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Unusual Angiographic Changes in an Internal Carotid Artery Pseudoaneurysm After Infection in the Deep Neck Space

—Case Report—

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Abstract

A 48-year-old man presented with a pseudoaneurysm at the cervical portion of the left internal carotid artery (ICA) secondary to infection in the deep neck space. Magnetic resonance (MR) imaging demonstrated enhancement of the wall of the ICA and a pseudoaneurysm, considered to be sequelae of infection spread. ICA occlusion occurred on the next day resulting in sudden onset of right hemiparesis and motor aphasia. The ICA pseudoaneurysm shrank gradually and his neurological deficits improved with conservative therapy. One month later, he presented with aneurysm regrowth. The common carotid artery was occluded with Guglielmi detachable coils to block arterial flow into the pseudoaneurysm. There were no neurological complications. Marked enhancement of the ICA wall on computed tomography and MR imaging may indicate the possibility of vascular complications such as rupture, pseudoaneurysm development, or ICA occlusion, and consequent neurological deficits. ICA occlusion caused by spread of infection in the deep neck space may cause accelerated coagulopathy due to ICA wall inflammation.

Key words: internal carotid artery, pseudoaneurysm, infection

Introduction

Infections in the deep neck space are characterized by the spread of inflammation leading to potential complications because of the unique anatomical relations between the adjacent structures.[1,5,8,15] The fascia separating the neck spaces does not provide anatomical barrier functions, so infections can involve several of the prevertebral, parapharyngeal, retropharyngeal, and carotid spaces.[1,9,15] Extension of the infection into the carotid or jugular vessels can result in potentially severe neurological deficits.[1,5,8,15] Although the use of antibiotics has reduced the complication rate of neck space infections,[1,9,20] occurrence may still be life-threatening.[18] Vascular complications of such infections are rare, but the natural history of untreated cases indicates a poor prognosis. Therefore, early detection and discovery of carotid artery involvement are important.[9,18,20]

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We present a rare case of internal carotid artery (ICA) pseudoaneurysm caused by infection in the deep neck space.

Case Report

A 48-year-old man was referred to our hospital with swelling, redness, and tenderness on the left side of the neck in May 2002. His medical history revealed no obvious abnormality. His body temperature was 36.9°C on admission. Laboratory examinations demonstrated white blood cell count of 7200/mm^3, C-reactive protein level of 0.4 mg/dl, and erythrocyte sedimentation rate of 36.0 mm/1 hr and 86.0 mm/2 hrs, suggesting chronic active inflammation. We suspected deep neck infection and administered antibiotics.

Computed tomography (CT) and angiography suggested an aneurysm at the cervical portion of the ICA. CT showed the aneurysm wall as irregularly enhanced and surrounded by hematoma (Fig. 1A).

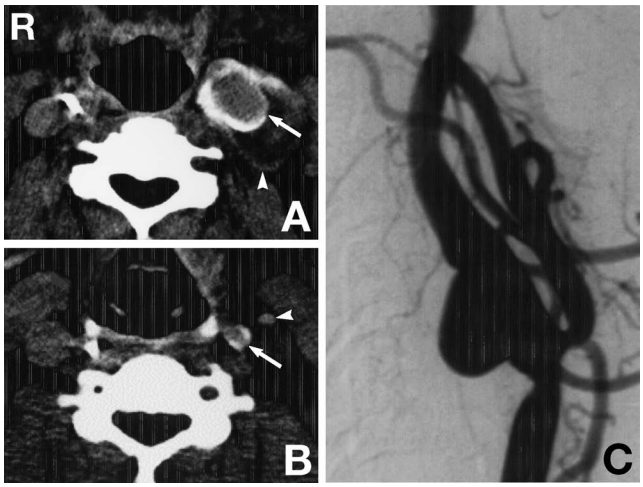

Fig. 1 **A, B: Computed tomography scans of the neck, (A) obtained at admission showing an aneurysm at the cervical portion of the internal carotid artery (ICA) (*arrow*), irregular enhancement of the ICA wall with a partial defect, and surrounding hematoma (*arrowhead*), and (B) after angiography showing marked enhancement of the ICA wall (*arrow*) but not the external carotid artery wall (*arrowhead*). C: Left common carotid angiogram showing stenosis of the common carotid artery and the irregularly-shaped aneurysm at the cervical portion of the ICA. The diagnosis was pseudoaneurysm.**

