

# Restoration of a stent-assisted thrombectomy-related middle cerebral artery stenosis: a case with 3 years' follow-up

Pinar Gelener<sup>1,2</sup> , Süha Halil Akpınar<sup>3</sup> 

<sup>1</sup>Department of Neurology, University of Kyrenia, School of Medicine, Kyrenia, North Cyprus

<sup>2</sup>Department of Neurology, Near East University, School of Medicine, Nicosia, North Cyprus

<sup>3</sup>Department of Radiology, Near East University, School of Medicine, Nicosia, North Cyprus

## Abstract

Early and late-onset stent-assisted thrombectomy (SAT)-related stenosis after endovascular interventions for acute stroke treatment is an entity that is infrequently discussed in the currently available literature. Herein, we present the restoration of SAT-related middle cerebral artery (MCA) stenosis after 3 years in a 65-year-old female patient who underwent endovascular SAT for acute MCA occlusion.

**Keywords:** Acute stroke, stent assisted thrombectomy, thrombectomy related stenosis

## INTRODUCTION

Endovascular thrombectomy is now the standard of care for patients with acute ischemic stroke within the therapeutic time limit for revascularization. Newer generations of devices such as stent retrievers and aspiration catheters are increasingly available, thus improving the clinical outcome while reducing complication rates (1). However, early or late-onset stenosis in the treated vessels has been studied in only few reports (2-4). In a high-resolution intracranial vessel wall-magnetic resonance imaging (MRI) study including patients undergoing stent-assisted mechanical thrombectomy for acute ischemic stroke, arterial wall signal abnormality and thickening secondary to microscopic damage and repair was shown over time. This entity was regarded as 'post-thrombectomy appearance' (5). In animal models, severe disruption of the intima and delayed hyperplasia of this layer was confirmed on histology specimens (6).

Here, we report the restoration of progressive stent-assisted thrombectomy (SAT)-related middle cerebral artery (MCA) stenosis in computed tomography angiography (CTA) after three years follow-up.

## CASE PRESENTATION

A 65-year-old female patient had undergone SAT due to acute left MCA occlusion. On admission, the patient had right-sided hemiplegia and aphasia with an National Institutes of Health Stroke Scale score of 19. An 8-Fr balloon guiding catheter was used to access the distal cervical ICA, and the occlusion site was reached with a Transend 14 microguidewire (Stryker, USA) and a Rebar 2.8-mm microcatheter (ev3, Covidien, USA), and SAT was performed with a 5\*30 mm Solitaire 2 stent (6 x 30 mm, ev3-Covidien, USA). Complete recanalization of TIC1 3 was obtained in the left MCA after four passes of SAT. During the thrombectomy procedure, a focal 1-cm-long internal carotid artery (ICA) dissection occurred at the distal cervical ICA where the balloon guiding catheter was inflated. The patient was discharged with mRS 1 on the 7<sup>th</sup> day.

At the early follow-up of her ICA cervical segment dissection, during the 8<sup>th</sup>-week follow-up magnetic resonance angiography (MRA), she was diagnosed as having focal distal MCA-M1 stenosis, which progressed to significant stenosis (70+%) at the SAT site, as demonstrated by second follow-up imaging obtained using CTA (Figure 1).

