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Case Report

A Case of Hyperacute Stroke Secondary to Carotid Artery Dissection Treated with Wire Recanalization Alone, without Carotid Stenting

Jae Guk Kim* and Soo Joo Lee

Department of Neurology, Eulji University Hospital, Eulji University, School of Medicine, Daejeon, Republic of Korea, South Korea

***Corresponding Author:** Jae Guk Kim, Department of Neurology, Eulji University Hospital, Eulji University, School of Medicine 95 Dunsanseo-ro, Seo-gu, Daejeon, 35233, Republic of Korea, South Korea.

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Abstract

We describe a 40-year-old male with hyperacute stroke due to dissection-associated carotid occlusion. Carotid occlusion remained after intravenous thrombolysis. During an additional intra-arterial procedure, the carotid artery was recanalized with wire passage alone. Although the patient had a long, multi-segment carotid dissection, we chose medical therapy without carotid stenting because patency of the internal carotid artery was apparent in delayed angiography images. The identification of luminal patency in carotid dissection is important for the selection of treatment with endovascular stent placement in patients with hyperacute stroke.

Keywords: *Carotid artery dissection; Stroke*

Carotid artery dissection, which is a major cause of stroke in young adults, is caused by hereditary connective tissue diseases that cause structural defects in the blood vessel walls or external factors such as trauma or excessive neck movement [1]. It is commonly treated with anti-platelet or anticoagulation medication for 3 - 6 months [2]. However, if there is no response to antithrombotic treatment, if the symptoms worsen, or if vessel occlusion develops due to carotid artery dissection, intravascular procedures, such as stent insertion, are considered for recanalization of the affected artery. We report here on acute phase treatment along with the cerebral angiogram findings of a patient with a stroke caused by dissection-associated carotid artery occlusion.

Case

A 40-year-old male presented with sudden onset of dysarthria and left hemiparesis. He had no particular family or previous medical history, other than hypothyroidism on thyroxine replacement, and a 30-pack-year smoking history. He denied trauma, excessive neck movement, or massage, although he did report a dull headache in the right frontal and temporal regions 3 days prior to presentation, for which he took painkillers.

The patient's blood pressure on presentation was 140/80 mmHg; there was no audible cardiac murmur or carotid bruit. On neurological examination, initial National Institutes of Health Stroke Scale (NIHSS) score was 12, with left central facial palsy, moderate dysarthria, left upper limb Medical Research Council strength grade I, left lower limb strength grade II, and decreased sensation on the left side. Computed tomography angiography of the brain performed two hours after the onset of symptoms showed right proximal carotid artery occlusion (Figure 1A) without acute bleeding. The patient was diagnosed with acute ischemic stroke and given an intravenous thrombolytic agent (rt-PA). Subsequent diffusion-weighted magnetic resonance imaging of the brain revealed ischemic stroke in the right middle cerebral artery (rMCA) territory (Figure 1B). Cerebral angiography was performed with additional intra-arterial thrombolysis, and showed tapered, flame-like occlusion of the right proximal internal carotid artery (ICA) (Figure 2A). Recanalization of the occlusion of right

proximal ICA to reperfuse the rMCA territory was scheduled; passage of a microwire was attempted, but was unsuccessful. Therefore, a Terumo wire was passed through the area of occlusion, and carotid artery angiography was performed with the guide catheter positioned in the proximal ICA. An intimal flap and ectasia were observed in the distal cervical carotid artery, as well as stenosis and a double lumen in the petrous segment of the ICA (Figure 2B). Diagnosis of acute cerebral ischemia secondary to carotid artery dissection was made, and stent insertion was considered. However, pharmacological treatment and close observation were selected, for the following reasons: 1) antiplatelet agents were contraindicated for 24 hours because an intravenous thrombolytic agent was used; 2) rMCA territory reperfusion was achieved through the resolution of the proximal ICA occlusion; and 3) delayed angiography showed good maintenance of blood flow in the dissected region of the carotid artery.

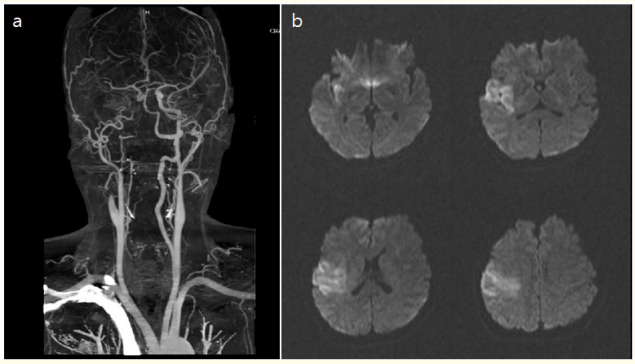

Figure 1: *Brain CT angiography (CTA) and diffusion weighted image (DWI).*
(A) Brain CTA shows that right proximal ICA is occluded and right MCA flow is preserved by collateral flow via anterior communicating and ophthalmic arteries.
(B) DWI shows regional high signal intensities at the right insula, frontoparietal cortex, and some white matter areas, suggesting acute right MCA territory infarction.

