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# Unusual Delayed Hydrocephalus After Bare Platinum Coil Embolization of an Unruptured Aneurysm

## —Case Report—

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## Abstract

**A 69-year-old woman developed hydrocephalus after the embolization of an incidentally detected unruptured large internal carotid artery aneurysm with bare platinum coils. Endovascular embolization resulted in near-total aneurysm occlusion. She complained of mild headache 18 hours after the procedure and magnetic resonance (MR) imaging performed on the 6th post-treatment day revealed wall enhancement and perianeurysmal brain edema. Follow-up MR imaging showed recanalization of the aneurysm and gradual ventricular enlargement. She presented with typical symptoms of hydrocephalus including disorientation, gait disturbance, and urine incontinence at 7 months post-embolization. We removed 30 ml of cerebrospinal fluid by lumbar tap, which improved her condition without symptom recurrence for 2 months. She did not require shunt placement. Post-interventional hydrocephalus is known in patients with unruptured aneurysms embolized with bioactive second-generation coils. This case shows that hydrocephalus can occur after aneurysm embolization with bare platinum coils without intracranial hemorrhage.**

Key words: hydrocephalus, endovascular embolization, unruptured aneurysm, bare platinum coil, complication

## Introduction

Endovascular coil embolization is less invasive and effectively prevents bleeding from intracranial aneurysms,[7,14,19] but a major disadvantage is aneurysm recurrence.[4,11,14] Biologically active detachable coils have been developed to reduce the recurrence rates and achieve more durable occlusion. However, adverse effects such as symptomatic local inflammation, aseptic meningitis, and communicating hydrocephalus have occurred in patients treated with polymer polyglycolic-lactic acid-coated coils (Matrix, Boston Scientific, Fremont, Calif., U.S.A.; Cerecyte, Micrus Endovascular, San Jose, Calif., U.S.A.) and hydrogel-coated coils (HydroCoil, MicroVention, Aliso Viejo, Calif., U.S.A.).[2,3,5,6,9,10,12,13,16] In contrast, no cases of hydrocephalus after embolization of unruptured aneurysms with bare platinum coils have been reported. Here we present a patient with aneurysm recurrence and hydrocephalus after embolization of a large unruptured internal carotid artery aneurysm with bare platinum coils.

## Case Report

A previously healthy 69-year-old woman was admitted for endovascular treatment of an incidentally detected aneurysm of the left internal carotid artery (18.8 × 16.1 × 15.3 mm) (Fig. 1A). Magnetic resonance (MR) imaging revealed no aneurysmal thrombus formation and no perianeurysmal brain edema, and no evidence of subarachnoid hemorrhage, ventricular obstruction, or neoplasm.

Three days prior to the procedure administration of

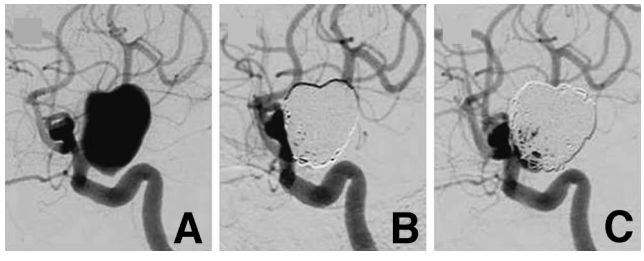


**Fig. 1 Left internal carotid angiograms, lateral view. Angiogram obtained before coil embolization showing a large saccular aneurysm on the internal carotid artery (A). Post-embolization angiogram revealing occlusion of the aneurysm except for a small saccular filling with contrast material (B). Angiogram showing coil loosening and aneurysm recanalization 7 months post-treatment (C).**

