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ORIGINAL ARTICLE

# Impact of atherosclerotic changes of carotid vessels on long-term outcome in relatively young patients with acute coronary syndrome

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## Abstract

**Background:** Complex stenoses of coronary vessels as well as unstable plaques are part of the widespread atherosclerotic process.

**Aim:** The possible association between the incidence of unstable coronary artery disease (CAD) and the morphology of carotid artery wall and cardiovascular events (MACE) was assessed prospectively in a two-year follow-up study.

**Methods and results:** Ninety-seven consecutive patients, aged under 60, admitted to hospital with suspected acute coronary syndrome (ACS) were included. Angiography was performed in all patients. Coronary artery disease was confirmed in 78 patients. This was the CAD(+) group. In 19 patients, coronary arteries were normal. This was the CAD(–) group. In all cases, carotid ultrasound was performed before discharge and at two-year follow-up, with evaluation of carotid arteries wall morphology: carotid intima-media thickness (CIMT) in far distal wall of common carotid artery and the presence of plaques. Carotid atherosclerosis was defined as CIMT > 0.9 mm or incidence of plaques; MACE was defined as death, ACS, stroke or need for urgent coronary revascularisation. Sixty patients from the CAD(+) group met the carotid atherosclerosis criteria. This was named the CAR(+) subgroup; 18 patients with normal carotid morphology comprised the CAR(–) subgroup. During the two years, MACE occurred only in the CAD(+) group (22 events). There was no statistical difference in the MACE-free survival curve of the CAR(+) and CAR(–) subgroups ($p = 0.91$).

**Conclusions:** The presence of atherosclerotic process in carotid region coexists well with the incidence of CAD; however, it does not determine prognosis after ACS.

**Key words:** acute coronary syndrome, carotid atherosclerotic plaque, carotid intima-media thickness

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## INTRODUCTION

Carotid intima-media thickness (CIMT) is a non-invasive, useful tool of early endothelial dysfunction assessment that can be easily performed by carotid artery B-mode ultrasound. As endothelial dysfunction is a systemic disorder, and a key variable in the pathogenesis of atherosclerosis, evaluation of carotid wall morphology is increasingly used in clinical research. Increased CIMT is thought to be an early atherosclerotic index [1–3]. In clinical practice, the measurement of CIMT is not yet performed as a routine investigation, and whether or not to use this parameter as an alternative to other conventional risk factors remains a matter of debate. Some authors, in comparing ultrasound evaluation of CIMT and quantitative coronary angiography, have found a relatively poor correlation [4–6].

On the other hand, in 2008, the American Society of Echocardiography published a consensus statement on the clinical use of carotid ultrasound for cardiovascular (CV) risk assessment [7]. Most of the studies performed so far have considered patients with stable coronary artery disease (CAD) or subjects at risk for CV diseases. The purpose of our study

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was to assess whether carotid atherosclerosis could be linked with an unfavourable long-term prognosis in a relatively young population with acute coronary syndrome (ACS).

## METHODS

Ninety-seven consecutive patients admitted to hospital with a suspicion of ACS were enrolled into the prospective observation. All patients gave informed consent and local ethics committee approval for the study was obtained. Inclusion criteria were: age less than 60 years and clinical manifestation typical for ACS.

After urgent coronary angiography and troponin levels measurements, patients were divided into two groups: a group with confirmed CAD — CAD(+), 78 patients, and a group with normal coronary vessels — CAD(–), 19 patients. Percutaneous coronary intervention (PCI) of one or more vessels was performed in 69 (88%) cases in the CAD(+) group.

Before discharge, every patient underwent ultrasound imaging of the carotid arteries using 7.5 MHz linear probe (VIVID 7, GE). Distal 10 mm far wall of left and right common carotid arteries (CCA), proximal to the carotid bulb, were examined with the measurement of CIMT.

A number of plaques was asessed in CCA and internal carotid arteries. Trained sonographers, blinded to the patients' coronary status, performed all scans. CIMT was defined as the distance between the leading edge of the lumen-intima echo and the leading edge of the media-adventitia echo. The mean values of three measurements were calculated. Maximum CIMT values from left or right CCA were used for further analysis. Carotid plaque was defined as the presence of focal wall thickening that is at least 50% greater than that of the surrounding vessel wall or as a focal region with CIMT greater than 1.5 mm protruding into the lumen that is distinct from the adjacent boundary. The total number of visualised plaques was calculated. Carotid atherosclerosis was defined as CIMT > 0.9 mm or a presence of plaques according to the European Society of Hypertension definition [7]. Additionally, high-sensitivity C-reactive protein (hsCRP) level was measured before discharge.

