic options.

Results: Only rare cases of local perianeurysmal inflammation have been reported in literature. Clinical symptoms are heterogenous, up to focal seizures or symptoms of acute hydrozephalus. Pathophysiological, thrombembolism, local inflammatory and mass are possible aetiological factors. However, overall long-term prognosis is good.

Conclusion: Neurovascular compression syndrome after intracraniel aneurysm coiling is a rare and possibly delayed complication. With regard to various causes, diagnostic and therapeutic options should be considered.

Keywords: Neurovascular; Intracranial aneurysma; Inflammatory; Vertebrobasilar circulation

Abbreviations: ADC: Apparent Diffusion Coefficient; CSF: Cerebrospinal Fluid; CT: Computed Tomography; CTA: Computed Tomography Angiography; DSA: Digital Subtraction Angiography; DWI: Diffusion-Weighted Imaging; FLAIR: Fluid Attenuated Inversion Recovery; FLASH: Fast Low Angle Shot; IV: Intravenous; MCA: Middle Cerebral Artery; MRA: Magnetic Resonance Angiography; MRI: Magnetic Resonance Imaging; PCA: Posterior Cerebral Artery; rtPA: Recombinant Tissue Plasminogen Activator; TCD: Transcranial Doppler Sonography; TOF: Time-of-Flight

Introduction

Endovascular treatment of aneurysms using detachable coils has evolved as a preferred alternative to classical surgical clipping to prevent rupture and subarachnoidal haemorrhage. For aneurysms located within the vertebrobasilar circulation, in particular, this treatment is first choice. Coil-induced closure of the aneurysm sac finally may reduce the lesion's mass effect on surrounding tissue with a consequent reduction in neurological symptoms, if symptoms due to mass effect occurred. Most reported complications of endovascular coiling include coil displacement, aneurysm rupture or puncture, artery dissection, and cerebral embolism. Only anecdotal information exists on progression of aneurysm size, local inflammation, and abscess formation after endovascular treatment. First, we present the case of a 49-year-old woman in whom a complex set of complications arose after aneurysm coiling and discuss current views on the subject and possible therapeutic options for rare side effects. In a second case we report about a 62-year-old man suffering from reduced consciousness, hemiparesis and dysarthria about three weeks after successful coiling of two intracranial aneurysms.

Patients and Methods

Case 1: A 49-year-old woman was admitted to the hospital for mild right-sided hemiparesis due to an ischemic stroke in the subcortical white matter of the left hemisphere, which was classified as a lacunar stroke. During a diagnostic workup an asymptomatic aneurysm of the basilar artery was found (Figure 1). Diagnostic intra-arterial digital subtraction angiography (DSA), performed before interventional treatment, revealed that the aneurysm measured 9 × 6 mm and was accompanied by a small daughter aneurysm. Coils were placed in successful in both aneurysms. At the end of the procedure, a small coil loop could be seen to protrude from the larger compartment overlapping the small side aneurysm (Figure 1). Eight hours after the intervention the patient developed bilateral mydriasis, which was followed by a progressive loss of consciousness. Emergency cerebral CT scanning with CTA ruled out the presence of aneurysm bleeding or ruptures but showed partial obliteration of the P_1 section of both posterior cerebral arteries (PCAs). IV treatment with the gpIIb/IIIa inhibitor (tirofiban) was introduced. Immediate DSA confirmed the findings of the CTA with intraluminal sparing of contrast media, which were suggestive of fresh thrombotic material in both PCAs (Figure 2). After local thrombolytic treatment with 4 mg rtPA, the patient recovered well. MRI revealed multiple small ischemic lesions with haemorrhagic transformation within the cerebellum and PCA territories; however, the patient showed no neurological deficits and was discharged from the hospital 5 days later. The patient was sent home on a regimen of acetylsalicylic acid (100 mg/day) and low-molecular-weight heparin (certoparin) for thrombosis prophylaxis. One week later the patient presented at the emergency room with progressive headaches and undulating double vision as well

