## Stroke due to Late In-Stent Thrombosis Following **Carotid Stenting**

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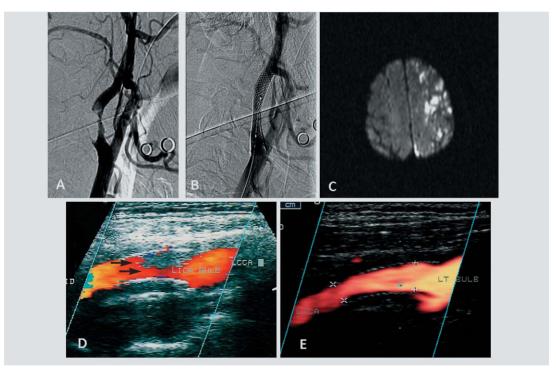


Figure 1. Digital subtraction angiography showed a high-grade stenosis of the left internal carotid artery (A). The stenosis was resolved after the carotid stenting (B). Diffusion-weighted image showed multiple areas of restricted diffusion in the left middle and anterior artery territories, suggesting embolic infarctions (C). Carotid duplex showed an iso-echogenic thrombus at the proximal end of the left ICA stent, arising from the carotid artery proximal to the stent and extending to the stent segment (arrow) (D). The thrombus disappeared at follow-up duplex one month after the stroke (E).

A 71-year-old man received carotid artery stenting (CAS) to the left internal carotid artery (ICA) after repeated minor ischemic strokes, with symptoms including transient right arm numbness, right arm weakness, and blurred vision in the left eye. Fundus examination showed central retinal artery occlusion. Brain magnetic

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resonance image (MRI) showed infarction at the left temporo-occipital lobes and severe stenosis at the left ICA. Angiography confirmed the severe ICA stenosis (Fig. 1A), and stenting to the left ICA was performed smoothly (carotid Wallstent 7mm x 40 mm) (Fig. 1B). He was discharged with nearly no neurological sequela except left-sided blurred vision. Dual antiplatelet therapy with aspirin 100 mg and clopidogrel 75 mg per day was prescribed for 3 months and then shifted to aspirin monotherapy. The post-stent ICA followed by carotid duplex 6 months later was uneventful.

Ten months after CAS, he sustained acute onset with progression of right hemiparesis and aphasia. Brain MRI showed multiple infarcts at the left middle and anterior cerebral artery territories, suggesting embolism (Fig. 1C). Carotid duplex showed a thrombus with isoechogenicity at the proximal end of the left ICA stent, arising from the carotid artery proximal to the stent and extending to the stent segment (Fig. 1D). In-stent thrombosis complicated with embolic stroke was considered, and we started anticoagulation therapy with heparin infusion, then bridged to warfarin 3 days later. His symptoms resolved gradually, and he was discharged with only mild right limbs clumsiness and aphasia. Follow-up carotid duplex one month later showed instent thrombus regressed (Fig. 1E), and his functional status recovered to his baseline.

Acute in-stent thrombosis is a well-known complication within one month after CAS, and it can be resulted from inadequate antiplatelet treatment, hypercoagulable state, procedure related local vascular injury, early stent restenosis or stent underexpansion<sup>(1)</sup>. Delayed onset instent thrombosis after CAS is less frequently mentioned <sup>(2,3)</sup>, although it is a widely-discussed complication after coronary stenting. Chronic inflammation is thought to play a role in the mechanism of late coronary stent thrombosis, and premature antiplatelet therapy interruption is one of the main predictors<sup>(4)</sup>.

In conclusion, stroke due to late in-stent thrombosis, although uncommon, may still occur in patients receiving regular antiplatelet therapy after CAS. Anticoagulation may be an effective treatment in acute stage of in-stent thrombosis related stroke.

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