
Carotid calcification on panoramic radiographs: An important marker for vascular risk

Stanley N. Cohen, MD,^a Arthur H. Friedlander, DDS,^b Desmond A. Jolly, BA,^c and Lesley Date, BA,^c Los Angeles, Calif

CEDARS-SINAI MEDICAL CENTER, UCLA SCHOOL OF MEDICINE, UCLA SCHOOL OF DENTISTRY, AND VA WEST LOS ANGELES HEALTHCARE CENTER

Objective. The objective of this study was to determine whether carotid calcifications are harbingers of future vascular events.

Study design. Between 1986 and 2000, 71 patients were found to have carotid artery calcifications on routine panoramic films. Medical records were reviewed for vascular risk factors existing before and vascular end points subsequent to the radiographs.

Results. The mean age of our patients was 68 years. Sixty-one (86%) had preexisting vascular risk factors, 73% with multiple risk factors. Forty-one end points occurred in 29 patients. The average time to an end point was 2.7 years. The end points included myocardial infarction (8, 11%), stroke (5, 7%), death (11, 15%), revascularization procedures (8, 11%), transient ischemic attack (2, 3%), and angina (7, 10%). Twenty-three patients (34%) had major end points of myocardial infarction, stroke, or death.

Conclusions. Carotid calcifications identified on panoramic radiographs are powerful markers for subsequent vascular events. Patients found to have carotid calcification on panoramic radiographs should be referred for cerebrovascular and cardiovascular evaluation and aggressive management of vascular risk factors.

(*Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2002;94:510-4)

Currently, there are 4.6 million Americans suffering the effects of an ischemic stroke and 12.6 million living with the ravages of coronary artery disease.¹ In 1998 almost 960,000 Americans died of these vascular disorders.¹ Common to both disorders is the atherosclerotic process of plaque formation in which fatty substances, cholesterol, platelets, cellular waste products, and calcium are deposited in the inner lining of the artery. The presence of atheromatous plaque in the carotid artery of clinically asymptomatic individuals is often associated with the later development of clinically evident cerebrovascular disease (transient ischemic attack [TIA] and stroke), coronary artery disease (angina and myocardial infarction [MI]), and death.²⁻⁴ Calcium deposits have been identified in the carotid arteries in a majority of patients with symptomatic cerebrovascular disease by using computed tomography.⁵ Calcium deposits have also been noted on the plain radiographs

(eg, cervical spine) of patients with angiographically demonstrated carotid atheromatous disease.⁶

Calcifications in the cervical carotid arteries were found on the panoramic radiographs of 3% to 4% of patients older than 55 years who were free of neurologic symptoms (Figs 1 and 2).⁷⁻⁹ They may appear as nodular radiopaque masses or radiopaque vertical lines inferior or posterior to the angle of the mandible. Calcifications in the carotid arteries must be distinguished from other radiopacities in this area such as the hyoid bone, epiglottis, stylohyoid ligament, submandibular gland sialolith, phleboliths, triticeous cartilage of the lateral thyrohyoid ligament, superior cornu of the thyroid cartilage, and cervical lymph nodes.¹⁰ These distinctions can be confidently made by using a combination of radiographic criteria and clinical examination.

Two case reports and 1 small study (10 patients) published in the dental literature have shown that carotid calcifications on panoramic radiographs are associated with highly variable degrees of stenosis.^{8,11,12} The results of large clinical trials published in the medical literature show general agreement that properly selected patients with 70% or greater carotid stenosis have a much better prognosis with carotid artery surgery, patients with 50% to 69% stenosis have only a slightly better prognosis with surgery, and those with less than 50% stenosis have a better prognosis without surgery.^{13,14} The extent of stenosis of most patients with carotid calcifications on their panoramic radio-

^aDirector, Clinical Stroke Program, Cedars-Sinai Medical Center, Professor of Neurology, UCLA School of Medicine.

^bAssociate Chief of Staff/Graduate Medical Education, Professor In Residence, Oral and Maxillofacial Surgery, UCLA School of Dentistry.

^cStroke Service, VA West Los Angeles Healthcare Center.

Received for publication Oct 31, 2001; returned for revision Feb 12, 2002; accepted for publication Mar 4, 2002.

© 2002, Mosby, Inc.

