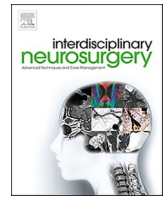




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## Delayed middle cerebral artery occlusion following endovascular coiling of carotid terminus aneurysm

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## ABSTRACT

Endovascular coil embolization of intracranial aneurysms is a relatively safe and effective alternative to traditional clip ligation. While bare platinum coils have long been used with good success, newer bioactive coils have been developed since approximately 2002 in an attempt to improve occlusion rates. Although unexpected complications such as aseptic meningitis, seizures, new-onset headaches, hydrocephalus, cranial nerve palsies and delayed visual compromise have been described following the use of both bare platinum coils and bioactive coils, we have not yet seen reports of delayed large vessel occlusion.

We describe an unusual case of an uncomplicated, elective endovascular coil embolization of a right internal carotid artery (ICA) terminus aneurysm using a combination of bare platinum coils and HydroCoils leading to the delayed occlusion of the proximal M1 segment of right middle cerebral artery (MCA) with no clinical sequelae.

### 1. Introduction

Endovascular coil embolization has proven to be a relatively safe and robust option for the treatment of intracranial aneurysms, and it has largely supplanted traditional clip ligation methods in many institutions [1–3]. In an effort to improve the durability of coil embolization of intracranial aneurysms, bioactive coils have been introduced as an alternative or adjunct to the traditional bare platinum coils [4]. Bioactive coils consist of a platinum core supplemented with either polyglycolic acid (*Cerecyte coil*), polyglycolic acid/lactide (*Matrix<sup>2</sup> coil*), or hydrogel (*HydroCoil*) [5]. Several studies have since demonstrated promising results with the addition of bioactive coils [6–10], though the optimal combination of bioactive and platinum coils remains unclear [11]. More recently, the HEAT trial demonstrated that small-to-medium aneurysms treated with at least 90% HydroCoils resulted in significantly less recurrence when compared to bare platinum coils without increased harm at two years [12].

The majority of coiling-related complications of elective endovascular procedures tend to occur intraoperatively or are detected shortly after the reversal of anesthesia [13]. Well recognized complications of intracranial aneurysm coiling include intraoperative aneurysm rupture, thromboembolism and stroke, and arterial dissection. More recently, there have been an increasing number of reports of perianeurysmal

edema sometimes with MRI enhancement suggesting a local inflammatory reaction. Such reactions are often self-limiting, though their implication in a number of unexpected complications such as aseptic meningitis, new-onset headaches and hydrocephalus cannot be excluded [14].

We describe a case of a 65-year-old female who underwent coil embolization of an unruptured right ICA terminus aneurysm and follow up imaging demonstrating complete occlusion of the proximal M1 segment of the right MCA.

### 2. Case illustration

#### 2.1. History and presentation

A 65-year-old female presented with left hand numbness for two weeks that started on the dorsal part of the hand and ascended up her arm to the shoulder. Her symptoms progressed to include fine motor difficulty and slight left-sided weakness of elbow flexion and extension, as well as wrist flexion and extension. Her past medical history is significant for hypertension, dyslipidemia, osteoarthritis and a previous history of smoking. An urgent CT/CTA showed normal parenchymal imaging with no mass lesion, infarct or hydrocephalus, however, the CTA revealed an incidental right ICA terminus aneurysm measuring 6.6

