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Spontaneous Dissection of the Cervical Internal Carotid Artery: Correlation of Arteriography, CT, and Pathology

George R. Petro¹
 Gordon A. Witwer¹
 Edwin D. Cacayorin¹
 Charles J. Hodge²
 Carl E. Bredenberg³
 Michael S. Jastremski⁴
 Stephen A. Kieffer¹

Spontaneous dissection of the internal carotid artery is being recognized as a more frequent cause of acute neurologic deficit, particularly in young persons. Saccular pseudoaneurysm formation may be an associated finding, especially in the presence of tortuosity (coiling) of the cervical internal carotid artery. Of eight patients with nine vessels demonstrating internal carotid artery dissection on arteriography, pseudoaneurysms were found in five arteries. Four of the five pseudoaneurysms occurred in tortuous (coiled) arterial segments. Thin-section contrast-enhanced dynamic incremental CT showed close agreement with the findings on selective arteriography and provided additional information on the presence and configuration of arterial wall thickening as well as the extent of the pseudoaneurysm. Our experience indicates that CT may play an important role in the diagnosis, management, and follow-up of this lesion.

Spontaneous hemorrhagic dissection of the internal carotid artery (ICA) is becoming more widely appreciated as a cause of transient ischemic attack (TIA), or stroke, particularly in relatively young patients [1-4]. Diagnosis is usually based on demonstration by arteriography of a long, tapered, eccentric narrowing of the cervical ICA beginning above the common carotid bifurcation and extending superiorly to the level of the base of the skull [2] or occasionally into the carotid canal [4]. Saccular pseudoaneurysm formation may occur as a result of dissection [4], and sequential arteriograms often demonstrate rapidly changing patterns as the process proceeds to resolution, pseudoaneurysm formation, or further stenosis and occlusion [1, 2].

CT of the neck has recently been suggested as an accurate and relatively noninvasive method for diagnosis and sequential assessment of occlusive changes affecting the common carotid bifurcation and the proximal ICA [5, 6]. We have studied eight patients with spontaneous ICA dissection using sequential arteriography; dynamic thin-section CT of the neck was obtained in five individuals in order to more completely assess both luminal and mural changes in this still incompletely understood lesion.

Materials and Methods

Eight patients with spontaneous cervical ICA dissection were diagnosed on arteriography, including four women and four men, aged 23 to 54 years. Clinical presentations included initial head and neck pain followed by TIAs in two patients, TIAs in two (one with a remote history of severe neck pain), and acute hemispheric stroke in three. Two patients also manifested an ipsilateral incomplete Horner's syndrome. In one patient who suffered a single focal seizure and was being evaluated for possible intracranial mass, contralateral dissection with pseudoaneurysm was an incidental finding on arteriography.

All eight patients underwent percutaneous transfemoral selective common carotid arteriography. Arteriography was performed promptly after clinical presentation in five patients, after 5 weeks of intermittent symptoms in one, and 2 years after the likely initial event in another. Follow-up arteriograms were obtained in five patients at 2 weeks to 14 months after the initial study.

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¹ Department of Radiology, SUNY Health Science Center of Syracuse, 750 E. Adams St., Syracuse, NY 13210. Address reprint requests to G. R. Petro.

² Department of Neurosurgery, SUNY Health Science Center of Syracuse, Syracuse, NY 13210.

³ Department of Surgery, SUNY Health Science Center of Syracuse, Syracuse, NY 13210.

⁴ Department of Critical Care and Emergency Medicine, SUNY Health Science Center of Syracuse, Syracuse, NY 13210.

