Management of Carotid Artery Occlusion

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Many authors have drawn attention to occlusive disease of the carotid arteries as a cause of cerebral ischemia. Although total carotid artery occlusion may be caused by different disease entities, by far the most frequent cause remains atherosclerosis. However, because of uncertainty about the pathophysiology of symptomatic internal carotid artery (ICA) occlusion, there has been controversy surrounding its proper management.

Natural History of Carotid Artery Occlusion

Early studies gave a good overall idea of the prognosis following carotid artery occlusion, but in many cases the information was incomplete. These studies were limited by 1) the inclusion of patients with moderate to severe neurological deficits whose prognosis for survival may differ from that of less severely affected individuals and in whom it may be difficult to detect new ischemic events in a vascular territory where a major stroke has already occurred, 2) no information on the exact localization of recurrent ischemic events during follow-up, and 3) the inclusion of patients treated with either anticoagulants or carotid surgery, both of which may influence morbidity and mortality.

Recently, more complete data have become available on this condition. In general, prospective studies of carotid artery occlusion suggest a higher stroke rate during follow-up than retrospective series, on the order of 5% or more per year. This rate is similar to the one found in the subgroup of patients with ICA occlusion treated medically in the Extracranial-Intracranial (EC-IC) Bypass Study. Prospective analysis is a more sensitive investigative tool that permits the detection of minor strokes or reversible ischemic neurological deficits that may go undetected in a retrospective study, especially in those cases in which there is no permanent impact on the functional status of the patient. In our study the exclusion of patients with a minor stroke not affecting functional status reduced the annual stroke rate from 5% to 3% ipsilateral to the occluded artery, which compares well with the rates found in retrospective series. Overall, the risk of stroke and vascular mortality in patients with ICA occlusion appears similar to or slightly less than that for patients presenting with transient ischemic attacks (TIAs) or minor strokes due to all causes.

Several studies have addressed the relation between the severity of carotid atheroma and prognosis. Although Harrison and Marshall have suggested that the risk of subsequent stroke and mortality is greatest in patients with complete carotid artery occlusion, as opposed to patients with less severe disease, others have been unable to confirm these findings. In a 10-year follow-up of 158 patients with angiographically proven arterial disease, we were unable to correlate the severity of the atherosclerotic lesion with the rate of ipsilateral ischemic events. Again, patient selection may explain some of these variations. Differences in stroke severity and the occurrence and management of vascular risk factors might explain variations in the mortality rate.

The amount of functional recovery after cerebral infarction due to unilateral ICA occlusion is variable. Waltimo et al followed a group of 155 patients for a median period of 53 months. Forty-five percent of their patients were independent in activities of daily living, 22% required some assistance, and 11% were totally disabled. Of note is the fact that their population was relatively young, with a median age of 53 years.

Few authors have addressed the prognostic significance of bilateral ICA occlusion. Wortzman et al, in a small group of patients, reported a poor outcome with a high rate of functional limitation and mortality. However, their population included patients with initial severe neurological deficits. In contrast, a more recent report has suggested a much better prognosis when prospective follow-up is restricted to a group of preselected patients, excluding those with severe deficits at presentation. Nonetheless, patients with bilateral ICA occlusion have a higher rate of cerebrovascular events and mortality when compared to patients with unilateral ICA occlusions.

Pathogenesis of Cerebral Ischemia

Both thromboembolic and hemodynamic mechanisms have been invoked in the etiology of cerebral ischemic events related to carotid occlusion. Emboli can originate from different sources. Acute carotid occlusion associated with atherosclerotic disease almost invariably complicates high-grade
stenosis. In this setting embolization could occur just prior to complete occlusion when remaining flow is presumably most turbulent. Another source of embolus is the propagation of thrombus that extends distally from the origin of the ICA to the first high-flow collateral, usually the ophthalmic artery. The collateral flow originating from the contralateral side or the posterior circulation via the circle of Willis may break off parts of the thrombus and produce distal embolization. Emboli can be large and cause proximal vascular obstruction with extensive corticosubcortical infarction or they may be smaller, causing obstruction of more distal vessels and producing a more limited area of infarction. Emboli may also originate from atheromatous disease of the external and/or common carotid arteries or even from the remaining "stump" of the ICA. These in turn can "paradoxically" shed to the retina or cerebrum via the external carotid artery along the ophthalmic arterial collateral pathway.