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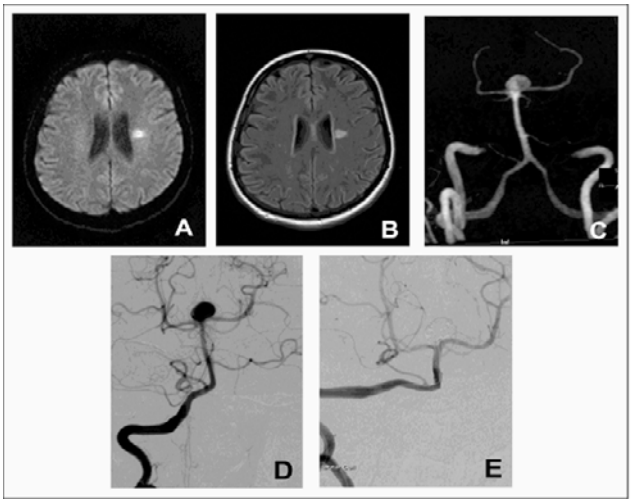

Figure 1: First case: 49-year-old woman after lacunar stroke (Figure 1A+B) with two-asymtomatic aneurysm of the basilar artery (Figure 1C+D). After coiling of both (Figure 1E) the patients developed bilateral mydriasis and loss of consciousness.

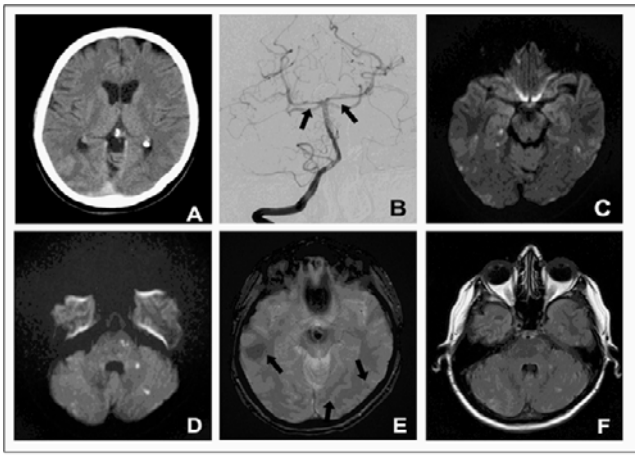

Figure 2: Native CT scan ruled out intracranial bleeding (Figure 2A). DSA and MRI scan revealed ischemic lesion in vertebrobasilar territory, PCA (Figure B-F).

as right-sided mydriasis (Figure 3). A complete workup showed only a mildly elevated red blood cell count in CSF obtained from lumbar puncture and no sign of a disturbance in the blood-brain barrier. MRI revealed mesencephalic oedema on FLAIR sequence without changes on DWI or ADC (Figure 4). Over the course of the next week, the patient's double vision worsened due to left-beating nystagmus (Figure 3). Despite escalated anti-inflammatory and anti-oedematous therapy consisting of steroids and glycerol, follow-up MRI showed progressive mesencephalic oedema and, finally, evidence of ischemic stroke (Figure 4). The patient's symptoms gradually stabilized. She was transferred to a neurological rehabilitation centre with persistent double vision and was given antithrombotic therapy consisting of clopidogrel and certoparin. At the 3-month follow-up examination, the woman's oculomotor palsy was no longer detectable, but her intermittent double vision while looking to the left still persisted (Figures 3C and D). MRIs indicated a small post-ischaemic mesencephalic defect with no further evidence of oedema.

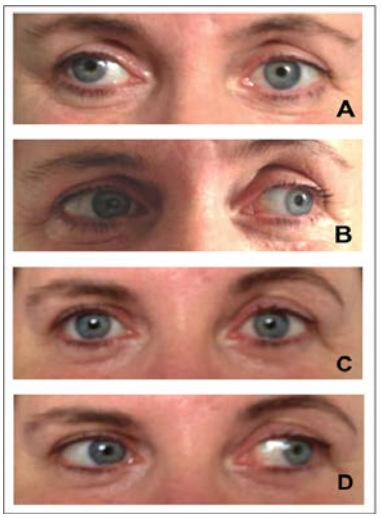

Figure 3: After one week undulating double vision and right sided mydriasis (A-D) without ischemic or inflammation correlate with the first MRI scan.