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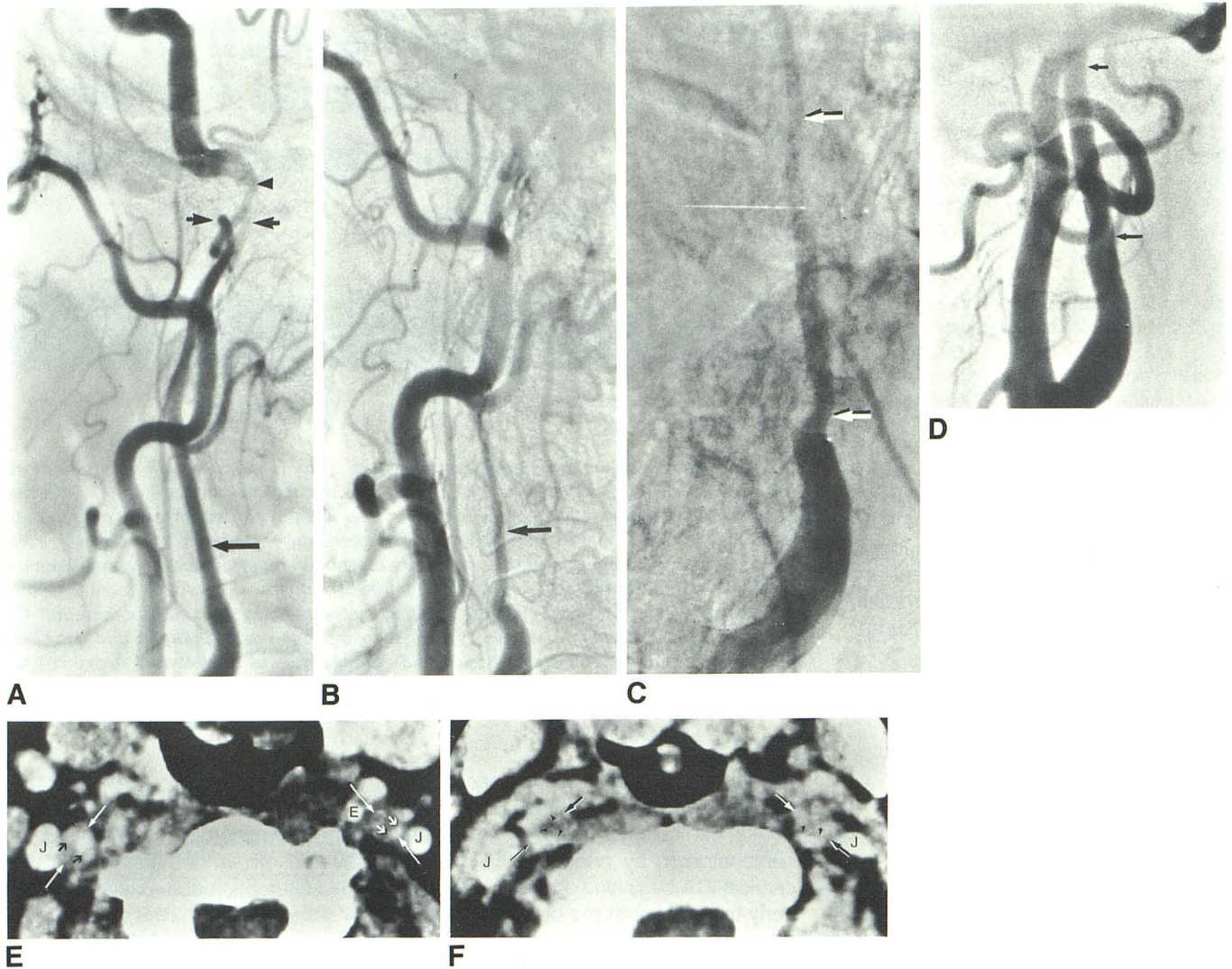


