Spontaneous resolution of occlusive lesions of the carotid artery

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The authors review the literature concerning spontaneous resolution of occlusive lesions involving the carotid artery. They add four cases illustrating three pathological vascular processes that may resolve spontaneously, namely, arterial dissection, atheromatous lesions at the carotid bifurcation, and an arteriopathy involving the intracranial vessels which is poorly understood at this time.

KEY WORDS • atherosclerotic plaque • carotid artery dissection • internal carotid artery • cerebral ischemia • carotid occlusion

The pathogenesis of occlusive lesions of extracranial and intracranial cerebral vessels is incompletely understood. Reluctance on the part of physicians to perform multiple angiograms in a group of potentially high-risk patients has limited our understanding of the natural history of these lesions. It is widely assumed that the atheromatous process accounts for the greatest proportion of human disease of middle and late life, and the process of atheromatous build-up is progressive.

Recent studies have emphasized that the occlusive plaque is in constant dynamic change. Atheromatous material fractures away from the plaque and is carried into the cranial circulation even in asymptomatic cases. Large ulcerative lesions heal spontaneously. Fibrin bodies develop, presumably secondary to platelet disruption, distal to sites of significant obstruction and these also break free and embolize. Thrombi also develop at sites of intimal damage, leading to distal occlusion via the embolization process.

The single angiogram, while extremely helpful in identifying the site and perhaps the degree of vessel narrowing, offers little information about the activity of the occlusive process. We present four cases with lesions of the carotid artery of varied etiology in which complete or partial resolution was demonstrated by angiography.

Case Reports

Case 1

This 58-year-old black man was in good health until 4 weeks prior to admission when he had the sudden onset of numbness in the left hand. Approximately 1 week after the onset of numbness, he developed weakness in the hand which progressed to involve the entire left arm. Two weeks prior to his admission, the left side of his face also became numb. He also described increased sensitivity on the left side of the body, with hypersensitivity to cold, heat, pin prick, and touch. The lower extremities were not affected.

Examination. General physical examination was unremarkable. Blood pressure was 148/86 mm Hg. The patient was alert and oriented in all spheres. The cranial nerves were normal with the exception of an incomplete left homonymous hemianopsia. There was moderate global weakness in the left arm, and normal tone and strength in the left leg. Pin prick, vibratory, and temperature appreciation were impaired in the left hand. There were no cerebellar signs.

Routine laboratory investigation, including cholesterol and erythrocyte sedimentation rate, was normal. The electrocardiogram was within normal limits. Computerized tomographic (CT) scan showed a low-density defect in the high anterior parietal convexity, with abnormal enhancement following the injection of contrast material. The pattern was consistent with a recent infarct.

Angiography showed marked narrowing of approximately 90% of the vessel diameter 1 cm below the bifurcation of the right intracranial internal carotid artery (ICA) into the middle and anterior cerebral arteries. There was also a narrow horizontal segment of the anterior cerebral artery, and narrowing of the
ICA at the level of the siphon (Fig. 1 left). However, the major portion of the middle and anterior cerebral arteries appeared normal. An arch injection demonstrated occlusion of the left vertebral artery at its origin.

Operation. An extracranial-intracranial (EC-IC) bypass procedure was performed in which the right superficial temporal artery was anastomosed into the temporal branch of the middle cerebral artery. Postoperatively, the patient had no additional deficit or complications. However, repeat angiography of the external system 15 days later indicated no perfusion of the middle cerebral artery branches via the bypass. At that time there was residual stenosis of the right ICA at the siphon.

The patient was again admitted 15 months later. He had noted gradual improvement in function of the left arm. There was residual left homonymous hemianopsia with slight left facial weakness. There was also increased tone in the left hand and left fingers. Carotid angiography indicated that the narrowing previously seen in the carotid siphon had now largely disappeared. Also, the narrow segment of the horizontal portion of the right anterior cerebral artery had widened considerably (Fig. 1 right).

Case 2

This 42-year-old woman was admitted immediately following sudden onset of right hemiparesis and aphasia. Her deficit cleared overnight and recurred twice the following day for 15 to 20 minutes on each occasion. She had a history of migraine headache, but had no headache or neck pain with the current episode. She had been taking birth control pills.

Examination. The initial neurological examination was within normal limits. Hemoglobin, white blood cell count, erythrocyte sedimentation rate, and sequential multiple analysis (SMA-24) profile were within normal limits. No cervical or ocular bruits were heard. Four-vessel angiography (Fig. 2 left) showed a tapered narrowing of the left carotid artery above the bulb, with irregular narrowing to the base of the skull. The artery was normal distal to this point.

