International Journal of Neurosurgery
2019; 3(1): 9-12
http://www.sciencepublishinggroup.com/j/ijn
doi: 10.11648/j.ijn.20190301.13
ISSN: 2640-1940 (Print); ISSN: 2640-1959 (Online)

Case Report

Perianeurysmal Edema After Embolization with Flow Diversion

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To cite this article:
Mehrnoush Gorjian, Scott Raymond, Matthew Koch, Aman Patel. Perianeurysmal Edema After Embolization with Flow Diversion. *International Journal of Neurosurgery.* Vol. 3, No. 1, 2019, pp. 9-12. doi: 10.11648/j.ijn.20190301.13

Received: June 23, 2019; **Accepted**: August 4, 2019; **Published**: August 19, 2019

Abstract: Flow diversion is an endovascular technique for embolization of intracranial aneurysms using a semi-porous stent to that redirects blood flow away from the aneurysm and is a scaffold for vessel remodeling. With flow diversion, aneurysms close slowly over 6-12 months as endothelial growth into the stent covers the neck of the aneurysm. Flow diversion is the preferred treatment for unruptured, large, paraclinoid aneurysms, which are otherwise challenging to treat with conventional open surgical or endovascular techniques. Post-embolization perianeurysmal edema (PAE) is an uncommon complication that occurs weeks to months after embolization and manifests as brain parenchymal edema surrounding the treated aneurysm. The clinical presentation is varied and includes headache, seizure or focal neurologic deficit. Frequently, PAE is misdiagnosed as an embolic stroke, which is a more common post-embolization complication and has some clinical and imaging overlap. PAE can be differentiated from ischemia by the absence of restricted diffusion and aneurysm wall enhancement on post-contrast Magnetic Resonance Imaging (MRI). PAE was initially described following coil embolization, but has subsequently been observed after flow diversion alone or with adjunctive coiling. Post-embolization PAE presumably results from rapid aneurysm thrombosis, endothelial cell necrosis, and the ensuing inflammatory reaction, which spreads to the adjacent brain parenchyma. Early recognition of PAE is critical to initiate appropriate therapy.

Keywords: Perianeurysmal Edema, Flow Diversion Stent, Coil Embolization, Intracranial Aneurysm

1. Introduction

Flow diverting stents (FDSs) are a popular endovascular method for treating intracranial aneurysms, especially wide neck or morphologically complex giant aneurysms not amenable to other interventions. FDSs redirect blood flow away from the aneurysm and provide a scaffold for neointimal growth across the neck of the aneurysm. FDSs may be used alone or in conjunction with coils. Prior to placement of a FDS, patients are started on dual-antiplatelet medication to limit the thromboembolic risk. [1]

Flow diversion is relatively safe and has a risk profile similar to other endovascular techniques including coil embolization [2, 3]. The most common perioperative complications are thromboembolic or access related [4]. Recently a number of rare delayed complications have been described including foreign body reaction after particulate embolization of the microcatheter [5] and post-embolization perianeurysmal edema (PAE).

PAE is an uncommon complication that was first reported in patients with large intracranial aneurysms following coil-embolization [6, 7], and more recently, in patients undergoing flow diversion alone or with adjunctive coils [8]. PAE typically presents as headache, seizure or focal neurologic deficit weeks to months after embolization. [9, 10] Imaging demonstrates edema in the brain tissue contacting the aneurysm, which will often demonstrate wall enhancement [12]. Although the precise mechanism is unknown, it is hypothesized that the edema represents an inflammatory reaction related to rapid aneurysm thrombosis and wall necrosis. [8, 11].

Steroids are the treatment of choice, although a large

fraction of patients do not respond and deteriorate despite maximal medical therapy [include reference]. Because of the nonspecific presentation, PAE can be mistaken for FDS-related thromboembolic stroke, delaying initiation of anti-inflammatory medications.

We present two cases of post-embolization PAE following flow diversion with adjunctive coil embolization. We discuss the imaging features, treatment strategies, and clinical implications of PAE.