Fig. 1.—39-year-old man with left hemispheric ischemia and left incomplete Horner's syndrome after sneezing. **A**, Initial left common carotid arteriogram (lateral view) several hours after onset of symptoms demonstrates diffuse, smoothly tapering stenosis (*long arrow*) of cervical internal carotid artery with sparing of its most proximal segment. Maximal narrowing is noted in distal cervical segment (*short arrows*) with abrupt return to normal caliber within proximal carotid canal (*arrowhead*). **B** and **C**, Follow-up left common carotid arteriogram at 2 weeks. Lateral (**B**) and late arterial phase anteroposterior (**C**) views show more severe flow limiting narrowing of left cervical internal carotid artery (*arrows*). **D**, Another follow-up left common carotid arteriogram (anteroposterior projection) after 10 days of further anticoagulation demonstrates marked improvement in lumen diameter (*arrows*). **E**, On day of presentation,

axial CT with intravenous contrast at a level 2 cm above common carotid bifurcation displays eccentric luminal narrowing of internal carotid artery bilaterally, more pronounced on left side. *Long arrows* define external margins of both internal carotid arteries. Luminal compromise is due to mural thickening posterolaterally on right and anteromedially on left. (*Short arrows* indicate interface between opacified lumen and mural thickening; **E** = external carotid artery, **J** = internal jugular vein.) **F**, At level of C2 body, CT shows that intramural hematoma has spiraled medially on right side and anteriorly on left. Intramural hematoma has caused both luminal compromise (*arrowheads*) and enlargement of external dimension of vessels (*arrows*) as compared with lower level (**E**).

CT of the neck was obtained in five of the eight patients. In two patients, rapid-sequence, contiguous, 1.5-mm-thick axial images were obtained from C1 through C4 using a 100-ml rapid intravenous bolus injection with subsequent drip infusion of meglumine iohalate 60% (Conray 60, Mallinckrodt). Three patients were scanned using overlapping 5-mm-thick sections (3-mm table incrementation) during a prolonged intravenous bolus injection of 150 ml of Conray 60. Reformating of axial scan data was carried out in various paraxial and oblique planes. The thickness of the arterial wall (not discernible on axial CT images of the neck in normal individuals) and the size of the opacified lumen were evaluated on the axial CT images.

Four patients subsequently underwent segmental resection of a

saccular pseudoaneurysm of the high cervical ICA; gross and histologic analyses of the resected specimens were correlated with CT and arteriographic findings. No patient with internal carotid stenosis alone was treated surgically.

Results

Arteriography

Initial selective arteriograms demonstrated findings consistent with spontaneous dissection in one ICA in seven patients and in both ICAs in one patient. In seven of the nine

involved vessels, the arterial lumen was narrowed, and in two of these the narrowing was associated with an adjacent pseudoaneurysm. In two vessels, no luminal narrowing was evident but saccular pseudoaneurysms were demonstrated in the high cervical region.

Six of the seven ICAs with luminal narrowing demonstrated long stenotic segments extending to or beyond the skull base. The luminal narrowing in four vessels was smoothly tapered, becoming progressively more stenotic distally; the narrowing was relatively uniform and symmetric in two arteries and was

