Atheromatous Pseudo-occlusion of the Internal Carotid Artery

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Between 1978 and 1988, the diagnosis of atheromatous pseudo-occlusion of the internal carotid artery was made in 34 patients by angiography. Results of noninvasive tests were abnormal in 33 of the 34 patients examined. Twenty-five patients had carotid endarterectomy, and the other nine were treated medically. Four of the 34 patients (12%) had significant complications, two related to angiography and two to surgery. Twenty-three of the 25 operated patients were seen in long-term follow-up; 19 (83%) were found to have a patent operated vessel by noninvasive testing. None of the 23 operated patients followed up suffered recurrent neurologic deficits following surgery; two had distant contralateral strokes. Three of the nine patients treated medically (33%) experienced delayed ipsilateral stroke. This study shows that the risks associated with angiography and surgery for atheromatous pseudo-occlusion are significant and are higher than previously reported.

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Highly stenotic but patent internal carotid arteries (ICAs) can easily be misdiagnosed as occluded by both noninvasive testing and by angiography. Blood flow distal to a site of extreme stenosis may be so minimal that it remains undetected. The term “atheromatous pseudo-occlusion” was first used by Lippman et al in 1970. They described layering of contrast material along the dependent posterior wall of the ICA distal to a site of high-grade stenosis at the time of angiography. Other descriptive terms used to categorize the angiographic appearance of such extremely stenotic lesions are the poststenotic slim sign, the string sign, and the nearly occluded carotid artery. Once identified, urgent carotid endarterectomy seems to be automatically accepted as appropriate for pseudo-occlusion. However, surgery for pseudo-occlusion should be attempted only if surgically treated patients are shown to have better prognoses than those treated medically and if long-term patency of the operated vessel is high. These issues have not been previously addressed.

This report describes our experience with 34 patients with atheromatous pseudo-occlusion. All 34 underwent selective carotid angiography, and 25 subsequently had carotid endarterectomy. We report on the complications associated with both procedures. We also report the long-term clinical outcome and long-term vessel patency of the operated vessel in an attempt to address the issue of appropriate management of patients with nearly occluded carotid arteries.

Subjects and Methods

From July 1978 until July 1988, 1,092 successive patients underwent angiography for suspected extracranial cerebral vascular disease. The diagnosis of pseudo-occlusion was made in 34 patients (3%), 16 men and 18 women, average age 66 (range 47–78) years. Presenting symptoms were transient ischemic attacks (TIAs) in 17, stroke in 14, and nonlocalizing symptoms in one; two patients were asymptomatic. Risk factors included coronary artery disease in 19, diabetes in 16, hypertension in 22, peripheral vascular disease in 12, and hyperlipidemia in three. Three patients had undergone previous endarterectomy of the pseudo-occluded vessel.

All 34 patients had noninvasive testing prior to angiography. The noninvasive testing regimen evolved over the 10-year period. For the past 6 years, the routine noninvasive series has included B-scan ultrasound imaging, direct Doppler (continuous-wave [CW] and pulse wave) examination with spectral analysis, oculoplethysmography (OPG), and periorbital Doppler examination (PD). As we have previously reported, noninvasive studies cannot reliably distinguish pseudo-occlusion from occlusion of the ICA. Doppler-shifted ultrasound techniques measure the change that occurs when a signal of known frequency is reflected from a moving target. When directed at the carotid artery, the...
Doppler signal shift is proportional to the velocity of the erythrocytes within that part of the vessel. With increasing stenosis, erythrocyte velocity at the point of maximal stenosis increases. However, with extreme degrees of stenosis, blood flow across the stenosis decreases dramatically, with a corresponding fall in erythrocyte velocity. Doppler instruments contain high-pass filters to screen out low-amplitude echoes such as those generated by the movement of the carotid artery wall. When blood flow is sufficiently low, the Doppler shift generated by the movement of the erythrocytes will fall below this threshold and go undetected. Furthermore, the number of moving aggregates of erythrocytes in the ICA may be too few to generate a signal of sufficient energy to be distinguished from random background noise. Thus, for both pseudo-occlusion and occlusion, the results of Doppler study will be identical and will suggest the absence of blood flow. For both pseudo-occlusion and occlusion, B-scan ultrasound imaging will demonstrate thrombus within the ICA and will suggest complete occlusion. The results of the indirect tests (OPG and PD) will be significantly abnormal with both pseudo-occlusion and occlusion. Our noninvasive criteria for both pseudo-occlusion and occlusion of the ICA were 1) absence of any frequency shift in the returning Doppler signal (no blood flow), 2) decrease of peak systolic velocity in the ipsilateral common carotid artery compared with that of the opposite side, 3) ultrasonic visualization of a pattern of increased echoes in the diseased ICA, 4) absence of pulsatile motion, 5) abnormal OPG results on the side of the diseased vessel, and 6) reversal of blood flow in the ipsilateral supraorbital or supratrochlear arteries.