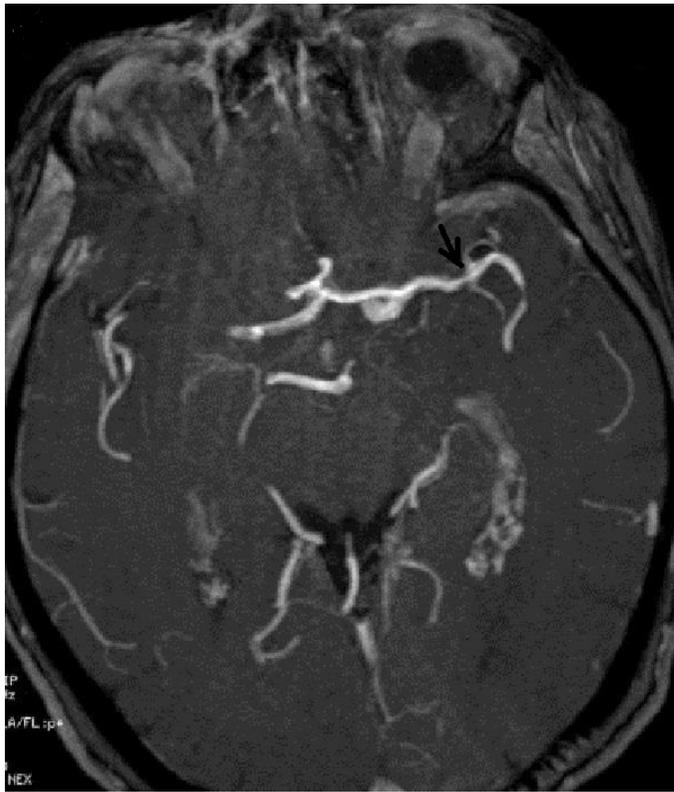
**You may cite this article as:** Gelener P, Akpınar SH. Restoration of a stent-assisted thrombectomy-related middle cerebral artery stenosis: a case with 3 years' follow-up. *Neurol Sci Neurophysiol* 2019; 36(3): 171-3.

**Corresponding Author:** Pinar Gelener **E-mail:** drpinargelener@gmail.com **Submitted:** 5 September 2018 **Accepted:** 26 January 2019

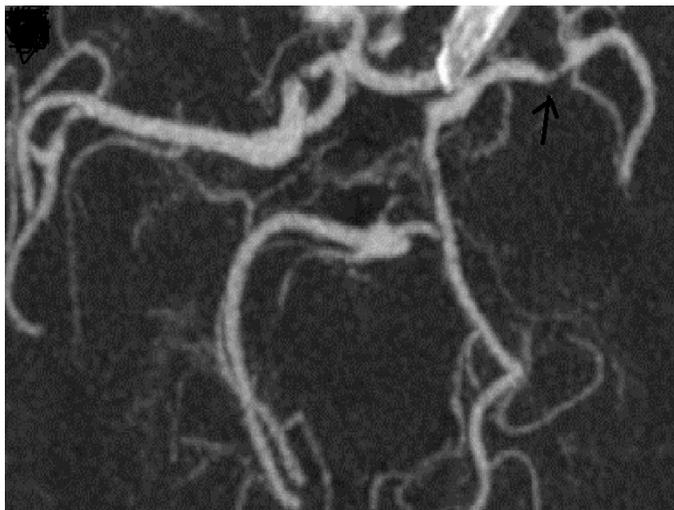


OPEN ACCESS

**Figure 1.** Focal distal MCA-M1 stenosis (70+%) at the SAT site on CTA  
MCA: middle cerebral artery; SAT: stent-assisted thrombectomy; CTA: computed tomography angiography

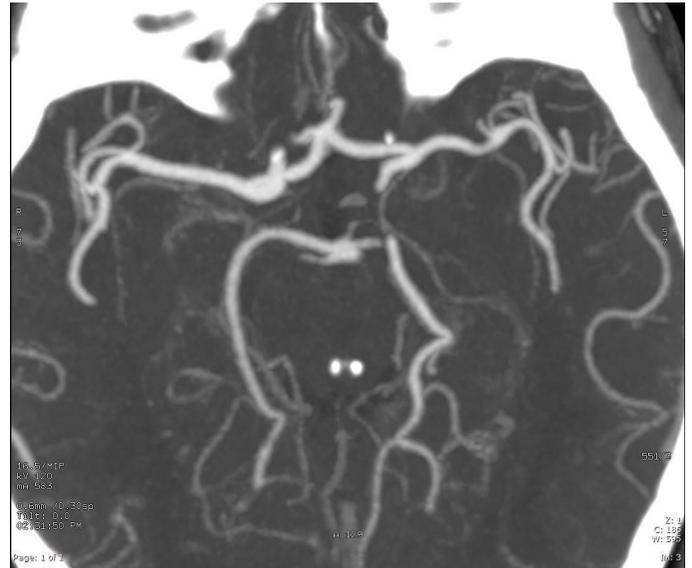


**Figure 2.** The stenosis which had reached a critical level of 90% after 1 year on CTA imaging  
CTA: computed tomography angiography



