Computed Tomography of the Cervical Carotid Artery: Significance of the Lucent Defect

Antonio Culebras, MD, Rafael Magaña, MD, and Edwin D. Cacayorin, MD

To investigate the clinical significance of lucent defects in computed tomography (CT) scans of the cervical carotid artery plaque, we studied 95 patients with recent symptoms of hemispheric ischemia. Using multiple linear regression analysis, we estimated the strength of the association of symptoms with laterality of carotid artery lucent lesions, stenoses, and ulcerations observed in CT scans. Hemispheric symptoms correlated strongly with ipsilateral carotid lucent lesions (partial $p<0.025$) and with ipsilateral severe ($>75\%$) carotid stenosis (partial $p<0.025$). Carotid artery ulcerations had a weaker association (partial $p<0.1$), and stenoses of mild and moderate degrees showed none. The overall performance of all three plaque complications was highly significant ($p<0.005$). The lucent defect indicates a morphologic change in the carotid plaque that plays an important role in the development of symptoms of hemispheric ischemia. This is in agreement with the notion that the lucent defect is the image of intraplaque hemorrhage and/or necrosis, which are complications central to the development of symptomatic carotid disease. (Stroke 1988;19:723–727)

High-resolution computed tomography (CT) of the cervical region permits a low-risk evaluation of the carotid vessels and atheromatous plaque formations, including intramural and extramural histopathologic plaque complications. In contrast-enhanced cervical CT images, carotid vessels are located immediately lateral and posterior to the pharyngeal cavity and medial to the sternocleidomastoid muscle (Figure 1). The internal carotid artery is usually found lateral and posterior to the external carotid, although occasional variations occur. In normal individuals, CT images of the vascular wall merge with the image of the contrast-filled lumen and are not observed as a distinct structure. Carotid atheromatous plaques are identified by the irregular, often constricted and commonly calcified contour of the vascular wall, encircling a contrast-filled lumen. Irregularities are more prominent at the carotid bifurcation or in the proximal segment of the internal carotid artery. In CT images of the cervical carotid plaque, it is possible to identify extramural morphologic changes such as stenosis, ulceration, and occlusion, as well as intramural lesions manifested by hyperdense changes indicative of calcification, and lucent, hypodense defects (Figures 2 and 3). Lucencies indicate an intraplaque histologic change that can be observed only by CT. They are suggestive of arterial subintimal hemorrhage or plaque necrosis, which are both complications that play an important role in the development of cerebral ischemia. Should that be the case, plaque lucencies could serve as markers or predictors of risk of stroke.

From the Departments of Neurology (A.C., R.M.) and Radiology (E.D.C.), State University of New York Health Science Center and the Veterans Administration Medical Center, Syracuse, New York.


Address for reprints: Antonio Culebras, MD, Neurology Service (127), VAMC, 800 Irving Avenue, Syracuse, NY 13210.

Received September 14, 1987; accepted December 4, 1987.
increments of 2.5–3.5 mm. Scanning time was 9.6 seconds at 160 mA/sec, and interscanning time was between 1 and 2 seconds. The average calculated radiation dose to the skin was 2.3 rad.

The following definitions of CT scan images were adhered to. Lucent lesion (Figures 2 and 3): a discrete, well-defined region in the carotid wall that appears darker than (hypodense to) the surrounding parenchyma. Lucencies may be punctate, linear, semilunar, or circumferential. Punctate lucencies measuring ≤1 mm in diameter and marginally hypodense lucencies regardless of their size were not tabulated as lucent defects. Stenosis (Figures 2 and 3): a reduction in the cross-sectional diameter of the carotid artery lumen expressed as percent stenosis (residual plaque lumen/carotid artery diameter × 100). Stenoses were subdivided in three groups: mild, 0–49%; moderate, 50–74%; and severe, 75–99%. There is close correspondence between severity of luminal compromise seen on cervical CT and arteriographic findings. Ulceration: an outpouching of contrast material adhered to. Lucent lesion (Figures 2 and 3): a discrete, well-defined region in the carotid wall that appears whiter than (hyperdense to) the surrounding parenchyma and contrast-enhanced lumen. Lucencies, stenoses, and ulcerations were identified and measured in CT scans obtained with narrow window settings, whereas calcifications were best demonstrated with wide window settings. CT studies were reviewed and reported by neuroradiologists experienced in reading cervical carotid scans. 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 identifies variables in a stepwise manner that have the best mathematical fit with laterality of symptoms.

