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Concomitant carotid plaque development and brachial artery diameter enlargement: A retrospective, recall-based study in postmenopausal women

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Abstract *Background and aim:* To verify if the carotid plaque development is concomitant to brachial artery diameter enlargement, in healthy postmenopausal women.
Methods and results: This is a retrospective, recall study. We enrolled 40 postmenopausal women, selected from a database for the period 2000–2008, not affected by subclinical carotid atherosclerosis and without risk factors for cardiovascular disease. At the recall visit, carotid and brachial duplex scan was again obtained.

The incidence of plaque was 30% after a mean follow-up period of 60 months. There were no differences in baseline characteristics between subjects developing carotid atherosclerosis and subjects who did not, except for the brachial diameter change, follow-up and heart rate. The logistic-regression analysis confirmed that only brachial diameter change resulted to be correlated with the development of carotid atherosclerosis.
Conclusion: Brachial artery diameter increase is concomitant to carotid plaque development. Vascular enlargement could not be a focal change but a systemic process associated with atherosclerotic plaque development. Brachial diameter could be a tool with a predictive significance.

Abbreviations: CAD, carotid atherosclerosis development; BAD, brachial artery diameter; CIMT, carotid intima-media thickness; IMT, intima-media thickness; SBP, systolic blood pressure; DBP, diastolic blood pressure.

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It was suggested that arterial enlargement may be a systemic process [1] associated with several cardiovascular risk factors [1], intimal media thickness (IMT) [1–3] and carotid atherosclerosis [3]. It was also associated with specific conditions as metabolic syndrome, diabetes and obesity [4–9]. In particular, several studies showed that morphological changes of brachial artery diameter (BAD) could be a better indicator of the extent and severity of coronary artery disease rather than other vascular indices assessed non-invasively [10–12]. Other studies showed that BAD has predictive significance in the stratification of cardiovascular risk [13]. Thus, the BAD measurement has the potential to become a tool in cardiovascular event prediction [1,11]. Since the absence of follow-up study we aimed to verify, by a retrospective study, if the development of carotid plaques, a well-known morphological marker of cardiovascular risk, is associated with BAD change, in the form of diameter enlargement, in apparently healthy postmenopausal women.

Methods

This is a population-based retrospective study, performed in Caucasian postmenopausal women referred to the Menopause Clinic of the University of Catanzaro. We used baseline data of the Clinic database from years 2000 to 2008, to recruit participants from 45 to 75 years of age. We enrolled postmenopausal women, defined as having no natural menses for at least 1 year and serum Follicle stimulating hormone (FSH) level more than 40 IU L^{-1}, at baseline. Exclusion criteria were cardiovascular disease, arterial hypertension, severe obesity, diabetes mellitus and current smoking. Subjects affected by metabolic syndrome, defined according to Adult Treatment Panel III (ATP III) criteria [14], were also excluded from the final analysis. The participants were also not affected by subclinical carotid atherosclerosis, on the basis of a baseline carotid ultrasound evaluation. A total of 40 postmenopausal women, who met these criteria, were enrolled in this study. All of them were recalled to collect their medical history and to verify the eventual presence of risk factors for cardiovascular disease, as well as for a new physical examination, laboratory tests and vascular ultrasound evaluation.

Physical examination included the assessment of blood pressure (BP) and anthropometric measurements. The body mass index (BMI) was calculated as weight (in kg) divided by square of height (in m^2). Clinic BP was obtained in both arms in the supine patients, after 5 min of quiet rest, with a mercury sphygmomanometer. A minimum of three BP readings were taken. Venous blood was collected after overnight fasting, into vacutainer tubes (Becton & Dickinson) and centrifuged within 4 h at every visit. Serum glucose, creatinine, total cholesterol, high-density lipoprotein-cholesterol, triglycerides, insulinaemia and C reactive protein (CRP) were measured by standard laboratory techniques. Quality control was assessed daily for all determinations. The following criteria were used to define the cardiovascular risk factors: diabetes: fasting blood glucose $>$ 126 mg dL^{-1} or treatment for diabetes; hypertension: systolic BP $>$ 140 mmHg and/or diastolic BP $>$ 90 mmHg; severe obesity: BMI = $>$36 kg m^{-2}.

The study was approved by the local ethical committee and all subjects provided informed written consent in accordance with principles of the Declaration of Helsinki.

Ultrasound

To detect carotid atherosclerosis, a carotid duplex scan, already performed in all patients at baseline visit, was repeated at the recall visit as well as an ultrasound measurement of BAD. In particular, the subjects underwent B-mode ultrasonography of the extracranial carotid arteries by use of a high-resolution ultrasound instrument (ATL, HDI 5000) with a linear array multifrequency transducer. The examinations were performed by the same ultrasonographer blinded to clinical information for both carotid and brachial artery (BA) at baseline and at follow-up examination. Electrocardiogram (ECG) leads were attached to the ultrasound recorder for continuous heart rate monitoring. Both common (CCA) and internal carotid arteries were evaluated. Plaque was defined as an echogenic focal structure encroaching into the vessel lumen of at least 50% of the surrounding IMT value [15]. Stenosis was defined as a peak systolic velocity $>$ 120 cm s^{-1} and occlusion was defined as the absence of Doppler signal. According to these criteria, subjects were considered as normal if no lesion was detected, or with carotid atherosclerosis development (CAD) when a plaque, stenosis or occlusion was detected in at least one segment of common, bifurcation or internal carotid artery at follow-up. The mean of three left and right longitudinal segments of IMT, defined as the distance between the intimal–luminal and the media–adventitial interfaces, was measured as previously described [16]. The coefficient variation of the IMT measurements was 3.3%. To evaluate artery diameters, images were magnified and depth and gain settings were set to optimise the image of the vessel wall, in particular, the media–adventitia interface ('m' line). The longitudinal image of the BA of the non-dominant arm was obtained proximal to the antecubital fossa wherever the best ultrasound image could be obtained. The end-diastolic diameter of the vessel, defined as the distance between near-wall and far-wall junctions of the media and adventitia, was measured over four cardiac cycles using digital callipers, and the average was then calculated. The coefficient of variation of this method was 5%. According to the BAD change between the baseline and the final value of BAD, we divided the population into two groups, defined as 'progressors' (if their diameter resulted increased) and 'stable' (if their diameter resulted unchanged). The pulsatile diameter of carotid and BA was also calculated as the difference between systolic and diastolic diameter; the coefficient of variation of the method was 7%.

Statistical analysis

Descriptive statistics were reported as mean $\pm$ standard deviation (SD) for continuous variables and as percentages for categorical variables. A *t*-test was performed to compare the mean of groups with and without carotid atherosclerosis at the recall visit. Logistic-regression analysis was used to estimate odds ratio of carotid