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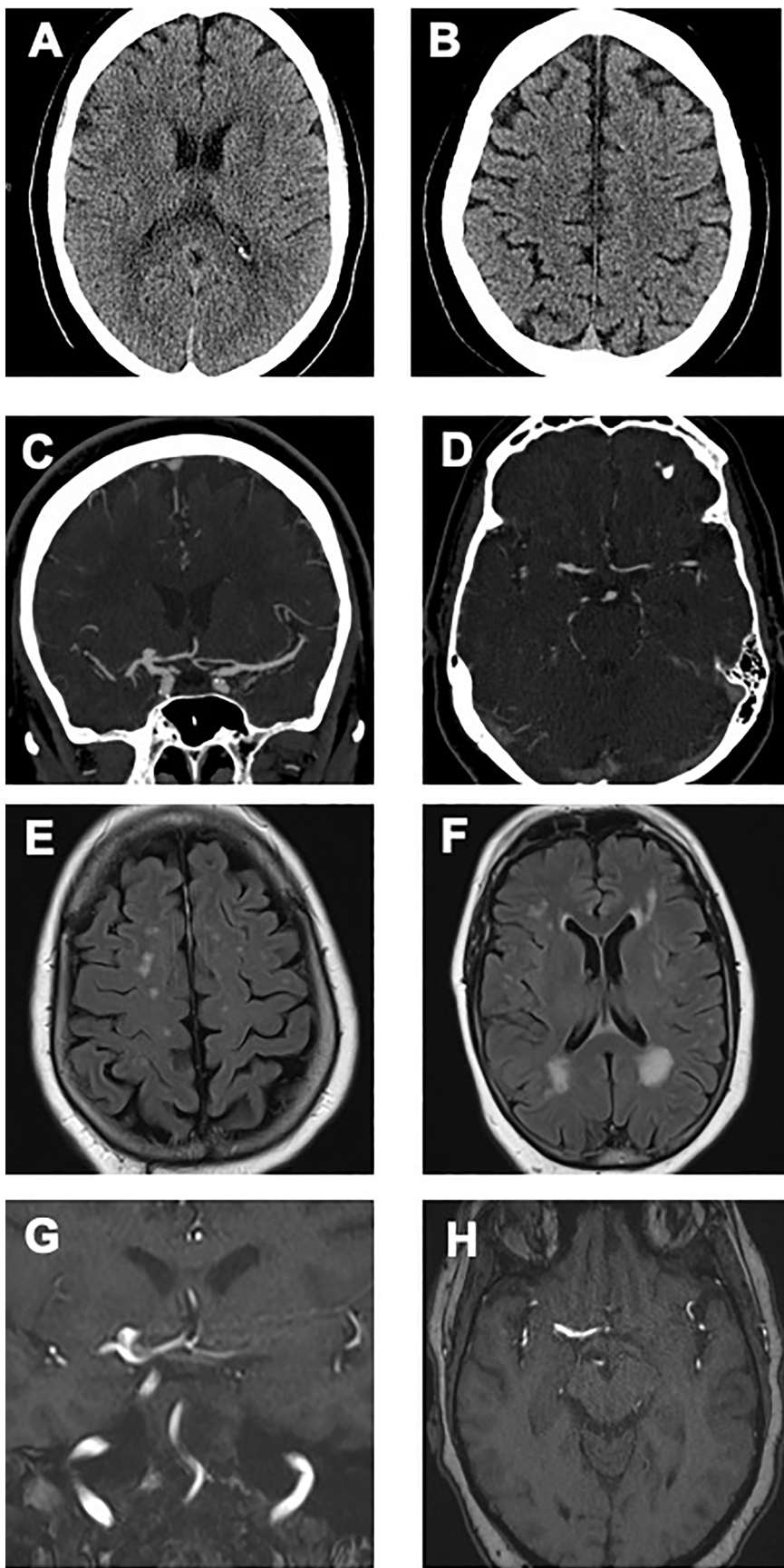
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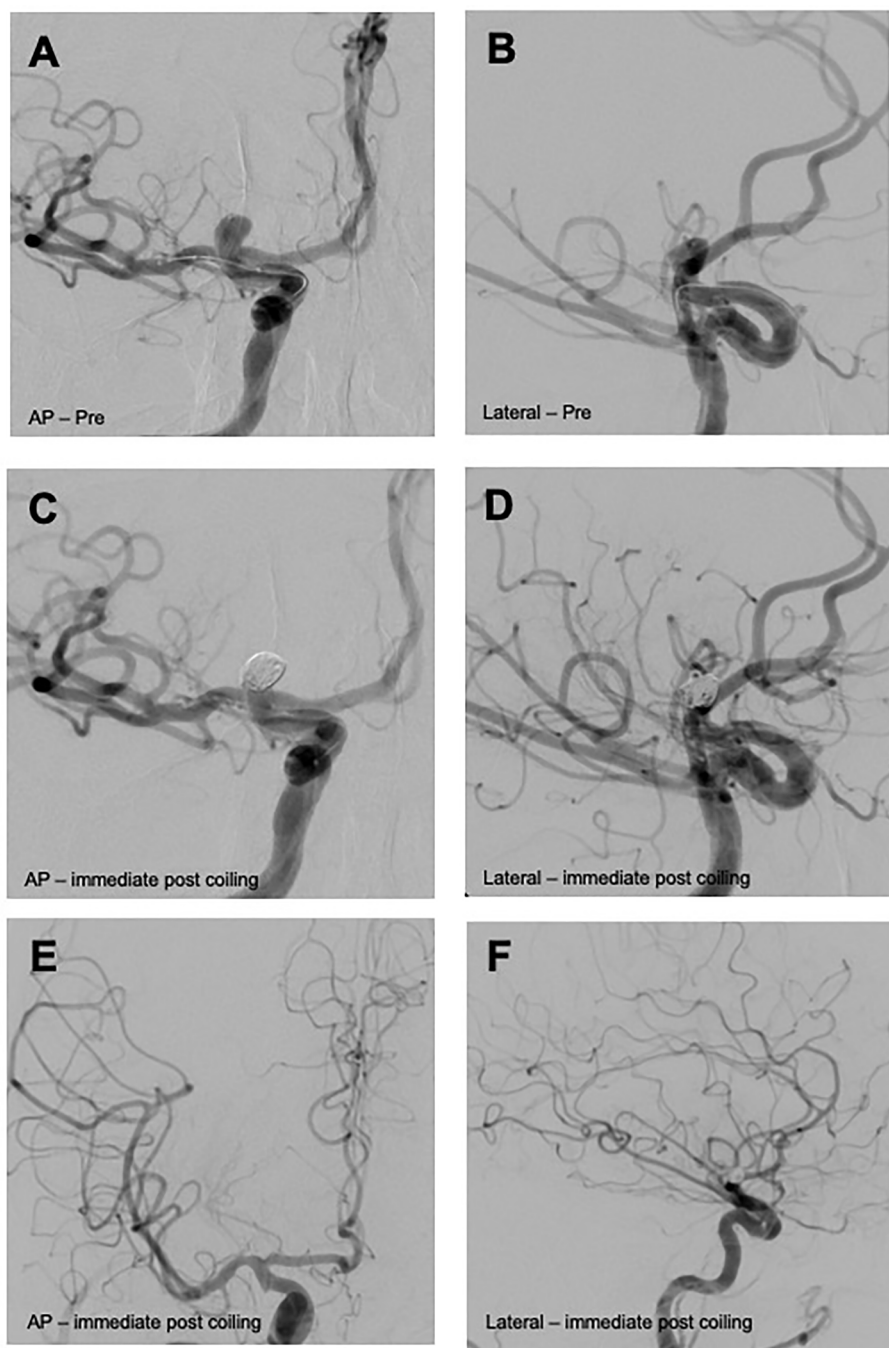
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**Fig. 1.** A 65-year-old female presents with an unruptured right ICA terminus aneurysm. Initial neuroimaging with CT/CTA. (A and B) axial non-enhanced cranial CT scan showing no obvious ischemic infarcts and normal ventricular calibre. (C and D) CT angiogram with coronal [C] and axial [D] views demonstrate an unruptured right ICA terminus aneurysm measuring approximately 5.5 mm and normal calibre of the right ICA, MCA and ACA. (E-H) The patient underwent follow up neuroimaging 3 months later with a non-enhanced cranial MRI/MRA showing a slight interval enlargement of the aneurysm. (E and F) axial T2 weighted sequences demonstrating bilateral supratentorial, subcortical white matter hyperintensities. (G and H) MRA time of flight (TOF) imaging with coronal (G) and axial (H) sequences demonstrates a small interval increase in size of the right ICA terminus aneurysm. Note: normal calibre of the distal supraclinoid right ICA, and proximal M1 segment of the right MCA and proximal A1 segment of the right ACA.



