Atheromatous pseudo-occlusion of the internal carotid artery

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In the past year, three patients were referred for microvascular bypass surgery for relief of symptoms secondary to an apparently occluded internal carotid artery (ICA). Careful review of the late films of their initial arteriographic series or repeat arteriography with a specialized technique revealed a thin trickle of contrast medium flowing antegrade through a region of extreme stenosis. This thin line of contrast material ascended slowly to meet the column of contrast medium in the cavernous carotid segment that was filling by collateral circulation. Surgical exploration of the neck in these patients revealed a patent but collapsed ICA distal to a localized atheromatous plaque. These patients have been asymptomatic following carotid endarterectomy. This distinctive angiographic appearance may be described as "atheromatous pseudo-occlusion." Once recognized, carotid endarterectomy is the logical treatment of choice.

KEY WORDS • carotid artery • internal carotid artery • thrombosis • carotid endarterectomy • transient ischemic attack • cerebral angiography • pseudo-occlusion • blood-flow velocity

Since the advent of microvascular bypass surgery for cerebral ischemia, neurosurgeons are consulted with increasing frequency for the management of symptomatic internal carotid artery (ICA) occlusion. After undergoing careful review, some of these patients are relieved of their symptoms by operations on the carotid arteries in the neck. Surgical procedures included endarterectomy of the contralateral carotid artery, ligation of the ICA stump,1,2 and endarterectomy of the external carotid artery, either as the only procedure or in preparation for subsequent bypass surgery.4,6 Moreover, there is a group of patients with total occlusion of the cervical ICA who are amenable to re-establishment of flow by endarterectomy, either because the occlusion is recent or because adequate collateral vessels have kept the petrous and cavernous portions of the carotid artery patent.18,19,23

In the last year, we have encountered three patients in whom a very high-grade stenosis of the origin of an ICA was initially misdiagnosed as total occlusion. These patients were considered for microvascular bypass surgery because of persistent symptoms. Either on careful inspection of their initial angiograms or on repeat arteriography by our group, they were found to have a thin trickle of contrast material coursing through the cavernous portion in an antegrade fashion, affirming the patency of the artery. All three patients obtained relief of symptoms following carotid endarterectomy.

The term "atheromatous pseudo-occlusion" may be used to distinguish this angiographically visualized entity from the somewhat similar appearance seen in cases of markedly raised intracranial pressure, intracranial ICA occlusion, dissection of the ICA, and severe hypoplasia of the ICA. This entity may not only escape angiographic detection, but, once diagnosed, its therapeutic implications are not necessarily obvious.

Case Reports

Case 1

This 71-year-old man collapsed while attempting to rise from a chair. When examined in the emergency room of a regional hospital shortly thereafter, he was unresponsive to verbal stimulation and had a dense left hemiparesis. He subsequently recovered consciousness, and his weakness began to resolve. At this point, he was transferred to our hospital.

Examination. He was alert and oriented, with a blood pressure of 174/74 mm Hg. A right carotid bruit
was present. He had a left hemiparesis, worse in the face and arm. There were no reflex abnormalities or sensory deficits. Computerized tomography (CT) performed on admission revealed a nonenhancing low-density lesion in the right frontoparietal region, consistent with cerebral infarction. Ophthalmodynamometry revealed a markedly reduced pressure on the right side. Over the next 24 hours, the neurological deficit improved significantly, and he was left with minimal hemiparesis.

Transfemoral common carotid angiography, performed 6 days after onset of the deficit, was initially thought to show occlusion of the right ICA in the neck (Fig. 1 left). There was stenosis of the right external carotid artery and filling of the right anterior cerebral artery through vessels collateral to the ophthalmic artery. The left common carotid injection showed no opacification of the right anterior communicating artery. The patient was considered for bypass surgery. However, subsequent review of the later phase angiographic films revealed a narrow stream of contrast material coursing very slowly antegrade through the ICA (Fig. 1 center and right).

