Computed Tomographic Study of Cervical Carotid Calcification

Antonio Culebras, MD, Carlos Otero, MD, José R. Toledo, MD, and Bruce S. Rubin, MS III

Contrast-enhanced computed tomography of the neck permits low-risk evaluation of morphologic complications of carotid plaque, including (and foremost) calcification. To investigate the patterns and clinical significance of calcified deposits in the cervical carotid arteries using computed tomography we studied 40 patients with unilateral symptoms of hemispheric ischemia. Calcium deposits observed in the external, internal, and common carotid arteries were oriented in space and individually scored. We found calcified deposits in 39 patients. Stepwise multiple regression of the data provided evidence strongly suggesting a correlation between advancing age and calcium scores. Calcium was more heavily concentrated in the posterior half of arteries (p<0.01), in particular of the internal and common carotid arteries, and always in relation to an atheromatous plaque, suggesting a causative link to hemodynamic forces within the arteries. There was no difference in calcification scores between symptomatic and asymptomatic sides, indicating that calcium deposits do not contribute to the development of symptoms. (Stroke 1989;20:1472–1476)

Contrast-enhanced computed tomography (CT) of the neck permits low-risk evaluation of morphologic changes of the carotid plaque, including calcification.1,2 Standard radiographs of the cervical region show occasional ill-defined images, suggestive of calcium deposits that are difficult to localize and evaluate. Definition improves in conventional angiographic studies of the cervical carotid arteries, but images of calcified deposits remain unsatisfactory for systematic evaluation. On the other hand, CT studies of the neck performed according to the technique prevalent in our medical center and described before1 can reveal clear and well-defined images of calcified deposits in arterial walls that allow precise anatomic localization and analysis. With this method, we investigated the patterns and clinical significance of mural calcified deposits in the cervical carotid arteries of patients with symptoms of hemispheric ischemia.

Subjects and Methods

We reviewed CT scans of the neck in 40 consecutive patients (average age 63.4, range 42–82 years) for the evaluation of symptoms of hemispheric ischemia occurring within the preceding 3 months; we excluded patients with bilateral events. All 40 patients were studied with CT of the head, and most were also studied with conventional cerebral angiography and duplex ultrasonography.

A GE 9800 scanner (Milwaukee, Wisconsin) and a Picker 1200 SX scanner (Pittsburgh, Pennsylvania) were used to obtain high-resolution images of the neck between the C2 and C6 vertebral bodies. Dynamic scans were taken after an intravenous bolus injection of 100–150 ml of 60% meglumine iothalamate (Conray 60, Mallinckrodt Inc., St. Louis, Missouri). Overlapping 5-mm-thick CT sections were obtained in rapid succession in the axial plane at automatic table increments of 2.5–3.5 mm. Scanning time was 9.6 seconds at 160 mA/sec, and interscanning time was 1–2 seconds. The average calculated radiation dose to the skin was 2.3 rads.

Calcification was defined as a discrete, well-circumscribed region in the carotid wall that appeared whiter than (hyperdense to) the surrounding parenchyma and contrast-enhanced lumen (Figure 1). Calcified deposits were optimally (although not exclusively) demonstrated in images obtained with wide window settings (Figure 2). The presence of calcium deposits was analyzed in the common, internal, and external carotid arteries of both sides. The segment of neck studied always included the carotid bifurcation. Calcium deposits were oriented in the anatomic plane and were scored according to their morphologic characteristics as punctate, 1 point; quadrantic, 9 points; semilunar, 18 points; and circumferential, 36 points (Figure 3). Each arterial circumference was divided in the anterolat-
FIGURE 1. Computed tomogram of cervical region showing near-circumferential calcification of distal left common carotid artery (CC) in 65-year-old man with left hemispheric stroke. Contrast material fills lumen of neck vessels. CA, calcified arterial wall; LM, contrast-enhanced lumen; IJ, internal jugular vein; EJ, external jugular vein; SCM, sternocleidomastoid muscle.

