

# Endovascular management of symptomatic cerebral aneurysm thromboembolism due to pre-aneurysmal arterial stenosis

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## Abstract

**Background:** Pre-aneurysmal internal carotid steno-occlusive disease resulting in cerebral intra-aneurysmal thrombosis and subsequent embolic infarction has not been previously described.

**Conclusion:** Antithrombotic treatment for dissolution of the thrombus and pre-aneurysmal stent angioplasty followed by Pipeline embolization flow diverter placement through the aneurysm is a safe and feasible management option.

## Keywords

Pipeline embolization device, cerebral aneurysm, thromboembolism

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## Introduction

Cerebral aneurysmal thromboembolic phenomenon is rare and the etiology is often related to large aneurysms that promote intra-aneurysmal stasis. Pre-aneurysmal internal carotid steno-occlusive disease with intra-aneurysmal thrombosis and subsequent embolism resulting in an infarct has not been described. Endovascular management using the combination of a balloon-mounted stent and a flow diverter device preceded by antithrombotic dissolution of the thrombus has not been previously described.

## Case presentation

A 46-year-old female with a past medical history of rheumatoid arthritis, diabetes mellitus, and anemia secondary to dysfunctional uterine bleeding (DUB) presented to the emergency department (ED) with a chief complaint of headache associated with right arm paresthesia. Asymptomatic anemia with a hemoglobin of 6.3 mg/dL was thought to be due to her DUB and was not characterized by change in her blood pressure. A head computed tomography angiogram (CTA) showed a 10 mm left paraclinoid thrombosed aneurysm (Figure 1(a)). The head CT not shown identified no other abnormality. Aspirin 81 mg was commenced and she was discharged with resolution of her symptoms, awaiting definitive treatment of

her DUB. She represented to the ED with right upper extremity paresis 2 months later. A CTA of the head showed partial recanalization of the left paraclinoid aneurysm (Figure 1(b)). The study also identified thrombus within the left supraclinoid internal carotid artery (ICA) (Figure 2(a)). Magnetic resonance imaging (MRI) of the brain showed a left parietal lobe acute infarct in the vascular territory of the left inferior division middle cerebral artery (Figure 2(b)). She received intravenous heparin for thrombus dissolution, prior to endovascular intervention. This was monitored by serial prothrombin times to assure therapeutic levels. A head CTA 10 days later while the patient remained on heparin showed a completely recanalized left ICA aneurysm with resolution of thrombus at the left supraclinoid ICA (Figure 2(c)). Pre-embolization digital subtraction angiography (DSA)