Operation. Since the ICA appeared to be patent and collateral flow to the right hemisphere was very minimal, exploration of the right carotid bifurcation was elected. There were no pulsations in the ICA, but upon gentle palpation it was soft and pliable. After clamping, an arteriotomy was made in the common and internal carotid arteries. An extremely stenotic ragged plaque extended 1 cm superiorly along the ICA, producing nearly total occlusion. No visible thrombus was present in the lumen. The endarterectomy was then completed without difficulty.

Postoperative Course. The patient showed no increase of his neurological deficit. A CT scan performed 1 week postoperatively showed the size of the

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FIG. 1. Right common carotid angiography. Left: Oblique view of the neck, early arterial phase, showing contrast material in the proximal 2 cm of the internal carotid artery (arrow), suggesting occlusion of that vessel. Center and Right: Lateral films of the upper neck and skull base taken 5 seconds (center) and 9.5 seconds (right) after the start of the injection, demonstrating a narrow stream of contrast material progressing cephalad through the internal carotid artery (solid arrows). The supraclinoid segment (open arrow) is reconstituted via the ophthalmic artery (arrowhead, center).
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FIG. 2. Right common carotid angiography, lateral view of the neck. Left: Film at 1.5 seconds following injection. The contrast material is seen only in the proximal 3 cm of the internal carotid artery (arrow), simulating occlusion. Center and Right: Films at 3 seconds (center) and 4 seconds (right) show progression of the contrast medium through the internal carotid artery (arrows), which is not completely identifiable at the base of skull.

Infarct to be unchanged; there was no evidence of hemorrhage into the infarct. The patient has been followed for 10 months and has had no new symptoms. His left-sided deficit has resolved completely. Ophthalmodynamometry in the immediate postoperative period and subsequently showed equal pressures in both retinal arteries.

Case 2

This 69-year-old physician had experienced several right hemispheric transient ischemic attacks. In November, 1978, he had a longer lasting episode, which resulted in slight weakness of the left upper extremity and nominal aphasia. Arteriography was interpreted as showing complete occlusion of the right ICA. Oral anticoagulation therapy was started at this point. Two weeks before admission to our hospital, he had another stroke that resulted in further left-sided deficit. A CT scan demonstrated a small right parietal infarct. The patient was referred for microvascular bypass surgery.

Examination. The patient was admitted to Presbyterian-University Hospital, and cerebral angiography was repeated. On right common carotid injection, the ICA appeared occluded 3 cm distal to its origin on the initial films (Fig. 2 left). There was considerable external carotid collateral circulation to the distal ICA, with opacification of the supraclinoid and cavernous portions of the ICA on the early films. The petrous and cervical portions of the artery were not seen on the films obtained within the first 2 seconds after injection. Delayed serial films of the neck (Fig. 2 center and right) and head (Fig. 3), however, revealed a thin trickle of contrast material progressing antegrade through the ICA. The petrous carotid artery also filled through external carotid collaterals. The circulation of the right hemisphere was mainly derived through collateral vessels from the vertebro-basilar and external carotid arterial systems.

Operation. We elected to undertake an endarterectomy. The carotid bifurcation was very high, requiring exposure of the ICA beyond the level of the first cervical vertebra. Electroencephalographic (EEG) monitoring was used throughout the procedure. We were prepared to use an external carotid artery shunt if EEG slowing appeared during the procedure, but this was unnecessary. On gentle palpation, a hard plaque was identified at the bifurcation, extending into the proximal ICA. The remainder of the palpable ICA was soft and pliable, suggesting patency. After isolating the internal, external, and common carotid
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arteries with tapes, a DeBakey clamp was placed on the ICA just distal to the plaque. A Heifetz aneurysm clip was then placed on the ICA as far distally as possible. An incision was made into the lumen of the artery between the two clamps. The Heifetz clip was then removed to reveal moderate backflow from the distal portion. The clip was reapplied, and the DeBakey clamp was then removed; there was a slight trickle of blood coursing through the ICA, indicating that the vessel was still patent proximally. Since the backflow from the distal segment was not satisfactory, a No. 5 Fogarty catheter was very gently passed cephalad for about 6 cm until resistance was first felt. The catheter was then inflated and drawn backward to extract a small clot. When this procedure was repeated, no further clot was obtained. This maneuver improved the backflow considerably. The Heifetz clip was reapplied, the common and external carotid arteries were clamped, and an endarterectomy was then performed in the usual fashion.

