

PRACTICAL PEARL



# Plastic in the Brain: Delayed Recognition of Progressive Unilateral Hemispheric Lesions

Ali Daneshmand, Karl N. Krecke and Eelco F. M. Wijdicks\*

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## Abstract

**Background:** Flow diverters are increasingly used to treat complex ruptured intracranial aneurysms. Most complications are ischemic and seen early after placement.

**Methods:** We present a patient with 3 year duration of neurologic symptoms and seizures as a result of lesions associated with a inflammatory response to embolized polymer coating.

**Results:** Over a period of 3 years MRI abnormalities were noted with substantial gadolinium enhancement of the stent but with resolution after corticosteroids.

**Conclusion:** Polymer embolization associated with a flow diverter may cause fluctuating unilateral hemispheric lesions and become symptomatic. Inflammatory response to a foreign body (polymer strands) can be successfully treated with corticosteroids.

**Keywords:** Endovascular treatment, Aneurysm, Flow diverter, Complications

Flow diverters have become a new staple of interventional neuroradiology. The device is used sparingly and mostly to acutely repair complexly configured, ruptured cerebral aneurysms. Neurointensivists are aware of acute—largely ischemic—complications but, we suspect, are less familiar with late complications, which occasionally are serious enough to require readmission to the neurosciences intensive care unit. We present a unique and dramatic inflammatory response to embolized polymer coating causing transient neurologic symptoms over a number of years and eventually seizures.

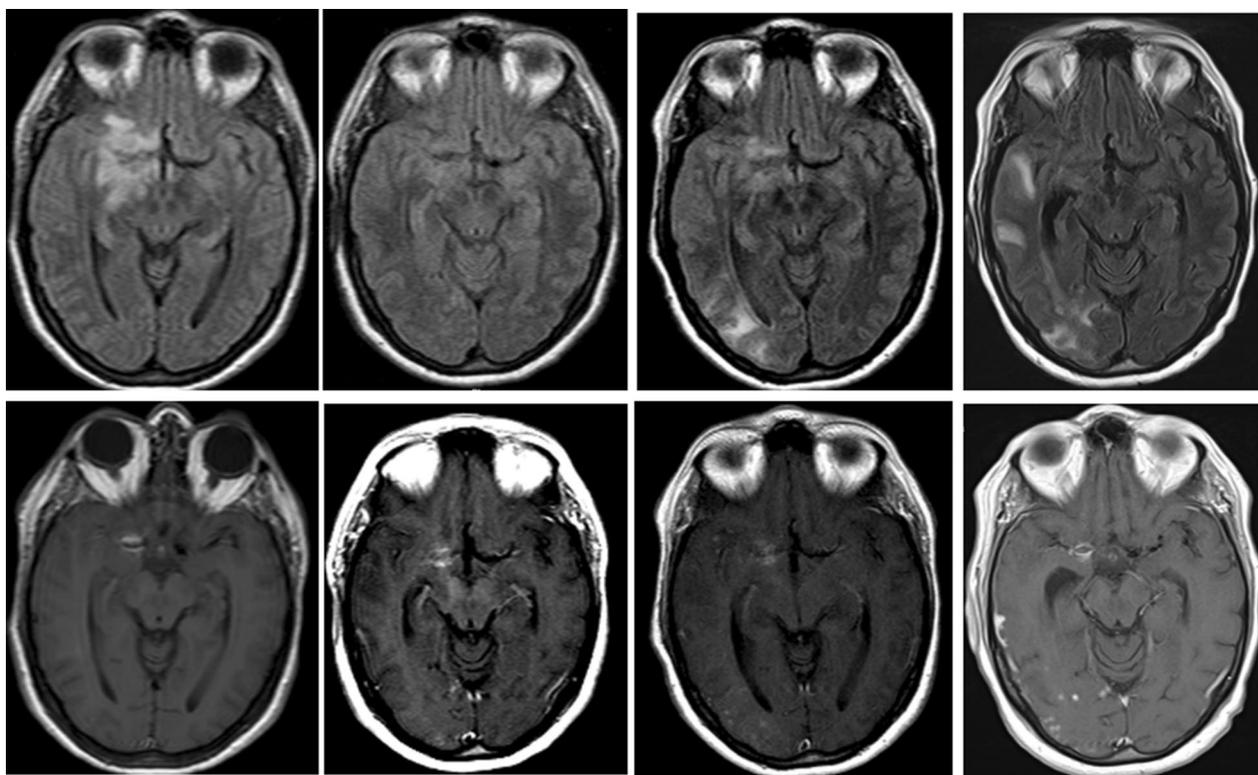
## Case Description

Three years prior to her visit at Mayo Clinic, a 55-year-old woman had a subarachnoid hemorrhage due to a ruptured aneurysm of the right supraclinoid carotid artery, for which she underwent a flow-diverter embolization (first-generation device PED) at an outside medical

center. A year later, she began experiencing intermittent left face and arm twitching several times a week and was started on valproic acid and levetiracetam. Within several months, she developed intermittent, progressive, left-sided numbness, followed by left hemiparesis and headaches. Over the course of three years, brain magnetic resonance imaging (MRI) demonstrated edema and persistent enhancement surrounding the flow diverter, prominent and confluent T2/FLAIR hyper-intensities, and multifocal enhancement in the cortico-subcortical junction, restricted to right carotid artery distribution (Fig. 1). Restricted diffusion was never evident.

