Intracerebral Hemorrhage after Endovascular Treatment of Unruptured Intracerebral Aneurysm: A Case Report and Review of the Literature

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Summary: Purpose: Intracerebral hemorrhage (ICH) that develops after treatment of unruptured intracerebral aneurysms is a rare complication. We present one case of ICH after endovascular treatment and discuss the possible pathophysiologic mechanisms and preventative strategies. Patient Case: A 51-year-old woman with left homonymous hemianopsia and a large paraclinoid (internal carotid-ophthalmic) aneurysm underwent flow diversion (FD) using the Pipeline Embolization Device and coiling. Several hours postoperatively, she had motor aphasia with mild right hemiparesis, and head computed tomography revealed an ipsilateral frontotemporal hematoma. Magnetic resonance angiography and digital subtraction angiography suggested a form of hyperperfusion syndrome, and conservative management resulted in almost complete resolution of symptoms. Conclusions: The etiology of ICH acutely following FD may be multifactorial due to dual antiplatelet therapy (DAPT) hyper-response and flow modification related to hyperperfusion and the Windkessel effect. Conservative management resulted in a good outcome. However, for severe hemorrhagic cases, platelet transfusion, discontinuation of DAPT to single antiplatelet therapy, and surgical intervention should be considered. Perioperative monitoring indicating antiplatelet hyper-response or radiographic hyperperfusion should direct strict blood pressure control and risk reduction precautions.

Key words: complications, endovascular treatment, intracerebral hemorrhage, flow diverter, unruptured intracerebral aneurysm

Introduction

Intracerebral hemorrhage (ICH) is a well-known complication following carotid endarterectomy and carotid artery stenting associated with the cerebral hyperperfusion syndrome. Here, we describe a rare case of ipsilateral parenchymal hemorrhage after endovascular treatment (EVT) of a large unruptured paraclinoid (internal carotid-ophthalmic) aneurysm.
carotid (IC)-ophthalmic) aneurysm and discuss the possible mechanisms and preventative strategies.

Case Report

A 51-year-old woman presented with a 3-year history of progressive left visual difficulty. Five months prior to admission, partial left homonymous hemianopsia on ophthalmological examination was identified, and she was referred to our institution for further testing. On admission, neurological examination was significant for left nasal hemianopsia with intact extraocular movements and no apparent diplopia. Three-dimensional digital subtraction angiography (DSA) revealed a large 24.3 mm × 21.6 mm unruptured left paraclinoid (IC-ophthalmic) saccular aneurysm with narrow neck (Fig. 1A).

Based on the symptomatic, high-risk condition, surgical treatment was recommended; however, the patient declined the option of direct clipping with proximal carotid ligation and M2-STA bypass and agreed to undergo EVT using flow diversion (FD) with coiling. Using a right femoral approach, one Pipeline Embolization Device (PED; Medtronic/Covidien, Irvine, USA) (4.25 mm × 20 mm) was placed in a telescoping fashion across the ophthalmic portion of the left internal carotid artery, followed by intra-aneurysmal coiling (875 mm) (Fig. 1B, C).

After preoperative dual antiplatelet therapy (DAPT) (aspirin 100 mg, clopidogrel 75 mg), intraoperative monitoring using VerifyNow revealed a PRU of 23 and ARU of 385. Immediate postoperative head computed tomography (CT) was unremarkable (Fig. 2A). Postoperatively, DSA revealed contrast stagnation with positive eclipse sign indicating effective FD, and no reversal of heparin was performed. Additionally, DSA demonstrated an iatrogenic carotid-cavernous fistula due to suspected wire perforation of the meningohypophyseal trunk (arrow Fig. 1C) with no sign of vascular damage from mechanical devices or extravasation at any other location. Despite meticulous intra- and postprocedural systolic blood pressure control (range, 100-120 mmHg), 7 h postoperatively, the patient had sudden vomiting with motor dominant aphasia and right hemiparesis (MMT 5-/5). Head CT revealed a left subcortical frontotemporal intrasylvian fissure ICH (Fig. 2B). Postoperative magnetic resonance angiography (MRA) and DSA suggested increased distal flow compared to preoperative MRA and DSA (Fig. 3, 4). Arterial spin labeling (ASL) and single-photon emission computed tomography (SPECT) were not performed. However, hyperperfusion syndrome was suspected, and after obtaining informed consent, conservative management consisting of blood pressure control, intravenous brain edema treat-
ment, and rehabilitation was selected, with the option of surgical hematoma evacuation in the event of increasing hemorrhage size. Despite persistent visual deficits, the patient was almost cleared of postoperative signs of aphasia and right hemiparesis within 2 weeks (mRS 1).