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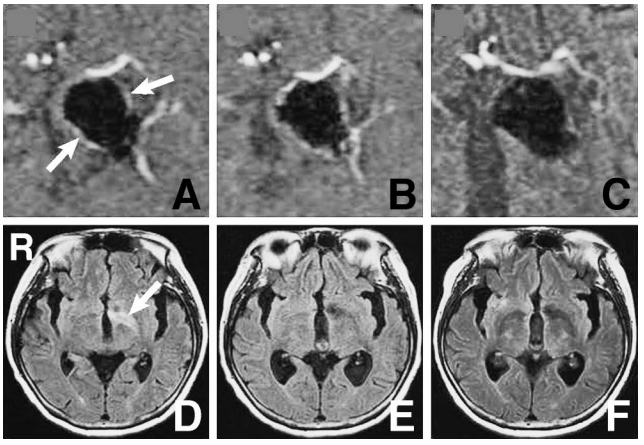


**Fig. 2 Axial gadolinium-enhanced time-of-flight (A–C) and fluid-attenuated inversion recovery magnetic resonance images (D–F) obtained 6 days (A, D) and 3 months (B, E) post-embolization, and after the appearance of hydrocephalus (C, F), showing wall enhancement and perianeurysmal edema 6 days post-embolization (A, D; *arrows*), which had progressed 3 months after treatment (B) and receded subsequently (C). Perianeurysmal edema had disappeared 3 months after treatment (E, F).**

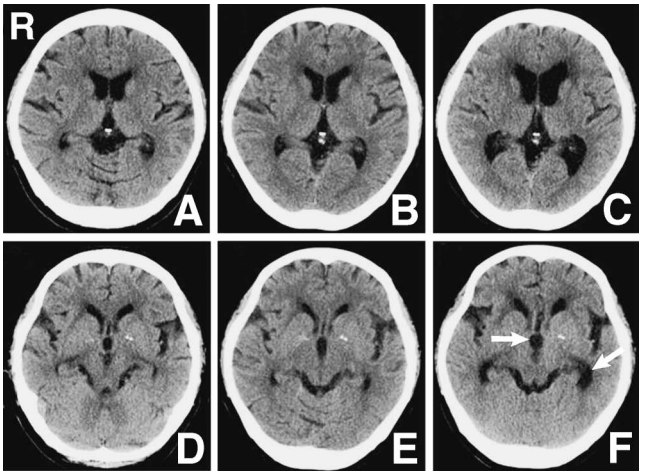


**Fig. 3 Computed tomography scans before (A, D), and 1 month (B, E) and 7 months (C, F) after endovascular coil embolization showing gradually progressing ventricular enlargement, with Evans index of 0.27, 0.29, and 0.32, respectively. Note that the inferior horn of the lateral ventricle and the third ventricle have obviously expanded (F, *arrows*).**

aspirin (100 mg/day) was begun. Endovascular occlusion was performed under general anesthesia and systemic heparinization. Fifteen Guglielmi detachable coils (GDC 18: 280 cm, GDC 10: 220 cm; Boston Scientific) and one 15-cm Orbit coil (Cordis, Miami Lakes, Fla., U.S.A.) with a total coil length of 515 cm were positioned in the aneurysm. Post-embolization angiography revealed occlusion of most of the aneurysm sac, but a small area around the neck showed opacification (Fig. 1B). No thromboembolic complications or aneurysm rupture occurred during the procedure.

She was afebrile and neurologically intact after the procedure, but complained of mild headache 18 hours later that had improved 7 days later without steroid administration. MR imaging on the 6$^{th}$ post-embolization day disclosed thin aneurysm wall enhancement on time-of-flight (TOF) images with gadolinium, which was seen more clearly 3 months later (Fig. 2A, B). Fluid-attenuated inversion recovery images showed extensive brain edema adjacent to the treated aneurysm on the 6$^{th}$ post-treatment day, which had disappeared 3 months after treatment (Fig. 2D, E). Computed tomography (CT) showed the lateral ventricles gradually enlarged and the Evans index (ratio of the maximum width of the frontal horns to the maximum width of the inner table of the cranium) increased from 0.27 at admission to 0.29 (Fig. 3A, B, D, E), but she exhibited no obvious symptoms of hydrocephalus.