The walls of the ICA and common carotid artery (CCA) also showed marked enhancement suggesting inflammation. The external carotid artery (ECA) wall was not enhanced (Fig. 1B). Angiography revealed stenosis of the CCA and an irregularly-shaped aneurysm arising from the ICA (Fig. 1C). The diagnosis was pseudoaneurysm. There were no abnormal neurological findings. Laboratory test showed increased sedimentation rate. Arterial blood culture was negative.

On the following morning, he suddenly manifested right hemiparesis and motor aphasia. Magnetic resonance (MR) imaging and MR angiography were performed immediately, and showed multiple hyperintense lesions on the T_2- and diffusion-weighted images, suggesting border zone infarction between the territories of the anterior and middle cerebral arteries (Fig. 2A). T_1-weighted MR imaging with gadolinium indicated marked enhancement of the ICA wall and pseudoaneurysm wall (Fig. 2B, C). MR angiography delineated ICA occlusion, and patent flow into the aneurysm and ECA (Fig. 2D).

Angiography performed 1 week later demonstrated ICA occlusion and preservation of the ECA flow

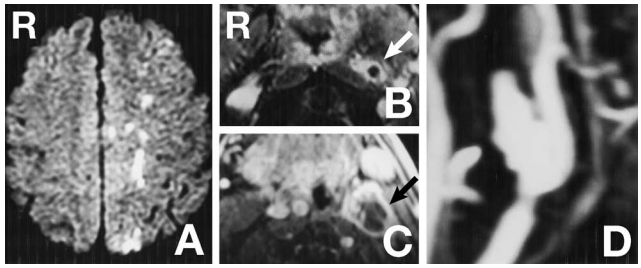

Fig. 2 **A: Diffusion-weighted magnetic resonance (MR) image obtained after the manifestation of hemiparesis and motor aphasia showing multiple hyperintense lesions suggesting border zone infarction between the territories of the anterior and middle cerebral arteries. B, C: T_1-weighted MR images with contrast medium showing marked enhancement of the internal carotid artery (ICA) wall (B, *arrow*) and pseudoaneurysm wall (C, *arrow*). D: MR angiogram demonstrating occlusion of the ICA distal to the aneurysm, and patent flow to the aneurysm and external carotid artery.**

(Fig. 3A), and 2 months later showed marked shrinkage of the aneurysm (Fig. 3B). The patient was then discharged without neurological deficits. However, he returned 1 month later complaining of recurrent neck swelling. Emergency CT and angiography revealed recurrence of the aneurysm and enhancement of the CCA wall (Fig. 3C). Laboratory data showed no signs of infection. Steroid therapy resulted in disappearance of the CCA wall enhancement on MR imaging. After confirming tolerance of the balloon occlusion test, CCA occlusion was performed with Guglielmi detachable coils. After the procedure, thrombosis of the aneurysm was found, but the left ECA remained patent via a shunt between the bilateral superior thyroid arteries (Fig. 3D). He was discharged without neurological deficits and antibiotic medication was continued for 2 months. The patient has not experienced aneurysm recurrence or inflammation during the 4-year follow up.

Discussion

In our patient, CT detected no indications of inflammatory change in the deep neck space and no damaged fat tissue. However, he presented with the typical clinical signs of deep neck infection on the left side of his neck such as swelling, redness, and tenderness, so we had to consider spread of the infection into the carotid space, which carries the risk of thromboembolism, occlusion, pseudoaneurysm