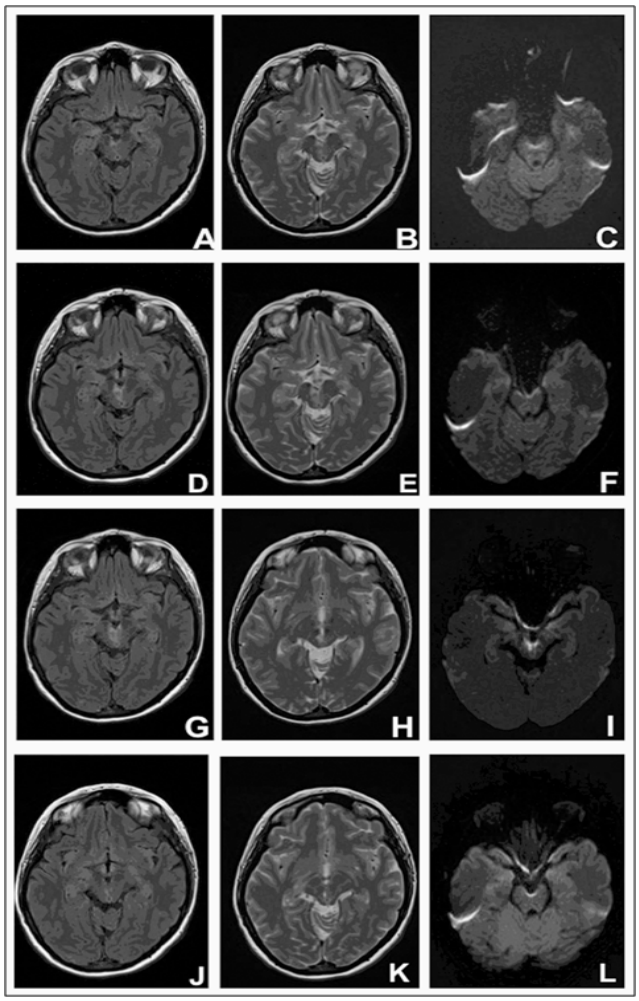

Figure 4: In follow up after one week with worsened symptoms enhanced mesencephalic oedema and ischemic stroke were seen (Figure 4A-L).

Case 2: A 62-year-old male patient presented with reduced consciousness, right-sided hemiparesis and dysarthria. About three weeks before onset, two intracerebral aneurysms were coiled without complications, one at the top of the basilar artery (Figures 5A and 5C) and another at the left MCA (Figures 5B and 5C). CT scan was negative for intracranial haemorrhage. CSF was also negative for detection of haemorrhage, but showed elevated cell count. Empiric antibiotic treatment with a regime of ceftriaxone and ampicillin, later furthermore metronidazole and fosfomycin, next to an anti-oedematous treatment with methylprednisolone was initiated. In further work-up, MRI scan showed regular position of both coil packages and a local lesion nearby the coil of the left ACM aneurysm (Figure 5D). A new ischemic lesion could not be detected. After medical treatment over two weeks, clinical recovery and reduction of the inflammatory lesion in MRI were seen (Figure 5E). The patient was discharged after restitutio ad integrum. Occasional amnestic aphasia lasting for few seconds could be correlated with pathological EEG transformation over the left temporal lobe. Under medical treatment in a 5-year follow up, the patient presented stable results in clinical examination, MRI and transcranial ultrasound.