Hemodynamic hypoperfusion is the second mechanism by which cerebral ischemia may occur. Acute ICA occlusion in the absence of adequate collateral circulation may cause a massive infarct. This result has been well described following therapeutic occlusion of the ICA or common carotid artery for a variety of intracranial disorders. Some patients with chronic ICA occlusion are vulnerable to sudden drops in mean arterial blood pressure because of poor collateral reserve.

Watershed infarcts traditionally have been associated with hemodynamic hypoperfusion along the border zones between the territories of major intracranial arteries but can also be produced by showers of microemboli shed from a mural thrombus prior to complete occlusion of the carotid artery.17 Some authors15 have suggested that mild neurological deficits and the occurrence of premonitory TIAs in ICA occlusive disease are more often linked to low distal perfusion states or hemodynamic causes, as opposed to the more severe deficits and fewer warning symptoms in patients with a presumed thromboembolic event. Although it is difficult to estimate the relative frequency of each of these mechanisms as a cause of cerebral ischemia, most would agree that thromboembolic phenomena are probably more frequent than hemodynamic mechanisms among patients with symptomatic ICA occlusion.15,16,18

Clinical Aspects

The spectrum of neurological deficits associated with ICA occlusion is wide and varies from asymptomatic to massive and lethal cerebral infarction. The presence of signs and/or symptoms in this setting is dependent on multiple factors including the available collateral circulation, atherosclerotic disease in other arterial territories, and vascular anatomic variations as well as systemic blood pressure, blood viscosity, and oxygen content. Certain authors19 have found a direct relation between the volume of cerebral infarction, the presence of good collateral flow, and the functional status of these patients. Although the clinical symptomatology accompanying ICA occlusion may be quite diverse and nonspecific, certain clinical presentations are more often associated with this condition: watershed infarctions of different types,20 orthostatic cerebral ischemia,21 and ischemic oculopathy.22

Diagnostic Measures

Selective biplane intra-arterial carotid angiography with suitable subtraction imaging is the most sensitive procedure for diagnosing complete carotid occlusion. The drawbacks of carotid angiography are well known and, apart from the inherent risk of morbidity, include the need for hospitalization, discomfort to the patient, and cost. For these reasons noninvasive studies are frequently used, especially as screening procedures. These techniques include direct and indirect methods of investigation.

One of the major difficulties encountered with noninvasive techniques is differentiating between a high-grade stenosis and a complete occlusion of the ICA. This distinction is important since the management of high-grade stenosis is different in many cases from that of complete ICA occlusion. Although a battery of noninvasive studies can be highly accurate in the initial evaluation of the patient with symptomatic carotid disease, it is generally accepted that conventional biplane intrarterial angiography (or intraarterial digital subtraction angiography [DSA]) is essential to confirm the presence of complete occlusion.23,24

Angiography is also invaluable for determining the adequacy of collateral flow and the presence of associated arterial lesions that could be the source of persisting symptoms. Intravenous DSA (IV-DSA) does not carry the same risk as a conventional intra-arterial study. Some authors suggest that IV-DSA can distinguish ICA occlusion from very tight stenosis, but others disagree.25

Therapeutic Considerations in ICA Occlusion

Medical

As with all types of atherosclerotic vascular disease, the control and elimination of risk factors, especially hypertension, is desirable for preventing ischemic events. In the acute phase, however, blood pressure reduction should be very gradual, especially in patients with carotid occlusion, in order to avoid worsening of the ischemic process secondary to decreased blood flow and impaired autoregulation. Antiplatelet and anticoagulant agents are used regularly in the management of patients with ischemic cerebrovascular disease including ICA occlusion. Although aspirin has been found to be of value in the prevention of cerebral ischemia in patients with TIAs and minor strokes, little information is available about its efficacy in patients with differing severities of arterial disease, especially in
patients with advanced vascular disease such as carotid occlusion. The role of anticoagulants is even less clear in this regard.

The status of hemodilution in acute ischemic stroke is still not definite. Recent reports have not been very encouraging, although they do not provide much information about the underlying vascular status of the patients in terms of either large-vessel disease or microcirculatory flow. It is conceivable that patients with carotid occlusion and reduced cerebral blood flow may respond more positively to this therapy. Attention has lately been focused on the use of tissue plasminogen activator (tPA) and other fibrinolytic agents in the treatment of acute occlusive cerebrovascular diseases. Preliminary results are encouraging, with high rates of recanalization. However, the role of thrombolytic therapy in acute ICA occlusion has not been established, and several trials are under way. Patients must be identified and treated within a few hours of stroke onset to minimize the risk of hemorrhagic transformation of the ischemic infarction. Other issues that need to be addressed include the best route of administration (either intra-arterial or intravenous), the type of agents (tPA or others), and determination of the best dose rate for maximum safety and efficacy.