2. Case Description

2.1. Case 1

A 56-year-old woman presented with one month of progressive ataxia and right-sided weakness. Brain MRI demonstrated a large basilar tip aneurysm and no signal abnormality in the adjacent brainstem (Figure 1). A diagnostic cerebral angiogram demonstrated a 3 cm superior and posteriorly projecting giant basilar tip aneurysm. Given her progressive neurological symptoms, she was admitted and started on dexamethasone and dual anti-platelet therapy. On hospital day 3, the patient underwent flow diversion with a Pipeline embolization device (PED) and adjunctive coiling. There were no immediate thromboembolic or hemorrhagic complications. The patient was extubated and recovered in the neuro Intensive Care Unit (ICU). She was discharged to inpatient rehabilitation post op day 6 at her pre-embolization baseline, notable for right-sided weakness (walking with a rolling walker), ataxia, and mild cognitive deficits including lack of insight. After a short inpatient rehab stay, she was discharged home.

She was seen in our clinic one month after embolization and had persistent right sided weakness: 4 out of 5 strength in the right upper and lower extremities and able to ambulate only short distances with a walker. Two months after embolization, she presented to the emergency room with 24 hours of worsening right-sided weakness (strength in the right upper and lower extremities 1 out of 5), new right facial droop and dysarthria. Brain MRI demonstrated T2/FLAIR hyperintensity involving the left thalamus, posterior limb of the internal capsule, and dorsal pons without associated restricted diffusion. MRA showed persistent filling at the base and along the inferior wall of the aneurysm. Post-contrast T1 weighted sequences showed enhancement of the aneurysm wall. She was admitted and started on 8 mg dexamethasone twice daily without significant improvement. A percutaneous gastrostomy tube was placed due to aspiration risk.

Three months after embolization, she presented from a skilled nursing facility with somnolence and no verbal output, following commands on the left side but 1 out of 5 weakness on the right. MRI at that time demonstrated unchanged edema in the left thalamus and pons (not shown). She had a protracted hospitalization complicated by an upper GI bleed, urinary tract infection, and aspiration pneumonia. Her exam progressively declined to being locked in. Repeat cerebral angiography 5 months after embolization demonstrated approximately 90% obliteration of the aneurysm with persistent filling at the base and mild coil compaction.

Given the progressive decline in her exam and unchanged edema in the adjacent left thalamus and pons, she underwent repeat embolization with an additional two PEDs extending from the basilar artery to the left PCA to overlap the previously placed PED. Brain MRI one week following the embolization did not show any significant change in the edema and mass effect along the left thalamus, basal ganglia, and medial left temporal lobe. At the time of discharge, the patient was able to open her eyes to voice and track the examiner with minimal movement in her extremities. Three and six month follow up MRIs were notable for slight increase in size of the basilar tip aneurysm/coil mass, with associated increase in the adjacent parenchymal edema. The patient's neurological exam remained unchanged.

