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Carotid artery remodeling in acute coronary syndromes: Correlation with high sensitivity C-reactive protein

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Background: The carotid intima-media thickness (IMT) may be a marker of advanced coronary atherosclerotic disease. Inflammation might promote the development of athero-sclerosis, & high levels of C-reactive protein (CRP) are associated with an increased risk of acute coronary events (ACE).

Objectives: To investigate the relationship between ultrasonographic features of carotid arteries (CA), left ventricular (LV) high sensitivity (hs) CRP, & coronary remodeling features of coronary plaques in acute coronary syndromes (ACS).

Methods: The study enrolled 150 patients with ACS. Within one week of the acute event, the common CA were examined using B-mode ultrasonography. The CA, intimal and adventitial diameter (IAD) & luminal diameter (LD) were measured. Carotid plaques were assessed. Ultrasensitive immunoaassay was used to measure serum levels of hs-CRP. All patients underwent coronary angiography & according to the presence or absence of plaque remodeling, coronary plaques (CCP), patients were divided into 3 groups (G), simple plaque G (I), solitary complex plaque G (II)&multiple complex plaque G (III).

Results: No significant difference was detected between GI&G II as regard IMAT, AADs-CRP levels (0.92±0.19 mm vs. 0.93±0.21 mm, 7.75±0.49 mm vs 7.86±0.5 mm, 0.72±1.5 mg/L vs 7.1±1.68 mg/L respectively, p<0.05 for all.) While these variables were significantly higher in G III when compared to those of G I (1.1±0.2 mm vs 0.2±0.19 mm, 8.1±0.48 mm vs 7.75±0.49 mm, 9.97±3.02 mg/L vs 7.1±1.5 mg/L respectively, p<0.001 for all. )&those of G II (1.1±0.1 mm vs 0.86±0.1 mm, 8.1±0.48 mm vs 7.86±0.5 mm, 9.97±3.02 mg/L vs 7.1±1.68 mg/L respectively, p<0.001 for all). Strong positive correlations detected between IMAT & IAD (r=0.67 in G I, r=0.77 in G II, r=0.66 in G III) also between IMAT&CRP levels (r=0.61 in G I, r=0.57 in G II, r=0.88 in G III), p<0.001 for all. The prevalence of PCR was significantly higher in GII (50.7%) when compared to G I (15.38%) or GII (15.9%) p<0.001. In multivariate analysis, multiple CCP, dyslipidemia&Diabetes Mellitus were the strongest independent predictors (OR 3.87, 2.92, 2.86 & 95% Cl [1.65-9.07], [1.83, 4.77], [1.61, 3.21], respectively).

Conclusions: In ACS, multiple CCP are associated with more evident inflammatory process with higher hs-CRP levels, and PCR suggesting that plaque vulnerability may be a systemic phenomenon.

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Inhibition of Interleukin-1 activity improves coronary flow, endothelial function and aortic wall properties: a randomized cross-over, placebo-controlled, trial

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Background: Interleukin-1 plays an important role in the pathophysiology and progression of inflammation and may be involved in regulation of coronary vasomotor tone. Anakinra, a human recombinant interleukin-1 receptor antagonist, has been used as an anti-inflammatory agent in patients with rheumatoid arthritis. We investigated the effects of anakinra on endothelial function, arterial wall properties and coronary flow.

Patients and methods: 20 patients (female 4 male, median age 49 yrs) with rheumatoid arthritis were randomized to receive a single injection of anakinra (150 mg sc) or placebo and after 48 hours (drug’s 5 half lives) the alternative treatment (placebo or anakinra respectively). Before and 3 hours after each s.c. injection, we assayed a) the time integral of the coronary flow velocity (CF-VTI), the coronary flow reserve (CFR) of the LAD after adenosine infusion, and aortic strain, by means of echocardiography, b) flow mediated dilatation (FMD) and NTG-mediated dilatation (NTG-MD) by ultrasound on coronary arteries (CA), levels of high sensitivity (hs)-CRP & the angiographic features of coronary plaques in acute coronary syndromes.

Results: During the acute phase of the study (pre- and 3 hours post injection), a significant improvement in FMD and in CF-VTI and aortic strain was observed. Anakinra reduced plaque vulnerability (PCR) compared to placebo arm, while there were no differences between FMD as measured on consecutive post-STEMI days (see table 1).

Conclusion: IL-1a receptor antagonism improves coronary flow, endothelial function and aortic wall properties: a randomized cross-over, placebo-controlled, trial

Table 1. FMD and NTG-MD in study groups

<table>
<thead>
<tr>
<th>Baseline Stable CAD</th>
<th>STEMI</th>
<th>Controls</th>
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<tbody>
<tr>
<td>FMD</td>
<td>9.6±6.7</td>
<td>7.8±5.3</td>
</tr>
<tr>
<td>NTG-MD</td>
<td>16.7±9.9</td>
<td>18.3±5.9</td>
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p<0.05

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Stunned endothelium assessed by FMD - does it exist?

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Some hypotheses have been put forward on transient episodes of ischaemia resulting in irreversible endothelial dysfunction, a phenomenon referred to as «stunned endothelium».

Aim: to measure endothelium-dependent brachial artery flow-mediated dilation (FMD) and NTG-mediated dilation (NTG-MD) by ultrasound on consecutive post-STEMI days after 3 min occlusion, and following the administration of NTG infusion, and aortic strain, by means of echocardiography, b) flow mediated dilatation (FMD) and NTG-mediated dilatation (NTG-MD) by ultrasound on consecutive post-STEMI days after 3 min occlusion, and following the administration of NTG infusion, and aortic strain, by means of echocardiography.

Methods: 172 subjects were examined: 130 patients with stable CAD (CCS II/III; mean age: 61.1±8.9 years), 27-STEMI patients (mean age: 61.2±10.9 years; STEMI: anterior wall n=19; inferior wall n=8; treatment: primary percutaneous coronary intervention performed in 2-6 hours from the onset of symptoms), and 15 healthy controls (mean age: 51.4±4.4 years). Clinical data, pharmacotherapy, concomitant diseases were all assessed. FMD was measured by high-resolution ultrasound as the percent change of brachial artery diameter after 3 min occlusion, and following the administration of 0.4 mg sublingual nitroglycerin (NTG-MD) - in the STEMI patients the measurements were performed on 1st, 2nd, 3rd day post myocardial infarction.

Results: The FMD values were comparable in stable angina and in STEMI patients; the results were significantly decreased in comparison to the control. There were no differences between FMD as measured on consecutive post-STEMI days (see table 1).

Conclusion: CAD patients show endogenous vasodilator dysfunction, which is comparable in stable angina and STEMI. STEMI does not affect FMD and CF-VTI was demonstrated. In addition, a further improvement in FMD and CF-VTI was demonstrated. In addition, a further improvement in FMD and CF-VTI was demonstrated.

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