Common Carotid Intima-Media Thickness and Risk of Stroke and Myocardial Infarction

The Rotterdam Study

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Abstract

Background Noninvasive assessment of intima-media thickness (IMT) is widely used in observational studies and trials as an intermediate or proxy end point for cardiovascular disease. However, data showing that IMT predicts cardiovascular disease are limited. We studied whether common carotid IMT is related to future stroke and myocardial infarction.
**Methods and Results** We used a nested case-control approach among 7983 subjects aged \( \geq 55 \) years participating in the Rotterdam Study. At baseline (March 1990 through July 1993), ultrasound images of the common carotid artery were stored on videotape. Determination of incident myocardial infarction and stroke was predominantly based on hospital discharge records. Analysis (logistic regression) was based on 98 myocardial infarctions and 95 strokes that were registered before December 31, 1994. IMT was measured from videotape for all case subjects and a sample of 1373 subjects who remained free from myocardial infarction and stroke during follow-up. The mean duration of follow-up was 2.7 years. Results were adjusted for age and sex. Stroke risk increased gradually with increasing IMT. The odds ratio for stroke per standard deviation increase (0.163 mm) was 1.41 (95% CI, 1.25 to 1.82). For myocardial infarction, an odds ratio of 1.43 (95% CI, 1.16 to 1.78) was found. When subjects with a previous myocardial infarction or stroke were excluded, odds ratios were 1.57 (95% CI, 1.27 to 1.94) for stroke and 1.51 (95% CI, 1.18 to 1.92) for myocardial infarction. Additional adjustment for several cardiovascular risk factors attenuated these associations: 1.34 (95% CI, 1.08 to 1.67) and 1.25 (95% CI, 0.98 to 1.58), respectively.

**Conclusions** The present study, based on a short follow-up period, provides evidence that an increased common carotid IMT is associated with future cerebrovascular and cardiovascular events.

**Key Words:** atherosclerosis • ultrasonics • risk factors

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**Introduction**

Prospective follow-up studies have recently been initiated in which high-resolution B-mode ultrasonography of the carotid arteries is used to study atherosclerosis in populations at large.\(^1\)\(^2\)\(^3\)\(^4\)\(^5\)\(^6\)\(^7\) Carotid B-mode ultrasonography provides for evaluation of lumen diameter, intima-media thickness, and presence and extent of plaques. An increased cross-sectional carotid intima-media thickness was associated with unfavorable levels of established cardiovascular risk factors.\(^1\)\(^2\)\(^3\)\(^4\)\(^5\)\(^6\)\(^7\) prevalent cardiovascular disease,\(^7\)\(^8\)\(^9\) and atherosclerosis elsewhere in the arterial system.\(^10\)\(^11\)\(^12\) There is a growing belief that carotid intima-media thickness can be regarded as an indicator of generalized atherosclerosis\(^12\) and that it may be used as an intermediate end point or proxy end point in observational studies and trials as a suitable alternative for cardiovascular morbidity and
mortality. Indeed, a limited number of intervention studies showed reduced progression of intima-media thickness in subjects treated with lipid-lowering drugs compared with a placebo group. However, to apply these measurements with confidence, there is an urgent need for quantitative data to show that increased intima-media thickness and progression of intima-media thickness are related to future cerebrovascular and cardiovascular diseases. Presently, such information is very limited. So far, only one study has shown that an increased maximum common carotid intima-media thickness is related to an increased risk of myocardial infarction.

In the present study, we set out to examine the association between common carotid intima-media thickness and incident stroke and myocardial infarction among men and women participating in the Rotterdam Study.

**Methods**

**Population**

The Rotterdam Study is a single-center, prospective, follow-up study on disease and disability in the elderly in 7983 subjects aged ≥55 years living in the suburb of Ommoord in Rotterdam, Netherlands, as detailed elsewhere. Baseline data for each subject were collected from March 1990 through July 1993 in a home interview and two visits at the research center. The overall participation rate of those invited for the study was 78%. The study was approved by the Medical Ethics Committee of Erasmus University, and written informed consent was obtained from all participants.