Results

The first post-interventional set of complications observed in the first patient still has not been explained satisfactorily. Enhanced clotting during the endovascular procedures and the primary stroke made us screen for a coagulation abnormality, which seems likely but nevertheless remains unproven. There was no thrombotic event in the patient's history nor in her family. All known and measurable coagulopathies including Fabry's disease were ruled out. The embolic pattern of the lesions on DWI and the fact that the lesions were located significantly below the aneurysm neck make it unlikely that the coils caused the thrombus formation. We therefore propose underlying endothelial hypersensitivity as a possible cause of the lesions, which would also reflect the early haemorrhagic transformation of these lesions as seen in (Figure 2E). Double vision lasting only for seconds to a few minutes is more likely explained by a malfunction of the third cranial nerve caused by pulsation of the coiled aneurysm; this is similar to the neurovascular compression syndromes known to occur with the 5th cranial nerve. In 1991 Kwan et al. reported an increase in aneurysm size due to what they called the "water-hammer effect" [1] of pulsatile blood flow from the basilar artery. In the present case there was no evidence of an increase in aneurysm size, incomplete coil packing, or space between aneurysm and coils, which makes this hypothesis unlikely. We discussed the possibility of a neurovascular compression syndrome and were considering placing the patient on antiepileptic medication to treat her symptoms when the last set of complications began. Over time we observed progressive neurological symptoms, which could be explained by an impairment of the tractus lemniscus medialis. At that point in our diagnostic investigation, neurovascular compression as the sole pathological mechanism appeared unlikely. The damage to the tractus was best explained by local inflammation of the aneurysm wall leading to mesencephalic oedema. Horie et al. observed similar oedema, but in their patients the aneurysms had not been completely obliterated [2]. Overall, the precise mechanism leading to obliteration of an aneurysm is still unclear, with opposing data obtained in humans [3,4] and mice [5] concerning the endothelium at the neck of the aneurysm (Table 1). Considering a normal response of thrombosis, the release of inflammatory cytokines after platelet activation [6] in a setting of critical-size of space occupying aneurysm could lead to an inflammatory response, which in turn leads to even greater thrombosis: [7,8] a vicious circle of acute thrombosis and worsening of neurological symptoms. Treatment options in this case seem to be high doses of steroid medications, as reported by Stracke et al. [9]. In the first reported case the presumed inflammation proved to be sterile, with no evidence of bacterial contamination of the coil package [10] as confirmed by an analysis of the patient's CSF. In the end we believe that a small enlargement of the aneurysm following coiling first caused a neurovascular compression syndrome and, over time, produced mesencephalic oedema via sterile neurovascular inflammation. In the second case also an inflammatory lesion after coiling might be responsible for diverse neurological deficits. The combination of local inflammatory lesion in MRI with contrast enhancement and elevated cell count in CSF is also suspect of post-interventional abscess, which could not be excluded retrospectively. Elevated CSF cell count can be found in local inflammation as well. Finally, medical treatment with antibiotical and anti-inflammatory regime was successful. Possible reasons for neurovascular inflammation after coiling or implementation of other devices and stents might be contamination of the material, mechanical irritation or hypersensitivity.

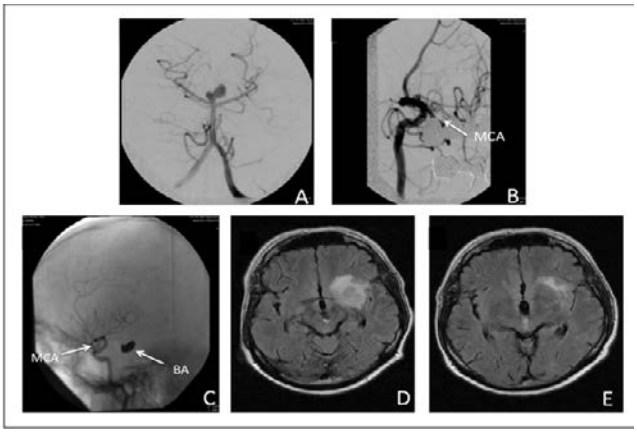

Figure 5: Case 2. 62-year-old patient with two intracerebral aneurysms one at the top of BA, another at left MCA (Figure 5A-C). Three weeks after coiling upcomin neurological deficites. MRI showed perianeurysmal inflammatory lesions responsive to antibiotic and antioedematous treatment.

However, only rare information about this complication exists in literature. Skolarus et al. describe white matter changes after aneurysm coiling and hypothesize an exuberant inflammatory response related to implementation of polyglycolic-polylactic acid coils, however, bioactive coils were not used in this case [11]. Despite escalating anti-inflammatory and anti-oedematous therapies, the oedema persisted and eventually a small mesencephalic stroke occurred. Whether this stroke was caused by obliteration of small perforating arteries or traction of arteries due to the mass effect of the aneurysm, or whether it was due to direct compression or indirect compression resulting in venous congestion is speculative [12,13]. Especially in the first case obliteration of the aneurysm led to a more pronounced inflammatory mass effect. This is in contrast to the effect itself mostly described in the literature: Alleviation of compression by reducing pulsation of the aneurysm wall [14,15]. We were fortunate that we never were forced to consider the *ultimo ratio* treatment option-bilateral occlusion of the vertebral artery [16]. In addition, coil removal to prevent a progressive unstoppable mass effect was never part of our discussion [17,18].

