Magnetic Resonance Findings in Spontaneous Dissection of the Cervical Internal Carotid Artery

—Case Report—

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Abstract

The magnetic resonance (MR) appearance of a spontaneous dissection of the cervical internal carotid artery (ICA) in a 53-year-old male is described. Cerebral angiograms demonstrated a long-segment stenosis of the left cervical ICA beginning above the common carotid bifurcation and extending to the skull base ("string sign"). T1-, T2-, and proton density-weighted MR images of the upper neck revealed a high-intensity crescent mass expanding the arterial wall and narrowing the arterial lumen of the left ICA. This high-intensity mass was considered to represent the mural hematoma of the involved ICA. Gradual improvement of the dissection was confirmed by both angiography and MR imaging. Cerebral angiograms have shown pathognomonic findings such as double lumen and intimal flap in only some patients with ICA dissection. Our experience suggests that MR demonstration of the mural hematoma is specific and important for diagnosis and follow-up in cases of spontaneous dissection of the cervical ICA.

Key words: internal carotid artery, dissection, magnetic resonance imaging, angiography

Introduction

Spontaneous dissection of the cervical internal carotid artery (ICA) is now considered more common than previously thought, although the etiology remains obscure.7,10,14) Transient ischemic attack (TIA) or completed stroke are the most frequent symptoms in patients with ICA dissection.6,10,17) Cerebral angiography is the usual diagnostic method, with the most frequent appearance of "string sign" beginning above the common carotid bifurcation and extending to the skull base, although this is not considered pathognomonic.5,10,16,18,21) Here, we report serial magnetic resonance (MR) imaging of a patient with ICA dissection confirmed by angiography.

Case Report

A 53-year-old male was in good health until April 24, 1990, when a neck pain suddenly developed on the left without associated trauma. After several hours, the symptoms resolved. On April 26, he had an episode of transient weakness of the right upper extremity. He was admitted to our hospital the next day.

Neurological examination revealed no abnormalities. No carotid bruit was observed. MR imaging (1.5-Tesla SIGNA; General Electronic Medical Systems, Milwaukee, Wis., U.S.A.) demonstrated no abnormal intensity area in the brain, but showed a high-intensity mass narrowing the lumen of the left cervical ICA on the proton density-weighted image (TR = 3000 msec, TE = 30 msec) (Fig. 1). Cerebral angiograms showed a tapered stenosis of the left cervical ICA beginning above the common carotid bifurcation and extending to the skull base (Fig. 2). No other abnormality was found in the carotid and
vertebrobasilar arterial systems. T1- (TR = 500 msec, TE = 20 msec), T2- (TR = 3000 msec, TE = 80 msec), and proton density-weighted (TR = 3000 msec, TE = 30 msec) MR images of the upper neck 14 days after onset revealed that the high-intensity crescent mass (arrow) in the left cervical ICA. This high-intensity mass expanded the arterial wall and narrowed the true lumen of the left ICA.

The 133Xe inhalation method single photon emission computed tomographic (133Xe-SPECT) scans demonstrated normal regional cerebral blood flow and reactivity to acetazolamide.

Antiplatelet aggregation therapy (ticlopidine chloride 200 mg/day) was started. Follow-up MR images 4 and 7 weeks after onset revealed a gradual reduction in the size of the mural high-intensity mass, and gradual improvement in both the expansion of the arterial wall and narrowing of the arterial lumen (Fig. 4 upper, middle). He was discharged 2 months after onset.

Repeat cerebral angiograms 5 months after onset showed significant improvement of the left ICA dissection (Fig. 5). MR images 7 months after onset revealed normal appearance of the left ICA (Fig. 4 lower). He has experienced no further ischemic symptoms.

Discussion

The cause of spontaneous ICA dissection remains unknown. Possible causes include dissection initiated by subintimal hemorrhage or primary disruption of the intima overlying the atheromatous plaque initiating hemorrhagic dissection into the plaque. Minor trauma such as nose blowing or repeated cough may be important in some cases.

Cervical ICA dissections usually cause cerebral ischemic symptoms beginning in the ipsilateral neck or head. Incomplete Horner's syndrome (oculosympathetic paresis) caused by the involve-
Follow-up MR images 4 (upper) and 7 weeks (middle) after onset, showing gradual decrease in the mural hematoma in the left cervical ICA. The mural hematoma is not visible 7 months after onset (lower).

Cerebral ischemic symptoms have two mechanisms: emboli arising from thrombus within the true lumen at the point of dissection, and a marked decrease in the caliber of the true lumen.
causing reduced cerebral blood flow. Petro et al.\(^\text{17}\) suggested that the cause of ischemic stroke is more often embolic than hemodynamic. In our case, the TIA probably resulted from thromboembolism because \(^{133}\text{Xe}-\text{SPECT}\) scans revealed normal cerebral blood flow and reactivity to acetazolamide in the ipsilateral cerebral hemisphere.\(^\text{11}\)

Stenotic ICA dissections frequently partially or completely resolve with time.\(^\text{3,5,7,10,16,17,21}\) The long-term outcome is favorable\(^\text{18}\) and recurrence of ICA dissection is very rare.\(^\text{1}\) However, progression of stenosis, occlusion, and the formation of pseudoaneurysm may occur.\(^\text{3,5,7,10,17}\) Angiography has often demonstrated residual damage in the healed carotid artery.\(^\text{10,18}\) Therefore, anticoagulants or antiplatelet drugs are required to prevent thrombosis in the arterial lumen. Surgery is indicated for patients with recurrent ischemic attack, pseudoaneurysm, or hemodynamic compromise due to severe ICA stenosis.\(^\text{14}\)

Angiography is the most reliable modality for diagnosis of spontaneous dissection. Among the various angiographic findings, the most frequent is the "string sign," a long-segment stenosis starting about 2–3 cm above the common carotid bifurcation and extending throughout the extracranial portion of the ICA.\(^\text{5,10,16-18,21}\) However, this finding is not specific and is difficult to differentiate from atherosclerosis, fibromuscular dysplasia, or neoplastic or inflammatory lesions. Unfortunately, pathognomonic findings such as double lumen and intimal flap occur in only a minority of patients with cervical ICA dissection.

The MR appearance of spontaneous carotid or vertebral artery dissection in the subacute stage includes high-intensity crescent mass expanding the arterial walls, and luminal narrowing of the artery on both T1- and T2-weighted images,\(^\text{2,8,12,19,20}\) as in the present case. Gomori et al.\(^\text{20}\) and Zimmerman et al.\(^\text{23}\) reported that a higher signal intensity on both T1- and T2-weighted images indicates the extracellular methemoglobin in the subacute clot, and this high-intensity mass represents the mural hematoma in the dissecting artery. In the present case, the mural hematoma of the left ICA was present from 4 days to 7 weeks after spontaneous dissection. Angiographic indications of improvement in the ICA dissection correlated with reduction in the size of the mural hematoma on MR images. Neither mural hematoma nor expansion of arterial wall are present on the MR images of patients with atherosclerotic carotid artery disease.

Our experience suggests that MR demonstration of mural hematoma is pathognomonic of cervical carotid artery dissection, and this is specific and important for diagnosis and follow-up in patients with spontaneous dissection of the cervical ICA.

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