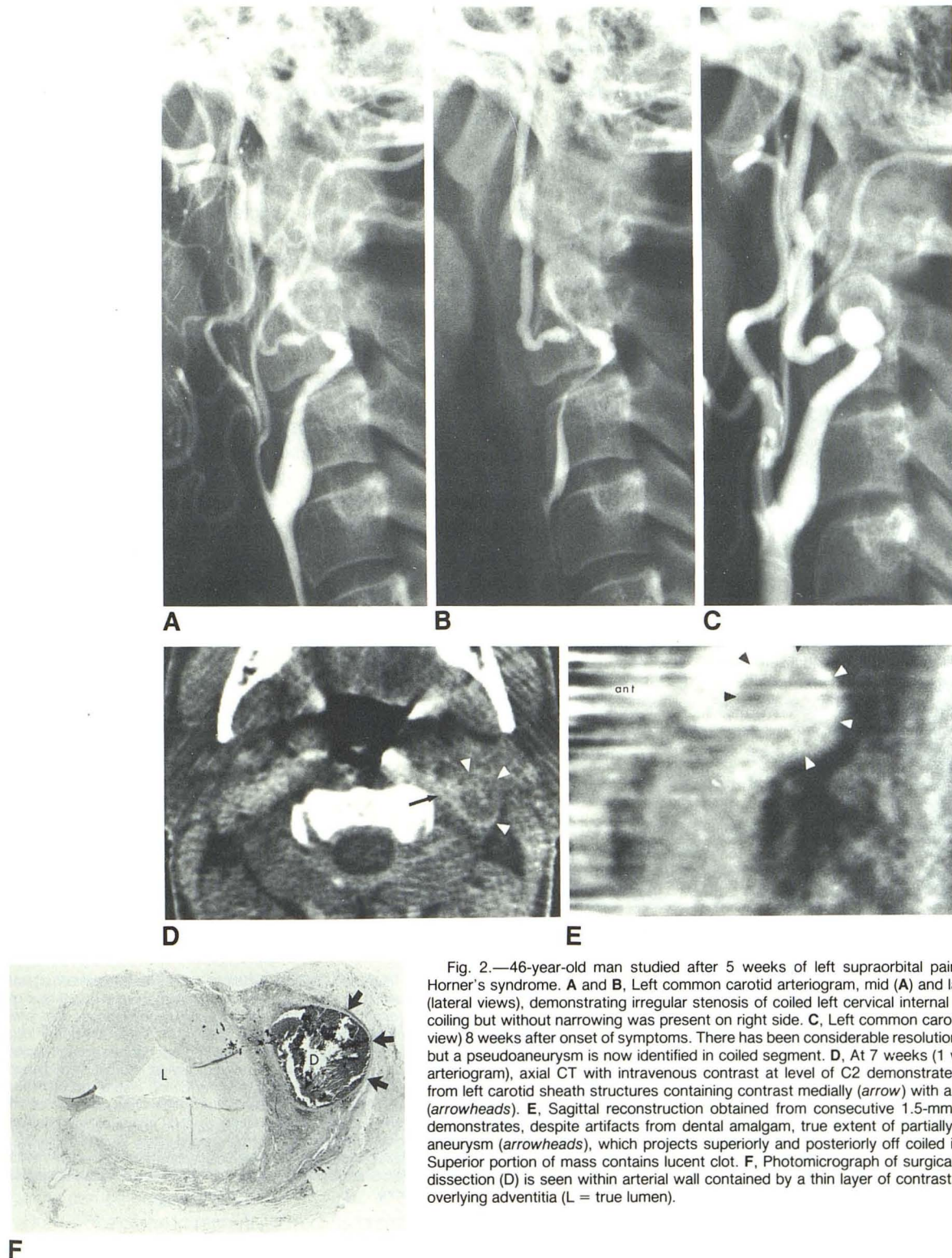


Fig. 2.—46-year-old man studied after 5 weeks of left supraorbital pain and left incomplete Horner's syndrome. **A** and **B**, Left common carotid arteriogram, mid (**A**) and late (**B**) arterial phases (lateral views), demonstrating irregular stenosis of coiled left cervical internal carotid artery. Similar coiling but without narrowing was present on right side. **C**, Left common carotid arteriogram (lateral view) 8 weeks after onset of symptoms. There has been considerable resolution of luminal narrowing, but a pseudoaneurysm is now identified in coiled segment. **D**, At 7 weeks (1 week before follow-up arteriogram), axial CT with intravenous contrast at level of C2 demonstrates a mass inseparable from left carotid sheath structures containing contrast medially (*arrow*) with a lucent region laterally (*arrowheads*). **E**, Sagittal reconstruction obtained from consecutive 1.5-mm-thick axial slice data demonstrates, despite artifacts from dental amalgam, true extent of partially thrombosed pseudoaneurysm (*arrowheads*), which projects superiorly and posteriorly off coiled internal carotid artery. Superior portion of mass contains lucent clot. **F**, Photomicrograph of surgical specimen. Portion of dissection (**D**) is seen within arterial wall contained by a thin layer of contrast medium (*arrows*) and overlying adventitia (**L** = true lumen).

eccentric in two vessels (Fig. 1A). In two arteries, the stenosis was not progressively tapering but rather varied in degree with focal areas of irregularity, which, in one patient, were located within a tortuous (coiled) segment of the cervical ICA (Figs. 2A and 2B). Luminal stenosis extended in continuity from the cervical through the intracranial and cavernous segments of the ICA in one patient in whom the supraclinoid ICA was completely occluded, presumably by an embolus. Stenosis of the seventh ICA began 3 cm above the common carotid bifurcation and extended to the midportion of the cervical ICA, which was severely coiled.