Angiography was performed at 7 months post-treatment (Fig. 1C). Follow-up TOF imaging with gadolinium showed aneurysm recurrence. Three days later she presented with gradually progressing disorientation, gait disturbance, and urine incontinence, and she was hospitalized again 2 days later because she could not walk unaided. CT showed expansion of the lateral ventricles and Evans index of 0.32 (Fig. 3C, F). MR imaging disclosed no acute stroke or perfusion anomalies, and no aneurysm wall enhancement or brain edema (Fig. 2C, F). Lumbar tap withdrew 30 ml of cerebrospinal fluid (CSF), and subarachnoid pressure was 13 cm$H_2O$. The CSF was clear, and protein and glucose concentrations were 51 mg/dl and 84 mg/dl, respectively. The white blood cell count was 4 lymphocytes/mm$^3$, and no organisms were identified in any cultures. Blood test showed no inflammatory reactions such as leukocytosis or elevated C-reactive protein. She was able to walk and urinate 6 hours after the lumbar tap, and her mini-mental state examination score improved from 12 to 28 points 3 days after the withdrawal of CSF. MR imaging continued to show ventricular enlargement, but her symptoms did not recur over the course of 2 months and shunt placement was not required. She was discharged without neurological deficits and she is being followed up for further embolization in the event of aneurysm recurrence.

## Discussion

Several cases of hydrocephalus after aneurysm embolization with bioactive second-generation coils such as Matrix and HydroCoils have been documented (Table 1).[2,5,6,10,12,13] The reported incidences of hydrocephalus after embolization with bare platinum coils, Matrix coils, and HydroCoils are 0%, 0%, and 17.6%, respectively,[6] and 2 other patients developed post-interventional hydrocephalus after aneurysm embolization with both Matrix coils and HydroCoils.[13] No post-embolization hydrocephalus occurred in a series of 126 patients with 143 unruptured aneurysms treated with bare platinum coils.[10] The present case of hydrocephalus without intracranial hemorrhage after aneurysm embolization with

**Table 1 Summary of patients showing hydrocephalus after endovascular coil embolization for intracranial unruptured aneurysms**

| Author (Year) | Age (yrs), Sex | Aneurysm location, Size | Occlusion grade | Coils | Cerebrospinal fluid | | | Adverse effects (time from treatment) | | | | | Shunt insertion |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Protein (mg/dl) | Glucose (mg/dl) | Cell (/mm$^3$) | Meningismus or headache | Wall enhancement | Perianeurysmal edema | Hydrocephalus | Aneurysm recurrence | |
| Meyers et al. (2004)[13] | 52, F | Poph, large | complete | BC, HC, MC | 101 | 57 | 51 | yes (26 hrs) | yes (26 hrs) | yes (26 hrs) | yes (NA) | no | yes |
| Berenstein et al. (2006)[2] | 75, F | Poph, large | residual aneurysm | BC, HC | | * | | NA | NA | NA | yes (9 wks) | NA | yes |
| | 46, M | ICA, small | residual aneurysm | BC, HC | | * | | NA | NA | NA | yes (6 wks) | NA | yes |
| | 79, M | PCoA, large | residual neck | BC, HC | | * | | NA | NA | NA | yes (1 wk) | NA | yes |
| Im et al. (2007)[10] | 56, F | ICA, small | NA | BC, HC | 43 | 48 | 1017 | yes (18 hrs) | NA | NA | yes (6 mos) | no | yes |
| Deshaies et al. (2007)[5] | NA | oph, large | NA | HC | NA | NA | NA | yes (NA) | NA | NA | yes (NA) | NA | yes |
| Kang et al. (2007)[12] | 67, F | ICA, small | NA | BC, HC | | ** | | NA | NA | NA | yes (NA) | NA | no |
| | 64, F | ICA, small | NA | BC, HC | | ** | | NA | NA | NA | yes (NA) | NA | yes |
| | 67, F | ICA, small | NA | BC, HC | | ** | | NA | NA | NA | yes (NA) | NA | yes |
| | 41, M | ICA, large | NA | BC, HC | | ** | | NA | NA | NA | yes (NA) | NA | no |
| | 51, F | BA, small | complete | BC, HC | | ** | | NA | NA | NA | yes (NA) | NA | yes |
| Fanning et al. (2008)[6] | 56, M | BA, small | residual aneurysm | BC, HC | NA | NA | NA | no | no | no | yes (30.6 mos) | no | yes |
| | 67, F | Poph, large | residual aneurysm | BC, HC | NA | NA | NA | yes (NA) | yes (NA) | yes (NA) | yes (11.8 mos) | no | no |
| | 45, F | BA, large | complete | BC, HC | NA | NA | NA | yes (NA) | no | no | yes (7.8 mos) | no | no |
| Present case | 69, F | ICA, large | residual aneurysm | BC | 51 | 86 | 4 | yes (18 hrs) | yes (6 days) | yes (6 days) | yes (7 mos) | yes (3 mos) | no |