**Carotid Arteries**

To measure carotid intima-media thickness, ultrasonography of the common carotid artery, carotid bifurcation, and internal carotid artery of the left and right carotid arteries was performed with a 7.5-MHz linear-array transducer (ATL UltraMark IV). On a longitudinal, two-dimensional ultrasound image of the carotid artery, the anterior (near) and posterior (far) walls of the carotid artery are displayed as two bright white lines separated by a hypoechoic space. The distance between the leading edge of the first bright line of the far wall (lumen-intima interface) and the leading edge of the second bright line (media-adventitia interface) indicates the intima-media thickness. For the near wall, the distance between the trailing edge of the first bright line and the trailing edge of the second bright line at the near wall provides the best estimate of the near-wall
intima-media thickness (Fig 1). In accordance with the Rotterdam Study ultrasound protocol, a careful search was performed for all interfaces of the near and far walls of the distal common carotid artery. When an optimal longitudinal image was obtained, it was frozen on the R wave of the ECG and stored on videotape. This procedure was repeated three times for both sides. The actual measurements of intima-media thickness were performed off-line. From the videotape, the frozen images were digitized and displayed on the screen of a personal computer using additional dedicated software. This procedure has been described in detail previously. In short, with a cursor, the interfaces of the distal common carotid artery were marked across a length of 10 mm. The beginning of the dilatation of the distal common carotid artery served as a reference point for the start of the measurement. The average of the intima-media thickness of each of the three frozen images was calculated. For each individual, the common carotid intima-media thickness was determined as the average of near- and far-wall measurements of both the left and right arteries. The readers of the ultrasound images from videotape were unaware of the case status of the subject. Results from a reproducibility study of intima-media thickness measurements among 80 participants of the Rotterdam Study who underwent a second ultrasound scan of both carotid arteries within 3 months of the first scan have been published elsewhere. In short, mean differences (SD) in far-wall intima-media thickness of the common carotid artery between paired measurements of sonographers, readers, and visits were -0.005 mm (0.09), 0.060 mm (0.05), and -0.033 mm (0.12), respectively.

Figure 1. Characteristic longitudinal, two-dimensional ultrasound image of the distal common carotid artery. A.W. indicates anterior (near) wall of the carotid artery; P.W., posterior (far) wall. Arrows from top to bottom indicate the leading edge of the intima-lumen interface at the near wall, the lumen-intima interfaces, and the media-adventitia interface at the far wall, respectively.

Off-line, the common carotid artery and the carotid bifurcation were evaluated from tapes for the presence (yes/no) of atherosclerotic lesions on both the near and far walls of the carotid arteries. Plaques were defined as a focal widening relative to adjacent segments, with protrusion into the lumen composed either of only calcified deposits or a combination of calcification and noncalcified material. The size or extent of the lesions was not quantified. A reproducibility study on the assessment of plaques in the carotid bifurcation among 166 participants revealed a $k$ of 0.59 for the left carotid artery, 0.65 for the right carotid artery, and 0.60 for plaques on either side, indicating moderate agreement.
Cerebrovascular and Cardiovascular Risk Indicators

A history of myocardial infarction and stroke at baseline was assessed on the basis of answers to the questions "Did you ever suffer from a myocardial infarction for which you were hospitalized?" and "Did you ever suffer from a stroke, diagnosed by a physician?". A subject's smoking status was classified as current, former, or never smoker. At the research center, height and weight were measured and body mass index (kg/m²) was calculated. Sitting blood pressure was measured at the right upper arm with a random-zero sphygmomanometer. The average of two measurements obtained on one occasion, separated by a count of the pulse rate, was used in the present analysis. Hypertension was defined as a systolic blood pressure $\geq 160$ mm Hg, a diastolic blood pressure $\geq 95$ mm Hg, or current use of antihypertensive drugs for the indication of hypertension. Diabetes mellitus was considered present when subjects currently used oral blood glucose–lowering drugs or insulin.

A nonfasting venipuncture was performed with the use of a 21-gauge butterfly needle with tube (Surflo winged infusion set, Terumo). Serum total cholesterol was determined by use of an automated enzymatic procedure. HDL cholesterol was measured similarly, after precipitation of the non-HDL fraction with phosphotungstate magnesium.

Incident Cerebrovascular and Cardiovascular Disease

In the Rotterdam Study, information on incident fatal and nonfatal events is obtained from the general practitioners (GPs) working in the study district of Ommoord. The GPs involved report all possible cases of both stroke and myocardial infarction to the Rotterdam research center. Events are presented as coded information according to the International Classification of Primary Care (ICPC). The ICPC codes for acute myocardial infarction and cerebrovascular accidents are K75 and K90, respectively. With respect to the vital status of the participants, information is obtained at regular intervals from the municipal authorities in Rotterdam, and death of a participant is reported as code A96 by GPs. The GPs whose practices are computerized send ICPC codes of participants of the Rotterdam Study on computer file to the Rotterdam Study data center on a regular basis. Follow-up data on computer file encompass $\approx 85\%$ of the Rotterdam Study cohort. When an event or death has been reported, additional information is obtained by interviewing the GP and scrutinizing information from hospital discharge records in case of admittance or referral. After consideration of all available information, some of the stroke events and myocardial infarctions initially suspected and reported by the GPs were not classified as such. For example, GPs are instructed to report cases of subdural hematoma (K90), which is not considered a stroke by a neurologist. Also, some subjects were reported to have died of a possible cerebrovascular accident, although a cardiac cause could not be excluded according to the GP. Understandably, some reported myocardial infarctions proved to be cases of angina pectoris, whereas others were eventually diagnosed as congestive heart failure.