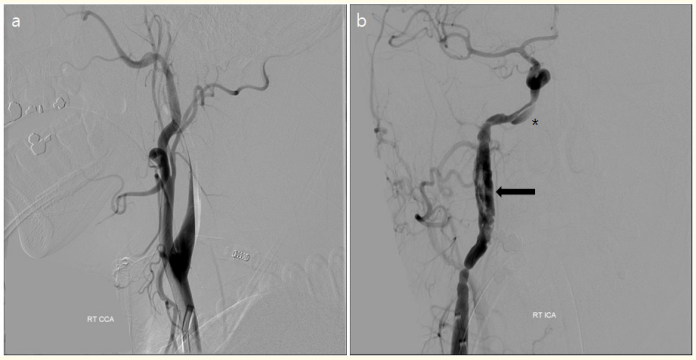

Figure 2: *Digital subtraction angiography.*
(A) The lateral view of right ICA injection demonstrates tapered, flame-like narrowing and occlusion in the proximal portion of the ICA, suggesting dissection.
(B) Double lumen (*) at the petrous ICA and post-stenotic ectasia with intimal flap (arrow) at the distal cervical ICA are observed in the anterior-posterior view of right ICA injection after passage of occlusion site with Terumo wire.

Post-admission testing including blood counts, biochemistry, lipid panel, and urinalysis were normal, except for confirmed asymptomatic hypothyroidism (plasma triiodothyronine concentration 77 ng/dl, plasma free thyroxine concentration 1.55 ng/dl, and thyroid-stimulating hormone 0.33 ng/dl). No particular finding was noted on transthoracic echocardiography or electrocardiography. Moreover, no finding was noted in blood tests for vasculitis. There was no change in the stenosis of the petrous segment of the ICA on magnetic resonance angiography (MRA) performed on the 5th day of admission (Figure 3A). The patient was maintained on anticoagulation therapy with heparin. He was discharged on the 10th day of admission with NIHSS score of 4, with numbness of the left hand, mild dysarthria, and left central facial palsy. There was no change in the intimal flap and ectasia in the proximal cervical ICA on MRA performed 3 months after the onset of symptoms, but stenosis of the petrous segment of the ICA was improved (Figure 3B); the modified Rankin scale score was 1.

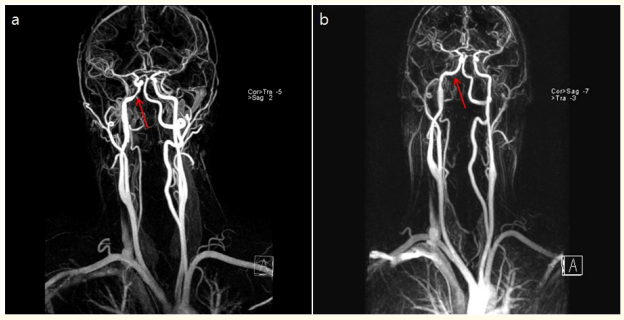

Figure 3: *Follow-up magnetic resonance angiography (MRA).*
(A) MRA on the 5th hospital day shows segmental ectasia at the right distal cervical artery and moderate stenosis at the petrous ICA.
(B) Segmental ectasia at the right distal cervical artery remains, but petrous ICA stenosis is improved on MRA after 3 months.

Discussion

Arterial dissection caused by the separation of the intimal layer can induce intraluminal stenosis or occlusion through intramural hematoma and false lumen formation. Further extension of the damage to the tunica adventitia can cause a dissecting aneurysm or cerebral hemorrhage [1]. Carotid artery dissection commonly presents with unilateral headache or neck pain associated with Horner's syndrome, though it can manifest with severe neurological deficits [1]. Treatment of carotid artery dissection is usually conservative, with antiplatelet or anticoagulant agents [2], and there is no significant difference in efficacy reported between the two agents [3].

The patient in this case report presented with a hyperacute stroke two hours after the onset of neurological symptoms. As the safety and efficacy in patients with carotid artery dissection is known [4], we administered an intravenous thrombolytic agent after confirming the absence of cerebral hemorrhage on brain computed tomography. Subsequent cerebral angiography of the right proximal ICA occlusion showed multi-segment carotid artery dissection. Stenting was considered due to a high risk of re-occlusion from the stenosis of the petrous segment of the ICA as well as the intimal flaps observed in the distal cervical ICA.

Generally, intravascular or surgical recanalization, such as stenting, is indicated in patients with carotid artery dissection on a limited basis, if the patient has vascular occlusion, if the neurological symptoms progress despite pharmacological treatment, or if dissecting aneurysms appear or persist [2]. In patients who present with hyperacute stroke secondary to dissection-associated carotid artery occlusion, such as that reported in this study, more aggressive treatment should be considered. However, it is difficult to choose between intravascular procedures and pharmacotherapy, as only limited research, including case reports on the differences between the two

methods in terms of efficacy and safety, is available, with no randomized studies [5-7]. The patient reported in this paper had a high risk of intra-stent thrombosis, as antiplatelet therapy was contraindicated due to intravenous thrombolysis. Moreover, the occluded area was successfully recanalized using a Terumo wire, and there was no observable re-occlusion of the dissected carotid artery or progression of stenosis on delayed cerebral angiography. Therefore, we chose conservative pharmacological therapy over stenting.

The most important factor in dissection-induced acute stroke is early revascularization by opening the occluded vessels. This case report shows that good prognosis can be achieved without aggressive endovascular treatment of stent insertion, in patient with hyperacute stroke due to dissection-related carotid occlusion, provided that the artery occluded due to carotid dissection is recanalized, and there is no decrease in blood flow in the anterior circulatory system on delayed cerebral angiography.

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