At the annual follow-up under dual antiplatelet treatment (clopidogrel and aspirin), the stenosis of the MCA where the SAT was performed was progressive and it had reached a critical level of 90%, as revealed with CTA imaging (Figure 2). Although she was put on dual antiplatelet treatment, initially for the non-occlusive ICA dissection, it was not enough to prevent ICA occlusion, but the MCA flow was restored as

**Figure 3.** Restoration of the MCA stenosis on CTA  
MCA: middle cerebral artery; CTA: computed tomography angiography



a result of a patent anterior communicating artery and posterior communicating artery. Clopidogrel was stopped after two years follow-up because her clinical status was stable, but this resulted in a minor stroke attack. Consequently, she was followed under dual antiplatelet treatment and the 3<sup>rd</sup>-year follow-up unexpectedly showed near-total restoration of the MCA stenosis under 3D CTA imaging (Figure 3).

As a result, the presented patient had no stenosis or atherosclerosis in the MCA during and immediately after SAT; however, she developed M1 stenosis that was observed on the 63<sup>rd</sup> day, which reached up to 70-75% at the 6<sup>th</sup> month and was resolved in the third year.

Verbal consent was taken from the patient for this case study.

## DISCUSSION

Stent retrievers are widely used in the treatment of acute ischemic stroke after successful results were obtained in the latest trials. However, there are limited data about early or late-onset SAT-related stenosis and their long-term outcomes. Although the data is limited, it has been shown that vascular stenosis of treated vessels, particularly in the MCA region, may develop during or soon after the endovascular intervention, which is presumed as vasospasm of the vessel as a result of the interventional procedure (1-3).

A thrombectomy procedure may incite trauma and the response of the vessel wall to injury may cause de novo stenosis. If this maintains, long-term luminal narrowing may be associated with recurrent ischemic events, as in our case. Long-term evaluation of the luminal changes of intracranial vessels through neuroimaging, although rare, is important after endovascular interventions for these cases (4, 5).

In the SWIFT trial, it was stated that angiographic vasospasm rates were 16.4% with MERCI and 22.5% with Solitaire devices, but none of these were screened for progression to stenosis (7). In another study, delayed stenosis in the intracranial vessels following endovascular treatment for acute stroke was confirmed using MR angiography in 8.8% of patients (3). Gascou et al. identified early MCA stenosis in 3.47% of patients, which was associated with higher mortality (8). In an angiographic follow-up study, vasospasm occurred in 26% of occluded vessels immediately after mechanical thrombectomy for acute ischemic stroke. Follow-up DSA on the 107<sup>th</sup> day revealed de novo stenosis in 3.4% of patients who were clinically asymptomatic (4). Macke et al. reported two asymptomatic patients with delayed stenosis following the use of stent retrievers in acute stroke intervention in the 5<sup>th</sup> and 6<sup>th</sup> months in the vertebrobasilar region (9).

According to different studies, one of the mechanisms for de novo stenosis is the multiple procedural clot removal attempts, which may further disrupt an unstable plaque and irreversibly damage the vessel wall (8). Another point is that the MRA and CTA can easily detect intracranial stenosis when arteries remain permeable, but are insufficient to diagnose an underlying intracranial stenosis when the vessel is occluded. This study successfully depicted the lumen and walls of the MCA using high-resolution magnetic resonance imaging (HRMRI). Specifically, the plaque surface irregularity and the remodeling ratio on HRMRI were highly suggestive of an unstable plaque (10, 11). Another mechanism revealed is that repeated SAT attempts may increase the risk of intimal disruption (2, 12). However, the study by Kurre et al. revealed no relationship between this entity and the number of thrombectomy passes and dissections (4). Also, the underlying silent intracranial dissection, which spontaneously resolved after dual anti-aggregant therapy and could not be detected by neuroimaging MRA, can be speculated to have a role in the mechanism for this presented case. Another possible mechanism is the vasoprotective and beneficial effects of clopidogrel that was added to aspirin therapy on endothelial nitric oxide bioavailability and the inflammatory status (13).