The CAD(+) group was then divided into two subgroups: patients with carotid atherosclerosis as defined above: CAR(+) and patients with normal carotid vessels: CAR(–). The population was observed for two years. Patients received pharmacological treatment according to the current guidelines. The incidence of major adverse cardiovascular events (MACE), defined as death, acute myocardial infarction (MI), stroke, or need for urgent revascularisation, was recorded. Two year follow-up included B-mode ultrasonography of carotid vessels.

### *Statistical analysis*

Statistical analysis was carried out using Statistica 7.0 Pl software. All continuous variables are shown as mean ± SD and categorical variables as absolute numbers or proportions. Continuous data were compared with the Student's t-test or Mann-Whitney U test, categorical variables with the $\chi^2$ test or Fisher exact test, and Spearman correlation was used to assess relationships between continuous variables. A Kaplan-Meier survival curve was drawn. A p value of $< 0.05$ was considered statistically significant. Univariable model of logistic regression was performed.

## RESULTS

Some baseline characteristics of the CAD(–) and CAD(+) groups as well as the CAR(+) and CAR(–) subgroups are shown in Tables 1 and 2.

Carotid atherosclerosis was found in the CAD(–) and CAD(+) groups in six (32%) and 60 (77%) patients, respectively ($p = 0.0001$). Mean CIMT was significantly higher in the CAD(+) group ($p = 0.002$). In the CAD(+) group, the mean number of plaques was 2.8 ± 3.7, whereas in the CAD(–) group, only one patient had one plaque in carotid bulb ($p = 0.05$).

During the two-year follow-up, we observed 22 MACE only in the CAD(+) group: 17 (28.3%) in the CAR(+) subgroup and five (27.8%) in the CAR(–) subgroup. The difference was not statistically significant. Almost all events (20) were the need for urgent revascularisation, either PCI or coronary artery bypass grafting. Two patients died in the CAR(+) subgroup, in both cases due to cardiac reasons (Tables 3, 4).

Measurements at the two-year follow-up visit revealed that CIMT was significantly higher in the CAD(+) group, as well as the CAR(+) subgroup: 1.15 *vs* 0.8; $p = 0.002$ and 1.21 *vs* 1.0; $p = 0.01$; respectively.

Correlation of baseline parameters with the incidence of MACE during the two-year follow-up was studied in univariate analysis. Logistic regression model (only univariable was possible to use) included traditional CV disease risk factors such as age, gender, body mass index, hyperlipidaemia, diabetes, smoking, history of PCI, and MI carotid atherosclerosis. After adjustment by regression analysis, presence of CAD, diagnosis of MI, prior aspirin and statin use, as well as history of PCI, were significant variables associated with a poor prognosis. The presence of carotid atherosclerosis did not significantly worsen patients' prognosis in our study. The results of univariable analysis are shown in Table 5. In the Kaplan-Meier event-free survival curves, no differences were found between the CAR(+) and CAR(–) subgroups (Fig. 1).

## DISCUSSION

High resolution B-mode ultrasound is an easy, non-invasive technique used to assess atherosclerosis in superficial arteries. CIMT, defined as the distance between blood-intima and media-adventitia of carotid wall, is suggested to be an early marker of widespread atherosclerotic process [1, 8–13]. On the other hand, it is well accepted that atherosclerosis can be considered as a chronic vascular inflammatory disease [14].

**Table 1.** Baseline characteristics of the CAD(+) and CAD(–) groups

| | CAD(+) (n = 78) | CAD(–) (n = 19) | P |
|---|---|---|---|
| Age [years] | 52 ± 7 | 49 ± 10 | 0.51 |
| Gender (male) | 57 (73%) | 9 (47%) | 0.003 |
| Body mass index | 27 ± 4 | 26 ± 2 | 0.37 |
| Total cholesterol [mg/dL] | 193 ± 48 | 157 ± 30 | 0.003 |
| Triglycerides [mg/dL] | 162 ± 102 | 120 ± 58 | 0.03 |
| HDL-cholesterol [mg/dL] | 41 ± 11 | 46 ± 13 | 0.02 |
| LDL-cholesterol [mg/dL] | 122 ± 39 | 87 ± 25 | 0.0006 |
| Diabetes | 15 (19%) | 1 (5%) | 0.09 |
| Hypertension | 58 (74%) | 6 (31%) | 0.0002 |
| Smoking | 50 ( 64%) | 4 (21%) | 0.0007 |
| Prior aspirin use | 34 (44%) | 6 (31%) | 0.25 |
| Prior statin use | 33 (42%) | 7 (37%) | 0.74 |
| History of MI | 21 (27%) | 0 | 0.001 |
| History of PCI | 16 (20%) | 0 | 0,03 |
| Recent PCI | 69 (88%) | 0 | 0.0001 |
| LVEF [%] | 51 ± 9 | 58 ± 4 | 0.001 |
| CAR(+) | 60 (77%) | 6 (32%) | 0.0001 |
| CIMT [mm] | 1.0 ± 0.2 | 0.8 ± 0.2 | 0.0002 |
| No of plaques in carotid arteries | 2.8 ± 3.7 | 1 (1 patient) | 0.05 |
| hsCRP [mg/L] | 19.2 ± 20 | 2.8 ± 3.0 | 0.001 |