**Fig. 2.** Digital subtraction angiographic images of the aneurysm before and after balloon-assisted coil embolization of the right ICA terminus aneurysm. A and B: working projections used for the aneurysm coiling demonstrating the angioarchitecture of the saccular aneurysm and parent vessel. C and D: Successful coil embolization of the aneurysm via a balloon-assisted approach. (E and F): Post-coiling anteroposterior (E) and lateral views (F) demonstrating occlusion of the aneurysms and persevered flow in the right ICA, ACA and MCA proximal and distal vessels. Note: normal calibre of the right ICA, ACA and MCA.

mm  $\times$  4.5 mm with a 3.5 mm neck. The aneurysm displayed an irregular appearance and projected anterosuperior with a small daughter sac projecting medially and slightly increased in size with a follow-up MRI/MRA completed a few months after the initial CTA [Fig. 1]. Given the size and morphology of the aneurysm, as well as her associated risk factors, the patient was offered definitive treatment through endovascular coil embolization.

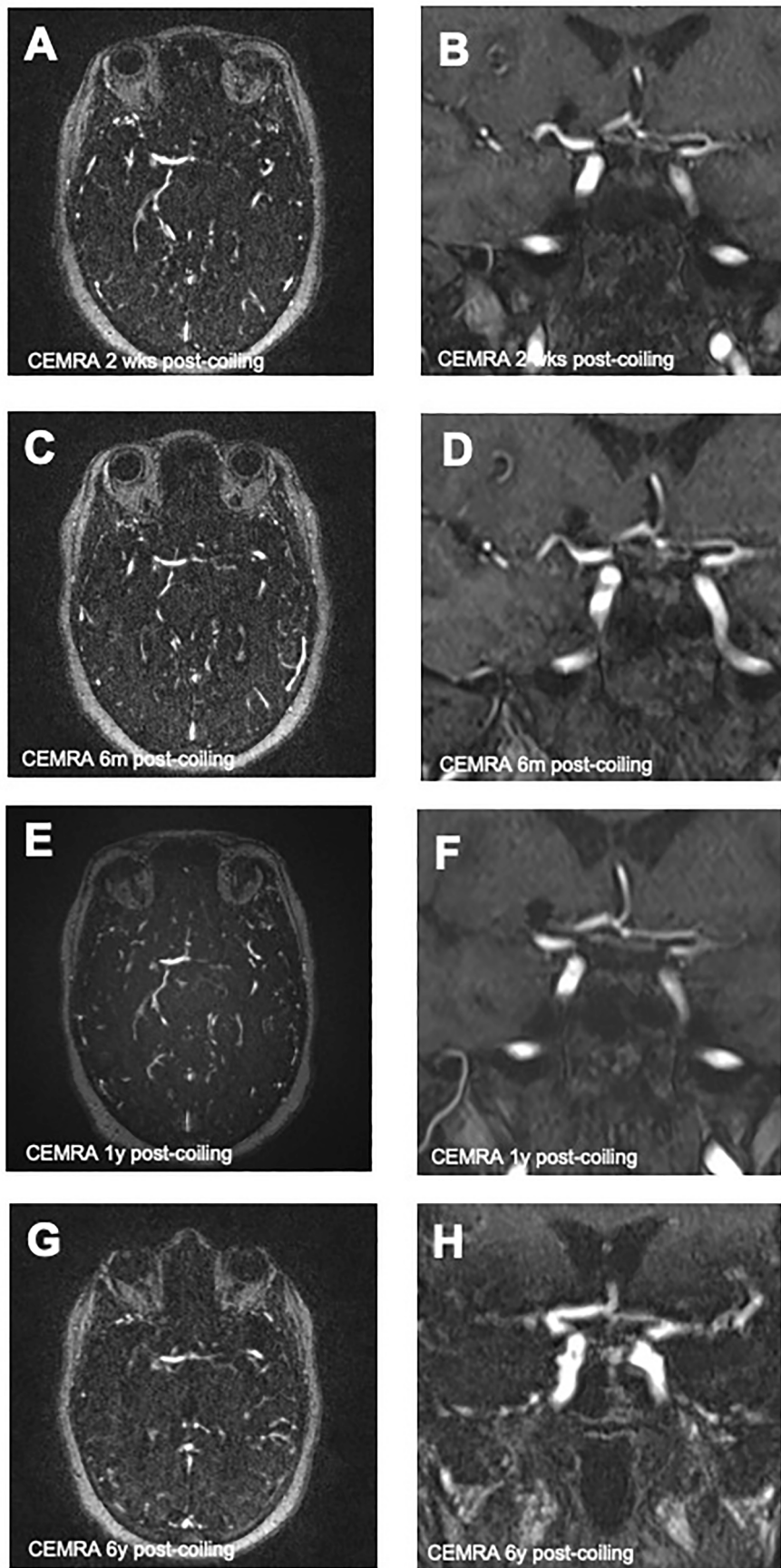
### 3. Endovascular treatment

The patient underwent a diagnostic cerebral angiogram and balloon-assisted coil embolization of unruptured right ICA terminus aneurysm without complication [Fig. 2]. A combination of bare platinum coils and HydroCoils were used. She was discharged from hospital on post-

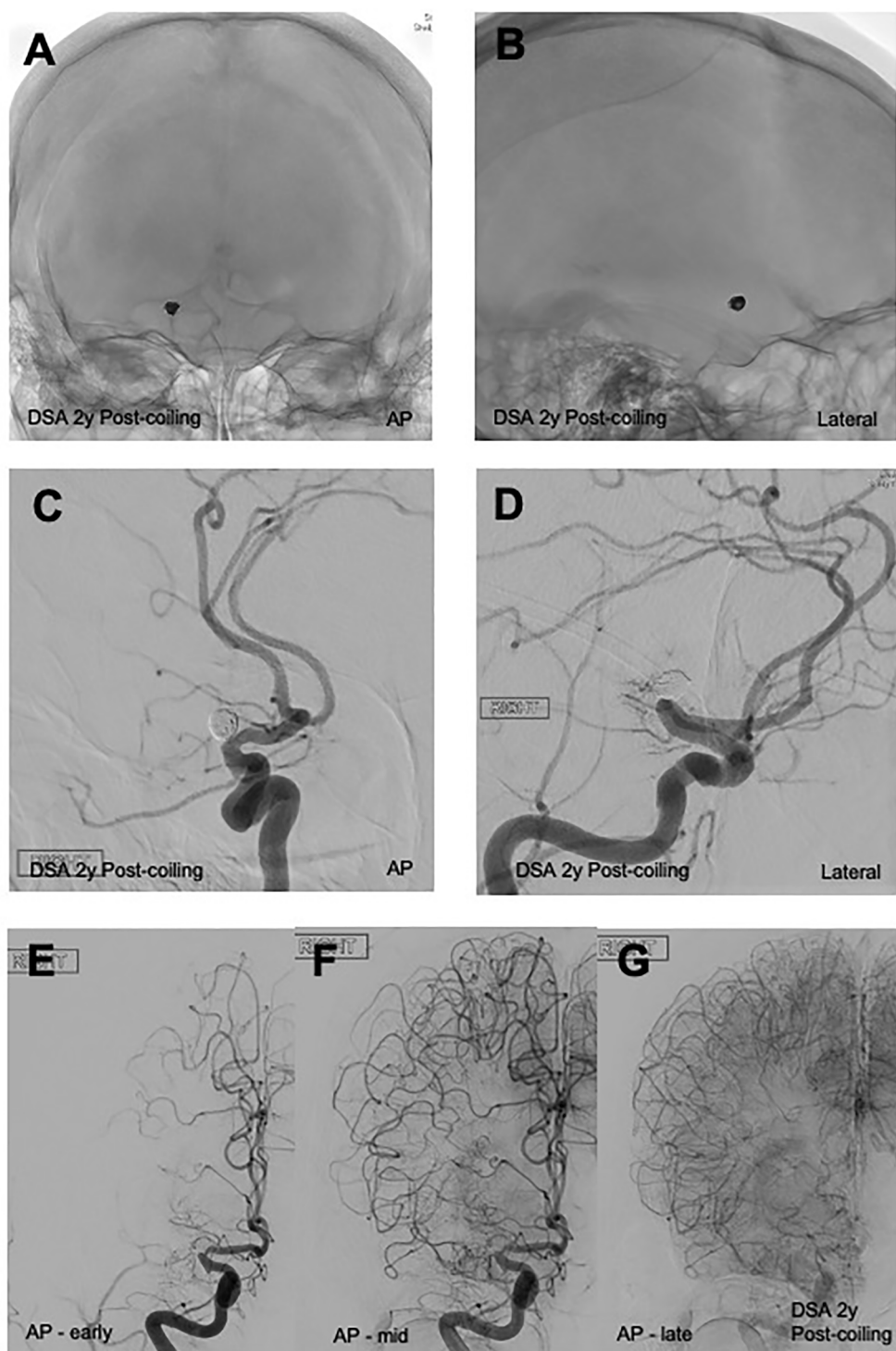
operative day one with a Modified Rankin Scale score of 0, and an NIH Stroke Score of 0.