At the present time aspirin remains the first step in the medical management of patients with ICA occlusion. In addition to lowering stroke risk, aspirin provides protection against cardiovascular events, which are the most frequent cause of mortality in these patients.

Surgical

The EC–IC Bypass Study Group identified and followed for an average of 55.8 months 808 patients with unilateral and 57 with bilateral ICA occlusions. Medical and surgical groups were similar for baseline characteristics and length of follow-up. Both of these subgroups actually did worse with surgery than with medical therapy. Although the investigators could not explain these findings, they proposed that the external carotid artery and the surgical bypass could have acted as a potential route for embolization.

Some have claimed that the most common cause of stroke in these patients was thromboembolic and that the study failed to identify appropriately patients with hemodynamic causes, who could possibly benefit from surgery. Even in the subgroup of patients in the EC–IC trial who had the poorest collateral supply on angiography, presumably the ones who would benefit most from revascularization, no surgical benefit could be found.

Positron emission tomography (PET) scanning remains the ultimate tool for determining regional cerebral blood flow (rCBF), regional cerebral metabolic rate of oxygen consumption (rCMRO₂), and oxygen extraction fraction (OEF). The combination of low rCBF, low rCMRO₂, and high OEF are diagnostic of “misery perfusion” and theoretically would identify cerebral tissue at risk of infarction. Recent evaluations of small groups of patients before and after EC–IC arterial bypass, with PET studies comparing them with either historical or medically treated controls, have failed to demonstrate a significant improvement in oxygen metabolism and on follow-up have also failed to show clinical differences in stroke rate or functional recovery. On the basis of this evidence, one should be cautious about inferring a benefit from any other surgical procedure that presumably increases cerebral blood flow distal to an occluded carotid artery, including contralateral endarterectomy. Perhaps remaining but yet unproven indications for EC–IC bypass surgery in the context of ICA occlusion include some patients with ischemic oculopathy or with PET-documented misery perfusion not responding to current medical therapy.

There is general agreement that thromboendarterectomy in the presence of an acute or chronic ICA occlusion is associated with a very high morbidity and mortality rate. However, Meyer et al reported a series of 34 patients with profound neurological deficits who underwent emergency thromboendarterectomy and of whom 38.3% made an excellent or good recovery. The mortality of 20.6%, although high, approximates the expected mortality with medical therapy.

The role of ipsilateral external carotid endarterectomy in both increasing collateral flow and removing a potential source of emboli is not well defined. Gertler and Cambria have gathered a collective review of the available literature, which includes 195 external carotid endarterectomies. The best results were obtained in patients who presented with amaurosis fugax or cerebral TIAs. However, these reports lacked adequate controls, and no firm conclusions can presently be drawn about the efficacy of this procedure in stroke prevention. The same remarks also apply to the removal of a remaining proximal ICA stump (stumpectomy) with or without the simultaneous performance of an external carotid endarterectomy.

Still more uncertainty underlies the various reconstructive procedures for common carotid artery occlusion because the natural history and clinical implications are even less well known.

Conclusions

Certain clinical presentations (e.g., watershed syndromes) are more suggestive of carotid occlusion and in many cases probably represent the clinical expression of a low-perfusion state. The choice of the proper diagnostic tools is important in confirming the presence of ICA occlusion and ruling out a high-grade stenosis (pseudo-occlusion), which often may require a different therapeutic approach. To date, intra-arterial angiography remains the procedure of choice.
Based on the available literature, a diagnosis of ICA occlusion probably carries a prognosis similar in terms of morbidity and mortality to less severe carotid lesions; however, the outcome may be slightly worse in patients with bilateral ICA occlusions.

The first line of treatment in patients with symptomatic ICA occlusion with no or minor deficits should probably be medical. The control of risk factors and the use of aspirin in this setting would seem reasonable options, although newer medical therapies now under investigation may provide added benefit or alternatives. Although subgroups of patients with ICA occlusion presenting with TIAs or minor strokes may show some improvement in certain hemodynamic and/or metabolic functions after surgical revascularization, the question still remains whether these improvements are sufficient to reduce the risk of subsequent cerebral infarction and to improve the overall outcome in these patients.

In the context of acute carotid artery occlusion, certain therapeutic approaches, including the use of fibrinolytic agents and thromboendarterectomy, may prove beneficial and should be further assessed.

References


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