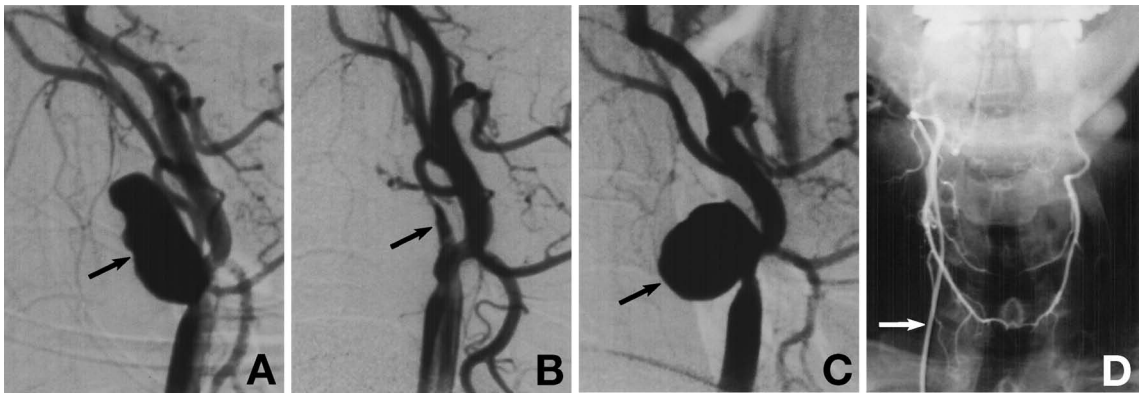

Fig. 3 Serial angiograms of the left common carotid artery (lateral view), (A) obtained 1 week after the manifestation of hemiparesis and motor aphasia showing occlusion of the internal carotid artery, and patent flow to the pseudoaneurysm and the external carotid artery (ECA) (*arrow*), (B) 2 months later showing remarkable shrinkage of the aneurysm (*arrow*), (C) at readmission showing regrowth of the aneurysm (*arrow*), and (D) preserved flow to the ECA through a shunt between the bilateral superior thyroid arteries, with the catheter (*arrow*) placed in the right superior thyroid artery.

development, and rupture, resulting in extracranial hemorrhage.[9,15,17,18]

Both CT and MR imaging of our patient demonstrated marked contrast enhancement of the walls of the ICA and CCA suggesting inflammatory reactions. The underlying mechanism probably involved spread of the inflammation to the ICA which induced a local response by various cytokines, resulting in endothelial cell dysfunction and swelling. Consequently, the ICA became permeable and was thus remarkably enhanced by contrast medium. MR imaging of a patient with a retropharyngeal abscess showed enhancement of the wall and narrowing of the lumen of the ICA, suggesting spasm and arteritis that reflected both infection and arterial wall inflammation.[7] In our case, the walls of the ICA and CCA were enhanced, but that of the ECA was not enhanced. Therefore, we concluded that flow in the ECA was preserved after ICA occlusion. We treated our patient preoperatively with prednisolone to avoid exacerbation of the inflammation as a consequence of intravascular embolization with detachable coils. Preoperative administration of adrenocorticosteroids effectively controls inflammatory reactions.[19]

The ICA occlusion in our patient probably resulted from the production of inflammatory cytokines in endothelial cells or leukocytes induced by the ICA response, which transformed the endothelial cell function from anti-thrombotic to thrombogenic. CT showed remarkable enhancement of the ICA wall, suggesting an inflammatory response. Decreased expression of thrombomodulin,[11] prostacyclin,[4] and nitric oxide[16] facilitated activation of endothelial cell-induced thrombogenesis in the ICA by inflammatory cytokines, such as tumor necrotizing factor-α and interleukin-1,[2] induced by infection in the deep neck space.

Surgical ligation of the affected artery is the definitive treatment,[9,10,17] by methods such as balloon catheter occlusion,[13] resection,[6,14] and reconstruction.[8,14] However, suture of the aneurysm is the most problematic complication in surgical ligation or repair, so that recently endovascular therapy has been used as a surgical adjunct and has improved patient outcome.[3,12,15,20] The object of endovascular therapy is to prevent hemorrhage or embolism, and reduce the mass effect on the surrounding structures. The most difficult problem with intravascular embolization is the placement of foreign materials into the focus of the inflammation. For this reason, prolonged antibiotic therapy may be indicated after embolization.[3]

Infection originating in the deep neck space may become life-threatening in patients with ICA involvement, so careful attention must be paid to marked enhancement of the ICA wall on CT and/or MR imaging.

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