1079-2104/2002/\$35.00 + 0 7/16/125580

doi:10.1067/moe.2002.125580



Fig 1. A standard panoramic radiograph that has been scanned and digitized to enhance the reader's visualization of the atherosclerotic process. The patient is a 53-year-old man with calcified carotid plaques visible in the right neck below the hyoid bone and in the left neck overlying the hyoid bone (*arrows*).



Fig 2. A standard panoramic radiograph that has been scanned and digitized to enhance the reader's visualization of the atherosclerotic process. The patient is a 69-year-old man with calcified carotid plaque in the right neck overlying the hyoid bone and in the left neck overlying and superior to the hyoid bone (*arrows*).

graphs remains unknown as does their prognosis for cerebrovascular and cardiovascular outcomes. Therefore, we retrospectively studied the risk of symptomatic vascular disease and death subsequent to the identification of carotid calcification on panoramic radiographs.

METHODS

During the years 1986 to 2000 a random sample of 2000 radiographs was collected from male outpatients older than 55 years who were being treated by the dental service at the Sepulveda campus of the VA Greater Los Angeles Healthcare System. Eighteen hundred seventy-nine ($n = 1879$) of these radiographs were of good image quality (not overexposed or underex-

posed) and showed an area of interest that extended 1.5 cm inferior and 2.5 cm posterior to the cortical rim of the midpoint of the mandibular angle. Patients with either unilateral or bilateral carotid calcifications on these radiographs were identified.

All patients were radiographed through use of the Panelipse II (Gendex Corp, Des Plaines, Ill) panoramic x-ray system. The unit was operated at 4 mA; the peak kilovoltage ranged from 70 to 80 depending on the estimate of the subject's jaw size. Kodak Dental Film/Ektamat: DFG-5 (Kodak, Rochester, NY) was used. The exposed radiographs were processed according to the manufacturer's directions. An automatic developer (A/T 2000; Air Techniques Inc, Hickville, NY) was used.

Table I. Patient characteristics at the time of the panoramic radiograph

Risk factor	N (%)
History of MI	13 (18.3%)
History of stroke	6 (8.5%)
History of TIA	4 (5.6%)
History of angina	13 (18.3%)
History of CHF	3 (4.2%)
History of abnormal ECG	22 (31.0%)
Diabetes mellitus	16 (22.5%)
Hypertension	38 (53.5%)
Hyperlipidemia	26 (36.6%)
History of smoking	39 (54.9%)
Obesity (body mass index >30)	15 (21.1%)
Patients with multiple risk factors	52 (73.2%)

The radiographs were examined in subdued ambient light through use of transmitted light from a standard viewing box and a rheostat-controlled 75-W bulb ("hot" light) for the presence of calcified carotid artery atheromas.

Institutional Review Board approval was obtained to review charts of patients found to have carotid artery calcifications. Charts were reviewed for a history of preexisting vascular risk factors including history of MI, stroke, TIA, angina, congestive heart failure, diabetes mellitus, cigarette smoking, or electrocardiographic abnormality. Charts were also reviewed for evidence of preexisting hypertension (defined by a physician's diagnosis of hypertension), hyperlipidemia (defined by a physician's diagnosis of hyperlipidemia), or obesity (defined as a calculated body mass index greater than 30).

In addition, the charts were reviewed for end points occurring subsequent to the dental radiographs. End points included death from any cause, MI, a revascularization procedure (coronary artery bypass graft, coronary angioplasty, or carotid endarterectomy), angina, stroke, or TIA. End points occurring up until December 31, 2000, were included.

RESULTS

Carotid calcifications were identified on the panoramic radiographs of 71 male patients or 3.8% of the individuals in the sample of radiographs (N = 1879). The mean age of these patients at the time of their panoramic radiograph was 68 years. The average follow-up period was 3.6 years. Sixty-one of the patients (85.9%) were found to have vascular risk factors present at the time of their identifying radiograph. Multiple risk factors were identified in 73.2% of our patients. The patients' risk factor characteristics are summarized in Table I.

Table II. Number of patients with each end point

End point	No. of patients (%)
Major end points	
Death	11 (15.4%)
MI	8 (11.3%)
Stroke	5 (7.0%)
Total major end points	23 (32.4%)*
Other end points	
Revascularization (coronary angioplasty, coronary artery bypass graft surgery, carotid endarterectomy)	8 (11.3%)
TIA	2 (2.8%)
Angina	7 (9.9%)

*One patient had both stroke and death.