Postoperative Course. The patient made a good recovery postoperatively and has subsequently been free of neurological symptoms over a follow-up period of 7 months. Oculoplethysmographic and Doppler ophthalmic studies performed 2 months postoperatively strongly suggested patency of the ICA. Postoperative ophthalmodynamometry has also revealed equal retinal artery pressures.

Case 3

This 54-year-old man presented with recurrent episodes of right-sided amaurosis fugax of 5 months' duration. Cerebral arteriography performed at another hospital showed no flow through the right ICA. A significantly stenotic ulcerated atheromatous plaque was present in the contralateral common carotid bifurcation. In view of the precarious circulation, a left carotid endarterectomy was performed first without complications. However, the patient's pre-
senting symptoms persisted, and therefore he was considered for bypass surgery.

Examination. Angiography was repeated at Presbyterian-University Hospital to reassess his cerebral circulation before surgery. The left ICA, which had previously been operated on, was widely patent. Late subtraction films revealed an irregular, filiform stream of contrast material passing through an extremely stenotic origin of the right ICA (Fig. 4). On the basis of our previous experience with Cases 1 and 2, we suggested that carotid endarterectomy be performed rather than the contemplated microvascular bypass.

Operation. A right common and internal carotid endarterectomy was performed by the referring surgeon (Dr. Webster). A severely stenotic atheromatous plaque was excised from the origin of the ICA. The patient has been asymptomatic during a follow-up period of 4 months. Postoperative ophthalmodynamometry, oculoplethysmography, and Doppler ophthalmic studies indicate sustained patency of this vessel.

Discussion

Previously Documented Cases

The term “pseudo-occlusion” of the carotid artery was first used in 1960 by Newton and Couch to denote an appearance described earlier by Riishede and Ethelberg in patients with severely raised intracranial pressure. Macpherson subsequently reported 31 cases of "pseudo-occlusion," along with seven other cases of intracranial carotid occlusion. He observed that, on the first film of the series, the contrast column came to a tapered end in the neck in 12 cases of pseudo-occlusion. In the rest, the contrast material did not progress beyond the proximal branches of the intracranial carotid artery. Subsequent films showed very slow advance of contrast material in a layered fashion in some of his cases, without progress beyond the proximal branches of the intracranial carotid artery. He also noted that cases of intracranial ICA occlusion due to thrombosis or embolus produced an appearance similar to “pseudo-occlusion.”

Clark, et al., were the first to point out that atheromatous narrowing of the carotid artery can simulate the appearance of pseudo-occlusion. The surgical findings in three cases of angiographic carotid occlusion led them to a detailed study of their fourth case, which had angiographic characteristics similar to our cases. Their patient, in addition, had supraclinoid carotid stenosis. Kilgore and Fields have also briefly illustrated this phenomenon. The angiographic appearance of pseudo-occlusion in cases of extreme stenosis within an atheromatous plaque is caused by two factors. One factor is partial collapse of the lumen of the cervical carotid distal to the stenosis. This is presumably the result of a substantial reduction in intraluminal pressure. The second factor is layering of the slowly flowing dense contrast material along the dependent portion of the lumen. This further exaggerates the appearance of collapse of the distal lumen, and thus the actual size of the artery is larger than suggested by the angiograms (Fig. 5). The ICA fills earlier than the external carotid artery in the normal carotid circulation. However, in our patients, the external carotid artery filled much sooner than the proximal ICA, and arrival of contrast medium in the cavernous segment through the ICA’s was delayed as much as 9.5 seconds (Case 1).

