Endovascular treatment for symptomatic carotid artery dissection

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A 64-year-old man presented with a two-hour history of acute onset right hemiparesis. A computed tomography of the head showed no pathologic findings, and a carotid duplex scan revealed a left carotid artery dissection, as evidenced by double lumens originating from the proximal common carotid artery (A). The patient was treated with systemic heparin anticoagulation. However, he continued to experience worsening of neurologic symptoms 3 days later. As a result, endovascular intervention for the carotid dissection was performed.

A carotid angiogram confirmed that carotid dissection originated from the proximal left common carotid artery orifice (B [Cover]). Moreover, the dissecting false lumen extended distally into the internal carotid artery (arrow, B [Cover]). A 0.035-in guidewire (Boston Scientific) was placed in the true lumen in the common carotid artery, which was followed by the placement of a 10 × 50-mm Wallgraft (Boston Scientific) at the common carotid artery origin, which successfully excluded the dissection entry point. Completion angiogram showed complete obliteration of the false lumen with restoration of the carotid artery circulation (C). The patient had a complete resolution of his neurologic symptoms. Follow-up carotid duplex scan at 16 months showed normal carotid artery anatomy without residual dissection.

DISCUSSION

Spontaneous dissection of the carotid artery is an uncommon phenomenon, with a reported incidence ranging from 1.8% to 2.5%. Clinical consequences of this condition include progressive stenosis, occlusion, or pseudoaneurysm formation. Operative intervention in an acute carotid dissection usually results in clinical failure. Consequently, anticoagulation has been the treatment of choice in clinically stable carotid artery dissection, since it can achieve resolution and healing of the dissection. Infrequently, disease progression can still occur in a small subset of patients due to carotid occlusion or continual cerebral infarction, resulting in unstable neurologic symptoms.

Endovascular therapy for carotid dissection was performed in our patient because of his worsening neurologic symptoms despite full anticoagulation. We placed a self-expanding Wallgraft at the proximal common carotid artery to exclude the dissection entry point. A covered stent was chosen for our patient, as opposed to a self-expanding stent, because it may permit a greater degree of exclusion of the dissection entry point when placed in the true carotid lumen. The full recovery of our patient with resolution of his neurologic symptoms underscores the potential utility of endovascular carotid therapy in selected patients.

REFERENCES


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