MI — myocardial infarction; PCI — percutaneous coronary intervention; LVEF — left ventricular ejection fraction; CAR — carotid atherosclerosis criteria; CIMT — carotid intima-media thickness; hsCRP — high-sensitivity C-reactive protein

**Table 2.** Baseline characteristics of the CAR(+) and CAR(–) subgroups

| | CAR(+) (n = 60) | CAR(–) (n = 18) | P |
|---|---|---|---|
| Age [years] | 53 ± 9 | 46 ± 5 | 0.04 |
| Gender (male) | 47 (78%) | 10 (55%) | 0.02 |
| Body mass index | 27 ± 3 | 27 ± 6 | 0.30 |
| Total cholesterol [mg/dL] | 194 ± 50 | 180 ± 36 | 0.64 |
| Triglycerides [mg/dL] | 162 ± 104 | 164 ± 100 | 0.83 |
| HDL-cholesterol [mg/dL] | 41 ± 11 | 42 ± 11 | 0.95 |
| LDL-cholesterol [mg/dL] | 123 ± 40 | 118 ± 37 | 0.60 |
| Diabetes | 15 (25%) | 0 | 0.02 |
| Hypertension | 47 (78%) | 11 (61%) | 0.14 |
| Smoking | 37 (62%) | 13 (72%) | 0.41 |
| Prior aspirin use | 28 (47%) | 6 ( 33%) | 0.31 |
| Prior statin use | 25 (41%) | 8 (44%) | 0.8 |
| History of MI | 17 (28%) | 4 (22%) | 0.6 |
| History of PCI | 12 (20%) | 4 (22%) | 0.83 |
| Recent STEMI [%] | 33 (55%) | 13 (72%) | 0.61 |
| Recent NSTEMI [%] | 15 (25%) | 1 (5.5%) | 0.04 |
| CAD extent (no. of vessels) | 2 ± 1 | 2 ± 1 | 0.88 |
| Recent PCI [%] | 53 (88%) | 16 (89%) | 0.94 |
| CIMT [mm] | 1.14 ± 0.22 | 0.76 ± 0.01 | 0.0001 |
| No of plaques in carotid arteries | 3.2 ± 3.7 | 0 | 0.004 |
| hsCRP [mg/L] | 19.5 ± 25 | 18.1 ± 17 | 0.4 |

STEMI — ST-elevation myocardial infarction; NSTEMI — non-ST-elevation myocardial infarction; CAD — coronary artery disease; other abbreviations as in Table 1

**Table 3.** Two-year follow-up: carotid intima-media thickness (CIMT) and major adverse cardiovascular events (MACE) in the CAD(+) and CAD(–) groups

| | CAD(+) (n = 78) | CAD(–) (n = 19) | P |
|---|---|---|---|
| MACE [%] | 22 (28) | 0 | 0.01 |
| CIMT [mm] | 1.15 ± 0.3 | 0.8 ± 0.1 | 0.002 |

**Table 4.** Two-year follow-up: carotid intima-media thickness (CIMT) and major adverse cardiovascular events (MACE) in the CAR(+) and CAR(–) subgroups

| | CAR(+) (n = 60) | CAR(–) (n = 18) | P |
|---|---|---|---|
| MACE [%] | 17 (28) | 5 (28) | 0.73 |
| CIMT [mm] | 1.21 ± 0.3 | 1.0 ± 0.2 | 0.01 |