### 4. Postoperative course

The patient remained clinically well throughout her postoperative course, with follow-up MRI with MR Angiography at two weeks showing no evidence of aneurysm recanalization or stroke, and no signs of new stenosis of the right MCA [Fig. 3]. However, at her routine six month and one-year follow-up, CEMRA showed a progressive stenosis and eventually complete radiographic occlusion of the right M1 segment. Fortunately, the patient remained completely asymptomatic and displayed no focal neurological deficits. A repeat cerebral angiogram was performed and confirmed complete occlusion of the proximal M1 segment of the



**Fig. 3.** Post coiling enhanced and non-enhanced MR angiography at 2 weeks (A and B), 6 months (C and D), 1 year (E and F) and 6 years (G and H) showing progression to complete occlusion of the right M1 segment of the MCA. Axial Contrast enhanced images (A, C, E and F). Non-enhanced MRA TOF coronal views (B, D, F, and H). Note: the MRI multimodal sequences at 2 weeks, 6 months and 1 year did not demonstrate any significant perianeurysmal edema, inflammatory reaction or ischemic infarcts (images not shown).



**Fig. 4.** Follow up DSA performed 2 years post right ICA terminus aneurysm coiling showing complete occlusion of the right M1 segment of the MCA and normal contour and calibre of the right supraclinoid ICA and A1 segment of the right ACA. A and B: Non-subtracted AP (A) and lateral (B) views of the skull demonstrating a stable coil mass with no evidence of coil migration. C and D: Magnified AP (C) and lateral (D) views demonstrating no opacification of the previously coiled right ICA terminus aneurysm. E – G: selective early, mid and late arteriograms of the right anterior cerebral circulation show a complete occlusion of the M1 segment of the right MCA with robust pial/leptomeningeal collaterals from ACA to MCA. Robust pial collaterals were also seen from the right PCA to MCA after selective injection of the dominate vertebral artery (not shown).

right MCA. Robust pial collaterals were noted from the right anterior cerebral artery (ACA) to the right MCA territory, as well as from the posterior cerebral artery (PCA) to the MCA. There was no residual filling of the right ICA terminus aneurysm, and the right supraclinoid ICA and right A1 segment of the ACA were of normal caliber. Interestingly, there were also numerous small vessels around the vicinity of the occluded right M1 in keeping with a possible underlying diagnosis of a Moyamoya-like arteriopathy [Fig. 4]. Now in her seventh decade of life, the patient continues to be asymptomatic and is independent with regards to her activities of daily living.