A myocardial infarction was considered to have occurred when (1) the event led to a hospitalization, and the hospital discharge record comprised a diagnosis of a new myocardial infarction based on signs and symptoms, ECG recordings, and repeated laboratory investigations during hospital stay (definite myocardial infarction) or (2) a subject died within 1 hour after onset of symptoms (sudden death) without having been hospitalized, and the GP reported a cardiac source as the most likely cause of death (probable myocardial infarction).
Because 25% to 30% of subjects who suffer an acute stroke are not hospitalized in The Netherlands, all suspected cerebrovascular events reported by the GPs were submitted for review to a neurologist (P.J.K.). On the basis of all information, including symptoms and signs obtained by interviewing the GP or, in case of hospital referral, by reviewing hospital data, the neurologist classified the events as definite, probable, or possible stroke. The present analysis is restricted to outcomes in which a stroke most likely did occur in the opinion of the neurologist. For the present analysis, an incident stroke was considered to have occurred when one of the following criteria was met: (1) the event led to a hospitalization, and the hospital discharge record indicated a diagnosis of a new stroke, such clinical diagnosis being based on signs and symptoms as well as neuroimaging investigations during hospital stay (definite stroke); (2) in case of no hospitalization, signs and symptoms associated with the event obtained from the GP records and interview were highly suggestive of a stroke according to the neurologist (P.J.K.) (probable stroke); or (3) in case of out-of-hospital death, the GP reported that the cause of death was a cerebrovascular accident, and a cardiac cause was judged by the GP to be highly unlikely (probable stroke).

**Selection of Case Subjects and Sampling of Control Subjects**

Ultrasonography of the carotid arteries was performed in 5965 of the 7983 subjects. In particular, for subjects who had their baseline Rotterdam Study examination at the end of 1992 or in 1993, ultrasonography could not always be performed due to the restricted availability of ultrasonographers. For reasons of availability and completeness of information on cardiovascular events, we restricted the present study to follow-up events registered by GPs who had computerized follow-up procedures (coverage of nearly 85% of the cohort). This resulted in a cohort of 5130 subjects from which the case and control subjects were drawn. The mean duration of follow-up was 2.7 years. Participants who were registered with GPs who had a computerized follow-up procedure were on average 5 years older than those associated with GPs without computerized follow-up procedures. Sex, systolic and diastolic blood pressures, total and HDL cholesterol levels, presence of diabetes mellitus, and history of angina pectoris, stroke, or myocardial infarction did not differ significantly between the two groups.

A total of 140 subjects with an incident cerebrovascular accident and 125 subjects with myocardial infarction were reported by the GPs. After review of all available information, 103 strokes were considered to be definite or probable strokes, whereas 101 myocardial infarctions were found to be definite or probable events. For these subjects only, intima-media thickness was quantified from the stored images. Data on intima-media thickness could be obtained from the stored images on videotape of 99 subjects with a myocardial infarction and 95 subjects with a stroke (71 definite and 24 probable strokes).

At the time of the present analysis, intima-media thickness had been quantified for a random sample of 1715 of the 5965 subjects who underwent a carotid ultrasonography. Several cross-sectional analyses from the Rotterdam Study based on these data have been reported. The measurement of intima-media thickness from stored images is an ongoing process. Control subjects were drawn from this random sample of 1715 subjects. A subject was eligible as a control if (1) he/she was registered with a GP with computerized patient files, (2) he/she remained free from myocardial infarction or stroke during follow-up, and (3) images of common
carotid intima-media thickness were available on videotape for the subject. The total number of control subjects was 1373 (Fig 2).

**Figure 2.** Schematic presentation of the selection of case and control subjects. GP indicates general practitioner; IMT, intima-media thickness.

