Spontaneous Dissecting Aneurysm of the Internal Carotid Artery

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Abstract:
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A case of spontaneous dissection of the internal carotid artery is presented and the ten other reported cases are reviewed. It is most common in males aged 35 to 41 years. Of eight patients with neurological symptoms, two had transient hemiplegia and five of six first seen with a severe neurological deficit had prodromal symptoms. Angiography revealed a typical “string sign.” A method of surgical treatment is illustrated. The importance of emergency angiography and surgery in patients with acute onset of progressive symptoms of cerebral vascular disease is emphasized by this case.

The surgical specimen was studied pathologically by serial section. At its origin the dissection measured 3 mm in width and 0.3 mm in length. Proximally the dissection was in the media but distally it lay at the medial-adventitial interface. There was no evidence of cystic medial necrosis but the muscle and elastic tissue of the media had a disorganized arrangement.

Dissecting aneurysm of the internal carotid artery and its major intracranial branches is a relatively rare occurrence. The lesion may develop spontaneously or may be related to external trauma to the head and neck or to carotid puncture for angiography or may extend from a more proximal dissection.

The present case, the eleventh reported patient with a spontaneous dissecting aneurysm of the internal carotid artery, is unusual in that the patient was successfully treated surgically and a detailed microscopic examination of the origin of the dissection was made.

Case Report
A right-handed bus driver, aged 41, was admitted to the Massachusetts General Hospital because of right hemiparesis and inability to speak of about six hours’ duration.

Two days before admission the patient, while in the bathroom, suddenly became weak and noted a film obscuring the vision in his left eye. He called to his wife who found him kneeling on the floor. He said he had had two similar episodes before. After the symptoms subsided he visited his physician who found the examination normal except for a slightly unsteady gait. He was then well until the next morning when after breakfast he noted inability to let go of the breakfast tray with his right hand or move the right leg. His speech was normal. Thirty minutes later he made his way to the bathroom with obvious dragging of his right leg. His speech remained clear. He went to sleep late that evening and at two o’clock the next morning, the day of admission, he fell trying to get out of bed, mumbled something, and vomited. At 6 A.M. he again fell getting out of bed and could not speak, and his right side was paralyzed.

An episode of neck pain had been treated successfully with traction five months before admission. He was thrown from a boat three months before admission without unconsciousness, neck symptoms, or apparent sequelae. There was no history of syphilis.

EXAMINATION
He was alert, but did not speak, and had an obvious right hemiparesis. The pulse was 84, blood pressure was 120/80, and temperature was

Stroke, Vol. 3, July-August 1972
SPONTANEOUS DISSECTING ANEURYSM

There was no evidence of pulmonary, cardiac, or abdominal disease. The peripheral pulses were present. The common carotid pulsations were 2+, but the left carotid pulse was diminished high in the neck. There was no bruit. The preauricular pulses were intact. The patient nodded yes to all questions whether appropriate or not, made no verbal responses, but obeyed simple commands with his left side. He reacted to threat, and followed the examiner in his left visual field but not his right. The pupils were equal and reacted to light and on accommodation. The extraocular movements were full to the right only, with doll’s head maneuver. Reaction to pain on the right was decreased, the right lower face was weak, and shrug was absent on the right. There was no spontaneous movement of the right extremities. On painful stimulus the right upper extremity remained immobile, and there was only slight withdrawal movement in the right lower extremity. The deep tendon reflexes were 3+ on the right and 2+ on the left, and the plantar reflexes were extensor. EKG and chest x-ray were normal.

ANGIOGRAPHY

An emergency left carotid angiogram disclosed an irregular narrowing of the internal carotid artery in the neck extending from approximately 1 cm distal to the bifurcation to the base of the skull (figs. 1 and 2).

OPERATION

The left internal carotid artery was immediately explored under general anesthesia. During the operation EKG and intra-arterial blood pressure were monitored continuously. A neosynephrine infusion was used when necessary to maintain the systolic blood pressure between 120 and 150 mm Hg. During the time of occlusion the pressure was elevated to a minimum level of 170 mm Hg.

At surgery the carotid vessels presented a striking picture. The internal carotid was dilated at the bifurcation, but 1.5 cm distally there was a constriction. No evidence of atheroma could be seen or palpated. Just beyond the constriction, the internal carotid artery was markedly dilated, bluish in color, and looped anteriorly. The bluish discoloration and dilatation extended to the base

FIGURES 1 and 2

Left carotid angiogram. The origin of the internal carotid artery is dilated. An irregular string of contrast media is seen in the residual lumen which is compressed by thrombus. At the base of the skull, the abnormality terminates abruptly.