Discussion

ICH has been identified as a unique postoperative complication of FD with an incidence of 2−3%\(^9\). Our patient demonstrated typical ipsilateral subcortical hemorrhage, with a slightly early onset; however, 24% of post-FD ICHs was reported in the first 24 h\(^9\). Although the etiology of ICH after EVT remains unknown, suggested mechanisms include DAPT, hypertension, hemorrhagic thromboembolic transformation (including polyvinylpyr-

![Immediate postoperative CT and 7 hours postoperative CT](image)

**Fig. 2** Head CT performed immediately postoperatively (A) was unremarkable; however, 7 h postoperatively, the patient had sudden vomiting with motor dominant aphasia and mild right hemiparesis. Head CT (B) shows left subcortical frontotemporal intrasylvian fissure intracerebral hemorrhage.

![Preoperative MRA and Postoperative MRA](image)

**Fig. 3** Postoperative magnetic resonance angiography (MRA) shows slightly increased distal left middle cerebral artery flow compared to preoperative MRA.

rolidone sheath), and flow modifications\(^1\(^3\)\(^5\)\(^10\)\(^11\). ICH after FD has been explained by the Windkessel effect involving intra-aneurysmal flow redirection with lower proximal arterial compliance and greater distal pulse pressure\(^3\), a phenomenon possibly unrelated to aneurysm size\(^1\(^2\)\(^9\)\(^11\). Cerebral hyperperfusion has also been postulated in clipping large or giant aneurysms, where increased intraoperative Doppler ultrasound flowmetry and increased flow intensity on postoperative perfusion CT, MRA, and SPECT were observed\(^6\)^\(^7\). In our case, ICH may have been caused by factors such as clopidogrel hyper-response (lower PRU has been associated with hemorrhage\(^4\)), operative time, and hemodynamic stress due to the Windkessel effect, as suggested by radiographic hyperflow on postoperative MRA and DSA, although this
could have been due to effects of intra-aneurysmal turbulent flow or contrast dilution, respectively. The outcome was good perhaps due to strict blood pressure management. Our case was limited as representing the first case in our early series of mostly cavernous sinus FD cases.

Current recommendations for severe cases of ICH after EVT with mass effect include standard surgical hematoma evacuation and decompressive craniectomy. Preoperative platelet transfusion, discontinuation of clopidogrel, and antiplatelet monitoring have been recommended; however, there is no consensus regarding how to adjust the preoperative antiplatelet regimen based on monitoring results\(^1\). Additionally, as demonstrated in our case, suspected increased distal flow on postoperative MRA and DSA could indicate potential postoperative complications of hemorrhage and guide stricter postoperative blood pressure management. Perioperative transcranial ultrasound, ASL, and SPECT could be useful.

**Conclusions**

The etiology of acute ICH after FD and coiling for a large unruptured IC-ophthalmic aneurysm may be multifactorial, which is related to DAPT hyper-response and post-procedural flow modification involving the Windkessel effect and cerebral hyperperfusion. Conservative treatment resulted in a good outcome; however, for severe hemorrhagic cases, platelet transfusion, discontinuation of DAPT to single therapy, and surgical intervention should be considered. Perioperative monitoring indicating hyperdynamic flow should be followed by strict blood pressure control.

**Declaration of conflicts of interest**

None of the authors or co-authors have any conflict of interest to declare.
References


要旨

目的：未破裂動脈瘤治療後の脳内出血はまれな合併症である。われわれは血管内手術後に生じた1例を経験し、その発生機序と予防策について検討したので報告する。

症例：51歳、女性。左同名半盲にて発症した大型の内頚動脈眼動脈分岐部動脈瘤であった。治療はflow-diverter (Pipeline™ Embolic Device: PED)とコイルにて行った。術後数時間の右片麻痺と運動性失語を認め、CTにて同側前頭側頭葉に脳内出血を認め、血管撮影では術前に比べて血流が増加しているようにみえたが、出血による症状は経過観察のみで消失した。

まとめ：本例では、抗血小板剤の過剰反応と、Windkessel効果に伴う過灌流症候群に類似した血行動態の変化によって出血が誘発されたと考えられた。本例のようなハイリスク症例に対して高度流動化効果の強いフローダイバーターを用いた血管内治療を行う際は、適切な周術期のリスク管理が重要である。特に、周術期の抗血小板機能モニタリングと過灌流リスクの画像評価は、慎重な血圧管理を含めた術後出血性合併症予防のために重要である。