Because of ongoing seizures, the patient sought a second opinion, but before evaluation, she developed a generalized tonic-clonic seizure and vomiting and was urgently transported to our emergency department. She had a marked respiratory and lactic acidosis (pH, 6.9; lactate, 16.4 mmol/L; PCO<sub>2</sub> > 100 mmHg) and was emergently intubated for hypercapnic respiratory failure. The seizure was aborted with intravenous lorazepam, and the patient was loaded with intravenous levetiracetam. Continuous electroencephalogram showed frequent

\*Correspondence: wijde@mayo.edu  
Division of Critical Care Neurology and Radiology, Mayo Clinic, 200 First Street SW, Rochester, MN 55905, USA



**Fig. 1** Serial MRIs show progression of disease. The MRIs are obtained 7, 14, 25, 39 months, respectively, after placement of the flow diverter. Upper row: FLAIR sequence. Lower row: T1 sequence showing contrast enhancement. All lesions resolved after repeated MRI following corticosteroid treatment

right-lateralized periodic discharges and brief subclinical seizures. She was extubated the next day, without major neurologic deficits. Examination revealed only a mild left-arm drift with some degree of impaired proprioception. After review of the MRI series, she was treated with intravenous methylprednisolone and continued on oral prednisone. Follow-up MRI one month later showed lesion resolution and no further enhancement of the stent. A cerebral angiogram showed irregularities within the stent, possibly caused by intimal hyperplasia.

### Discussion

There have been several reports of foreign-body emboli after endovascular treatments with coated devices [1–3]. The inflammatory response to embolized hydrophilic polymer coating has been reported days to months after the initial placement. In comparison with prior reported cases, our patient experienced slow progression and delayed recognition, likely due to fluctuation of her symptoms.

MRI findings are non-diagnostic enhancing brain lesions with possible white- and gray-matter involvement with lesions rarely restricted on diffusion weighted

imaging. The proclivity in gray–white matter-junction and vascular-border zone isolated to treated vascular territory supports a micro-embolic phenomenon. Some lesions on MRI produce rim-enhancing patterns and have been interpreted as aseptic abscess evolving to a foreign-body granuloma.

The pathology of biopsied lesions has been described; studies found strands of polymers but also demonstrated an archetypal inflammatory response to a foreign body. Autopsy reports have shown more details with predominant intravascular histiocytic giant cell and neutrophil response with micro-thrombus formation.

Several cases of polymer embolization associated with a marked inflammatory response have been treated with variable doses of corticosteroids. The treated cases have shown brain-lesion resolution several weeks after treatment. Long-term management remains undefined. Flow-diverter coating has changed over time with improved thrombogenicity, but it is unclear whether this also affects polymer embolization.

The recognition of progressive neurological symptoms, including new-onset focal seizures, has been increasingly linked to an inflammatory response to polymer

micro-emboli. Although embolic stroke after vascular intervention is usually attributed to atheroembolic and thromboembolic events, iatrogenic embolization of polymer coating should be considered in suspected cases. Why embolization continues despite expected well-developed neointimal hyperplasia remains unclear. Resolution of the lesions and enhancement after a short course of prednisone was documented, but long-term outcome is not yet known. High-resolution MRI sequences and gadolinium administration are necessary to clinch the diagnosis. We suspect underreporting particularly when symptoms are minor and fluctuating. Our case took 3 years to be accurately diagnosed—others have made forceful arguments about the need to better understand the effects of intravascular polymer coating of metal stents placed in patients [4, 5].

**Author Contribution**

Drs. AD, KNK, EFMW Analyzed data, drafting and critical revision.

**Source of Support**

None.

**Compliance with Ethical Standards****Conflict of interest**

The authors declare that they have no conflict of interest.

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**References**

1. Cruz JP, Marotta T, O'Kelly C, et al. Enhancing brain lesions after endovascular treatment of aneurysms. *AJNR Am J Neuroradiol.* 2014;35:1954–8.
2. Shapiro M, Ollenschleger MD, Baccin C, et al. Foreign body emboli following cerebrovascular interventions: clinical, radiographic, and histopathologic features. *AJNR Am J Neuroradiol.* 2015;36:2121–6.
3. Giordan E, Brinjikji W, Lanzino G. Intracranial foreign body reaction after endovascular procedures. *Neurology.* 2018;90:296–7.
4. Mehta RI, Mehta RI, Solis OE, et al. Hydrophilic polymer emboli: an under-recognized iatrogenic cause of ischemia and infarct. *Mod Pathol.* 2010;23:921–30.
5. Metha RI, Mehta RI. Hydrophilic polymer embolism: an update for physicians. *Am J Med.* 2017;130:e287–90.