**Table 5.** Univariable analysis of some baseline parameters' influence on incidence of major adverse cardiovascular events

| Factor | P | OR [95% CI] |
|---|---|---|
| Age | 0.49 | 1.02 [0.96–1.07] |
| Gender (male) | 0.96 | 0.97 [0.31–3.04] |
| Body mass index | 0.21 | 1.08 [0.95–1.25] |
| Total cholesterol | 0.11 | 0.99 [0.98–1.07] |
| Triglicerydes | 0.28 | 1.00 [0.99–1.01] |
| HDL-cholesterol | 0.11 | 0.96 [0.92–1.01] |
| LDL-cholesterol | 0.19 | 0.99 [0.98–1.00] |
| Diabetes | 0.09 | 2.80 [0.85–9.18] |
| Hypertension | 0.33 | 1.80 [0.52–6.27] |
| Smoking | 0.64 | 1.28 [0.44–3.73] |
| Prior aspirin use | 0.029 | 3.06 [1.08–8.70] |
| Prior statin use | 0.019 | 3.32 [1.16–9.46] |
| Prior MI | 0.02 | 2.65 [1.21–9.08] |
| Prior PCI | 0.03 | 3.43 [1.07–11.0] |
| Recent MI | 0.005 | 4.60 [1.54–13.78] |
| LVEF | 0.06 | 0.95 [0.91–1.00] |
| CAD(+) | 0.007 | 4.01 [1.40–11.54] |
| CAR(+) | 0.96 | 1.03 [0.31–3.40] |
| hsCRP | 0.25 | 1.02 [0.99–1.04] |

OR — odds ratio; CI — confidence interval; other abbreviations as in Table 1

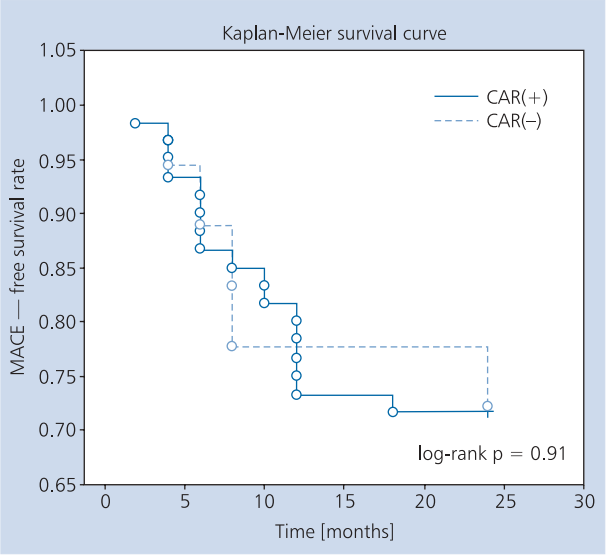


**Figure 1.** Major adverse cardiovascular events (MACE)-free survival curves for the CAR(+) and CAR(–) subgroups

Inflammatory cytokines are responsible for the activation of endothelial cells, favouring the attachment of circulating monocytes to the endothelium, thus enhancing their migration into the vascular intima-media layer [15–17]. A consequence of this process is an increase of arterial IMT, which is therefore considered a highly sensitive marker of atherosclerosis progression [18, 19]. The main CAD risk factors, such as hypercholesterolaemia, smoking, hypertension and diabetes, have been shown to cause endothelial dysfunction and thickening of carotid intima-media in asymptomatic patients, and even in children [20–23]. The authors of some multi-ethnic studies have recommended measuring carotid or other peripheral arterial IMT in all asymptomatic men aged above 45 and women aged above 55 as a guide to clinical decision making [24]. The American Heart Association also supports the recommendation of careful IMT investigation as an additional, valuable risk factor assessment [25].

Nevertheless, only a few studies concerning the relationship between carotid artery morphology and further prognosis in patients with ACS have been published so far. However, no recommendations for examining carotid arteries in this specific group exist.

In one recently published paper, the authors concluded that in patients with ACS, the morphology of carotid plaque is significantly different and more prone to fibrous cap rupture than carotid plaque in patients with stable coronary disease. Nevertheless, similarly to our results, no difference in the number of plaques as well as in the CIMT between 'unstable' and 'stable' patients was found [26]. Another interesting publication concerning the possibility of using CIMT as a prognostic tool in ACS revealed that although in patients with non-ST elevation myocardial infarction (NSTEMI), CIMT was significantly higher than in healthy subjects, this fact did not alter the clinical outcome in prospective observation [27].

Our study showed that neither CIMT nor the number of carotid plaques adds extra significant information to risk stratification and futher prognosis in patients with ACS. Although patients without carotid atherosclerosis were significantly

younger, did not suffer from diabetes, and had less NSTEMI, these facts did not change the CV risk. In the univariable regression model (no satisfactory multivariable model was possible to create) the only significant variables related to CV risk were: prior MI, prior PCI, recent MI as well as prior diagnosed CAD (with concomitant prior aspirin and statin use). Even hsCRP levels were not significant tools for risk stratification, which is in accord with the recently published results of the ASCOT study, where a significant drop in hsCRP levels did not predict a decrease in CV events [28].

