Ulcerated Atheromatous Plaques of the Carotid Artery Bifurcation

BY J. B. GOMENSORO, M.D., V. MASLENIKOV, M.D., J. A. DE BONI, M.D., G. LAGUARDIA DE PEREZ, M.D., J. PURRIEL, M.D., J. MEDOC, M.D., R. RODRIGUEZ BARROS, M.D., J. C. ABO, M.D., A. TENYI, M.D., AND N. AZAMBUJA, M.D.

Abstract: Ulcerated Atheromatous Plaques of the Carotid Artery Bifurcation

The clinical diagnosis of an ulcerated plaque at the bifurcation of the common carotid artery is based on: mid-carotid bruit, cholesterol or platelet emboli in the retina, transient ischemic attacks particularly with amaurosis fugax, and a reversible neurological deficit.

In this series, thrombosis in situ was the final phenomenon in the natural history of the ulcerated plaque.

In certain unusual instances it appears that the pathogenesis of a transient ischemic attack is a decrease in cardiac output (fall in systemic blood pressure) leading to a disproportionate decrease in cerebral blood flow in that portion of the arterial system distal to a pathologically narrowed artery.

The ultimate diagnosis of an ulcerated plaque depends upon the radiological description at arteriography and upon direct visualization by the surgeon.

Surgical treatment at the present time is the therapy of choice.

Ulcerated carotid plaques may be bilateral, and, occasionally, are found at other levels than the bifurcation of the common carotid; however, ulcerated plaques were not seen in the intracranial vessels.

Attention has been directed to atheromatous plaques in the first portion of the internal carotid artery as a potential source of retinal and cerebral emboli. These emboli are important in the pathogenesis of transient ischemic attacks (TIA). These may be cholesterol emboli, from the atheromatous portion of a plaque entering the circulation after there has been a break in the endothelium of the artery, or platelet-fibrin emboli which may arise from a variety of sources including the heart, aorta, cervical arteries, etc.

The current practice is to describe stenotic lesions in the internal carotid artery in terms of the degree of stenosis in percent and the length of the lesion in millimeters. An additional criterion is the character of the lesion, that is, ulcerated, smooth or rough. In 1963, Carlevaro and Azambuja described the radiological appearance of ulcerated plaques of the carotid arteries. Since 1966 systematic observations of the retina (preradiologically, postradiologically, and surgically) and careful neurological examinations have been correlated with the pathological description of the atheromatous plaques obtained at the time of surgical procedure or autopsy. The purpose of this paper is to evaluate the clinical and radiological methods of diagnosis, to further clarify the criteria for the diagnosis of ulceration of atherosclerotic plaques in the carotid arteries and to correlate and corroborate all of these data with the degree and character of the pathological lesions studied at the time of surgery or autopsy.

Methods

Thirty-seven patients admitted and studied at the Neurological Institute of Montevideo, Uruguay, constitute the basis for this report. Each patient had: clinical and neurological examinations, ophthalmological examination including tonography, electroencephalography, photoplethysmography, vertebral and carotid angiography, thromboendarterectomy (27 patients), subsequent study of 30 surgical specimens, and follow-up examination at six-month intervals.
ULCERATED ATHEROMATOUS PLAQUES

Results

Neurological signs on admission to the Neurological Institute were: (a) hemiparesis, 30 patients, (b) hemiparesthesias, seven patients, (c) dysarthria, six patients, (d) aphasia, six patients, (e) mental deterioration, five patients, and (f) convulsive seizures, one patient.

The ocular phenomena were: (a) five patients had unilateral transient blindness, and three of these had cholesterol and platelet-fibrin emboli while two had cholesterol emboli only (fig. 1), and (b) two patients had retinal ischemia.

Twenty-two patients had systemic hypertension, 32 had retinal atherosclerosis, four had diabetes, and nine had peripheral vascular disease.

Carotid arterial bruits were present in 21 patients, bilateral in 10 and unilateral in 11. These bruits were associated with arterial stenosis of less than 50% of the lumen in six instances, 50% to 89% of the lumen in 22, and 90% to 99% of the lumen in three.

Twenty-seven patients had four-vessel angiography, three patients had three vessels and in seven patients bilateral carotid studies were done. Thirty-nine ulcerated plaques were radiographically diagnosed in 34 patients. Twenty-two of these were in the left carotid artery and 19 in the right carotid artery. Five patients had solitary ulcerated atherosclerotic plaques, while among 32 patients the lesions were distributed as follows: occlusion of the internal carotid artery in eight patients, and stenosis in 24 patients. Associated stenotic lesions were observed in the vertebrobasilar system in 50% of the patients.