Our rationale for raising the possibility of pseudo-occlusion as opposed to complete occlusion after noninvasive testing was basically clinical. If results of the noninvasive test suggested occlusion and if symptoms occurred within the month preceding the noninvasive study and were appropriate to the abnormal vessel, pseudo-occlusion was suggested.

When pseudo-occlusion was suspected, the angiographic technique employed in 31 of the 34 patients was that first described by Countee and Vijayanathan. It was performed in the following fashion: 1) selective catheterization of the common carotid artery, 2) prolonged injection of high-dose contrast medium (4 ml Conray [Mallinckrodt, St. Louis, Missouri] 60/sec for 3–4 seconds, a total of 12–16 ml), 3) filming in the lateral view with coning to include both the carotid bifurcation and the supraclinoid carotid artery, 4) prolonged filming with 1 film/sec for 14 seconds, and 5) routine use of subtraction techniques. For the final three patients in this series, the angiographic technique used was selective catheterization of the common carotid artery with digital subtraction angiography (DSA) (4 ml Conray 30/sec for 3 seconds, a total of 12 ml; filming in the lateral projection with 2 frames/sec for 14 seconds).

![Figure 1](http://stroke.ahajournals.org/)

**Figure 1.** Right common carotid angiograms, lateral view, coned to include carotid bifurcation and supraclinoid carotid artery. Image at 12 seconds demonstrates thin trickle of contrast medium progressing cephalad to cavernous sinus (arrows).

The angiographic diagnosis of pseudo-occlusion was made when a thin, markedly delayed antegrade trickle of contrast medium without discernible washout on later films was visible in the ICA distal to a point of extreme stenosis (Figure 1). In most patients the contrast column had not reached the base of the skull by the 4-second film following injection. Patients presenting with high-grade stenosis but without layering or a significant delay in antegrade blood flow were not diagnosed as having pseudo-occlusion.

The decision to perform endarterectomy was made by the referring physician, based on the patient's clinical presentation and associated risk factors. No attempt was made to randomize patients with respect to surgery. In practice, patients underwent endarterectomy unless there were specific contraindications. Because most of these patients were acutely symptomatic, management was carried out on an urgent basis. All patients scheduled for surgery were heparinized immediately after angiography. Twenty-five patients had endarterectomy...
of the pseudo-occluded ICA performed by one of eight surgeons, 21 patients within 24 hours and four within 1 week after angiography. In no patient was any unusual technical difficulty encountered at surgery. The other nine patients had medical treatment of the pseudo-occlusion. Among the nine were two who had a significant angiographic complication. Of the remaining seven, four still underwent surgery; three had endarterectomy of the opposite ICA for 60–95% stenosis, and one was operated upon on the side of the suspected pseudo-occlusion but did not undergo endarterectomy after his surgeon decided, on direct inspection of the external surface of the vessel, that the ICA was not patent. The other three medically treated patients were considered to be inoperable due to severe cardiac problems.

Periodic follow-up noninvasive studies to determine vessel patency were performed after discharge. No patient had repeat angiography. At the time of follow-up noninvasive testing, a history of intervening neurologic symptoms was obtained. Clinical follow-up of patients was also accomplished by telephone conversation with the patient, the family, and the primary physician.

