Neurol Med Chir (Tokyo) 46, 434～437, 2006

Penetrating Atheroma in Cervical Carotid Artery Stenosis
—Case Report—

Ayumi NARISAWA*,**, Hiroaki SHIMIZU*, Mika WATANABE***, and Teiji TOMINAGA**

*Department of Neurosurgery, Kohnan Hospital, Sendai, Miyagi;
**Department of Neurosurgery, Tohoku University Graduate School of Medicine, Sendai, Miyagi;
***Department of Pathology, Tohoku University Hospital, Sendai, Miyagi

Abstract

A 61-year-old male presented with left hand motor weakness associated with cerebral infarction in the right frontal lobe. Right common carotid angiography demonstrated a 66% stenosis and carotid duplex scan demonstrated intermediate echogenic plaque, indicating typical carotid plaque. Carotid endarterectomy was performed 22 weeks after the ischemic onset. During exposure of the carotid artery, a soft and yellowish mass (5 × 5 mm) was observed in the lateral wall of the carotid bulbus, which was not covered with adventitia but with thin connective tissue. The mass was removed en-bloc with a small part of the surrounding arterial wall combined with ordinary endarterectomy. The artery was closed with a collagen-impregnated polyester patch graft (Hemashield patch™) to maintain adequate arterial lumen. Histological examination of the removed plaque confirmed that atheroma had protruded from the intima through the media as well as the adventitia and formed an extra-arterial mass. Such a case requires great care to dissect the carotid artery to prevent premature disintegration of the atheroma.

Key words: atherosclerosis, carotid endarterectomy, penetrating atheroma, plaque

Introduction

Cervical carotid artery stenosis resulting from atherosclerotic changes is one of the major causes of stroke, and accounts for 16–33% of all cases of ischemic stroke.[3,8] Atherosclerosis is usually characterized by the formation of intimal lesions called atheroma that protrude into and obstruct the vascular lumen. Atherosclerotic plaque originates in intimal injury.[9] Foam cells penetrate the injured wall and cholesterol accumulates. Atheromatous plaque is limited to the intima at first, and the media becomes weakened with the progression of atherosclerosis. The media is often involved but adventitial involvement is rare. When carotid endarterectomy (CEA) is indicated, one of the most important technical concerns is to dissect the intimal plaque but preserve the media and the adventitia as far as possible. If the media as well as the adventitia are invaded by the plaque, then special attention is needed not to injure the adventitia.

Received December 13, 2005; Accepted March 27, 2006

Here, we present a rare case of cervical internal carotid artery (ICA) stenosis caused by atheromatous plaque which penetrated the adventitia and formed a mass outside the artery. CEA required meticulous dissection and closure of the artery.

Case Report

A 61-year-old man presented with left hand motor weakness and was admitted to a neighboring hospital on the next day. T_2-weighted magnetic resonance imaging on admission showed a small hyperintense area in the right precentral gyrus (Fig. 1A). Conventional angiography showed a 66% right cervical ICA stenosis by the North American Symptomatic Carotid Endarterectomy Trial method (Fig. 1B). He was treated with heparin and then ticlopidine, then transferred to our hospital 40 days after the onset for possible surgical treatment.

On admission he presented with residual mild left hand motor weakness. He had a medical history of hypertension and hyperlipidemia. Carotid duplex study showed that the intima-media thickness was 4.9 mm in the right carotid bulbus. The echogenicity

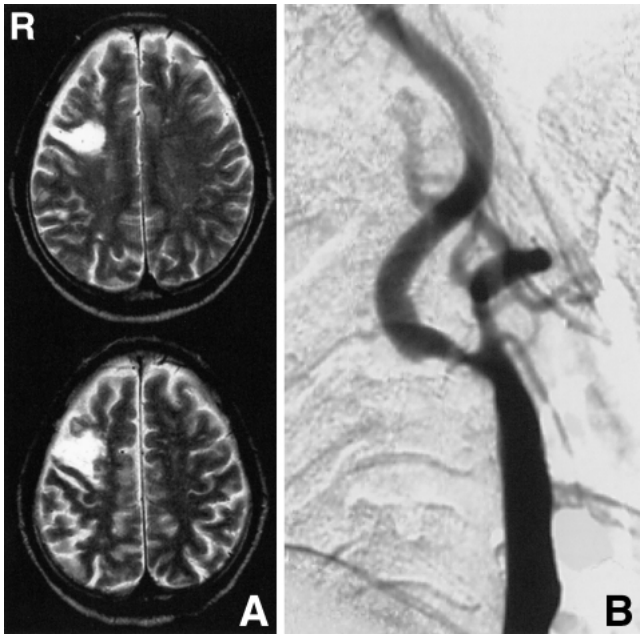

Fig. 1 **A: Preoperative T_2-weighted magnetic resonance images showing a small hyperintense area in the right precentral gyrus. B: Right common carotid angiogram showing a stenosis of 66% by the North American Symptomatic Carotid Endarterectomy Trial method.**