Clinical Status

There were four general categories of clinical presentation. These were: (1) transient ischemic attacks, 24 patients, (2) cerebral infarction (completed stroke), eight patients, (3) two patients on admission and three additional cases in long-term follow-up developed “atherosclerotic encephalopathy,” and (4) four patients were asymptomatic, carotid bruits constituting evidence of extracranial cerebrovascular disease.

Follow-Up

Follow-up observations ranged from one year to five and one-half years. Fifteen of the 27 patients treated with surgery were asymptomatic, while half of the ten patients medically treated were asymptomatic. Eight patients died: five with “atherosclerotic encephalopathy,” two during the immediate postoperative period, and one with cerebral infarction secondary to thrombosis of the internal carotid artery.

Angiographical Description of the Ulcerated Atheromatous Plaque

In the present material the radiographical findings are as follows: (a) less than 30% stenosis, eight plaques; 30% to 49% stenosis, 18 plaques; 50% to 89% stenosis, seven plaques; and one patient with radiological image of occlusion overlying an ulcerated plaque, and (b) the length of the atheromatous formation was 1 to 2 cm.

Three types of ulcerated lesions were observed. The first of these is an ulcerated plaque in the form of a sac or saccule which consists of a true cavity communicating with the arterial lumen through an aperture. The second was a rough ulcerated lesion with an irregular surface as apparent in figures 2 and 3. The third type was a smooth lesion appearing as a depression in the atheromatous surface with regular limits.

Pathological Changes of the Atheromatous Plaque

Twenty-four patients had plaques with an ulcer crater and in many instances friable thrombotic material was noted by the surgeon. These plaques were at the origin of the internal carotid artery. In 12 instances the ulcerated plaque was small, requiring careful observation by the vascular surgeon for detection but easily apparent to the pathologist.
Regardless of the scheme used to classify these arterial lesions, it is most important to conceive of each lesion as having the potential for change. Thus, a smooth plaque may remain quiescent or enlarge to create stenosis, may become rough and develop thrombus on the surface, may ulcerate or may heal. The traditional pathologist has been inclined to view each lesion as a static one, whereas clinical experience supports the concept that a lesion may take a variety of forms.

Figure 4 shows the evolution and metamorphoses of the plaque. The right column represents the clinically active lesions while the left column shows those mainly inactive.

Discussion
Clinical diagnosis of an ulcerated atheromatous plaque of the internal carotid artery is mainly dependent upon the detection of emboli in the ipsilateral retina. When an embolus is identified, the viewer cannot be certain about the time of occurrence of the embolic event; the embolus may have occurred weeks or months earlier. Re-inspection of that fundus may settle this issue—new emboli signify that the parent atheromatous lesion is currently active. Cholesterol and fibrin-platelet emboli may move and subsequently disappear. This phenomenon may explain the 9.5% frequency of retinal emboli in the carotid disease reported by Hollenhorst.4

Attacks of amaurosis fugax may imply the lodging of emboli and are almost solely associated with ipsilateral carotid lesions; however, the carotid lesion may not be an ulcerated one. Certain bruits heard over the origin of the internal carotid artery indicate carotid stenosis, but may or may not be associated with ulceration. The arteriographical findings often become important in finally establishing the diagnosis of ulceration of the plaque.

Treatment
If ulceration of a carotid atheromatous plaque is demonstrated, the treatment is surgical reconstruction of the artery, even though the patient is asymptomatic. If there is carotid stenosis, without ulceration, surgery may or may not be chosen, depending on a constellation of factors which include: (a) the presence or absence of neurological symptoms in the appropriate vascular distribution, (b) the quality of brain function (if the patient is hemiplegic, there is nothing to be gained by the surgery), (c) the status of the general cardiovascular system, and (d) the presence or absence of life-threatening systemic disease. Patients with transient ischemic attacks and a carotid lesion,
UlcERATED ATHEROMATOUS PLAQUES

**Figure 3**

Large right internal carotid artery ulcerated plaque, longer than usual (approximately 3 cm in its length).

UlcERATED or nonulcerated, make up the principle category where such surgery is indicated. In instances where there is severe stenosis produced by the plaque, thrombosis may begin and this early thrombus may constitute the source of embolic fragments.

**References**

Ulceraed Atheromatous Plaques of the Carotid Artery Bifurcation

Stroke. 1973;4:912-916
doi: 10.1161/01.STR.4.6.912

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1973 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/4/6/912

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org/subscriptions/