**Results**

Results of the initial noninvasive tests were significantly abnormal in 33 of the 34 patients studied, occlusion or pseudo-occlusion in 31 and 75–99% stenosis in two. In one patient, results of the noninvasive test were interpreted as normal. This occurred in the first year, prior to the introduction of B-mode imaging and pulsed Doppler studies.

Five of the 34 patients had cerebral complications at angiography. Three of the five patients experienced TIAs in the territory of the pseudo-occluded artery; symptoms in two cleared within minutes while the third patient, whose deficit worsened over the following 2 hours, underwent emergency endarterectomy and was completely well following surgery. Two of the five patients had strokes on the side of the pseudo-occluded vessel, one during angiography and one on the day after angiography. Neither patient underwent surgery, and both were left with significant neurologic deficits. To ensure that this high incidence of complications did not reflect the overall complication rate for unselected patients, a control group of 195 consecutive patients was studied prospectively as part of another investigation.11 Except for an episode of transient hypotension, no angiographic complications occurred in these 195 patients.

There were surgical complications in four patients. TIAs in two patients during the immediate postoperative period resolved within a few hours. Two patients experienced frank strokes. The first of these latter two patients had a history of stroke and previous endarterectomy in the territory of the pseudo-occluded vessel and presented with recurrent TIAs. This patient had a total occlusion of the contralateral ICA as well as bilateral vertebral artery stenosis and was left with mild hemiparesis. The second patient was experiencing multiple TIAs and had severe bilateral carotid artery disease. This patient had a stroke in the territory of the pseudo-occluded vessel at the time of surgery and died 2 weeks later of myocardial infarction. Again, to ensure that this complication rate was unique to this subset of patients, a retrospective study of 191 consecutive carotid endarterectomies done at our hospital was taken for comparison. The retrospective study demonstrated a perioperative stroke rate of 2.6% in diabetic patients and 0% in nondiabetic patients.12

Patients were followed to determine vessel patency and clinical outcome. Follow-up for vessel patency in all nine patients treated medically showed occlusion. Two operated patients were lost to vessel patency follow-up. The average follow-up time for the 23 remaining operated patients was 30 months (range 2 weeks to 73 months). Nineteen operated patients showed long-term patency of the previously pseudo-occluded vessel, and four had total occlusion. Thus, the patency rate following endarterectomy was 83%.

Two of the 34 patients were lost to follow-up for clinical outcome. The average long-term follow-up for clinical outcome among the 32 remaining patients was 34 months (range 1 week to 73 months). No operated patient experienced delayed neurologic complications in the operated hemisphere. Two operated patients did have contralateral strokes, one 6 months and one 24 months after surgery. Of the three medically treated patients who underwent endarterectomy of the opposite ICA, one had a major stroke in that portion of the brain supplied by the nonoperated pseudo-occluded vessel resulting in permanent neurologic disability 1 week following surgery. The second patient had a retinal artery occlusion resulting in monocular blindness on the pseudo-occluded side 11 months after surgery. The third patient had no further neurologic symptoms. The medically treated patient who underwent exploratory neck surgery but not endarterectomy had a severe stroke 3 months after surgery in the territory of the pseudo-occluded vessel. The three medically treated patients for whom surgery was thought to be contraindicated because of cardiac risk factors experienced no further neurologic deficits (Figure 2).

There were eight late deaths (average 16 months). Five occurred among the 23 operated patients and three among the nine medically treated patients. Six deaths were from cardiac causes, and one each from cancer of the lung and gastrointestinal bleeding.