eral, anteromedial, posterolateral, and posteromedial quadrants. Calcification points were prorated by quadrants to allow accurate scoring of lesions in which calcium deposits occupied more than one quadrant. The plane of the CT section showing the maximum amount of calcium was selected for final scoring of each artery. If calcium invaded \( \geq 50\% \) of a quadrant, a full score of 9 points was accorded; \(<50\% \) occupation received a score of 1 point for each spot of calcification within the quadrant. A

FIGURE 2. Computed tomogram of same structures as in Figure 1, shown with wide window setting ("bone window") to optimally reveal calcium deposits in arterial wall.
magnifying lens was used to identify all calcified vascular lesions on CT scans. The highest possible score for each artery was 36 points, and thus the maximum possible score for each patient was 216 points (36×6).

Mural lucencies, stenoses, and ulcerations were also identified and measured on CT scans obtained with narrow window settings, in accordance with methods and definitions previously established.1,2 Information relative to CT and clinical findings was fed into a computer program for stepwise multiple linear regression analysis of laterality of symptoms. This method identified variables that had the best mathematical fit with laterality of symptoms and was used to investigate the potential contribution of calcified deposits to the development of symptoms of hemispheric ischemia.

Results

We identified calcium deposits in 39 patients. The highest score was 117 points in a 60-year-old man with a history of inveterate smoking (138 pack-years) and one episode of transient ischemia on the left side; in this patient the heaviest concentration of calcium was found in the internal carotid arteries (circumferential bilaterally), which exhibited >75% lumen reduction. The only patient without calcium deposits was a 42-year-old woman with early evidence of atheroma formation in the internal carotid arteries (<50% lumen reduction) who had suffered a right hemispheric stroke.

Calcium deposits were abundant in the distal 1-cm segment of the common carotid artery and were consistently most abundant in the proximal 1-cm segment of the internal carotid artery; however, the difference between these two arteries was not significant. The external carotid artery was seldom calcified, and deposits in this artery did not occur without accompanying calcification in other arteries. The pattern of calcification between the left and right arteries was remarkably similar and was independent of their situation in the asymptomatic or symptomatic side. In fact, when the sums of the scores for the asymptomatic and symptomatic sides were compared, a close similarity also emerged (Figure 4).

The most characteristic pattern was that of calcified deposits in the distal 1 cm of the common carotid artery, extending into the bifurcation and invading the proximal 1 cm of the internal carotid artery. Calcium scores were higher in the posterior than in the anterior half of the carotid arteries (p<0.01, two-tailed t test; Figure 5), but there was no difference between the medial and lateral halves.

Linear regression analysis of the data indicated that calcification was more commonly associated with severe luminal stenosis (>75% reduction) (Figure 6) than with any other tabulated morphologic

---

**Figure 3.** Composite figure to show computed tomograms of four gradations of arterial mural calcification (arrows). A, punctate; B, quadrantic; C, semilunar plus quadrantic; D, circumferential.
complication of the plaque (mild stenosis [\(<50\%\) reduction], moderate stenosis [50–74\% reduction], mural lucency, and ulceration). As in previous studies,\(^2\) we found more instances of severe luminal stenosis of the carotid plaque in the symptomatic side (22) than in the asymptomatic side (15). Calcification was always observed in arterial segments in which atheromatous plaques usually occur and was commonly found in relation to other evidence of plaque formation such as thickening of the wall, any degree of stenosis, an irregular lumen, and a lucent defect.

Data analysis also indicated that calcium deposits did not contribute independently to the occurrence of symptoms, nor did deposits add risk to their appearance even when other morphologic complications were taken into account. Cigarette pack-years were not positively correlated with calcium scores. On the other hand, data analysis strongly suggested a direct correlation between advancing age and calcium scores in the carotid arteries.

