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■ Letter to the Editor ■

Endovascular Stenting for the Treatment of an Initially Asymptomatic Patient with Traumatic Carotid Artery Dissection

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Dear Editor:

Carotid dissection is a rare lesion after head injury and is often known to occur in the case of direct neck trauma. Although most of the carotid artery dissection occurs spontaneously, approximately 4% is associated with severe trauma [1]. For example, major blunt trauma resulting from high-speed motor vehicle accidents [2]. Clinical presentation encompasses a wide range of symptoms, often leading to a delay in diagnosis. Asymptomatic carotid artery injury may not be easily detected during clinical evaluation of head and neck trauma [3,4].

Case

A 78-year-old woman was admitted after being hanging while experiencing a cart wearing a scarf 1 day ago. She had a history of hypercholesterolemia. The neck computed tomography (CT) scan showed bilateral common carotid artery dissection (Figure 1A and B), with patent blood flow and hematoma with swelling in the pretracheal area (Figure 1C). However, the true and false lumen could not be clearly distinguished and the true lumen was not compressed (Figure 1D). There was no problem with perfusion through the bilateral internal carotid artery. The patient was hemodynamically stable and without neurological deficits. We planned follow-up CT after 3 days and the patient will be transfer to her home region if there is no change in the lesion.

Three days later, the patient complained of sudden severe dizziness. Patients were drowsy on the day of imaging follow-up with nausea and poor oral intake, and the Glasgow Coma Scale decreased from 4-5-6 to 3-4-6. Therefore, emergency transfemoral cerebral angiography and diffusion magnetic resonance imaging were performed. The magnetic resonance imaging showed multiple tiny high signal in-

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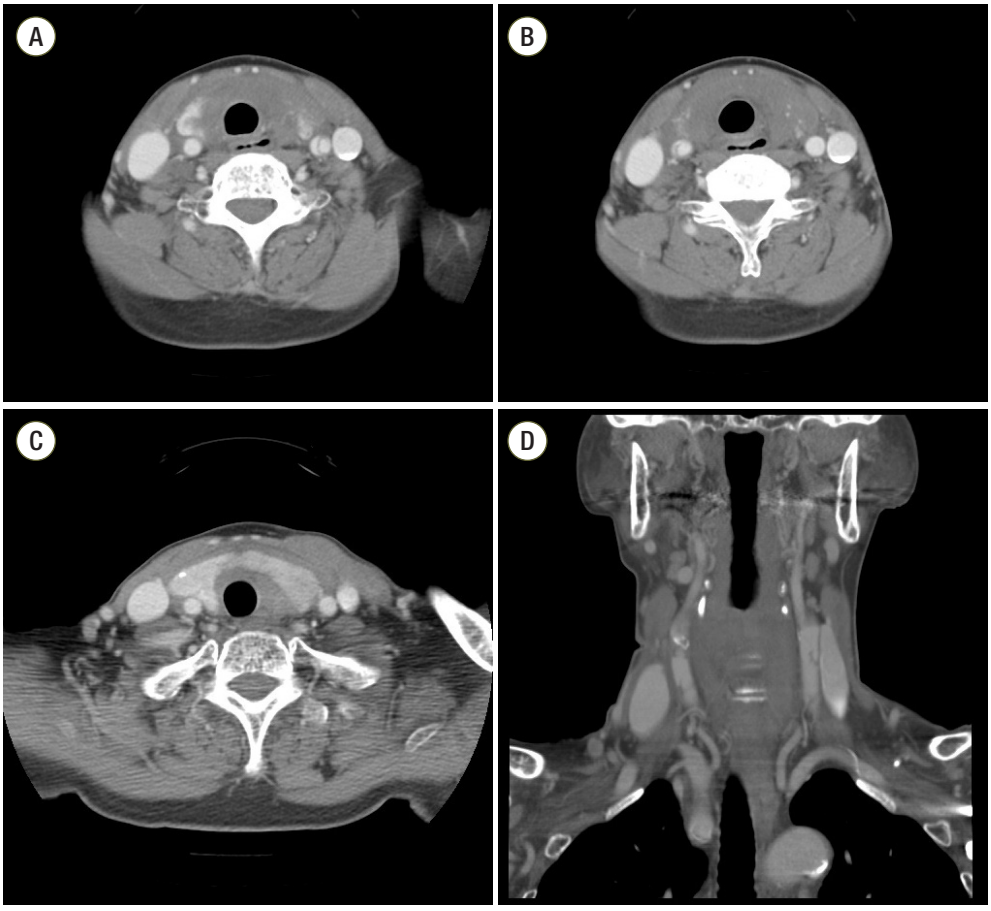