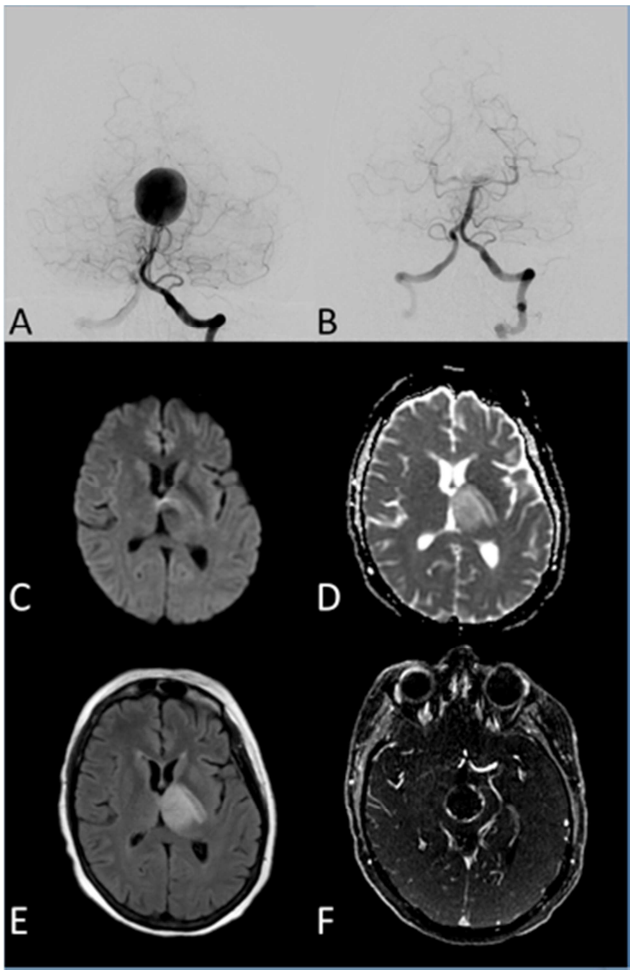

Figure 1. *Post-embolization PAE 2 months after coil embolization and flow diversion of a giant basilar tip aneurysm. (A) Pretreatment catheter angiogram in the Townes projection shows the 3 cm basilar tip aneurysm. (B) Immediate post-treatment angiogram in the same projection shows near complete obliteration of the aneurysm embolization with coils and a flow-diverting stent (Pipeline); there is residual contrast opacification at the aneurysm neck extending into the body (Raymond Roy Class 3). (C) DWI and (B) ADC show facilitated diffusion (no restriction) in the left thalamus, corresponding to the area of FLAIR hyperintensity (E). (F) Post-contrast T1 weighted image shows aneurysm wall enhancement.*

2.2. Case 2

A 60-year-old male with a history of hypertension and hyperlipidemia presented with new seizures. Brain MRI demonstrated a partially thrombosed aneurysm of the left middle cerebral artery with a rim of T2/FLAIR hyperintensity in the adjacent anterior temporal lobe (Figure 2). Cerebral angiogram demonstrated a 12.6 x 9.8 x 7.7mm wide neck left distal M2 middle cerebral artery aneurysm. The patient was started on dual antiplatelet therapy and underwent stent-assisted coil embolization of left MCA aneurysm utilizing the LVIS Blue intraluminal support device and coils. There were no immediate thromboembolic or hemorrhagic complications; the patient was discharged home on post op day 1 neurologically intact.

One week after embolization, he presented to the emergency room following a 10-minute episode of transient word-finding difficulty. Brain MRI demonstrated increased T2/FLAIR hyperintensity and mass effect in the adjacent temporal lobe, without restricted diffusion (Figure 2). Given that the patient's symptoms had resolved, corticosteroids were not given.

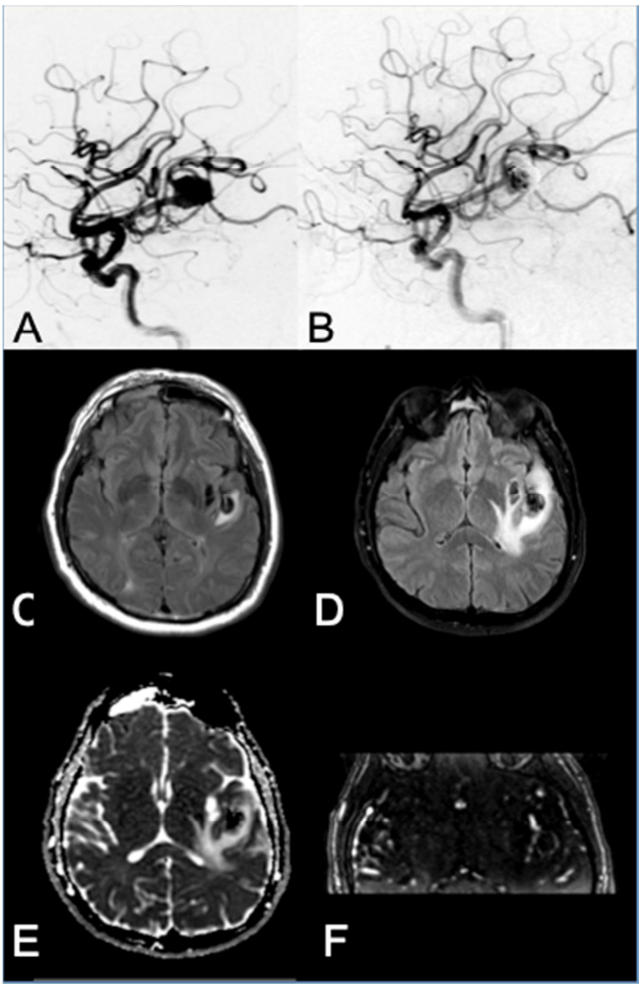

Figure 2. *Left M2 aneurysm in contact with parenchyma. Pre-embolization DSA (A), post-embolization DSA (B), FLAIR with parenchymal hyperintensity pre-embolization (C) and post-embolization (D), post-embolization ADC (E), and post-contrast T1 (F).*