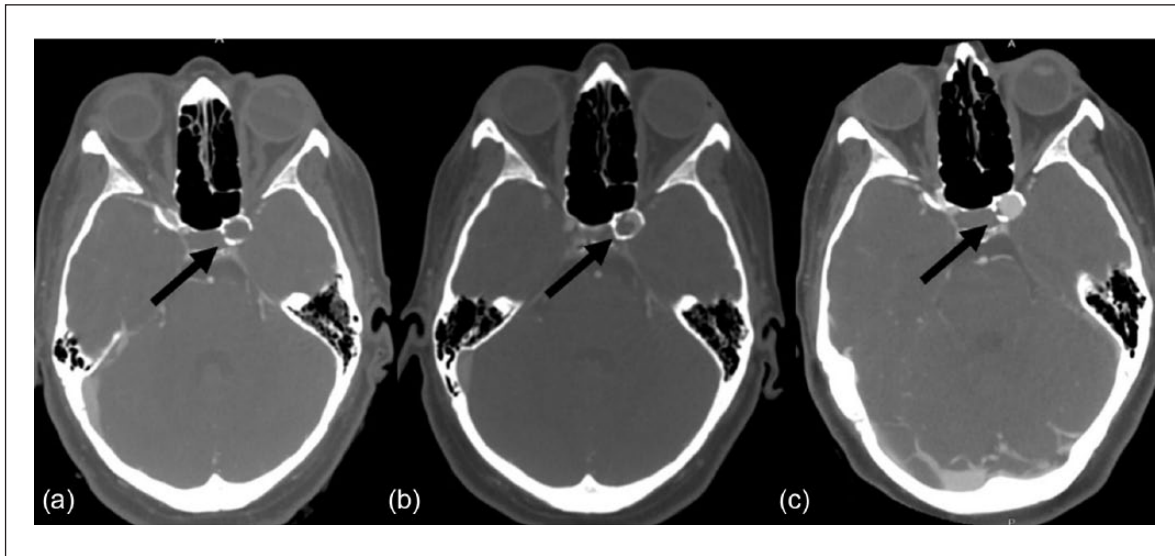
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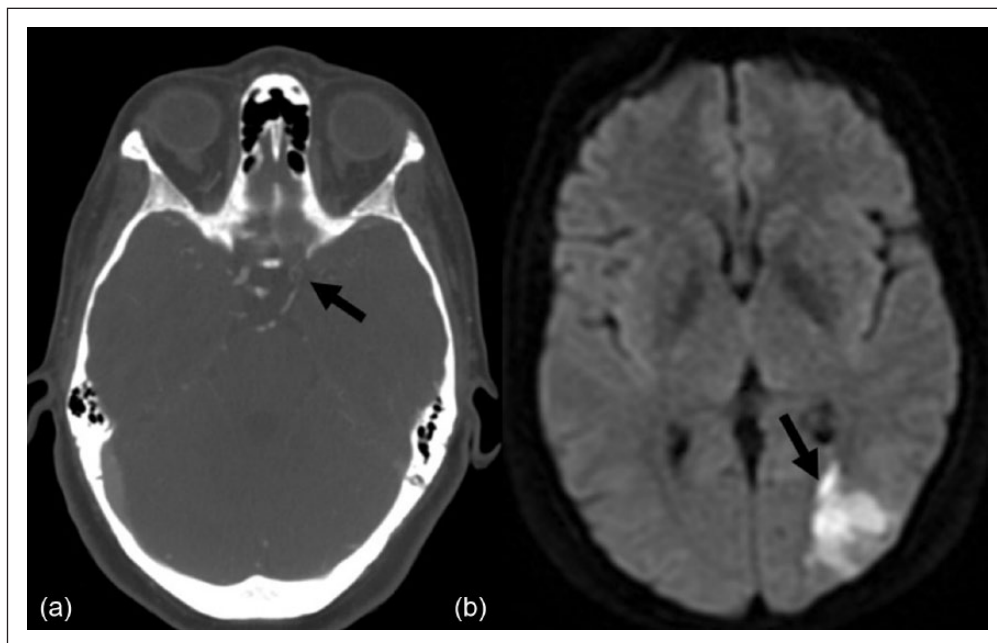
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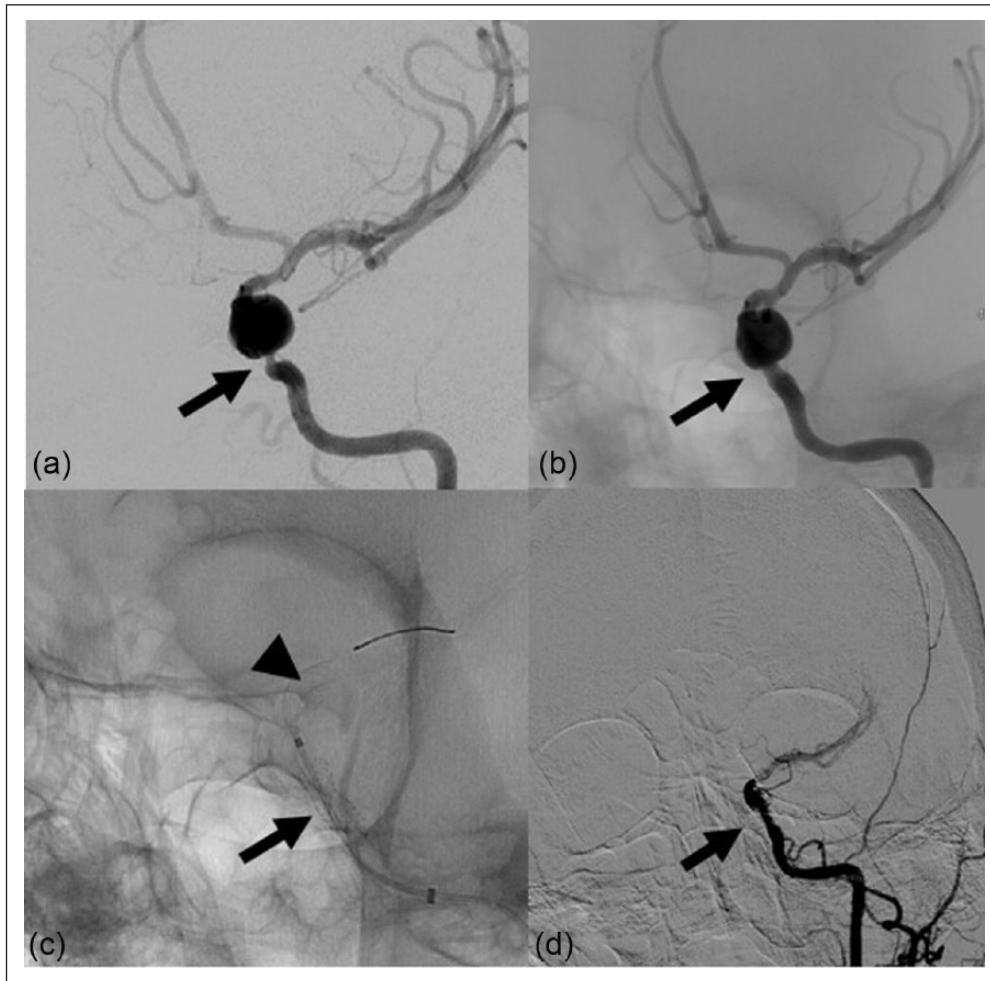
**Figure 1.** Axial CT demonstrating (a) thrombosed left paraclinoid aneurysm (arrow), (b) partial recanalization (arrow), and (c) complete recanalization after antithrombotic therapy (arrow).



