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 subarachnoid 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 heterogeneous, up to focal seizures or symptoms of acute hydrocephalus. Pathophysiological, thrombembolism, local inflammatory and mass are possible aetiological factors. However, overall long-term prognosis is good.

Conclusion: Neurovascular compression syndrome after intracranial 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; 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; TOF: 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 subarachnoid 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 present the case of a 49-year-old woman 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 P1 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.

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.

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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.
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 restitution 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. In 1991 Kwan et al. reported an increase in aneurysm size of an aneurysm, localization of the lesion, or already existing surgical clipping or endovascular treatment with respect to the critical mass effect. This is in contrast to the effect itself mostly described 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...
<table>
<thead>
<tr>
<th>Author</th>
<th>Case/study</th>
<th>Paper</th>
<th>Journal</th>
<th>year</th>
<th>Diagnosis</th>
<th>Treatment /problem</th>
<th>Treatment complication</th>
<th>#pat.</th>
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<tbody>
<tr>
<td>Vu Dang L</td>
<td>Case</td>
<td>Post-embolization perianeurysmal edema revealed by temporal lobe epilepsy in a case of unruptured internal carotid artery aneurysm treated with bare platinum coils.</td>
<td>J Neuroradiol.</td>
<td>2009</td>
<td>unruptured internal carotid artery (ICA) aneurysm</td>
<td>bare platinum coils</td>
<td>Seizures</td>
<td>1</td>
</tr>
<tr>
<td>I. Craven</td>
<td>Case</td>
<td>Symptomatic Perianeurysmal Edema Following Bare Platinum Embolization of a Small Unruptured Cerebral Aneurysm</td>
<td>AJNR Am J Neuroradiol.</td>
<td>2009</td>
<td>relatively small aneurysm (7 mm)</td>
<td>bare platinum coils</td>
<td>2 nocturnal seizures (tonic-clonic) and further daytime complex partial seizures</td>
<td>1</td>
</tr>
<tr>
<td>Fanning NF</td>
<td>Paper</td>
<td>Wall enhancement, edema, and hydrocephalus after endovascular coil occlusion of intradural cerebral aneurysms.</td>
<td>J. Neurosurg</td>
<td>2008</td>
<td>intradural cerebral aneurysms</td>
<td>bare platinum or modified platinum coils</td>
<td>Asymptomatic aneurysm wall enhancement perianeurysmal edema, and hydrocephalus, occurred in 18.6% of embolizations performed with bare platinum coils</td>
<td>181</td>
</tr>
<tr>
<td>Misaki K</td>
<td>Case</td>
<td>Unusual delayed hydrocephalus after bare platinum coil embolization of an unruptured aneurysm.</td>
<td>Neurol Med Chir (Tokyo).</td>
<td>2010</td>
<td>incidentally detected unruptured large internal carotid artery aneurysm</td>
<td>bare platinum coils</td>
<td>wall enhancement and perianeurysmal brain edema. Hydrocephalus including disorientation, gait disturbance, and urine incontinence at 7 months post-embolization</td>
<td>1</td>
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<tr>
<td>Horie</td>
<td>Case * Review</td>
<td>Progressive perianeurysmal edema induced after endovascular coil embolization. Report of three cases and review of the literature.</td>
<td>J. Neurosurg</td>
<td>2008</td>
<td>endovascular coil embolization</td>
<td>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</td>
<td>3</td>
<td></td>
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<tr>
<td>Marden FA</td>
<td>Case</td>
<td>Perianeurysmal edema with second-generation bioactive coils</td>
<td>Surg Neurol.</td>
<td>2008</td>
<td>unruptured, nongiant, saccular aneurysm</td>
<td>embolization with Matrix2 coils</td>
<td>perianeurysmal edema</td>
<td>1</td>
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<tr>
<td>Stracke CP</td>
<td>case</td>
<td>Severe inflammatory reaction of the optic system after endovascular treatment of a supraophthalmic aneurysm with bioactive coils.</td>
<td>AJNR Am J Neuroradiol.</td>
<td>2007</td>
<td>2 aneurysms of the right ICA</td>
<td>endovascular coiling</td>
<td>after a symptom-free interval, developed severe vision impairment, extensive edema in the central nervous tissue neighboring the treated aneurysm</td>
<td>1</td>
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<tr>
<td>Deus-Silva L</td>
<td>Case</td>
<td>Severe aggressive acute disseminated encephalomyelitis-like reaction after aneurysm coiling</td>
<td>Neurosurgery.</td>
<td>2010</td>
<td>incidental 4 x 8-mm anterior communicating complex aneurysm</td>
<td>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.</td>
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<td>Su IC</td>
<td>retrospective analysis</td>
<td>Aneurysmal wall enhancement and perianeurysmal edema after endovascular treatment of unruptured cerebral aneurysms</td>
<td>Neuroradiology</td>
<td>2014</td>
<td>wall enhancement and perianeurysmal edema on MRI after endovascular treatment were analyzed</td>
<td>Eighty-five (64.4 %) aneurysms had wall enhancement, and 9 (6.8 %) aneurysms had perianeurysmal brain edema</td>
<td>124</td>
<td></td>
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<tr>
<td>White JB</td>
<td>Case</td>
<td>But Did You Use HydroCoil? Perianeurysmal Edema and Hydrocephalus with Bare Platinum Coils</td>
<td>AJNR Am J Neuroradiol.</td>
<td>2008</td>
<td>Bare platinum coils</td>
<td>headache, dysequilibrium, abducens palsy, and internuclear ophthalmoplegia. Hemorrhage. Hydrocephalus</td>
<td>2</td>
<td></td>
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<tr>
<td>Ushikoshi S</td>
<td>Case</td>
<td>Aggravation of brainstem symptoms caused by a large superior cerebellar artery aneurysm after embolization by Guglielmi detachable coils-case report.</td>
<td>Neurol Med Chir (Tokyo)</td>
<td>1999</td>
<td>Guglielmi detachable coils</td>
<td>aggravation of right oculomotor nerve paresis and left hemiparesis</td>
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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 hydrocephalus 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 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, perianeurysmal 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 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.

### References