**Discussion**

The natural history of patients presenting with pseudo-occlusion of the ICA is unknown. Pseudo-occlusion represents a moment late in the time
Review of the literature reveals 62 reported cases of angiographically diagnosed atheromatous pseudo-occlusion of the ICA.1-10,13 Reports contain as many as 13 and as few as one case. Fifty-six of these 62 patients were symptomatic, and 54 had carotid endarterectomy. There were no reported complications of either angiography or surgery, suggesting that patients with pseudo-occlusion are at no particular risk when subject to aggressive management. However, in our series four patients experienced significant strokes with permanent deficits, two related to angiography and two to surgery. Review of other series that relate the complication rates of carotid endarterectomy to clinical presentation suggests that our experience is not unique. Sundt et al14 grouped patients according to preoperative evaluation of risk of carotid endarterectomy in a series that encompassed 342 operations. Neurologically stable patients without medical or angiographically determined risk factors (Group 1) had a risk for neurologic deficit at 1%. However, neurologically unstable patients (Group 4), into which category almost all of our patients would fall, had a 10% risk. Zurbrügg and his colleagues15 reviewed 200 reports that encompassed 16,858 endarterectomies and found a mortality of 1.3% and a permanent morbidity of 4.4% for patients presenting with TIAs and a mortality of 6.8% and a permanent morbidity of 10.2% for those presenting with stroke. Since their papers deal only with the risk of surgery, our combined rate of mortality and permanent morbidity for both angiography and surgery (12%) is not unexpected.

Our rate of serious complications would normally preclude endarterectomy.16 However, a number of studies have suggested that symptomatic patients with pseudo-occlusion or acute ICA occlusion are at increased risk for stroke.17-19 Certainly, the outcome in the seven medically treated patients having no angiographic complication supports the impression that these are patients at extremely high risk. Two of these seven had a major stroke and another lost vision in one eye, all appropriate to the side of the pseudo-occlusion.

There are at least four possible mechanisms of stroke with pseudo-occlusive disease. These mechanisms include hemodynamic ischemic insults, progressive propagation of thrombus to the intracranial arteries, “stump” emboli passing through the external carotid circulation, and emboli arising from clot formed at the site of evolving occlusion, so-called “secondary emboli.”20-24 Since pseudo-occluded vessels contribute little if any useful blood supply to the cerebral hemisphere, ischemic infarction caused by a decrease in the critical blood flow seems to be an unlikely mechanism for injury. The usual atrophic appearance of the cervical ICA distal to the site of maximum stenosis supports this concept. The justification of endarterectomy then is largely to prevent stroke from secondary emboli. All but two of our 34 patients were symptomatic, and, as discussed by Goldstone and Moore,25 the risk of not operating in these circumstances may be quite high. It can be argued that the high rate of mortality and morbidity associated with ICA occlusion may well justify every attempt to prevent the periocclusive state from progressing to the occlusive.26-28 Nevertheless, we found the complication rates for angiography and endarterectomy in patients with ICA pseudo-occlusion to be high and possibly to outweigh any benefit derived from surgery.

The patency rate among the 23 operated patients who were followed up was 83%. Of the four for whom the attempt to reopen their vessel failed, none suffered any postoperative complication. All four were found to have abnormal noninvasive test
results immediately following endarterectomy. None of the 19 with normal noninvasive test results immediately after surgery later occluded, nor did they experience any further neurologic deficit on the operated side. Two of the 19 patients who underwent successful endarterectomy later suffered a stroke in the opposite circulation, emphasizing the severity of the atherosclerotic process in this group.

Ringelstein et al described nine patients in whom the diagnosis of pseudo-occlusion was made prospectively with CW Doppler. These authors recommended careful examination of the region of the carotid bulb to detect a faint, sharp, and continuous hissing sound. This tone was stated to be characteristic of pseudo-occlusion. Our examination procedure with CW Doppler appears to parallel theirs, but we identified this finding in only two of our 34 patients. If we had depended upon the detection of a faint, nonpulsatile hiss detected with CW Doppler, we would have overlooked the diagnosis of pseudo-occlusion in 94% of our patients. Rushton and Kukora reported findings similar to ours and concluded that noninvasive tests could not reliably distinguish very-high-grade stenosis from total occlusion. This observation is important since the noninvasive findings may determine how aggressively the diagnosis of pseudo-occlusion is pursued at angiography.