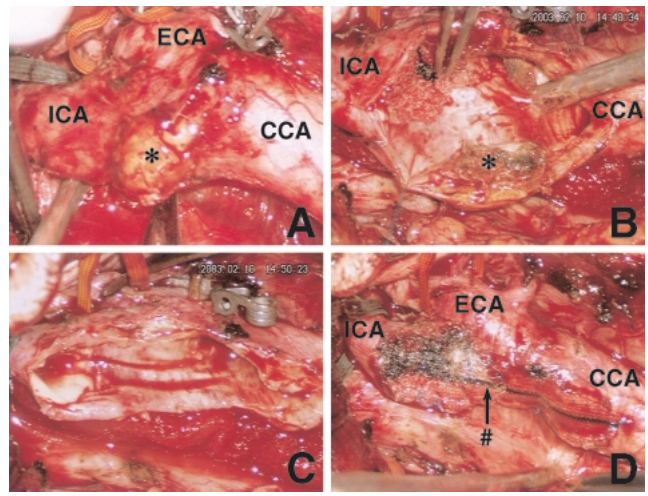

Fig. 2 **Intraoperative photographs of the right carotid artery. A: Appearance of the carotid artery before arteriotomy showing the atheromatous plaque (*asterisk*) projecting from the artery. B: After removal of the bulk of the atheroma showing the residual plaque penetrating the adventitia (*asterisk*) which was removed en-bloc. C: After removal of the protruding atheromatous plaque with the surrounding arterial wall. D: Final view showing arterial closure with a Hemashield patch graft (*sharp*). CCA: common carotid artery, ECA: external carotid artery, ICA: internal carotid artery.**

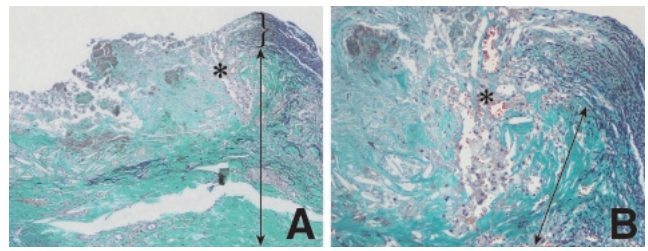

Fig. 3 **Photomicrographs of the protruding atheromatous plaque. A: Atheromatous plaque (*asterisk*) had interrupted the media (*brace*) and invaded the adventitia (*arrow*). Elastica-Masson stain, original magnification ×4. B: Cluster of foamy histiocytes and cholesterin (*asterisk*) had destroyed the collagen fiber of the adventitia (*arrow*). Elastica-Masson stain, original magnification ×10.**

was intermediate and there was no indication that the atheroma protruded out of the adventitia. CEA was planned according to the surgical indications for symptomatic patients.[6,7] However, since preoperative screening with coronary arteriography revealed severe stenosis of the left descending artery, percutaneous transluminal angioplasty was first performed in another hospital.

Twelve weeks after the coronary intervention and 22 weeks after the onset, CEA was performed under somatosensory evoked potential (SEP) and regional cerebral oxygen saturation (rSO_2) monitoring from the bilateral frontal and right temporal regions. On reaching the carotid bifurcation, a yellowish mass was observed protruding out of the vessel wall (Fig. 2A). The mass appeared to be fragile atheromatous plaque and the adventitia over the mass had disappeared over an area of 5 mm in diameter. The atheroma seemed to penetrate the adventitia and was only covered with loose connective tissue. Care was taken not to injure the fragile atheroma before the carotid artery could be dissected proximally and distally for temporary occlusion.

After systemic heparinization, endarterectomy was performed during temporary occlusion of the carotid system. The arterial incision was made longitudinally avoiding the protruding atheroma (Fig. 2B). Since SEP and rSO_2 monitoring detected no change, no shunt was used during the occlusion. The yellowish mass was totally removed from the

carotid wall to form an oval orifice surrounded by the endarterectomized media-adventitia (Fig. 2C). Then a collagen-impregnated polyester patch graft (Hemashield patch™; Boston Scientific, Ballybrit Business Park, Galway, Ireland) was used for arterial closure (Fig. 2D).

Histological examination of the resected carotid artery found marked atherosclerosis with thickening of the intima by fibrosis and atheroma plaque consisting of foamy histiocytes and cholesterin (Fig. 3). The media had completely disappeared and the adventitial collagenous tissue was penetrated by the atheroma in the yellowish mass, confirming that the mass was part of the atheroma.

The postoperative course was uneventful and he left hospital 14 days after the surgery with no new neurological deficit.

Discussion

One of the major hypotheses for the pathogenesis of atherosclerosis is called the ''response to injury'' hypothesis, which suggests that atherosclerosis is a chronic inflammatory response of the arterial wall initiated by injury to the endothelium.[9] Chronic endothelial injury leads to increased permeability, leukocyte adhesion, and higher thrombogenic potential. Monocytes and leukocytes migrate into the intima and are transformed into macrophages and foam cells. Then smooth muscle cells migrate from the media into the intima and proliferate in the intima. Finally, lipids accumulate within the cells as well as extracellularly.