Both the layering and collapse phenomena have been previously demonstrated in experimental and clinical studies. Fox and Hugh have shown in hydraulic models that the flow in the ICA is likely to be laminar. They believed that the rapid injection of contrast material produced a temporarily turbulent flow. When either the injection pressure was low or the
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Fig. 5. Schematic drawing of a highly stenotic ("pseudo-occluded") internal carotid artery showing layering of contrast material in the collapsed segment distal to the stenosis. Such layering creates the radiographic impression of an "apparent lumen" somewhat narrower than the actual lumen in its collapsed state. C. carotid = common carotid artery; ext. = external carotid artery; int. = internal carotid artery.

circulation was delayed, layering between the two fluids was observed, the heavier fluid flowing along the dependent portion of the vessel.

Debaene\textsuperscript{7} studied cerebral blood flow and circulation time in 163 patients who had had rapid serial angiography. Their patient population was composed chiefly of adults with hydrocephalus or subarachnoid hemorrhage or those in deep coma from other reasons. In these patients, layering of the contrast medium was found to correlate well with reduced cerebral blood flow and to correlate moderately with reduced circulation time. In these cases, there was initial prompt filling of the entire length of the cervical ICA, followed by extended layering for about 3 seconds. This pattern of layering was thus quite different from that observed in patients with atheromatous pseudo-occlusion. Debaene also studied nine patients with severe stenosis of the carotid artery, but they did not demonstrate any layering although the flow was reduced to less than half of the normal value in six. Presumably, blood flow must be reduced to an extreme degree for the phenomena of layering and collapse to occur.

In 1970, Lippman, et al.\textsuperscript{14} noted that the ICA may appear narrowed distal to a region of severe stenosis. They labeled this phenomenon "poststenotic carotid slim sign." The authors attributed the narrowing to the greatly diminished perfusion pressure beyond the stenotic region. In 1974, Houser, et al.\textsuperscript{13} (from the same group) reported a total of 13 cases from their series of patients with carotid disease who underwent surgical correction. In patients with the "slim sign," the preoperative cerebral blood flow was markedly depressed, and did not decrease further during clamping of the artery at surgery. The patients were monitored by EEG, and an intraoperative shunt was not required. Following carotid endarterectomy, however, cerebral blood flow increased markedly. This suggested to the authors that in the group with the "slim sign" the blood flow through the cervical carotid had been markedly diminished preoperatively, and that the major contribution to the hemispheric circulation was through collateral vessels from the external carotid artery and circle of Willis. The published angiograms of the patients with the "carotid slim sign" resemble those of the patients we are reporting here. However, our cases appear to have a much greater narrowing of the angiographically visualized cervical ICA. In addition, in each of our patients, the early films of the cervical carotid series showed the typical appearance of carotid occlusion, which was not the case in patients with the "slim sign." Pseudo-occlusion may thus be considered an extremely severe degree of the "slim sign."

Most recently, Countee and Vijayanathan\textsuperscript{8} reported a series of five patients with ICA occlusion who underwent successful endarterectomy. In these patients, preoperative angiography showed a thin line of contrast material in the cervical carotid artery, extending in an anterograde fashion for a short distance from the bifurcation. The distal portion of the ICA was kept patent by external carotid collateral vessels. However, all these arteries were reported to be occluded at surgery. The published arteriograms of these authors look similar to ours; however, in our cases, the column of contrast material in the ICA progressed from the neck to meet the intracranial carotid column filled through collateral vessels. In addition, in our cases, patency of the stenotic segment was surgically verified. In another recent report describing their experience with external carotid endarterectomy, the same authors\textsuperscript{5} reported a case of severe ICA stenosis with angiographic and surgical findings similar to our cases. They mentioned that they have seen four additional examples of this phenomenon. It is uncertain whether these are the same patients that were interpreted as having complete carotid occlusion in their initial paper.
Angiographic Considerations