Twenty-nine patients (40.8%) had 41 end points during the follow-up period. A breakdown of the end points is summarized in Table II. Twenty-three patients (32.4%) had major end points of MI, stroke, or death (1 patient suffered both stroke and death). Twenty-six of the 61 patients (43%) with established vascular risk factors at the time of their radiograph went on to have an end point. Three of the 10 patients (30%) with no preexisting vascular risk factors had an end point during follow-up. The difference is not significant as determined by a chi-square test of independence.

DISCUSSION

Previous reports of carotid calcifications identified on panoramic radiographs made the association between the calcifications and underlying carotid atheromas, but their clinical significance was often not documented.⁸ Recently, Woodworth et al¹⁵ reported on the value of carotid calcifications as a predictor of vascular death in a homogeneous population of Native Americans of the Gila River Pima Indian community in Arizona. They found that these calcifications were a significant predictor of cardiovascular death.

Our study showed that 41% of patients with carotid calcifications had at least 1 end point, with a 15% death rate during an average 3.6-year follow-up. The average time to an event was 2.7 years.

Our study was a retrospective review of medical records. As such, it has problems inherent in retrospective studies. It is probable that we have not accounted for all of the end points in our patients because some of them received additional healthcare outside of the Veterans Affairs system, and it is possible that some of them may have suffered end points at an outside facility. Therefore, our finding of documented end points in 41% of patients should be considered a conservative estimate.

There are 3 potential explanations of why carotid

calcifications appear to carry such an ominous cerebrovascular and cardiovascular prognosis:

1. Carotid calcifications are a sign of underlying significant carotid stenosis.
2. Carotid calcifications are simply another indication of a heavy risk factor burden.
3. Carotid calcifications are an independent risk factor for cerebrovascular and cardiovascular disease.

Prior work reporting a similar population of 295 veterans (mean age, 64 years) without a history of cerebrovascular accident or TIA identified 10 individuals (mean age, 69 years) with a panoramic radiograph showing calcified carotid artery atheromas. Carotid duplex scanning of these 10 individuals showed that the underlying degree of stenosis ranged from only 25% to a rarely encountered 70%.⁹ Therefore, it is unlikely that most patients in this study with comparable carotid calcification would have had high-grade stenoses (greater than 70%). In addition, high-grade carotid stenoses would not explain the high ratio of angina and MI to TIA and stroke end points (37% vs 17%). Therefore, we believe it is unlikely that the poor prognosis is due to the calcifications being a sign of underlying significant carotid stenosis.

Our patients carried a heavy risk factor burden. Eighty-six percent of our patients had at least 1 established vascular risk factor, and 73% had more than 1. In another study of 444 male veterans with asymptomatic carotid stenosis, the risk factor burden was similar to that found in our patients.¹⁶ In that study the 4-year rate of MI, stroke, and death was 52%.³⁻⁴ In each study, all patients had evidence of large artery atherosclerosis in addition to the majority having a heavy burden of other established risk factors. However, in our study the patients with calcifications and no other risk factors had a comparable rate of end points to those with 1 or more risk factors. This tends to lend credence to the importance of the calcifications and suggests that the high rate of end points is not due to the calcifications being just another indication of heavy risk factor burden.

The mean age of our patients was 68 years. Monitoring patients in this age bracket, we expect a certain percentage of vascular end points and death independent of carotid calcifications. It is possible that the advanced age of our patients is the risk factor driving the high rate of vascular end points and death. Further study needs to be done on a comparable group of risk factor laden patients without carotid calcification to determine whether it is the presence of the carotid atheromatous disease that is an independent risk factor.

It is also possible that carotid calcification identified on panoramic radiograph is an independent risk factor for generalized vascular disease and death. The study of Woodworth et al¹⁵ supports this conclusion in a homo-

geneous population of Native Americans. To confirm this hypothesis, a large prospective study in a heterogeneous population comparing age-matched patients with and without carotid calcifications would need to be performed. We are in the process of collecting data on age- and risk factor-matched male veterans. Once that study is complete, we will be able to determine with more certainty whether calcifications identified on the panoramic radiograph are true independent risk factors for vascular disease and death.

