Quantitative Computed Tomographic Volumetry after Treatment of a Giant Intracranial Aneurysm with a Pipeline Embolization Device

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Recently developed flow diverters, such as the pipeline embolization device (PED), allow for safe and efficacious treatment of giant intracranial aneurysms, with high occlusion rates and a low incidence of complications. However, incomplete obliteration after PED treatment may lead to aneurysm regrowth and delayed rupture. Herein, we report a case of a partially thrombosed giant aneurysm of the cavernous internal carotid artery that showed progressive recanalization at 1–3 months after application of a PED. We monitored inflow volume in the aneurysm by computed tomographic angiography (CTA) and computed tomographic volumetric imaging (CTVI). Based on the imaging results, rather than applying additional PED, we decided to make the switch from a dual antiplatelet medication to low-dose aspirin alone at 3 months after the treatment; complete obliteration of the aneurysm was noted at 21 months. Similar to the findings in this unusual case, CTA and CTVI may be useful follow-up methods for optimal management of patients with giant intracranial aneurysms after PED treatment.

Key Words: Intracranial aneurysm, therapeutic embolization, medical device, computed tomography, angiography

INTRODUCTION

Recently developed flow diverters, such as the pipeline embolization device (PED), allow for safe and efficacious treatment of giant aneurysms, with high occlusion and relatively low complication rates.¹⁻² Incomplete obliteration of giant aneurysms after PED treatment may lead to delayed aneurysm rupture; hence, monitoring of inflow volume by follow-up imaging is important. Herein, we describe a partially thrombosed giant aneurysm of the cavernous internal carotid artery (ICA) that showed dynamic inflow volume change after application of a single PED and discuss the possible causes and clinical significance of early recanalization of giant aneurysms after successful PED treatment.

CASE REPORT

A 62-year-old woman with headache after a head contusion was referred to our hospital. Brain computed tomographic angiography (CTA), magnetic resonance imaging (MRI), and digital subtraction angiography (DSA) revealed a partially thrombosed giant aneurysm (maximum diameter, 33 mm) in the right cavernous ICA (Fig. 1A, B, and C). The patient had no neurologic symptoms associated with the aneurysm. The patient was premedicated with aspirin (100 mg once daily) and clopidogrel (75 mg once daily) for 3 weeks prior to treatment. Platelet inhibition tests demonstrated acceptable responses to aspirin and clopidogrel. She was treated by using a 4×25 mm PED (Covidien, Irvine, CA, USA) that spanned the ICA terminus, crossed the aneurysm neck, and terminated at the horizontal cavernous ICA. Completion DSA revealed disrupted flow into the aneurysm, with redirection of the primary inflow jet axis toward the posteroinferior aspect of the aneurysm sac.
Immediate DynaCT confirmed optimal stent positioning across the aneurysm neck (Fig. 1F). The patient has consented to the submission of this case report for publication.

We assessed the patient’s condition using CTA and computed tomographic volumetric imaging (CTVI) after PED treatment. Conventional brain CTA was performed using a 256-slice multiple detector computed tomography (CT) scanner (Brilliance iCT; Philips Healthcare, Cleveland, OH, USA). The scanning parameters were as follows: 120 kVp, 140 mAs, 0.9-mm slice thickness with 0.45-mm overlaps, 0.33 second rotation time, 22-cm field of view, and a 512×512 pixel matrix. Volumetric images were reconstructed in a multimodality workstation (Extended Brilliance Workspace; Philips Healthcare) using semi-automated methods and the tissue segmentation tool as follows: 1) set the CT number to the volume of interest at a level of 1160 Hounsfield unit (HU) and a width of 2048 HU; 2) initially select the candidate region including the contrast-filled portions of the aneurysm, adjacent vessels, and the PED; 3) manually isolate and remove adjacent vessels on a section-by-section basis to refine the aneurysm; and 4) automatically calculate the volume of the contrast-filled portion of the aneurysm.