We reviewed the literature to see whether a surgical approach may have been a wiser treatment plan. We found no clear preference for surgical clipping or endovascular treatment with respect to the critical size of an aneurysm, localization of the lesion, or already existing

Author	Case/study	Paper	Journal	year	Diagnosis	Treatment /problem	Treatment complication	#pat.
Cohen JE	Case	Postembolization perianeurysmal edema as a cause of uncinate seizures.	J Clin Neurosci.	2012	giant unruptured supraclinoid aneurysm	endovascular embolization , bare coils and implantation of a flow diverterstent	Uncinated seizures, successfully treated by steroids	1
Vu Dang L	Case	Post-embolization perianeurysmal edema revealed by temporal lobe epilepsy in a case of unruptured internal carotid artery aneurysm treated with bare platinum coils.	J Neuroradiol.	2009	unruptured internal carotid artery (ICA) aneurysm	bare platinum coils	Seizures	1
I. Craven	Case	Symptomatic Perianeurysmal Edema Following Bare Platinum Embolization of a Small Unruptured Cerebral Aneurysm	AJNR Am J Neuroradiol.	2009	relatively small aneurysm (7 mm)	bare platinum coils	2 nocturnal seizures (tonic-clonic) and further daytime complex partial seizures	1
Fanning NF	Paper	Wall enhancement, edema, and hydrocephalus after endovascular coil occlusion of intradural cerebral aneurysms.	J. Neurosurg	2008	intradural cerebral aneurysms	bare platinum or modified platinum coils	Asymptomatic aneurysm wall enhancement perianeurysmal edema, and hydrocephalus, occurred in 18.6% of embolizations performed with bare platinum coils	181
Misaki K	Case	Unusual delayed hydrocephalus after bare platinum coil embolization of an unruptured aneurysm.	Neurol Med Chir (Tokyo).	2010	incidentally detected unruptured large internal carotid artery aneurysm	bare platinum coils	wall enhancement and perianeurysmal brain edema. hydrocephalus including disorientation, gait disturbance, and urine incontinence at 7 months post-embolization	1
Horie	Case + Review	Progressive perianeurysmal edema induced after endovascular coil embolization. Report of three cases and review of the literature.	J. Neurosurg	2008		endovascular coil embolization	progressive vasogenic brain edema surrounding a cerebral aneurysm. incomplete occlusion of larger aneurysms may lead to a disorganized intraluminal thrombosis, aneurysm pulsing, and intramural hemorrhage or inflammation	3
Marden FA	Case	Perianeurysm edema with second-generation bioactive coils	Surg Neurol.	2008	unruptured, nongiant, saccular aneurysm	embolization with Matrix2 coils	perianeurysm edema	1
Stracke CP	case	Severe inflammatory reaction of the optic system after endovascular treatment of a supraophthalmic aneurysm with bioactive coils.	AJNR Am J Neuroradiol.	2007	2 aneurysms of the right ICA	endovascular coiling	after a symptom-free interval, developed severe vision impairment, extensive edema in the central nervous tissue neighboring the treated aneurysm	1
Deus-Silva L	Case	Severe aggressive acute disseminated encephalomyelitis-like reaction after aneurysm coiling	Neurosurgery.	2010	incidental 4 x 6-mm anterior communicating complex aneurysm		severe brain edema induced by contrast media and resembling an aggressive acute disseminated encephalomyelitis-like reaction with global aphasia, dysarthria, right upper motor neuron pattern facial paresis, and right hemiplegia and hemianesthesia.	1
Sim KJ	Retrospective analysis	Intracranial aneurysms with perianeurysmal edema: Long-term outcomes post-endovascular treatment.	J Neuroradiol	2014		838 patients, endovascularly treated aneurysms from January 2001 to December 2012,	Ten of the aneurysms treated with endovascular therapy demonstrated perianeurysmal edema, mostly asymptomatic	838
Su IC	retrospective analysis	Aneurysmal wall enhancement and perianeurysmal edema after endovascular treatment of unruptured cerebral aneurysms	Neuroradiology	2014		wall enhancement and perianeurysmal edema on MRI after endovascular treatment were analyzed	Eighty-five (64.4 %) aneurysms had wall enhancement, and 9 (6.8 %) aneurysms had perianeurysmal brain edema	124
White JB	Case	But Did You Use HydroCoil? Perianeurysmal Edema and Hydrocephalus with Bare Platinum Coils	AJNR Am J Neuroradiol.	2008		Bare platinum coils	headache, dysequilibrium, abducens palsy, and internuclear ophthalmoplegia. Hemorrhage. Hydrocephalus	2
Ushikoshi S	Case	Aggravation of brainstem symptoms caused by a large superior cerebellar artery aneurysm after embolization by Guglielmi detachable coils--case report.	Neurol Med Chir (Tokyo)	1999		Guglielmi detachable coils	aggravation of right oculomotor nerve paresis and left hemiparesis	1