Results

Discrete lucent defects in the carotid plaque varied in size and shape from a linear to a semilunar or circumferential defect (Figures 2 and 3). Lucent lesions were commonly associated with calcium deposits and luminal compromise (Figures 2 and 3). In 95 carotid arteries appropriate to hemispheric symptoms, there were 31 lucent defects, 86 stenoses (mild in 43 arteries, moderate in 22, and severe in 21), and 13 ulcerations (Figure 4). In 95 asymptomatic carotid arteries, there were 12 lucent defects, 75 stenoses (mild in 56, moderate in 12, and severe in 7), and 7 ulcerations. The correlation between symptoms and ipsilateral lucent lesions was nominally significant (partial $p<0.025$) and equal in strength to that of symptoms and ipsilateral severe carotid artery stenosis (partial $p<0.025$). Ulcerations had a weaker association (partial $p<0.1$), and stenoses of mild and moderate degrees showed none. The partial $p$ value for lucent defects and severe carotid artery stenosis indicates that these complications added individually predictive value to the development of symptoms. The overall performance of all three plaque complications was highly significant ($p<0.005$). While positively correlated with each other, the plaque complications studied were not highly correlated, the correlation coefficient being $\leq 0.207$.

Discussion

The lucent defect observed in contrast-enhanced CT images of cervical carotid artery plaques manifests a morphologic change within the body of the plaque. In a previous study, we investigated the nature of lucent defects by histologically studying 15 surgical carotid artery specimens from symptomatic patients whose CT had shown a lucent defect on the side appropriate to the symptoms. In 13 patients, we found hemorrhagic material of varying age in the midst of thickened, lipid-laden plaques. In one specimen there was necrosis of the plaque, and in another there was prominent intimal thickening. We suggested that lucent defects demonstrated by CT in carotid artery walls represent subintimal hemorrhage, focal necrosis, excessive subintimal thickening due to lipid accumulation, or a combination of these abnormalities with hemorrhage predominating. According to the study by Leeson et al., markedly lucent defects most likely manifest intramural hemorrhage, whereas the nature of small, isodense to mildly hypodense focal mural thickening is likely fibrolipid deposition without excluding a component of hemorrhage. It remains to be determined why hemorrhagic debris, thrombus, and plain hemorrhage appear hypodense in CT images of the arterial wall.

Figure 1. Computed tomogram of cervical region at level of carotid bifurcation showing configuration of normal structures and their relations. Contrast material fills lumen of neck vessels. EJ, external jugular vein; SCM, sternocleidomastoid muscle; IJ, internal jugular vein; IC, internal carotid artery; EC, external carotid artery; CC, common carotid artery.
FIGURE 2. Computed tomogram of cervical region in neurologically asymptomatic patient. Top: Complicated plaque in proximal internal carotid artery with semilunar lucent defect in posterolateral segment (arrows), bordered posterolaterally by calcification (arrowheads) and causing moderate stenosis of lumen (open arrow). IJ, internal jugular vein; SCM, sternocleidomastoid muscle; EJ, external jugular vein. Bottom: Corresponding conventional angiogram shows moderate luminal stenosis of proximal internal carotid artery secondary to atheromatous plaque formation (arrows).

However, these are consistent with CT images of extracranial carotid dissecting hematoma and of recent occlusion of the carotid lumen, which also appear hypodense in cervical CT scans.