While selective carotid angiography with injection of medium high-dose contrast and prolonged filming was critical to the diagnosis of pseudo-occlusion, the possible contribution of angiography to the high complication rate we experienced cannot be overlooked. Injection of a large volume of contrast medium under pressure immediately proximal to fresh thrombus may well pose a significant hazard. As previously noted, no similar problems were reported by other authors. In 1984, we decreased our injected volume from 16 to 12 ml. It has been suggested that rapid-sequence axial computed tomography (CT) and intravenous DSA could be used to diagnose pseudo-occlusion without risk to the patient. We have no experience with the former and have been disappointed in our limited experience with the latter. Limited resolution, patient movement, misregistration artifacts, superimposition of vessels, and the inability to obtain a lateral view all pose significant problems in the use of intravenous DSA. Intra-arterial DSA, with selective injections of smaller volumes of contrast medium, should be able to replace high-dose delayed conventional angiography in patients with pseudo-occlusion, possibly lowering the incidence of angiographic complications. We studied our last three patients using only selective intra-arterial DSA, achieving good-quality studies without any adverse reactions.

Our retrospective study suggests that aggressive management of atheromatous pseudo-occlusion of the ICA carries a significantly worse prognosis than that reported by other authors. Of the 34 patients with pseudo-occlusion undergoing angiography, five (15%) had angiographically related ischemic symptoms. Of the 25 patients having endarterectomy, four (16%) experienced ischemic symptoms. Among our 34 patients with pseudo-occlusion, four (12%) were left with fixed neurologic deficits, two related to angiography and two to surgery. Unfortunately, the outcome for patients not undergoing surgery appears correspondingly bleak. Excluding the two patients who suffered irreversible ischemic deficits at the time of angiography, three of the seven medically treated patients later experienced significant deficits in the territory supplied by the pseudo-occluded vessel. None of these medically treated patients was heparinized.

Our study does demonstrate four facts: 1) pseudo-occlusion is a very unstable situation; 2) angiography and surgery represent a significant risk for patients with pseudo-occlusion; 3) endarterectomy is technically feasible, long-term patency of the operated vessel is high, and distant neurologic sequelae within the territory supplied by the operated vessel are rare; and 4) the major long-term risk for these patients is cardiac disease. The high complication rate of angiography and surgery must cause us to reflect on the proper management of these patients. We conclude that surgical intervention remains appropriate in part because the long-term outcome for patients successfully operated upon was good and in part because the suspected outcome in patients treated conservatively would not be as good. We recognize that we do not have adequate data to support this latter supposition. When surgery is contemplated, certain steps should be taken to lower the incidence of complications. The volume of contrast medium used at angiography should be reduced. DSA should be employed whenever pseudo-occlusion is suspected. In our most recent case of suspected pseudo-occlusion, which proved at angiography to be occlusion, we used only 6 ml Conray 30. Whether the use of intra-arterial DSA and small volumes of contrast medium are always adequate in this setting remains to be proven, but it seems reasonable to start the examination in this fashion. Additional injections with cut film can always be performed if necessary. Also, more attention needs to be focused on the neurologic status of these patients at the time of presentation. Management should be based on this, rather than on results of angiographic study. It is a surgical axiom that thrombus is best removed while still fresh. If patients are neurologically stable, or have had a neurologic deficit that has cleared, and the history and results of noninvasive tests suggest possible pseudo-occlusion, we recommend emergency angiography and endarterectomy. In patients with fluctuating or small "fixed" neurologic deficits, we would perform a CT or magnetic resonance imaging scan as the first step. If this shows no lesion or a small lesion, we would handle them as asymptomatic patients. In patients who present with a
significant or progressing neurologic deficit, we suggest that angiography and surgery be delayed until the patient is in stable condition. During the waiting period emboli should be prevented by anticoagulation, first with heparin and later, if necessary, with warfarin. Buchan and his colleagues suggested such an approach in the management of patients who presented with intraluminal thrombus in the carotid arteries because of the significant risks they found associated with emergency endarterectomy. Other authors recommend the same treatment for neurologically symptomatic patients with tight carotid stenosis. While it is unlikely to occur, to truly answer the question whether this is adequate therapy for pseudo-occlusion would require a prospective randomized study.

References

O’Leary et al Pseudo-occlusion of ICA 1173

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