Follow-up angiography was performed in five patients at 2, 3, 3 1/2, 20, and 60 weeks after initial evaluation and institution of anticoagulant therapy. In all three patients reevaluated within 4 weeks of clinical presentation, residual narrowing was still present but was much reduced in severity, with the configuration being similar to that seen on initial arteriography. However, the single patient with bilateral involvement initially showed a unilateral increase in stenosis on the symptomatic side at 2 weeks with subsequent improvement after 10 days of further anticoagulation (Figs. 1B–1D). The 20-week follow-up arteriogram in a patient with severe tapered narrowing and pseudoaneurysm formation revealed minimal residual stenosis. Although follow-up arteriography demonstrated improvement or complete resolution of luminal narrowing in all five patients so studied, concomitant enlargement of a pseudoaneurysm was noted in one individual, evolution of an apparent fusiform dilatation into a large saccular pseudoaneurysm was demonstrated in another, and a new pseudoaneurysm was identified in a third patient (Fig. 2C).

Saccular pseudoaneurysms were encountered in five of the nine involved arteries, three on the initial arteriogram and two on follow-up studies. Four of the five pseudoaneurysms arose on coiled arterial segments (Fig. 2).

Arteriography also revealed occlusion of the supraclinoid ICA in one patient and of branches of the middle or anterior cerebral arteries in four, all likely embolic in origin.

Computed Tomography

Dynamic incremental CT examinations were obtained in five patients (six vessels). The diameters and configurations of the opacified arterial lumina on CT were in close agreement with the findings on arteriography in all six. Thickness and configuration of the arterial wall, findings not appreciable on arteriography, were clearly depicted on CT (Figs. 1E and 1F). Mural thickening was demonstrated on CT in all five vessels exhibiting narrowing on arteriography. The mural thickening was eccentric in distribution and was noted to spiral around the vessel from level to level with attenuation values comparable to adjacent muscles. In a normal ICA, the arterial wall is not identifiable on CT.

Mural thickening causing enlargement of total vessel dimension was demonstrated on CT in three of six involved arteries with total external diameter (lumen and walls) increased from 1.5 to 3 times that of adjacent ipsilateral or similar contralateral ICA segments. The greatest degree of external widening was recorded in association with a pseudoaneurysm that was not evident on the initial arteriogram 2