Course. The patient was treated with intravenous heparin anticoagulation for 19 days and changed to Coumadin (warfarin) anticoagulation prior to discharge from the hospital on the 23rd day. She had several transient ischemic attacks during the first few days on heparin anticoagulation, but was then free of transient ischemic attacks during the 7 months of follow-up review. A four-vessel angiogram at 7 months was normal except for minimal intimal irregularity in the left ICA (Fig. 2 right).

Case 3

This 49-year-old man was brought to the emergency room in a comatose state following several seizures.
Spontaneous resolution of carotid occlusion

He was reported to have been drinking heavily the previous day. He regained consciousness in the emergency room, but remained hemiplegic on the right side and aphasic. One month earlier, he had undergone intermaxillary fixation of a fracture of the mandible sustained in a fight. He had no neurological deficit at that time.

Examination. He had total receptive and expressive aphasia, weakness of conjugate gaze to the right, right hemiplegia, hyperreflexia, and a Babinski sign. A CT scan showed a large low-density lesion in the left frontoparietal region compatible with an infarct. Right brachial and left carotid angiography showed occlusion of both ICA's just above their origins (Fig. 3 left). The patient was treated with daily 40-minute exposures to hyperbaric oxygen at 1.5 atmospheres absolute for 14 days, beginning 12 days after his stroke. He received intravenous heparin anticoagulation during this time.

Operation. He demonstrated improvement in neurological function, and EC-IC anastomosis was carried out on the left side after 14 days of intermittent hyperbaric oxygen exposure. Left carotid angiography 9 weeks later revealed intracranial filling through a stenotic ICA (Fig. 3 right). The EC-IC anastomosis was also patent. He had a residual mild right hemiparesis 4 months after his stroke.

Case 4

This 65-year-old man had the sudden onset of expressive aphasia and weakness of the right lower part of the face, tongue, and arm, with slight weakness in the right leg on the day of admission. Left carotid angiography showed total occlusion of the left ICA at the cervical bifurcation (Fig. 4 left). A right brachial angiogram showed an irregular plaque at the cervical carotid bifurcation, without significant stenosis. The patient received daily 40-minute exposures to hyperbaric oxygen at 1.5 atmospheres absolute, and an oral salicylate, 300 mg, four times a day for 2 weeks. During this time, his neurological deficit improved. Repeat left carotid angiography showed a narrowed but patent ICA (Fig. 4 right).

Operation. Left carotid endarterectomy was performed 16 days after the onset of neurological deficit. An extensively calcified arteriosclerotic plaque with an eccentric pinpoint lumen was removed from the cervical carotid bifurcation (Fig. 5).

Postoperative Course. The patient's preoperative improvement continued postoperatively so that at 1 year following his stroke the only neurological deficit was mild dysphasia and barely detectable impairment of the fine movements in the right hand. Angiography 4 months after carotid endarterectomy showed that the left ICA was patent (Fig. 6).
Discussion

As early as 1959, Luessenhop observed that the earlier angiography was performed after stroke, the higher the incidence of finding a major intracranial arterial occlusion. Similar observations have been made by Jacobsen and Skinhøj, Torvik and Jørgensen, Fieschi, and Zatz et al. A high early death rate and spontaneous lysis of certain occlusive lesions were assumed to be the explanation. Allcock reported normal vessels at repeat angiography in five of 10 patients who had previously been shown to have occlusion of the middle cerebral artery. In 1969, Fieschi and Bozzao reported "disobliteration" on follow-up angiograms in eight of 25 cases, with occlusion of the middle cerebral artery or its major branches, and in two of six cases of occlusion of the distal ICA. Clinicians over the years have observed the fragmentation and distal migration of emboli in the retinal vessels, and have come to expect that a high number of embolic occlusions will "disobliterate." Most of these reported lesions were assumed to be embolic in origin.

Recent reports have called attention to several cases of idiopathic arteriopathy of the larger cerebral arteries which were demonstrated to have resolved spontaneously at follow-up angiography. Mokri et al. reported three cases with angiograms suggestive, but not typical, of fibromuscular dysplasia of the carotid and vertebral arteries, and stenotic lesions of the carotid arteries resembling spontaneous dissections. Both the stenotic areas and the diffuse arteriopathy had partially or completely resolved at follow-up angiography. Day et al. reported resolution of stenotic lesions within the carotid siphon after proximal carotid endarterectomy in two patients. They considered these lesions to be embolic, and subsequently recommended that patients who are being considered for bypass procedures have angiography at intervals preoperatively to determine if spontaneous resolution has occurred. They did not comment on the possible role of long-term aspirin therapy on the basic vascular lesion, although both of their patients received this medication. Little et al. have also reported apparent arteriographic resolution of distal carotid narrowing following proximal carotid endarterectomy, although the lesions in their cases were thought to have been flow defects rather than structural lesions in the vessel walls.