Some more recent studies have pointed out that focusing only on CIMT examination does not give enough information for further prognosis and therapeutic implications. Hirano et al. [29], in a recently published paper, proved that it is not the extent of carotid sclerotic plaque, but above all its echolucency, that is crucial for prognosis estimation.

The same authors in another study observed that plaque size progression, rather than baseline CIMT, correlates well with future coronary events. A careful examination of plaque morphology, especially its dynamic progression during the observation period, may help in risk stratification [30].

The lack of a strong correlation of carotid atherosclerosis with a worse prognosis in our study may also be due to some limitations. Firstly, we measured IMT manually, by placing the cursor in the distal far wall of left and right CCA (depending on visualisation) and by determining the boundaries of CIMT interfaces. Maximum value of the mean left or right CIMT was then taken for further analysis. This manual approach may however be less precise and reproducible than computerised measurement, when an average of 100 points of measure in the distal segment of CCA is analysed [31]. Secondly, certain images were impossible to take into account because of very poor quality. Thus, we assume that some data may be underestimated. This is concordant with the results of other studies that have questioned the predictive value of CIMT evaluated only in the distal part of CCA, and pointed out the need to examine other arterial sites [32]. Additionally, though there is epidemiological data indicating that a value of CIMT at or above 1 mm at any age is associated with a significantly increased CV risk, some groups consider defining the threshold above which the risk begins to rise sharply. Finally, our study included a relatively small number of patients, thus a limitation of statistical power has to be acknowledged.

## CONCLUSIONS

Carotid atherosclerosis certainly participates in CV risk assessment in primary and secondary prevention. In our group of patients however, it was not an additional significant tool. The results of our study do not justify routine examination of carotid vessels in a young population with ACS. More data is needed in order to introduce this easily accessible diagnostic method into everyday clinical practice.

***Conflict of interest:*** *none declared*

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# Wpływ zmian miażdżycowych w tętnicach szyjnych na długoterminowe rokowanie w grupie relatywnie młodych chorych z ostrym zespołem wieńcowym

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## Streszczenie

**Wstęp:** Złożone zmiany miażdżycowe tętnic szyjnych i niestabilne blaszki miażdżycowe naczyń wieńcowych są częścią rozległego procesu miażdżycowego.

**Cel:** Celem pracy było ustalenie wpływu obecności miażdżycy tętnic szyjnych na występowanie dużych niekorzystnych zdarzeń sercowo-naczyniowych (MACE) u pacjentów po ostrym zespole wieńcowym (OZW) w prospektywnej, 2-letniej obserwacji.

**Metody i wyniki:** Do badania włączono 97 kolejnych chorych w wieku poniżej 60 lat, z podejrzeniem OZW, przyjętych na oddział kardiologii. U wszystkich pacjentów wykonano koronarografię. Obecność OZW potwierdzono na podstawie wyników koronarografii i badań enzymatycznych u 78 chorych: grupa CAD(+), podczas gdy u 19 pacjentów wykluczono OZW: grupa CAD(–). U wszystkich chorych przeprowadzono badanie ultrasonograficzne tętnic szyjnych przed wypisem ze szpitala oraz po 2 latach z oceną grubości kompleksu infima-media (CIMT) w tętnicy szyjnej wspólnej oraz obecności blaszek miażdżycowych. Miażdżycę tętnic szyjnych definiowano, gdy CIMT > 0,9 mm lub gdy stwierdzano blaszki miażdżycowe, a MACE obejmowały śmierć, OZW, udar lub potrzebę pilnej rewaskularyzacji wieńcowej. Kryteria obecności miażdżycy tętnic szyjnych spełniło 60 pacjentów z grupy CAD(+): podgrupa CAR(+), podczas gdy u 18 chorych z grupy CAD(+) stwierdzono prawidłową morfologię tętnic szyjnych: podgrupa CAR(–). W 2-letniej obserwacji MACE wystąpiły jedynie w grupie CAD(+) — 22 zdarzenia. Nie było istotnej różnicy między przeżywalnością wolną od MACE między podgrupami CAR(+) i CAR(–); p = 0,91.

**Wnioski:** U pacjentów poniżej 60. rż. obecność zmian miażdżycowych tętnic szyjnych często współistnieje z OZW, jednak nie wpływa istotnie na rokowanie w tej grupie chorych.

**Słowa kluczowe:** ostry zespół wieńcowy, miażdżyca tętnic szyjnych, kompleks intima-media

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