**Data Analysis**
Linear regression analysis was applied to evaluate the association between common carotid intima-media and potentially confounding cardiovascular risk indicators such as age, sex, body mass index, smoking, systolic and diastolic blood pressures, hypertension, total and HDL cholesterol levels, diabetes mellitus, and previous history of stroke and myocardial infarction. The association between common carotid intima-media thickness and incident myocardial infarction and stroke was evaluated by use of a logistic regression model. Analyses were performed with common carotid intima-media thickness used as a continuous variable (per SD) and as a categorized variable (based on quintile cutoff points of the distribution). Because the number of events in the lowest quintile was too limited, the lowest two categories were combined and used as a reference category. Whether the association differed with age or sex was evaluated by entering interaction terms into the model. The interaction terms did not reach statistical significance in the analyses for either myocardial infarction or stroke (probability values ranged from .17 to .76). Separate analyses were performed excluding subjects with a history of myocardial infarction or stroke. When not specified, all presented associations are adjusted for age and sex with corresponding 95% CIs.

**Results**
Table 1 describes general characteristics of the study subjects. Significant positive associations with common carotid intima-media thickness were found for age, male sex, body mass index, systolic blood pressure, hypertension, total cholesterol, diabetes mellitus, and a previous history of myocardial infarction or stroke. A significant inverse association was found for HDL cholesterol. The association with smoking was positive but did not reach the level of statistical significance ($P = .09$).

The risk of stroke increased gradually with increasing common carotid intima-media thickness. The odds ratio for stroke per SD increase (0.163 mm) was 1.41 (95% CI, 1.25 to 1.82). In men, the odds ratio per SD increase (0.172 mm) was 1.81 (95% CI, 1.30 to 2.51) and in women, an odds ratio of 1.33 (95% CI, 1.03 to 1.71) per 0.155-mm SD increase was observed. When subjects with a previous history of myocardial infarction or stroke were excluded, the odds ratios were 1.57 (1.27 to 1.94) for all subjects, 1.89 (95% CI, 1.29 to 2.77) for men, and 1.37 (95% CI, 1.02 to 1.83) for women. When differences in risk factors were allowed for, the associations were attenuated: 1.34 (95% CI, 1.08 to 1.67), 1.47 (1.08 to 2.02), and 1.14 (0.85 to 1.54), respectively. The associations with stroke in categories of common carotid intima-media thickness are presented in detail in Table 2.

The risk of myocardial infarction increased 43% per SD increase in common carotid intima-media thickness (odds ratio, 1.43; 95% CI, 1.16 to 1.78). Exclusion of subjects with a history of myocardial infarction and stroke revealed an odds ratio of 1.51 (95% CI, 1.18 to 1.78). The associations for first incident event were similar for men and women: 1.56 (95% CI, 1.12 to 2.18) and 1.44 (95% CI, 1.00 to 2.08), respectively. Additional adjustment for cardiovascular risk factors attenuated the magnitude of the associations and their statistical significance. Odds ratios were 1.25 (95% CI, 0.98 to 1.58) for all subjects, 1.25 (95% CI, 0.91 to 1.72) for men, and 1.26...
(95% CI, 0.89 to 1.79) for women. The association between intima-media thickness and risk of myocardial infarction did not show a clearly linear pattern (Table 2). The risk was particularly increased in subjects with an intima-media thickness in the upper quintile of the distribution (0.908 mm) relative to the risk in the reference category (0.75 mm); the odds ratio for first myocardial infarction was 2.32 (95% CI, 1.17 to 4.64).

Discussion

The findings in the present study indicate that an increased common carotid intima-media thickness is associated with future cerebrovascular and cardiovascular events in older subjects. These associations were independent of age, sex, and history of myocardial infarction or stroke. As expected, additional adjustment for cardiovascular risk factors attenuated the associations. It is important to realize that these levels of common carotid intima-media thickness do not reflect the presence of arterial stenosis in the common carotid artery and that arterial blood flow in the common carotid artery in these subjects is virtually normal.

Several aspects of the present study need to be addressed. Several biases that might affect the validity of the findings in our nested case-control study were excluded. First, exposure was measured without knowledge of the case-control status of the participant. Second, the outcome events were based to a large extent on documented medical information, which limits the extent of misclassification of the diagnosis. However, if such misclassifications are still present, the observed associations most likely represent an underestimation of the true associations because misclassification is likely to be nondifferential. Similarly, inclusion of subjects with silent myocardial infarctions or silent strokes in the control group might lead to attenuation of the associations with intima-media thickness. Third, validation studies in which ultrasonographically measured intima-media thickness was compared with histologically determined intima-media thickness showed that ultrasound is capable of accurately measuring intima-media thickness. Whether increased common carotid intima-media thickness itself reflects local atherosclerosis is still a subject of debate. It may merely reflect an adaptive response of the vessel wall to changes in shear stress, tensile stress, and blood flow and subsequent changes in lumen diameter, as has been suggested in particular for intima-media thickness <90 µm. Atherosclerosis is a disorder of the intima, and ultrasound imaging cannot discriminate between the intimal and medial layers of the vessel wall. The question is whether it matters very much if common carotid intima-media thickness does not represent local atherosclerosis. Several cross-sectional studies have
shown that increased common carotid intima-media thickness may be of use as a marker of atherosclerosis elsewhere in the arterial system, and the present findings show that an increased common carotid intima-media thickness confers an increased risk of cerebrovascular and cardiovascular diseases.