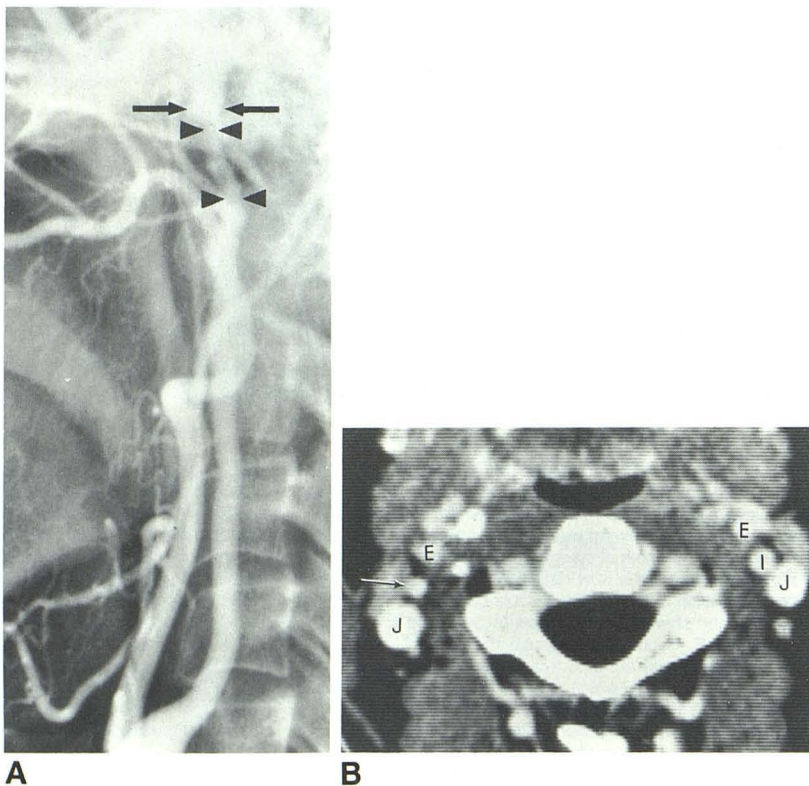


Fig. 3.—Overestimation of longitudinal extent of internal carotid artery dissection by angiography in a 39-year-old woman with right-sided headache, neck soreness, and intermittent transient ischemic attacks. A, Three days after initial symptoms, right common carotid arteriogram (lateral projection) shows diffusely narrowed cervical internal carotid artery with irregular, more severe narrowing distally (arrowheads) and abrupt reconstitution of lumen at its entrance into carotid canal (arrows). B, Proximally, axial CT image reveals relatively small lumen of right internal carotid artery (arrow) but without mural thickening to indicate dissection at this level (I = internal carotid artery, E = external carotid artery, J = internal jugular vein).

weeks earlier; axial CT images revealed an ill-defined, partially opacified, 2-cm-thick carotid sheath mass (Fig. 2D), but paraxial reformatted images better depicted the partially thrombosed pseudoaneurysm on a coiled ICA (Fig. 2E). Follow-up arteriography 1 week after the CT study confirmed the evolving pseudoaneurysm but underestimated its true extent (Fig. 2C).

In two dissected vessels, CT demonstrated mural thickening resulting in luminal compromise but without significant overall external enlargement. In one of these, CT not only confirmed the diffusely small lumen but also revealed mural thickening of only the distal half, indicating only distal dissection (Fig. 3). In another patient, CT confirmed the angiographic finding of a 3-mm saccular pseudoaneurysm on a tightly coiled ICA in which there was no luminal narrowing.

Clinical Course

Of the seven symptomatic patients in this series, four underwent successful surgical resection of their saccular pseudoaneurysms with reanastomosis, and three were treated with intravenous heparin. All seven initially symptomatic patients were either clinically improved or normal after surgery or heparinization, and none experienced recurrent neurologic deficits after therapy. One of the eight patients was asymptomatic from dissection, and no treatment was instituted.

Pathology

Gross and histologic examination of the four resected saccular pseudoaneurysms demonstrated intimal tears at the level of the proximal aspect of the pseudoaneurysm in all four. In each case, dissection occurred into the outer media (Fig. 2F) and, in two cases, extended into the plane between the media and the adventitia of the arterial wall. No identifiable predisposing atherosclerotic or other underlying pathologic changes were identified within the resected specimens.

Discussion

The cause of spontaneous hemorrhagic dissection of the ICA remains unknown. Affected individuals most typically present in the second through fifth decades of life with acute ischemic neurologic deficits [1-4]. If this is concurrent with or preceded by head or neck pain or occurs in association with an incomplete Horner's syndrome (as was the case in two of our patients) or with a subjective bruit, cervical internal carotid dissection must be strongly suspected [7].

Cervical ICA dissections have been reported after vigorous nose blowing [8], and a possible association with repeated coughing has been suggested [2]. One patient in our series experienced the onset of symptoms after an episode of repeated forceful sneezing, while another patient gave a history of recent chiropractic manipulation. In such instances, a causal versus a coincident association with apparently minor trauma can be suspected but not proven.