The lesion in Case 1 of our series most resembled
Spontaneous resolution of carotid occlusion

FIG. 4. Case 4. Left: Left carotid angiogram demonstrating total occlusion of the left internal carotid artery at the cervical bifurcation. Right: Left carotid angiogram 2 weeks later showing a narrow but patent internal carotid artery.

the lesion reported by Day, et al. Since there was no suspected proximal source for an embolus in this case, and since the lesion was not lodged at a bifurcation, we consider this lesion to be a primary arteriopathy, rather than of embolic origin. The additional finding of a similar lesion in the proximal anterior cerebral artery supports this position. This lesion did not resemble the lesions termed "idiopathic regressing arteriopathy" by Mokri, et al., which had angiographic characteristics suggestive of fibromuscular dysplasia with dissection.

The angiographic appearance of a dissection of the ICA is characteristic. The typical lesion is a tapered, irregular narrowing of the artery beginning 1 cm or more above the bifurcation; it may extend cranially for a variable distance and terminate as a pointed total occlusion, or it may extend as a fusiform segment to the base of the skull, where the artery regains its normal diameter as it enters the carotid canal. The irregular narrowed segment may be shorter. These arteries may show the elongation and tortuosity seen in fibromuscular dysplasia, and the irregularities of fibromuscular dysplasia may be seen in the carotid artery on the side opposite to the dissection. Accumulated experience with these lesions suggests that operative treatment is often unsuccessful, whereas spontaneous resolution, with restoration of normal or near normal arterial patency, is the rule and may occur in 80% to 90% of the cases. It is important, therefore, that the clinician make the correct diagnosis from the angiogram so that operation may be avoided.

Heparin anticoagulation therapy is the initial treatment of choice unless contraindicated by established cerebral infarction. The duration of anticoagulant therapy necessary, as well as the indications for the use of Coumadin, have not been clearly defined.

Arteriosclerosis involving the ICA is considered to be a progressive disease, although spontaneous recanalization of an occluded ICA has been reported. The incidence is not known. Keller, et al., reporting on 334 patients with severe obstruction of the supraaortic extracranial cerebral arteries, found evidence of spontaneous resolution of obstructive lesions in the ICA in five patients, in the brachiocephalic trunk in one, and in the proximal subclavian artery in one. Confirmation was by repeat angiography in one case,
by comparison of angiographic and operative findings in two cases, and by serial Doppler examination in the remaining four cases. To our knowledge, in none of the reported cases of spontaneous recanalization of ICA occlusion has the lesion been proven to be an arteriosclerotic plaque. Most of the reported cases were probably embolic or spontaneous dissections.17,21

One of the two cases in our series of partial resolution of lesions involving the cervical carotid bifurcation was found in a series of 15 patients who underwent follow-up angiography to evaluate the status of an EC-IC anastomosis in one hospital. Angiography was repeated in Case 4 after the unexpected finding in Case 3. Since follow-up angiograms are seldom performed once the diagnosis of carotid occlusion is made, the actual incidence of spontaneous resolution of occlusive cervical carotid lesions may be higher than has heretofore been expected.

Fox,9 Day, et al.,3 and Heros and Sekhar12 have each recommended interval angiography prior to EC-IC bypass to determine the persistence of the offending lesion. Their recommendation is based on experience with emboli, dissections, carotid siphon lesions of undetermined type, or in one case development of collateral circulation distal to an occlusion of the cervical ICA. We add that the atheromatous plaque occluding the cervical carotid bifurcation may partially resolve to permit flow through it in sufficient quantity to establish patency of the distal ICA. In these cases, although the offending lesion may still require surgical treatment, cervical endarterectomy should provide more effective revascularization than EC-IC anastomosis, and also removes a potential source for emboli to the brain which remains if the diseased carotid is patent.

Summary

Four cases have been presented in which spontaneous resolution of occlusive carotid lesions was documented by angiography. These cases include a spontaneous carotid dissection, two atheromatous occlusions involving the cervical carotid bifurcation, and a poorly defined arteriopathy involving the supraclinoid carotid. These cases suggest the following conclusions:

1. Certain occlusive carotid lesions are dynamic, with the processes of stenosis and repair proceeding at different rates. Thus, certain surgically inaccessible lesions may be amenable to medical therapy. Further efforts to define these lesions and establish treatment modalities are warranted.

2. The need for interval angiography before EC-IC anastomosis has been reinforced.

3. Physicians who treat patients with cerebrovascular disease should be familiar with the angiographic picture of known self-healing lesions such as carotid dissection, so that inappropriate operations may be avoided.

J. P. Kapp and R. R. Smith
Spontaneous resolution of carotid occlusion

References


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