3. Discussion

Post-embolization PAE is an uncommon complication after coil embolization or flow diversion that can present with mild nonspecific symptoms or focal neurological deficit depending on the location and extent of edema. PAE is primarily diagnosed by MRI, which demonstrates T2/FLAIR hyperintensity in the brain parenchyma adjacent to the aneurysm. Affected parenchyma typically demonstrates facilitated diffusion on diffusion-weighted imaging (DWI), in contrast to the restricted diffusion seen in acute ischemia. Post-contrast imaging may demonstrate aneurysm wall enhancement, although this finding is nonspecific and has been observed in up to 18% of cases after coil embolization. [7] Post-embolization aneurysmal wall enhancement in isolation is usually a self-limiting subclinical process and does not require treatment unless symptomatic and associated with edema. Some have suggested that progressive thickening of the enhancing wall may correspond to a higher risk of developing PAE. [12]

The size of the aneurysm is an important factor in development and degree of PAE in unruptured aneurysms. [13] In addition to surface area, the rate of aneurysm thrombosis may play a role. Berge et al, in a case series of 7 patients with post-embolization PAE, reported that acute partial thrombosis of the aneurysm was seen in all cases and patients with more rapid thrombosis had increased surrounding edema. [8]

PAE has been observed following a variety of embolization strategies including coil embolization alone [6, 7] and flow diversion with or without adjunctive coils, using the Silk flow diverter (Balt Extrusion, Montmorency, France) [8], or, in our cases, the Pipeline embolization device and LVIS blue intraluminal support device.

In aneurysms treated with coil embolization alone, PAE was associated with a higher coil packing density, presumably due to more rapid thrombosis [7]. Adjunctive coils are frequently used during flow diversion of larger aneurysm to promote more rapid thrombosis and protect against the increased risk of delayed post-flow diversion rupture. [14] The decision to use adjunctive coils during flow diversion of larger aneurysms must therefore be balanced against the unknown but likely increased risk of post-embolization PAE.

The first-line treatment for symptomatic patients with post-embolization PAE is steroid therapy. In Berge et al., four patients were started on a course of steroid therapy on the day of treatment for 3 weeks and 2 patients received steroids after exacerbation of their symptoms. The improvement of symptoms in both groups was observed around 20 days following the treatment. [8] There is no evidence that prophylactic steroid therapy pre-embolization or in subclinical phase prevents the development of PAE. [12, 15]

4. Conclusion

Interventionalists, neurologists, and neuro-intensivists

should be familiar with post-embolization perianeurysmal edema as an uncommon complication following flow diversion. Because of the nonspecific clinical presentation, ranging from headache to focal deficit, clinicians should rely on MRI with contrast to differentiate PAE from other more common post-embolization syndromes including ischemia and hemorrhage.. DWI and post-contrast T1 weighted sequences are critical for differentiating PAE from acute ischemia, and should be obtained for any patient presenting with seizure or focal deficit after aneurysm embolization.

Steroids are the standard treatment for post-embolization PAE, although in small case series they have had limited efficacy. Clinicians should have a low threshold to initiate steroids in patients with imaging proven edema. Further studies are needed to determine optimal treatment for post-embolization PAE.

Careful consideration should be given to treatment of large aneurysms in contact with parenchyma, especially adjacent to brainstem or eloquent areas of brain. In patients with neurological symptoms related to the mass effect of the aneurysm, surgical clipping can be considered as an option to decompress the mass effect. When an endovascular approach is selected, one needs to decide between the higher risks of delayed aneurysm rupture with isolated flow diversion versus higher risk of PAE (but lower delayed rupture risk) in coil assisted flow diversion.

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