## 5. Discussion

To our knowledge, this is the first case reported in the literature of an uncomplicated, elective endovascular coiling of an ICA terminus aneurysm resulting in the complete occlusion of the proximal MCA that remarkably did not result in any clinical sequelae, ischemic infarcts or new neurological deficits. As previously mentioned, we used a combination of bare platinum coils to create a structural framework within the aneurysm followed with Hydrocoils as finishing coils (“gel the neck”). At the time of the initial procedure, there was no evidence of coil herniation/migration into the parent artery. Furthermore, follow-up MRI/

MRA at two weeks did not demonstrate any significant perianeurysmal edema, inflammatory reaction or ischemic infarcts. As such, the precise etiology of the complete right M1 artery occlusion remains unclear. Of note, subsequent MRIs continued to show a progression of bilateral supratentorial periventricular and subcortical white matter lesions presumably due to chronic microvascular changes. There is a previously published case report that claims an association between the development of subcortical white matter changes after aneurysm coiling and an inflammatory response believed to be related to the use of polyglycolic-polylactic acid coils [15].

Although large trials such as HELPS and the GREAT trial have shown generally favourable safety profiles of bare platinum coils and bioactive coils [8,10], complications such as aseptic meningitis, seizures, new-onset headaches, hydrocephalus, cranial nerve palsies and delayed visual compromise have been implicated in both [16–18]. A recurring theme in the literature suggests that aberrant inflammation following endovascular coiling may play a significant role in the development of such complications though some degree of aneurysmal wall enhancement can mark the normal healing response. In one retrospective analysis of 124 patients who underwent endovascular coiling of intracranial aneurysms, 85 (64.4%) of aneurysms had wall enhancement and 9 (6.8%) had perianeurysmal brain edema with no reports of any of the aforementioned delayed complications. Higher aneurysm volumes and aneurysms embedded within the brain parenchyma were independently associated with increased wall enhancement [19]. The authors did not observe any significant difference in the degree of inflammatory change when either bare platinum coils or bioactive coils were used.

In those cases where significant perianeurysmal edema was a defining radiographic characteristic in patients presenting with delayed complications, high dose corticosteroids may have improved outcomes. For example, in one series of six patients presenting with delayed visual loss following coil embolization of paraophthalmic aneurysms, four were treated with both bare platinum and HydroCoils, one was treated with bare platinum coils alone, and one using only HydroCoils [19]. In two patients, visual improvement was seen with high-dose corticosteroids. Backhaus *et al.* describe a 62-year-old male presenting with decreased level of consciousness, right-sided hemiparesis and dysarthria three weeks following the coiling of a basilar tip aneurysm and a left MCA aneurysm [20]. MRI was negative for new ischemic lesions however significant perianeurysmal brain edema was noted. Following two weeks of antibiotic and steroid treatment, clinical recovery accompanied a reduction in the edema observed on MRI. Given the relatively high frequency of aneurysm wall enhancement post-coiling, it is probably wise to wait until symptomatic brain edema is encountered before initiating treatment with high-dose corticosteroids. Thus, it is imperative to have a well-established protocol to follow patients post aneurysm coiling with routine clinical and radiographic assessment including MRI/CEMRA at 2 weeks, and 6-, 12- and 24-months post-coiling. Further observational data and reporting of these findings will be useful with a focus on clinical correlation of such radiographic findings.

We did not treat our patient with high-dose corticosteroids as the early follow-up MRI images did not show significant perianeurysmal edema, and she remained asymptomatic throughout her post-procedural course. Moreover, investigations for vasculitis were negative, and her vascular risk factors were well controlled. Consideration of an EC-IC bypass was made though not offered given her excellent clinical status, despite the complete occlusion of her right M1 segment. Her subsequent long-term follow up imaging has not demonstrated further progression or involvement of any other extracranial or intracranial vessels. Although possible that the isolated arteriopathy report here is completely coincidental and unrelated to the aneurysm coiling, it is concerning given the close proximity of the coiled aneurysm to the occluded M1 segment and should be reported.

## 6. Conclusion

We have described an unusual case of an uncomplicated, elective endovascular coil embolization of a right-sided ICA terminus aneurysm leading to the delayed complete occlusion of the proximal M1 segment of the right MCA. Repeat MRI images continued to show progressive white matter changes which could potentially indicate an inflammatory response, however a definitive explanation for the phenomenon of large vessel occlusion could not be adequately elucidated.

## CRedit authorship contribution statement

**Robert G. Power:** Writing - original draft, Writing - review & editing. **Brian A. van Adel:** Conceptualization, Supervision, Writing - review & editing.

## Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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