Occlusions of the intracranial ICA or its branches consistent

with an embolic origin were identified on arteriography in five of our seven symptomatic patients. This suggests that the cause of the ischemic neurologic symptoms is often embolic in nature rather than purely hemodynamic. In their report of 42 patients with spontaneous ICA dissection, Houser et al. [4] noted a 15% incidence of intracranial arterial occlusions likely secondary to emboli on angiography.

Most stenoses (up to 80% of reported case series) due to cervical ICA dissection appear on follow-up angiography to partially or completely resolve with time [1, 4, 9, 10]. Follow-up angiography in six stenotic cervical ICA dissections in this series showed significant improvement or complete resolution in all six involved vessels after 2 to 20 weeks of anticoagulant therapy (Figs. 1 and 2). In one of these cases, however, the stenosis progressed to near occlusion before delayed resolution with further anticoagulation (Figs. 1A-1D). Progression to complete occlusion was reported by Ehrenfeld and Wylie [1] in one of six stenoses and by Houser et al. [4] in three of 15 stenoses at repeat angiography.

Saccular pseudoaneurysms were identified in five of nine internal carotid dissections (56%) in this series. The formation of a pseudoaneurysm as a sequela of arterial dissection and the subsequent course of this lesion is a dynamic and variable phenomenon. Fisher et al. [2] and Houser et al. [4] followed a total of 13 pseudoaneurysms with sequential arteriograms showing four to be unchanged, six to have decreased in size, three to have essentially resolved, but none to have enlarged. In two of our patients and in a case reported by Bostrom and Liliequist [11], enlargement of a pseudoaneurysm was documented on serial studies, and late development of a saccular pseudoaneurysm not evident on initial examination occurred in another patient in this series (Fig. 2). Delayed appearance of a pseudoaneurysm has been reported in approximately 5% of internal carotid dissections in larger series [2, 4, 12].

Marked tortuosity (coiling) of the cervical ICAs has been reported in 3% of a large series of carotid arteriograms [13]. Four of the five pseudoaneurysms in this series occurred on such coils, while none of the four dissected vessels with narrowing alone were coiled. While an association of tortuosity of the cervical portion of the ICA with dissection has been previously noted [1, 3], the association of pseudoaneurysm and ICA coiling apparent in this small series has not been previously stressed. It is our impression that demonstration of an aneurysm-like outpouching of the arterial lumen on a coiled midcervical ICA makes dissection a preferred diagnosis. When dissection occurs in an area of coiling, the likelihood of subsequent pseudoaneurysm formation may be significantly increased, and follow-up sequential imaging (arteriography or CT) would assume greater importance.

Thin-section dynamic incremental CT with contrast enhancement demonstrated in all six vessels so studied that CT accurately assessed lumen size. Mural thickening was demonstrated in the five vessels showing narrowing on arteriography. In one case, CT established the initial diagnosis of a partially thrombosed pseudoaneurysm, characterizing its true extent, which was underestimated on subsequent arteriography (Fig. 2).

Thin-section CT with intravenous contrast infusion has

demonstrated the occurrence and extent of intraplaque hemorrhage in patients with atherosclerotic involvement at the common carotid bifurcation [6]. Our experience indicates that CT may also play an important role in the diagnosis and management of hemorrhagic dissection of the ICA. The longitudinal extent of the dissection and the full extent of an associated pseudoaneurysm can be accurately discerned, a prerequisite in those cases requiring surgical intervention. CT may prove more accurate than arteriography in differentiating luminal narrowing secondary to dissection from luminal collapse due to spasm or marked diminution in flow (Fig. 3). In patients in whom the diagnosis of cervical ICA dissection remains in doubt after arteriography, CT can be supportive if enlargement of the external dimension of the stenotic vessel is demonstrated. CT may also be useful as an alternative to arteriography in the follow-up of an arterial dissection.

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