Carotid plaque lucent defects are observed in both symptomatic and asymptomatic arteries. In previous studies, we showed that lucencies of any kind, whether punctate, semilunar, or circumferential, and whether marginally or markedly hypodense, were present in 72% of symptomatic carotid arteries and in 38% of
contralateral asymptomatic arteries. With the application of more strict criteria in the present work and with exclusion of lucencies measuring ≤1 mm in diameter or lucencies only marginally hypodense to avoid simple fibrolipid plaque lesions, as suggested by Leeson et al, percentages dropped to 32% and 12%, respectively. The stepwise multiple linear regression analysis of the data in this study demonstrated a strong association between lucent defects and the development of appropriate symptoms of hemispheric ischemia (partial $p < 0.025$). This finding is in agreement with the idea that the lucent defect is the image of the

FIGURE 3. Computed tomogram of carotid artery in same patient as in Figure 2 two years later, shortly after developing symptoms of appropriate hemispheric ischemia. Top: Larger lucent defect (arrows) suggestive of hemorrhage, causing more severe stenosis (open arrow). Calcified posterolateral segment has also increased (arrowheads). U, internal jugular vein; SCM, sternocleidomastoid muscle; EJ, external jugular vein. Bottom: Corresponding conventional angiogram shows possible increase of stenosis (arrows) but gives no information on arterial intramural developments.
intraplaque hemorrhage, a leading complication in the development of symptomatic carotid disease. The lucent defect exhibits a weighted association with appropriate hemispheric symptoms similar to that of severe carotid artery stenosis observed in the same CT scans. In a recent study, Chambers and Norris indicated that severe carotid artery stenosis with a luminal compromise of >75%, measured with ultrasonography, is a powerful predictor of cerebral ischemic events in asymptomatic patients. However, in that study, 14 of 36 patients eventually became symptomatic in the absence of an appropriate severe carotid stenosis. This suggests that factors other than severe stenosis contribute to the development of symptoms, an idea also suggested by our study in which only 23% of patients with symptoms had severe stenosis. Since the lucent defect shows a weighted association with symptoms similar to that of severe carotid artery stenosis, it should therefore be interpreted as a previously ignored intramural factor contributing individually to symptoms, with a weight similar to that of severe stenosis.

In neurologically asymptomatic patients, the presence of lucent defects in CT images of the cervical carotid artery suggests a stage in the evolution of the carotid plaque that might be close to that of precipitation of symptoms. Prospective studies using CT scanning of the asymptomatic cervical carotid plaque should determine the value of the lucent defect as a predictor of outcome and a possible marker of risk for transient ischemic attack or stroke development. Thus, it could help identify a subset of asymptomatic patients at higher risk of stroke in whom a surgical modality of treatment, such as carotid endarterectomy, might be preferable. Furthermore, if the lucent defect indicates the presence of a plaque hemorrhage, the prophylactically intended, but highly empirical and scientifically unproven, practice of recommending aspirin therapy to asymptomatic individuals with carotid atheromatous lesions is likely contraindicated because aspirin could promote intraplaque hemorrhaging and plaque growth.

CT imaging of the cervical carotid artery is a relatively noninvasive and risk-free method of serially studying the carotid plaque. It might assist in obtaining data about the natural history of carotid artery lesions on which to base treatment options, which are lacking at the present time. Particularly pertinent to clinical decision-making is information about the intramural stage of evolution of the plaque and the presence of factors contributing to the development of symptoms, such as the lucent defect. This information might be attainable only by CT because ultrasonography and other noninvasive methods, while exposing severe stenosis, fail to image lucent defects. In addition, CT scanning of the extracranial carotid arteries can serve as a sophisticated and cost-efficient screening procedure for the selection of patients for conventional cerebral angiography.

Acknowledgments

The authors gratefully acknowledge the assistance of Paul R. Sheehe, ScD, Professor of Preventive Medicine, and William J. Mueller, Associate Professor of Bioelectronics and Computer Sciences, State University of New York Health Science Center at Syracuse.

References


Key Words • tomography, x-ray computed • carotid artery disease

FIGURE 4. Bar graph of lateralization of carotid plaque complications relative to asymptomatic and symptomatic sides. Numbers over boxes are number of complications found in computed tomograms.
Computed tomography of the cervical carotid artery: significance of the lucent defect.
A Culebras, R Magaña and E D Cacayorin

*) doi: 10.1161/01.STR.19.6.723

Stroke. 1988;19:723-727

Copyright © 1988 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/19/6/723

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/