Stroke, Vol. 3, July-August 1972
of the skull. By dividing the posterior belly of the digastric muscle, an excellent exposure of the internal carotid artery was achieved. An incision just distal to the constriction disclosed a thrombus. An 18-mm segment was resected, including the area of narrowing. Distally there were two distinct lumens—one with apparently normal intima and the other filled with firm thrombus which could not be removed (fig. 3). No back-bleeding was encountered at this time. A No. 4 Fogarty catheter was then inserted along the normal lumen beyond the area of dissection. The bag was inflated and the catheter withdrawn, removing the thrombus and dissected intima. Following this, brisk back-bleeding occurred. A 5-mm Dacron graft was then sewn in place (fig. 4). An intraoperative angiogram showed excellent circulation in both the cervical and intracranial internal carotid artery.

POSTOPERATIVE COURSE
Immediately postoperatively the right hemiparesis and aphasia persisted. The patient was alert, nodded yes both appropriately and inappropriately to questions, and did not speak. He had a right visual field defect to threat, weakness of the right face, weakness of the right arm more than the leg, and bilateral extensor plantar responses. He made steady progress and at the time of discharge three weeks after admission he could shave, dress himself, and walk without a cane. Elements of receptive and expressive dysphasia persisted. Subsequent examinations showed continued improvement and, when last seen, 20 months after surgery, the patient could use the right upper extremity in all daily activities. Writing was slow but legible. Gait was very satisfactory with only minimal limp. Simple calculations were done without difficulty. He could now do some reading. There was only minimal difficulty in expressing himself. The right visual field defect had improved.

PATHOLOGICAL FINDINGS
The surgical specimen, which included 10 mm of the internal carotid artery proximal to the dissection and about 8 mm of the dissected segment, was embedded in paraffin, sectioned serially and stained with hematoxylin and eosin, phosphotungstic acid hematoxylin, Verhoeff's elastic and Masson's trichrome stains (figs. 5 and 6). The dissection extended from the lumen

![Figure 3](https://example.com/figure3.png)

**FIGURE 3**
Location and extent of area of dissection.

![Figure 4](https://example.com/figure4.png)

**FIGURE 4**
SPONTANEOUS DISSECTING ANEURYSM

FIGURE 5
Site of break from lumen through inner lamina into main dissection whose external wall has been surgically incised. a. Lumen. b. Flaps of inner wall. c. Flaps of outer wall. (Verhoeff’s elastica stain × 29.)

FIGURE 6
Walls of dissection compartment 0.3 mm distal to the break out of the lumen. a. Lumen. b. Inner wall of dissection. c. Surgically incised outer wall of dissection at outer media, above, and at junction of inner two-fifths and outer three-fifths of the media, below. Note well-formed internal elastic lamina and very slight thickening of the intima. The dissection at this level involved about one-third of the circumference of the artery. (Verhoeff’s elastica stain × 29.)
through the arterial wall to the region between the media and the adventitia. While many of the anatomical details could be readily interpreted, there existed at the site of the primary penetration of the wall a structural abnormality of the inner media that was difficult to reconstruct.

As the artery was traced distally in the serial sections just proximal to the origin of the dissection the inner one-quarter of the media underwent reduplication, splitting off an extra well-formed inner lamina for about 4 mm of the circumference of the wall. This lamina, which consisted of intima, internal elastic lamina, and a thin layer of media, ended in a distal free border after extending only 0.5 mm along the artery, thus forming a tiny shallow pocket that was open to the main lumen at its distal edge. The point at which the dissection originated lay at one horizontal extremity of the free edge of the pocket, which appeared to have been torn away, creating a breach in the inner media of the main wall through which blood penetrated to reach the junction of the inner two-thirds of the media and the outer one-third and at a slightly more distal level to reach the medial-adventitial interface where the major dissection occurred. The gap in the inner media at the origin of the dissection consisted of a horizontal slit measuring only 0.3 mm in vertical extent and approximately 3 mm horizontally. It is remarkable that such a small hole in the intima of the internal carotid artery should permit so extensive and injurious a dissection.

The wall of the artery showed no definite recognizable, underlying intrinsic disease, but the muscle and elastic tissues of the media had an irregular disorganized arrangement rather than the usual laminar pattern. This at first appeared to be frankly abnormal, suggesting fibromuscular hyperplasia or dysplasia, but examination of 13 internal carotid arteries from routine necropsies disclosed two with an appearance similar to that of our case. Therefore, a study of further control material will be necessary before an adequate interpretation can be made, since this region of the internal carotid artery is difficult to reconstruct.