*Two of 3 patients had finding of an aseptic non-specific inflammatory process. **Elevated protein, normal glucose. and pleocytosis (32–3800/mm$^3$). BA: basilar artery, BC: bare platinum coils, HC: HydroCoils, ICA: internal carotid artery, MC: Matrix coils, NA: not available, oph: ophthalmic, PCA: posterior cerebral artery, PCoA: posterior communicating artery, Poph: paraophthalmic.

bare platinum coils is extremely unusual. MR imaging shows aneurysm inflammation after coil embolization, which is associated with post-treatment hydrocephalus, as aneurysm wall enhancement and perianeurysmal brain edema.[6,13] Histological study confirmed inflammatory reaction adjacent to the coil-embolized aneurysm wall.[1,8,15] The reported incidences of wall enhancement after embolization with bare platinum coils, Matrix coils, and HydroCoils are 18.6%, 25.7%, and 81.8%, respectively.[6] Perianeurysmal edema occurs as a result of the spread of wall inflammation to the surrounding brain.[6,9,16] The incidences of perianeurysmal edema after embolization with bare platinum coils, Matrix coils, and HydroCoils are 0%, 0%, and 29.4%, respectively.[6] These findings suggest that induction of aneurysm inflammation is less likely in the presence of bare platinum coils than bioactive coils, but aneurysm recurrence after embolization with bare platinum coils results in symptomatic brain edema and wall enhancement.[9,17] In our patient, follow-up imaging studies showed post-embolization aneurysm recurrence, wall enhancement, and ventricular dilation, suggestive of aneurysm inflammation.

Table 1 summarizes cases of post-embolization hydrocephalus in patients with unruptured aneurysms.[2,5,6,10,12,13] Large aneurysms are defined as aneurysms with a maximum sac dimension of at least 11 mm, and small aneurysms as those with smaller sac dimensions.[14] Two previous patients developed hydrocephalus after bare platinum coil embolization,[18] but we excluded these patients from our summary because they presented with posterior fossa hemorrhage and subarachnoid hemorrhage, respectively. Table 1 indicates that CSF analysis showed elevated protein levels and pleocytosis, but normal glucose levels and negative culture studies, indicating non-specific aseptic meningitis.[2,10,12,13] These CSF and MR imaging findings suggest that long-term aseptic meningitis is involved in the appearance of post-treatment hydrocephalus. Headache and meningismus after aneurysm coil embolization may be predictive of delayed hydrocephalus. The administration of glucocorticoids reportedly reduces the level of aneurysm inflammation.[3,5,9,12,16] Although we did not treat our patient with steroids, the effectiveness of steroid administration to prevent post-treatment hydrocephalus needs investigation. In our patient, CSF protein level was slightly elevated but no significant pleocytosis was present. Although MR imaging showed inflammatory responses reflected by wall enhancement and brain edema in the early stage after embolization, CSF analysis yielded no definite evidence of inflammation. We suggest that hydrocephalus in our patient improved after only one lumbar puncture because the aneurysm inflammation was milder than that seen after embolization with bioactive coils.

Post-embolization hydrocephalus can occur even after endovascular intervention with bare platinum coils. We suggest that patients with MR imaging evidence of wall enhancement and perianeurysmal brain edema after coil embolization be followed up to detect the delayed appearance of hydrocephalus.

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