The present analyses have been restricted to common carotid intima-media thickness measurements. This is because recording on videotape and quantification of intima-media thickness of the carotid bifurcation and internal carotid artery in the Rotterdam Study started at a later stage. Ultrasound images including intima-media thickness of the carotid bifurcation and internal carotid artery have been stored only after approximately the first 1500 subjects were enrolled in the study. Thus, the question of whether the results differ by arterial segment cannot be answered with the present analyses.

Unfavorable levels of cardiovascular risk factors have been associated with increased common carotid intima-media thickness and with stroke and myocardial infarction. In the present analyses, adjustment for cardiovascular risk factors reduced the magnitude of the associations between common carotid intima-media thickness and incident stroke and myocardial infarction. This may be expected because common carotid intima-media thickness, as an indicator of atherosclerosis, may be considered an intermediate factor in the causal pathway leading to stroke or myocardial infarction. In a strict sense, these risk factors should therefore not be considered as confounding variables of the association and in principle should not be controlled for in the analyses. However, when the main interest is to assess whether common carotid intima-media thickness predicts stroke and myocardial infarction independently of these risk factors, one may want to additionally adjust for these factors.

Data to show that common carotid intima-media thickness relates to future cerebrovascular and cardiovascular events are limited. Salonen and Salonen, in the one available study to date performed in a random sample (n=1257) of middle-aged Finnish men, reported that an increase of 0.1 mm in maximum common carotid intima-media thickness was associated with an 11% (95% CI, 6% to 16%) increase in the risk of myocardial infarction. Our results are in line with the Finnish findings, although direct quantitative comparison of the findings between studies is not possible due to differences in presentation of exposure measures. A recent report from Kuller and coworkers showed a considerably increased risk of cardiovascular morbidity and mortality for subjects with subclinical disease compared with subjects with no signs of subclinical disease. Subclinical disease was defined by a combination of ankle-brachial blood pressure, carotid artery stenosis, carotid wall thicknesses, ECG and echocardiography abnormalities, and the Rose questionnaire. These results are in accordance with our finding that among subjects free from symptomatic cerebrovascular and cardiovascular diseases, an increased intima-media thickness is associated with incident stroke and myocardial infarction.

We found a graded association of common carotid intima-media thickness with stroke but not with myocardial infarction. No clear explanation for this apparent difference can be given. The differences between heart and brain might influence the presence and extent of atherosclerotic lesions that give rise to symptoms. Symptomatic myocardial infarction may generally be caused by advanced large-vessel atherosclerosis. Atherosclerotic abnormalities in small coronary arteries may not lead to typical symptoms and may therefore remain undetected. Stroke, however, may be
due to large-vessel atherosclerosis, but atherosclerotic changes in small cerebral arteries (for example, due to elevated blood pressure) may also lead to symptoms suggestive of a stroke. This notion remains speculative, however, and needs to be confirmed in future studies.

The noninvasive assessment of common carotid intima-media thickness appears to provide a promising method to study atherosclerosis directly, at the level of the vessel, in populations at large. Intervention studies on the efficacy of lipid-lowering regimens in reducing progression of atherosclerosis have further shown the feasibility of application of these measurements in trials. The use of carotid intima-media thickness measurements as an indicator of generalized atherosclerosis is conditioned on the view that its measurement reflects cardiovascular disease risk. The present study lends support to this view and provides supportive evidence for the use of intima-media thickness measurements as an intermediate or proxy end point in observational and intervention studies as an alternative to the use of cardiovascular disease or death as an end point. At present, the clinical and therapeutic relevance of an increased carotid intima-media thickness measurement in an individual may be limited for that individual. However, observational studies and trials on the efficacy of certain treatment regimens using intima-media thickness as a primary outcome measure may yield important results that may have major implications for clinical practice.

In conclusion, the present study, based on a short follow-up period, shows that an increased common carotid intima-media thickness relates to future cardiovascular and cerebrovascular events. This study provides supportive evidence for the use of intima-media thickness measurements as an intermediate or proxy end point in observational studies and trials.

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