Endovascular treatments may present a risk of developing early and delayed stenosis in patients undergoing repeated SAT, particularly in the MCA. Detection of this phenomenon is important because stenosis at the SAT site may become symptomatic, as in our case.

**Informed Consent:** Verbal consent was taken from the patient for this case study.

**Peer-review:** Externally peer-reviewed.

**Author Contributions:** Supervision – S.H.A.; Data Collection and/or Processing – P.G., S.H.A.; Analysis and/or Interpretation – S.H.A.; Writing Manuscript – P.G.; Critical Review – S.H.A.; P.G.

**Conflict of Interest:** The authors have no conflicts of interest to declare.

**Financial Disclosure:** The authors declared that this study has received no financial support.

## REFERENCES

1. Chartrain AG, Awad AJ, Mascitelli JR, et al. Novel and emerging technologies for endovascular thrombectomy. *Neurosurg Focus* 2017; 42: E12. [\[CrossRef\]](#)
2. Akpınar S, Yılmaz G. Early middle cerebral artery stenosis following stent-assisted thrombectomy. *Interv Neuroradiol* 2015; 21: 337-340. [\[CrossRef\]](#)
3. Enomoto Y, Takagi T, Matsubara H, et al. Delayed stenosis in the intracranial vessels following endovascular treatment for acute stroke. *J Vasc Interv Radiol* 2015; 26: 1814-1819. [\[CrossRef\]](#)
4. Kurre W, Pérez MA, Horvath D, Schmid E, Bänzner H, Henkes H. Does mechanical thrombectomy in acute embolic stroke have long-term side effects on intracranial vessels? An angiographic follow-up study. *Cardiovasc Intervent Radiol* 2013; 36: 629-636. [\[CrossRef\]](#)
5. Power S, Matouk C, Casaubon LK, et al. Vessel wall magnetic resonance imaging in acute ischemic stroke: effects of embolism and mechanical thrombectomy on the arterial wall. *Stroke* 2014; 45: 2330-2334. [\[CrossRef\]](#)
6. Nogueira RG, Levy EI, Gounis M, Siddiqui AH. The Trevo device: preclinical data of a novel stroke thrombectomy device in two different animal models of arterial thrombo-occlusive disease. *J Neurointerv Surg* 2011; 4: 295-300. [\[CrossRef\]](#)
7. Akins PT, Amar AP, Pakbaz RS, Fields JD; SWIFT Investigators. Complications of endovascular treatment for acute stroke in the SWIFT trial with solitaire and Merci devices. *AJNR Am J Neuroradiol* 2014; 35: 524-528. [\[CrossRef\]](#)
8. Gascou G, Lobotesis K, Machi P, et al. Stent retrievers in acute ischemic stroke: complications and failures during the perioperative period. *AJNR Am J Neuroradiol* 2014; 35: 734-740. [\[CrossRef\]](#)
9. Macke JJ, Bellew MP, Hellinger FR Jr. Delayed stenosis following stentriever use in acute stroke intervention. *J Neurointerv Surg* 2015; 7: e17. [\[CrossRef\]](#)
10. Chung GH, Kwak HS, Hwang SB, Jin GY. High resolution MR imaging in patients with symptomatic middle cerebral artery stenosis. *Eur J Radiol* 2012; 81: 4069-4074. [\[CrossRef\]](#)
11. Marquering HA, Nederkoorn PJ, Smagge L, Gratama van Anandel HA, van den Berg R, Majoie CB. Performance of semiautomatic assessment of carotid artery stenosis on CT angiography: clarification of differences with manual assessment. *AJNR Am J Neuroradiol* 2012; 33: 747-754. [\[CrossRef\]](#)
12. Machi P, Costalat V, Lobotesis K, et al. Solitaire FR thrombectomy system: immediate results in 56 consecutive acute ischemic stroke patients. *J Neurointerv Surg* 2012; 4: 62-66. [\[CrossRef\]](#)
13. Heitzer T, Rudolph V, Schwedhelm E, et al. Clopidogrel improves systemic endothelial nitric oxide bioavailability in patients with coronary artery disease: evidence for antioxidant and anti-inflammatory effects. *Arterioscler Thromb Vascular Biol* 2006; 26: 1648-1652. [\[CrossRef\]](#)