Figure 1. Neck computed tomography scan showing dissections of both common carotid arteries, mid portion. (A) The 2.6-cm segmental intimal flap in the left common carotid artery. (B) A short segmental intimal flap in the right common carotid artery. (C) Diffuse hematoma around the thyroid gland in the anterior neck. (D) Coronal view showing both common carotid artery dissections.

tensities with acute lacunar infarct in both cerebral white matter (Figure 2). On the transfemoral cerebral angiography, the dissection was observed in distal parts of both common carotid arteries and flow flap. As a result, spontaneous healing could not be expected, and endovascular stent insertion was performed in both common carotid arteries (Figure 3). We inserted 10 mm × 60 mm sized self-expanding Nitinol stents on each side without embolic protection device or ballooning.

After stent insertion, we started antithrombotic therapy (clopidogrel 75 mg and aspirin 100 mg per day as oral medications). The patient was discharged about 2 weeks later, complaining only of pain in the anterior neck without any specific neurologic deficiency. The study was performed according to the Helsinki Declaration and ap-

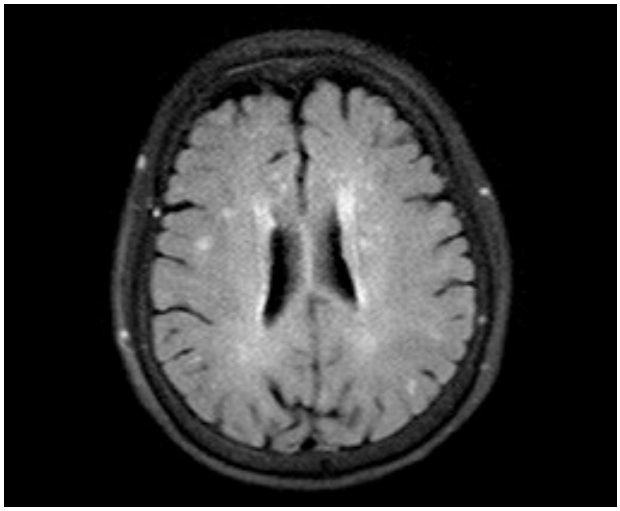

Figure 2. Brain diffusion magnetic resonance image showing a tiny diffusion-restricted lesion in the left frontal white matter. Multiple tiny high signal intensities in both cerebral white matter.

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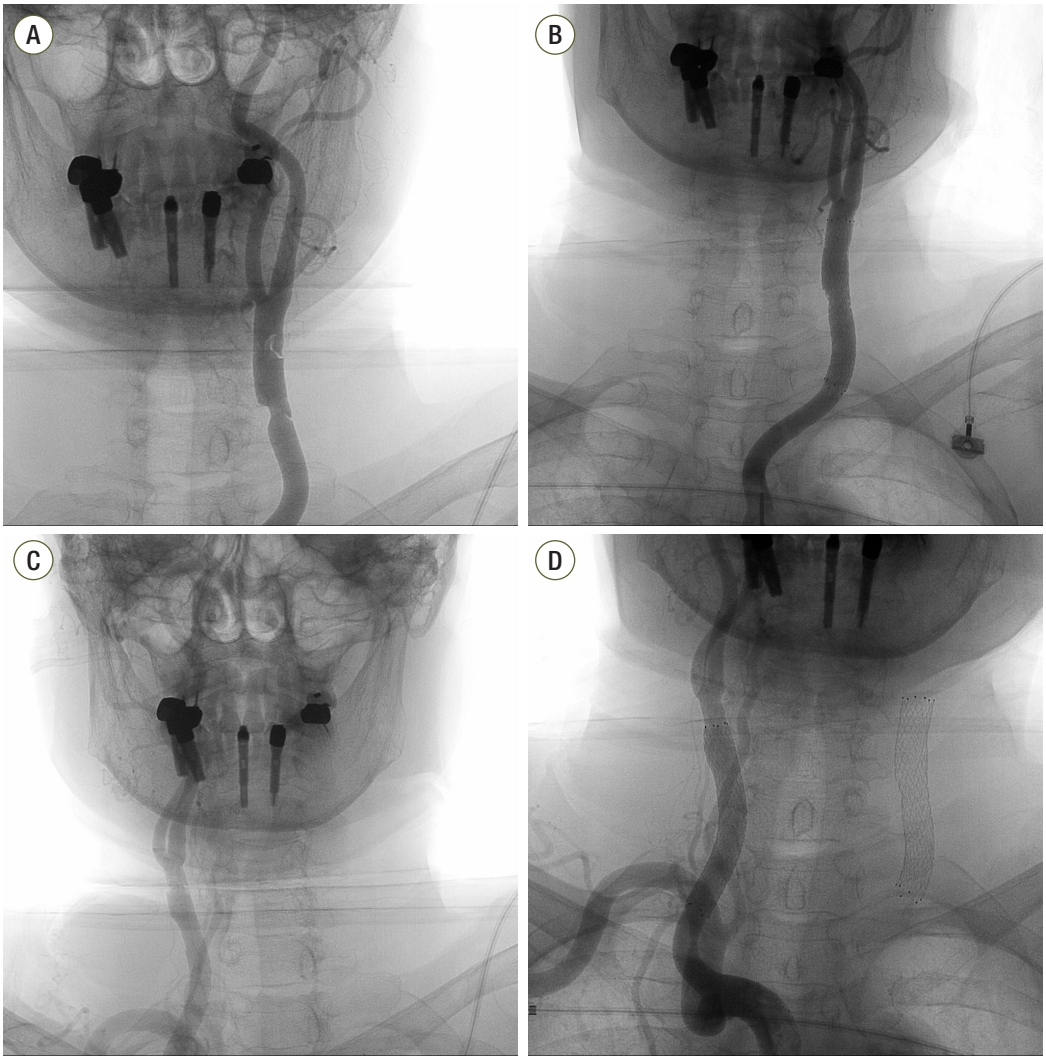