Discussion

Our study shows that calcification appears in carotid atheromatous plaques that are presumably active, although in itself this histologic complication may not contribute to the occurrence of symptoms of hemispheric ischemia. Calcification also occurs in inactive plaques contralateral to symptomatic carotid territories. In CT scans of the cervical region, calcification is a useful marker for the presence and location of a carotid plaque. Our study also shows that calcium is more heavily deposited in arteries with severe luminal stenosis and in the posterior half of the common and internal carotid arteries, in the 1-cm segment adjacent to the bifurcation. Furthermore, our study provides evidence strongly suggesting a direct correlation between advancing age and deposition of calcium in the carotid arteries.

Calcification is a histologic complication in the evolution of the atheromatous plaque; other complications are mural hemorrhage, ulceration, thrombosis, stenosis, and occlusion. Clinical manifestations of hemispheric ischemia appear when the carotid plaque becomes complicated, and each complication (except calcification) contributes independently to the risk of symptom development.\(^2\)\(^-\)\(^7\)

Histologic studies have shown that in the carotid siphon calcium is deposited in the elastic lamina, the thickened subintima, and the inner part of the media.\(^8\) Fisher et al\(^8\) found that only one or a few segments of the circumference were commonly involved, but occasionally a complete ring of calcification was found around the artery in the siphon. These authors further observed that shallow plaques were more heavily calcified than thick ones and that the thickest segment of a plaque was not usually calcified, whereas the adjacent thin rim of atherosclerosis might be. This observation led the authors to suggest that calcification limits the growth of a plaque. In that study\(^6\) there was a strong quantita-
tive parallel between calcification in the carotid siphon and advanced atherosclerosis in the carotid sinus, whereas calcification in the carotid sinus was always associated with atherosclerosis there. Fisher et al also found that the incidence and severity of carotid siphon calcification increased with age, an observation in keeping with ours of a probable link between advancing age and calcium deposition in the cervical carotid arteries.

The metabolic and hemodynamic determinants of calcium deposition in the carotid arteries remain unknown. Plaques are usually thickest near the midpoint of the lateral carotid sinus wall, opposite the point where shear stress is highest. Low shear facilitates disruption of laminar blood flow and favors margination and attachment of formed blood elements. In arterial sectors in which shear forces are low and the blood flow forms secondary vortices, a delay in particle clearance leads to an increased residence time of particles, with increased exposure to atherogenic agents, delay in renewal of nutritive compounds, and even hypoxia. The lateral carotid sinus wall has been shown to be a low-shear sector and one of the arterial segments most vulnerable to the formation of focal atherosclerosis. Histopathologic studies revealing that calcium tends to be deposited, not within the thickest segment of the plaque, but in the adjacent rim are in concordance with our results indicating that the heaviest calcium deposition occurs not in the lateral wall but in the posterior half of the vascular circumference. Preservation of laminar blood flow within the external carotid artery protects this vessel from the formation of plaques and from secondary calcification. The distal common carotid artery appears to be less vulnerable than the proximal internal carotid artery to the formation of plaques and to secondary calcification.

Although calcium deposition as an independent complication does not contribute to symptoms according to our results, calcium deposition has the potential to participate in embolic events as has been observed in episodes of amaurosis fugax in patients with calcific aortic stenosis. The presence of calcium deposition in the carotid plaque should nevertheless be acknowledged and tallied as an independent variable in studies correlating carotid plaque with the risk of occurrence of hemispheric symptoms since the natural history of this complication of atheroma formation remains largely unknown.

The development of therapeutic modes other than crude excision of the carotid sinus plaque, such as laser therapy and angioplasty, may necessitate the detailed preoperative evaluation of all plaque complications, including calcium deposition and its location within the carotid wall. CT of the cervical carotid arteries is the only method available to image calcified deposits and their anatomic relation to other plaque complications.

Acknowledgment

The authors gratefully acknowledge the assistance of Paul R. Sheehe, ScD, Professor of Preventive Medicine, State University of New York Health Science Center at Syracuse.

References


Key Words • carotid artery diseases • tomography, x-ray computed • calcium
Computed tomographic study of cervical carotid calcification.
A Culebras, C Otero, J R Toledo and B S Rubin

Stroke. 1989;20:1472-1476
doi: 10.1161/01.STR.20.11.1472

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/20/11/1472