Atheroma in the cervical carotid arteries is generally formed in the intima but often involves the media. However, atrophy of the underlying media with loss of elastic tissue in advanced plaque causes weakness of the arterial wall.[9] Unstable plaques of the carotid artery show histological evidence of collagen and elastic fiber fragmentation.[2] In our case, the histological findings showed that the media was interrupted and foamy histiocytes and cholesterin had extended from the adventitia into the connective tissue. Therefore, we suspected that such media and adventitia weakening may have been involved in the mechanism of atheroma protrusion.

In this case, even retrospective peer review found no preoperative radiological evidence to suggest that the atheroma in the carotid artery had penetrated the vessel wall. Methods which could depict such plaque advancement include carotid duplex scanning, intravascular ultrasonography,[1,5] black blood magnetic resonance imaging,[10] optical coherence tomography (OCT),[4] and others. The first three methods do not have sufficient resolution to demarcate the adventitia clearly at present. OCT is a new imaging method with a high resolution of approximately 10–20 μm. OCT imaging showed the intima, media, and adventitia in a case of coronary stenosis with good correlation of the intimal thickness measured by OCT and histological examination. However, the maximum depth of penetration of OCT is approximately 2.0 mm from the center of the catheter.[4]

The present case appears to be the first to illustrate penetrating atheroma in the carotid artery and shows that careful dissection of the carotid artery is essential if the carotid wall is unusually colored, to prevent premature disintegration of the fragile atheroma. Primary arterial closure may not be possible after removal of the plaque, so patch graft closure should be considered to maintain adequate arterial lumen. To minimize the temporary occlusion time, a patch graft should be prepared before carotid clamping.

References

1) Clark DJ, Lessio S, O'Donoghue M, Schainfeld R, Rosenfield K: Safety and utility of intravascular ultrasound-guided carotid artery stenting. *Catheter Cardiovasc Interv* 63: 355–362, 2004
2) Formato M, Farina M, Spirito R, Maggioni M, Guarino A, Cherchi GM, Biglioli P, Edelstein C, Scanu AM: Evidence for a proinflammatory and proteolytic environment in plaques from endarterectomy segments of human carotid arteries. *Arterioscler Thromb Vasc Biol* 24: 129–135, 2004
3) Kimura K, Kazui S, Minematsu K, Yamaguchi T; Japan Multicenter Stroke Investigator's Collaboration: Analysis of 16,922 patients with acute ischemic stroke and transient ischemic attack in Japan. A hospital-based prospective registration study. *Cerebrovasc Dis* 18: 47–56, 2004
4) Kume T, Akasaka T, Kawamoto T, Watanabe N, Toyota E, Neishi Y, Sukmawan R, Sadahira Y, Yoshida K: Assessment of coronary intima–media thickness by optical coherence tomography: comparison with intravascular ultrasound. *Circ J* 69: 903–907, 2005
5) Lockwood GR, Ryan LK, Gotlieb AI, Lonn E, Hunt JW, Liu P, Foster FS: In vitro high resolution intravascular imaging in muscular and elastic arteries. *J Am Coll Cardiol* 20: 153–160, 1992
6) Moore WS, Barnett HJ, Beebe HG, Bernstein EF, Brener BJ, Brott T, Caplan LR, Day A, Goldstone J, Hobson RW 2nd, Kempczinski RF, Matchar DB, Mayberg MR, Nicolaides AN, Norris JW, Ricotta JJ, Robertson JT, Rutherford RB, Thomas D, Toole JF, Trout HH 3rd, Wiebers DO: Guidelines for carotid endarterectomy. A multidisciplinary consensus statement from the ad hoc Committee, American Heart Association. *Stroke* 26: 188–201, 1995

7) National Institute of Neurological Disorders and Stroke Stroke and Trauma Division. North American Symptomatic Carotid Endarterectomy Trial (NASCET) investigators: Clinical alert: benefit of carotid endarterectomy for patients with high-grade stenosis of the internal carotid artery. *Stroke* 22: 816–817, 1991
8) Petty GW, Brown RD Jr, Whisnant JP, Sicks JD, O'Fallon WM, Wiebers DO: Ischemic stroke subtypes: a population-based study of incidence and risk factors. *Stroke* 30: 2513–2516, 1999
9) Schoen FJ, Cotran RS: The blood vessels, *in* Schmitt W (*ed*): *Robbins Basic Pathology, ed 7*. Philadelphia, Saunders, 2003, pp 325–360
10) U-King-Im JM, Trivedi RA, Sala E, Graves MJ, Gaskarth M, Higgins NJ, Cross JC, Hollingworth W, Coulden RA, Kirkpatrick PJ, Antoun NM, Gillard JH: Evaluation of carotid stenosis with axial high-resolution black-blood MR imaging. *Eur Radiol* 14: 1154–1161, 2004

Address reprint requests to: A. Narisawa, M.D., Department of Neurosurgery, Tohoku University Graduate School of Medicine, 1–1 Seiryo–cho, Aoba–ku, Sendai 980–8574, Japan.