**Figure 2.** (a) Axial CT demonstrates thrombus in right supraclinoid ICA (arrow) and (b) axial diffusion MRI sequence shows acute infarction left parietal lobe (arrow).

identified critical stenosis of the cavernous ICA proximal to the aneurysm, not discernible by CTA (Figure 3(a)). Five days prior to the anticipated date of procedure, oral clopidogrel 75 mg daily was commenced. On the day of the procedure, the patient's P2Y12 Verifynow assessment of platelet function demonstrated adequate inhibition. Balloon-mounted stent angioplasty of the pre-aneurysmal stenosis was performed with a drug-eluting Xience Alpine Stent (Abbott Vascular, Santa Clara, CA, USA). Precise stent placement

was performed across the stenosis only with minimal stent overlap into the aneurysm. A tandem Pipeline flow diverter embolization device (ev3, Irvine, CA, USA) was delivered through the resolved stenosis and aneurysm (Figure 3(b) and (c)). The embolization device was positioned across the aneurysm lumen and distal parent vessel to facilitate maximal flow diversion across the aneurysm. At 6-month follow-up, her right extremity paresis improved with complete occlusion of the aneurysm and no in-stent stenosis (Figure 3(d)).



**Figure 3.** (a) Oblique projection digital subtraction angiography (DSA) demonstrating critical stenosis proximal to the paraclinoid internal carotid artery aneurysm. (b) Oblique projection unsubtracted angiogram showing restoration of luminal caliber post stent angioplasty. (c) Oblique projection fluoroscopic spot view showing the drug-eluting stent across the stenosis (arrow) and delivery of the Pipeline embolization device (short arrow). (d) Oblique projection DSA demonstrating aneurysm obliteration (arrow) without recurrent pre-aneurysmal stenosis.

## Discussion

Cerebral aneurysms can develop spontaneously, as a result of trauma or infection, or from change in cerebral hemodynamics.<sup>1</sup> It is well known that aneurysms develop at arterial bifurcations and branch points, due to hemodynamic stress at these aforementioned areas.<sup>2</sup> High wall shear stress and high positive wall shear stress gradients can predispose to cerebral aneurysms.<sup>3,4</sup> High wall shear stress can lead to aneurysm formation specifically in the setting of focal stenosis proximal to the aneurysm, as identified in our patient. Stenosis greater than 40% by diameter causes non-physiologic high wall shear stress and wall shear stress gradients, which could theoretically lead to aneurysm formation.<sup>4</sup>

Cerebral aneurysms can present as subarachnoid hemorrhage, focal neurologic deficits from local mass effect, or rarely with transient ischemic attacks and ischemic strokes. Multiple series have reported ischemic events from unruptured

aneurysms to be between 3% and 11%. These ischemic events have been considered a result of thrombus formation within the aneurysm leading to embolic strokes. The larger the size of the aneurysm dome in relation to the aneurysm neck, the greater the possibility for thrombosis formation.<sup>5</sup>

It is well documented in the literature that thrombus formation within a cerebral aneurysm is related to turbulent flow.<sup>2</sup> To our knowledge, there has been no reports describing thrombus formation within an aneurysm as a result of proximal stenosis of the parent artery with subsequent embolic infarction. This case report describes varied pathological processes often considered independently, within the same artery, with a safe endovascular management approach.

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**Declaration of conflicting interests**

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

**Ethical approval**

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**Informed consent**

Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article.

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