Ozaki M	Case	Delayed hydrocephalus after embolization of unruptured aneurysms using bare platinum coils: report of 2 cases	AJNR Am J Neuroradiol.	2011	unruptured intracranial aneurysms	bare platinum coils	delayed hydrocephalus	2
Mitha AP	Case	Communicating hydrocephalus after endovascular coiling of unruptured aneurysms: report of 2 cases.	J Neurosurg.	2008	unruptured aneurysms	endovascular coiling, hydrogel-coated coils	delayed communicating hydrocephalus	2
Marchan EM	Retrospective	Hydrogel coil-related delayed hydrocephalus in patients with unruptured aneurysms.	J Neurosurg	2008	unruptured cerebral aneurysms	bare platinum (n=26) and hydrogel coils (n=29)	14% incidence (95% confidence interval 3.9-31.7%) of symptomatic hydrocephalus 2-6 months after the intervention with hydrogel coils	55
Turner RD	case	Delayed visual deficits and monocular blindness after endovascular treatment of large and giant paraophthalmic aneurysms	Neurosurgery	2008	paraophthalmic region aneurysm	coil embolization, hydrogel-coated and bare platinum coils	delayed onset of vision loss	6
Meyers PM	Case	Chemical meningitis after cerebral aneurysm treatment using two second-generation aneurysm coils: report of two cases.	Neurosurgery.	2004	large cerebral aneurysms	second-generation aneurysm coils	evelopment of symptomatic nonbacterial meningitis	2
Dönmez H	Case	Stroke secondary to aseptic meningitis after endovascular treatment of a giant aneurysm with parent artery occlusion	Cardiovasc Intervent Radiol.	2009	giant aneurysm	bare platinum coils	aseptic meningitis causing brain stem and cerebellar infarct	1
Studley MT	Case	Delayed thromboembolic events 9 weeks after endovascular treatment of an anterior communicating artery aneurysm: case report.	AJNR Am J Neuroradio	2002	anterior communicating artery aneurysm	12 Guglielmi detachable coils	Thromboembolic events, stroke	1

Table 1: Overview on literature.

neurological symptoms. In 1994 Van Halbach and Higash showed that endovascular treatment reduces a mass effect [19], whereas in 2004 Heran et al. reported the opposite, favouring surgical clipping in cases in which a mass effect was present [20]. Regarding critical anatomical locations, in 2011 Schuss et al. reported a small series of aneurysms affecting the visual system; these authors found that clipping could be superior to endovascular treatment in reducing a mass effect. Concerning treatment complications most available reports are cases describing clinical symptoms like seizures or symptoms of hydrozephalus after coiling. Three larger studies found post interventional wall enhancement and oedema of the coiled aneurysma in between 6.8 and 18.6% [21-35].

In summary, there are four main complications described in literature:

- Thrombembolism as a cause of stroke from aneurysmal neck
- Inflammatory response due to coiled and thrombosed aneurysma
- Mass effect
- Local inflammation by chemical ingredients or contamination of the coils. These cases emphasize again the necessity of multimodal treatment choices in patients with complicated aneurysms of the basilar artery that exert a mass effect, both with regard to the choice of treatment and the management of complications like seizures, perianuerysmal edema and hydrocephalus.

Conclusion

Local symptomatic perianeurysmal oedema with neurovascular compression syndrome is a rare complication of endovascular treatment and has to be considered when patients display neurological symptoms, especially in space-occupying aneurysms. The exact pathophysiology of this complication remains nebulous; however, contamination of the coil, thrombosis, local cytokine release by mechanical irritation and a specific reaction to the coil material may be responsible. Administration of corticosteroids, platelet inhibitors, and hyperosmolar therapy may be therapeutic and, overall, the long-term prognosis for these patients is good.

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Conflict of Interest

The authors report no conflicts of interest related to this study or preparation of the paper.

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