The preoperative inflow volume of the aneurysm was 3883.8 mm$^3$ (except the thrombosed portion, 100%). Progressive thrombosis was observed at 1 week after PED treatment: the inflow volume was 756.6 mm$^3$ (19.5%), but increased thereafter, 1385.4 mm$^3$ (35.7%) at 1 month, and 1565.7 mm$^3$ (40.3%) at 3 months (Fig. 2). CTA did not detect any stent foreshortening or endoleaks. Because the patient’s antiplatelet status presumably accounted partly for the reduced thrombosis and rather than applying additional PED, we switched from a dual anti-

![Fig. 1. Images of the partially thrombosed aneurysm pre- and post-stenting. (A) Initial T2-weighted magnetic resonance imaging shows a partially thrombosed (asterisk) giant aneurysm in the cavernous segment of the right internal carotid artery. (B and C) Preoperative digital subtraction angiography reveals a giant aneurysm with the inflow jet axis oriented posterosuperiorly toward the aneurysm dome on the sagittal plane. (D and E) Post-treatment completion angiography shows significantly decreased inflow to the aneurysm sac with redirection of the initial inflow jet axis toward the postero-inferior aspect on the sagittal plane. (F) DynaCT identifies the optimal stent position across the aneurysm neck.](image-url)
platelet medication to low-dose aspirin alone. The inflow volume steadily decreased to 954.7 mm$^3$ (24.6%) at 4 months and 413.6 mm$^3$ (10.6%) at 6 months. Based on the results of sequential CTA and CTVI, we again decided not to apply additional PED; instead, we continued to follow up the patient. The inflow volume further decreased at 15 months (124.5 mm$^3$, 3.2%), and complete obliteration was achieved at 21 months. The patient had no neurological events during the follow-up period.

**DISCUSSION**

Flow diverters reduce the extent of hemodynamic exchange between an aneurysm and parent artery, which promotes thrombosis within the aneurysm sac, and provide a framework for neointimization of the aneurysm neck. Immediate aneurysm exclusion is not expected; complete exclusion requires time. A recent review reported complete occlusion rates of 80% for small aneurysms, 74% for large aneurysms, and 76% for giant aneurysms 6 months after PED treatment.

Progressive aneurysm recanalization in partially thrombosed giant intracranial aneurysms after successful PED treatment is rare. Causes of intra-aneurysmal thrombus formation include reduced inflow velocity, reduced vorticity, and increased turnover time. Dual antiplatelet therapy (aspirin and clopidogrel) synergistically inhibits platelet aggregation, in-
stent thrombus formation within stents, and together with other events, thrombus formation within the aneurysm sac. Low-dose aspirin alone has modest effects, compared with aspirin plus clopidogrel.

Progressive recanalization may also result from immature thrombus formation following PED deployment. Unlike a thrombus after PED, a spontaneous thrombus in a giant aneurysm is regarded as a mural hematoma, owing to the nature of its formation. Factors contributing to spontaneous thrombus formation and growth include wall dissections, intramural hemorrhages, and intra-thrombotic vascular channels. Martin, et al. found that mural thrombus had relatively constant MRI features (T1 high and T2 low signal intensities) over a long period and, hence, suggested that mature thrombi are either relatively static or at equilibrium. In the present case, a preexisting mural thrombus showed T1 high and T2 low signal intensities on pretreatment MRI and may be a mature, rather than a fresh mural thrombus. Serial CTA revealed that recanalization occurred in the newly formed fresh thrombus, separate from the mural thrombus, and progressed toward the posteroinferior portion of the aneurysm sac, similar to the flow redirection (initially posterosuperior) observed on completion DSA. We speculate that recanalization resulted from both the antplatelet status caused by the dual antplatelet medication and the immature thrombus with inflow redirection.

The potential association between progressive recanalization and delayed aneurysm rupture is concerning. The incidence of rupture is higher in giant than in small or large aneurysms, and most ruptures occur within 1 month of PED treatment. Causes of delayed aneurysm rupture include wall shear stress at the aneurysm dome, unfavorable hemodynamic status, and autolysis of the aneurysm wall by thrombosis-induced proteases. Although yet to be established, we believe that progressive recanalization with redirected inflow may be a risk factor for rupture and a signal for further intervention.

Because DSA detects contrast flow in the partially thrombosed aneurysm, it likely underestimates the inflow volume after PED deployment, owing to contrast stasis and turbulent flow within the sac. Thus, CTA and magnetic resonance angiography are superior to DSA for evaluating inflow volume after PED treatment. CTVI provides quantitative volume in cubic millimeters and may have less inter-observer variation. Radiation exposure during CTA is concerning. In our case, the mean dose-length product of each CTA was 1269.2 mGy·cm, and the calculated total patient effective radiation dose was 24.0 mSv for 21 months (16.7 mSv for 1 year), which may be reasonable given the potential risk of delayed aneurysm rupture.

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