Angiography

Figures 1 and 2 illustrate the angiogram in our case. The dissection itself was occluded by a thrombus and, therefore, did not fill with contrast medium. The most striking finding was a long, irregular filling defect starting about 2 cm above the bifurcation and extending throughout the extracranial course of the internal carotid artery. The resultant long, narrow column of dye appears to be characteristic of dissection and might be referred to as “the string sign.” We have not seen a similar picture with occlusive disease due to atheroma.

Of the group of 11 cases in the literature, nine had angiography which usually showed the abnormality originating between 1.5 and 3.0 cm from the carotid bifurcation. Three showed a long, irregular filling defect in the internal carotid with no filling of the dissection (present case). One showed such a defect and at follow-up angiography two months later, the artery was occluded, two had occlusion of the internal carotid artery, one had localized 2-cm narrowing of the internal carotid artery, one showed a double lumen beginning 4 cm above the origin of the internal carotid, and one showed a localized outpouching from the wall of the internal carotid artery which had enlarged at repeat angiography one month later. In three reported cases and in our own, the dissection extended to the base of the skull.
SPONTANEOUS DISSECTING ANEURYSM

CLINICAL COURSE AND TREATMENT

Of the nine clinical cases, five died from massive cerebral infarction without treatment of the dissection. One of these patients with a major neurological deficit was treated with anticoagulants, and the deficit gradually progressed.

The patient presenting with subjective noise in the ear was treated surgically. Under hypothermia, osteotomy of the mandible was undertaken, and a 1.5-cm segment of the aneurysm was resected. The double lumen was obliterated at each end of the dissection, and an anastomosis was accomplished with an autogenous external carotid artery graft. Some postoperative neurological deficits were noted.

One patient with transient ischemic attacks was not treated, and follow-up angiography two months later showed occlusion of the internal carotid artery. The other patient with transient attacks of hemiparesis had resection and vein graft replacement of the distal two-thirds of the internal carotid artery. Exposure was accomplished by means of an osteotomy at the angle of the mandible.

Ours is the only case in which an internal carotid dissecting aneurysm in a patient with an acute neurological deficit has been surgically treated. The surgical technique illustrated in figure 4 consisted of resection of the origin of the dissection, re-establishment of the lumen of the internal carotid artery by removal of the dissected intima and thrombus in the region of the dissection using a Fogarty catheter, and replacement of the resection with a graft. Excellent exposure was achieved without dividing the mandible.

Our patient, who presented with a paralyzed right face and arm, weak right leg, and severe aphasia, had an excellent return of function following operation. Some reports indicate that in a patient with an acute major neurological deficit due to cerebral vascular occlusive disease, surgical intervention is contraindicated and, indeed, even angiography. It is our current opinion that patients with a history of progressive onset of disability should be studied angiographically as an emergency, and surgical treatment considered even when the deficit is severe unless signs of increased intracranial pressure are present.

PATHOLOGY

In the seven cases from the literature in which details were provided, the dissection lay in the outer layers of the media in four of seven instances and between the media and the adventitia in three. The dissections involved the whole circumference of the artery in one case, 80% in one, and about 50% in four cases. The dissection originated at varying distances from the origin of the internal carotid artery—1.0, 1.5, 2.0, 2.5, 3.0, and 4.0 cm. One at necropsy measured about 3 cm below the base of the skull. The site of the penetration was sometimes on the anterior wall, sometimes on the posterior, and a small intimal tear was identified in four cases. The dissection usually extended not more than a few millimeters proximal to the tear.

In seven of the ten previously reported cases, the involved artery on microscopic study showed cystic medial necrosis, usually with similar changes in the opposite carotid and the aorta. In one, fibromuscular dysplasia was present. One studied angiographically only showed coiling, tortuosity, and diffuse dilatation of the opposite internal carotid artery similar to that frequently seen in fibromuscular dysplasia. In one, arteriosclerosis was the basis. In our case, the elastica and muscle of the media had a disorganized appearance, resembling the descriptions given in many of the other cases, but two of our 13 control arteries showed a similar picture. In the cases in the literature, the process of cystic medial necrosis was not advanced. It is our viewpoint that a reliable interpretation of the alterations found in the elastic and muscular elements of the media of the internal carotid artery in the present case probably requires the examination of further control material.

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