Figure 3. Transfemoral cerebral angiography showing dissection of both distal common carotid arteries. (A) A movable flap was observed according to flow in the left common carotid artery without thrombus. (B) Flow patent after stent insertion in the left common carotid artery. (C) A movable flap was observed according to flow in the right common carotid artery without thrombus. (D) Flow patent after stent insertion in the right common carotid artery.

proved by the institutional review board of Jeju National University Hospital (No. 2016-12-009).

The incidence of carotid artery dissection induced by blunt trauma ranges from less than 1% to 3% [2]. The actual incidence can be even higher and remain undiagnosed. Risk factors for traumatic carotid artery dissection include intense physical activity, blunt injury, and penetrating neck trauma.

In general, about 10% of patients show immediate symptoms. In the first 24 hours after dissection, 55% had symptoms, and 35% had no symptoms for more than 24 hours after injury [5]. Patients with immediate symptoms usually have neurological deficits. Headache, including neck and facial pain, pulsatile tinnitus, decreased taste sensation, focal weakness, and migraine-like symptoms.

Carotid artery dissection begins to tear in one of the carotid arteries, and the blood enters the arterial wall and splits the layer. As a result, intramural hematoma or aneurysmal dillatation is created and this process can either be the source of miroemboli. This injury involves an initial intimal tear and exposes the thrombogenic subendothelial collagen, initiating platelet aggregation with

subsequent thrombus formation that can embolize the artery [6]. Therefore, some studies have recommended an initial antithrombotic regimen [7,8].

In the present case, the patient was unable to undergo antithrombotic therapy at the initial stage due to hematoma-induced neck swelling. On angiography, a thrombus was not present but a visible intimal flap was observed (Figure 3A and C), so the dissection was classified as grade II [9]. The management of low-grade (grade I and II) blunt carotid artery injuries remains controversial. However, antiplatelet agents or anticoagulants are used as first-line treatments, and endovascular stenting is generally reserved for symptomatic or higher-grade blunt injuries [6,10-13]. Low-grade blunt cerebrovascular injuries (BCVI) carry the low risk of cerebral infarction. Griessenauer et al. [14] searched the outcomes in 112 patients with BCVI. They concluded that most ischemic strokes occur before prescreening using CT angiography, and antiplatelet therapy. This indicates that subsequent imaging may not help prevent most ischemic strokes. The progression of injury was not changed by specific treatment or absence of treatment [13,15]. However, according to available literature, it is difficult to determine the actual rate of BCVI-related ischemic strokes. The incidence of BCVI-related ischemic stroke has been reported to be as low as 0.05% and as high as 50% [14,16-18]. At that time of admission, the risk of active bleeding or cerebral hemorrhage was low, but if the hematoma increased, dyspnea would occur and the patient's management might be difficult. Thus, we did not proceed with the initial antithrombotic therapy and proceeded after the endovascular procedure.

Fortunately, this patient had a temporary neurological symptom that disappeared after stent insertion. There were no new symptoms during the 2 weeks of hospitalization. In addition, the hemorrhage in the neck did not progress after antithrombotic therapy. Therefore, we could manage the patient with low-grade carotid artery dissection with endovascular stent insertion without any complication, despite the delayed initial antithrombotic therapy.

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