Whether the carotid calcification is a surrogate marker for established vascular risk factors or an independent risk factor for vascular disease is moot. Patients found to have these calcifications have a high frequency of end points in a relatively short period of time after the calcification is identified. If the first end points were most frequently TIA and angina, waiting for symptoms before referring for an evaluation and treatment would be prudent. However, in our series, MI, stroke, and death were the most common end points. In view of this, these patients should be expeditiously referred for appropriate evaluation and treatment of the carotid arteries, coronary arteries, and vascular risk factors.

CONCLUSIONS

Carotid artery calcifications found as incidental findings on panoramic radiographs are powerful markers for future cerebrovascular and cardiovascular events and death. Our data indicate that identification of these risk factors warrants expeditious referral for comprehensive vascular evaluation and appropriate treatment for prevention of subsequent heart disease and stroke.

REFERENCES

1. American Heart Association. 2002 Heart and stroke statistical update. Dallas: American Heart Association; 2002.
2. Executive Committee for Asymptomatic Carotid Atherosclerosis Study Group (ACAS). Endarterectomy for asymptomatic carotid artery stenosis. *JAMA* 1995;273:1421-8.
3. Chimowitz MI, Weiss DG, Cohen SN, Starling MR, Hobson RW, VA Cooperative Study Group 167. Cardiac prognosis of patients with carotid stenosis and no history of coronary artery disease. *Stroke* 1994;25:759-65.
4. Cohen SN, Hobson RW, Weiss DG, Chimowitz MI, VA Cooperative Study 167 Group. Death associated with asymptomatic carotid stenosis: long term clinical evaluation. *J Vasc Surg* 1993; 18:1002-9.
5. Culebras A, Otero C, Toledo JR, Rubin BS. Computed tomographic study of cervical carotid calcification. *Stroke* 1989;20: 1472-6.
6. Dobranowski ID, Franchetto AA, Jaeschke R. The relevance of detecting carotid artery calcification on plain radiograph. *Stroke* 1993;24:1330-4.
7. Friedlander A, Lande A. Panoramic radiographic identification of carotid arterial plaques. *Oral Surg Oral Med Oral Pathol* 1981;52:102-4.
8. Friedlander AH, Baker JD. Panoramic radiography: an aid in detecting patients at risk of cerebrovascular accident. *J Am Dent Assoc* 1994;125:1598-603.

9. Carter LC, Haller AD, Nadarajah V, Calamel AD, Aguirre A. Use of panoramic radiography among an ambulatory dental population to detect patients at risk of stroke. *J Am Dent Assoc* 1997;128:977-84.
10. Friedlander RH. Panoramic radiography: the differential diagnosis of carotid artery atheromas. *J Spec Care Dent* 1995;15:223-7.
11. Almog DM, Illig KA, Khin M, Green RM. Unrecognized carotid stenosis discovered by calcifications on a panoramic radiograph. *J Am Dent Assoc* 2000;131:1593-7.
12. Carter LC, Tsimidis K, Fabiano J. Carotid calcifications on panoramic radiography identify an asymptomatic male patient at risk for stroke. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1998;85:119-22.
13. North American Symptomatic Carotid Endarterectomy Trial Collaborators. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. *N Engl J Med* 1991;325:445-53.
14. Barnett HJM, Taylor DW, Eliasziw M, et al. Benefit of carotid endarterectomy in patients with symptomatic moderate or severe stenosis. *N Engl J Med* 1998;339:1415-25.
15. Woodworth W, Genco RJ, Knowler W, et al. Calcified carotid atherosclerotic plaque as a predictor of CVD death. *J Dent Res* 2000;79:524.
16. Hobson RW, Weiss DG, Fields WS, et al. Efficacy of carotid endarterectomy for asymptomatic carotid stenosis. *N Engl J Med* 1993;328:221-7.

Reprint requests:

Stanley Cohen, MD
Division of Neurology
Cedars-Sinai Medical Center
8700 Beverly Boulevard
Room 4159-4NW
Los Angeles, CA 90048
Stanley.Cohen@cshs.org

CALL FOR LETTERS TO THE EDITOR

A separate and distinct space for Letters to the Editor was established by Larry J. Peterson, editor in chief of *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontics* in his Editorial in the January 1993 issue.

Dr Peterson also encouraged brief reports on interesting observations and new developments to be submitted to appear in this letters section as well as letters commenting on earlier published articles.

Please submit your letters and brief reports for inclusion in this section. Information for Authors for the Journal appears in this issue of *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontics*.

We look forward to hearing from you.