Angiographic visualization of a vessel is influenced by several technical variables. The foremost is the selectivity of injection. The likelihood of demonstrating false occlusion is considered much greater when arch aortography is performed instead of high common carotid artery catheterization. Small catheters prove safer, since large catheters may wedge against the wall of the injected vessel impairing runoff of contrast material after injection, and promoting spasm or other local injury. A No. 5 French catheter has proved very satisfactory for selective cerebral angiography in adults. Excellent flow of the contrast material around the catheter is confirmed routinely by fluoroscopy of each vessel examined. Direct magnification (×2 to ×4) and serial filming with views prolonged over 4 to 10 seconds also prove quite important. Whenever there is apparent occlusion at the origin of the ICA on test injection of the contrast medium at fluoroscopy, or even in the initial film series, a prolonged film series is recommended. The lateral projection is usually more valuable, and should include the entire ICA from its origin to its intracranial bifurcation. Oblique films of the neck region, taken after fluoroscopic positioning to maximize visualization of the carotid bifurcation, are also valuable.

Atheromatous pseudo-occlusion is distinguishable from true occlusion of the carotid artery in which the distal portions of the artery are kept patent by collateral circulation. In these latter cases, no antegrade progression of the contrast material can be seen, even in later films. Any appearance of the contrast material in the cavernous or petrous portions of the ICA can be seen to coincide with external carotid opacification. An angiographic picture similar to atheromatous pseudo-occlusion is seen in dissecting carotid aneurysms, an appearance that Fisher, et al., have termed the “string sign.” However, patients with spontaneous carotid dissection characteristically have a normal carotid bifurcation, and the narrowing starts (in a funnel-like fashion) some 2 cm distal to the bifurcation. The lumen of the dissected artery usually recovers its normal caliber abruptly at the level of the petrosal segment of the ICA. Fungal granulomatous arteritis has been reported to produce diffuse narrowing of the ICA, but the bifurcation region was affected to the same extent as the cervical ICA’s in a diffusely irregular fashion. When the ICA is occluded distal to the ophthalmic artery, antegrade flow may persist through the ophthalmic artery, producing marked slowing of flow. Initial arteriograms may mimic the appearance of atheromatous pseudo-occlusion. Delayed films will distinguish the two, since, in the former, the bifurcation is relatively nonstenotic, the cervical carotid is not collapsed, and the site of distal occlusion can be identified. Recanalization of a thrombosed ICA may also be distinguished from atheromatous pseudo-occlusion by the characteristic irregular attenuated appearance.

Surgical Treatment

There are some theoretical considerations that suggest that carotid endarterectomy rather than microvascular bypass is the treatment of choice for atheromatous pseudo-occlusion. Carotid endarterectomy is technically a simpler procedure than microvascular bypass. Moreover, it can bring a greater quantity of blood to the cerebrovascular bed almost instantaneously. It is also possible that, in these cases of extreme stenosis, the symptoms are due to thromboembolism from the segment of the artery distal to the stenosis, where circulation is extremely slow. When this is the case, endarterectomy would eliminate the cause of symptoms. Microvascular bypass, on the other hand, may improve blood flow distally, but may have no effect on the “blind loop” which serves as the source of emboli. In Case 2, we were able to extract a clot from the distal segment, perhaps the cause of both the transient ischemic attacks and strokes.

This limited experience would suggest that endarterectomy is a safe and successful form of treatment in these patients. However, our follow-up period has been short (4 to 10 months), and long-term patency has not been proven. In all three cases, postoperative noninvasive ultrasonic studies and ophthalmodynamometry have strongly suggested patency of the endarterectomized vessel. Since these patients have been asymptomatic, there seems to be little reason to subject them to the potential risk of postoperative angiography.

Addendum

We wish to report a recent failure of attempted carotid endarterectomy for atheromatous pseudo-occlusion. The patient was different from the others in that the pathology was known to be present for at least a year prior to the time of referral, and at surgery the backflow from the distal internal carotid was poor, despite attempts to clear clots with a Fogarty catheter. Postoperative arteriography demonstrated an ICA occlusion. The patient has since had a microvascular bypass operation.

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References


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