Endothelium-Dependent and -Independent Vasodilation in Young Males with Previous Myocardial Infarction

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Flow-mediated vasodilation – a non-invasively measurable index of endothelial function – is a good predictor of cardiovascular risk. This study was undertaken to analyse the correlation between flow-mediated vasodilation and the severity of coronary artery disease in males less than 40 years of age and with myocardial infarction in their history. Coronary angiography demonstrated single-vessel disease in 16 patients (Group A) and multi-vessel disease in 12 (Group B). The control group comprised 14 healthy young males (Group C). Endothelium-dependent vasodilation produced by reactive hyperaemia, as well as nitroglycerine-induced, endothelium-independent vasodilation was appraised on the brachial artery, using a high-resolution duplex ultrasound device (ACUSON 128XP/10). Variations in vessel size recorded as prescribed by the protocol developed by Celermayer were expressed as percentage change compared to baseline.

Compared to controls, endothelium-dependent vasodilation was attenuated in patients with previous myocardial infarction (p < 0.01). The same applies to multi-vessel disease, in comparison to single-vessel disease. There was no difference between controls and postinfarction patients as regards nitroglycerine-induced vasodilation.

Endothelium-dependent vasodilation is diminished in young males with previous myocardial infarction, and the magnitude of this reduction is related to the severity of coronary artery disease. J Clin Basic Cardiol 2003; 6: 73–6.

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ever, indirect appraisal of vasodilatation can be performed as a substitute [1].

Endothelium-dependent vasodilation is triggered by the activation of receptors (through the binding of acetylcholine, bradykinin, P-substance etc.) or distension of the vascular wall (ie, shear stress).

Non-invasive testing involves measuring the changes induced by acetylcholine administration (or the fluctuations of blood flow (ie, FMV) – in the diameter of vessels with duplex ultrasound [6]). The brachial or the femoral superficial artery is used instead of the coronaries, and vasodilatation is induced by reactive hyperaemia.

FMV is a function of the NO-production of the endothelium – vasodilatation is attenuated or absent if NO-production is reduced (eg, in atherosclerosis). In advanced coronary disease, paradox vasoconstriction can occur instead of vasodilation.

As demonstrated by Celermajer et al. [7], FMV is low before the age of 40. In males, ED appears at the end of the fourth – or, in females, the fifth – decade of life. The manifestations of ED are apparent in the whole population older than 65 years of age.

Although myocardial infarction is most prevalent among middle-aged males, it is not uncommon before 40 years of age and even without preceding angina.

**Objectives**

This study was performed to answer the following questions:

1. Can ED be demonstrated in myocardial infarction occurring in the young?
2. Is the magnitude of ED related to the extent of coronary artery disease?

**Study Population**

The study population comprised male patients aged 40 years or younger who had suffered myocardial infarction during the preceding 3 months. In view of their young age, coronary angiography was performed on all participants – irrespective of the verification of myocardial ischaemia. Diabetic patients were excluded in order to obtain homogeneous study groups.

Twenty-eight male patients with a mean age of 35.3 ± 3.7 years were enrolled. Participants were stratified into one of two groups, according to the angiographically verified extent of coronary artery disease. The control group consisted of males less than 40 years of age, without apparent CV risk factors or any significant disease in their history:

- **Group A** – single-vessel disease: n = 16
- **Group B** – multi-vessel disease: n = 12
- **Group C** – healthy controls: n = 14

The demographic properties of the study population are summarized in Table 1. There were no significant differences between individual parameters of Groups A and B.

### Table 1. The demographic properties of the study population

<table>
<thead>
<tr>
<th></th>
<th>A</th>
<th>B</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>16</td>
<td>12</td>
<td>14</td>
</tr>
<tr>
<td>Age (mean)</td>
<td>34</td>
<td>37</td>
<td>30</td>
</tr>
<tr>
<td>Vessel disease</td>
<td>1</td>
<td>2–3</td>
<td>–</td>
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<tr>
<td>Hypertension</td>
<td>6</td>
<td>6</td>
<td>0</td>
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<tr>
<td>Cholesterol 6.0 mmol/l</td>
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<td>4.6 mmol/l</td>
<td></td>
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<tr>
<td>HDL cholesterol</td>
<td>1.05 mmol/l</td>
<td>1.02 mmol/l</td>
<td>1.12 mmol/l</td>
</tr>
<tr>
<td>LDL cholesterol</td>
<td>3.2 mmol/l</td>
<td>3.56 mmol/l</td>
<td>2.8 mmol/l</td>
</tr>
<tr>
<td>Smoking</td>
<td>6</td>
<td>5</td>
<td>0</td>
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</tbody>
</table>

**Methods**

Non-invasive, risk-free testing was done during the morning hours, in an air-conditioned office with constant ambient temperature (22 °C). Patients had rested in recumbent position for at least 10 minutes before the measurement. The consumption of alcoholic beverages, tea and coffee, as well as smoking was prohibited on the day of testing. At least 24 hours must have elapsed since the ingestion of the latest dose of calcium channel blockers, β-adrenergic receptor blockers, long-acting nitrates, or ACE inhibitors.

Ultrasound scanning was performed on the right forearm, using an ACUSON 128 XP/10 duplex scanner equipped with a 7.5-MHz linear transducer.

Changes in the diameter of the brachial artery (FMV) were measured approximately 4 centimetres above the cubital fossa. The mean of three readings was recorded.

Baseline values were recorded and then a 5-minute supra-systolic compression was applied with a sphygmomanometer cuff. Three readings were taken during the 2-minute period following the deflation of the cuff. Endothelium-dependent changes in the diameter of the vessel caused by hyperaemia-related fluctuations in shear stress were recorded. The return of parameters to baseline values was verified by an additional series of measurements performed after 30 minutes of relaxation. Then, 3 readings were taken 90 seconds after the sublingual administration of 0.5 mg nitroglycerine to appraise endothelium-independent vasodilatation.

**Statistical Analysis**

Percentage changes compared to baseline were analysed by paired t-test.

**Results**

1. No significant changes were detected between study groups as regards the diameter of the brachial artery or the velocity of blood flow at baseline.
2. Endothelium-dependent vasodilatation:
   a) FMV was significantly lower in postinfarction patients than in healthy controls (4.95 ± 6.1 % vs. 15.5 ± 6.8 %; p < 0.001).
   b) As shown by coronary angiography, FMV was significantly lower in multi-vessel, than in single-vessel disease (2.1 ± 42 % vs. 7.1 ± 86 %; p < 0.001).

3. Endothelium-independent vasodilatation:
   a) The magnitude of nitroglycerine-induced vasodilatation was similar in postinfarction patients and in controls; however,
   b) it was (not significantly) lower in multi-vessel, than in single-vessel disease (Figure 2).

4. In four of the 28 postinfarction patients, hyperaemia-induced vasoconstriction, instead of vasodilatation. These patients had severe hypercholesterolaemia, and multi-vessel disease evidenced by coronary angiography.

**Discussion**

Non-invasive measurement of FMV was first described by Celermajer et al. [7, 8], who demonstrated the attenuation of this phenomenon in (adult and paediatric cases of) familial hypercholesterolaemia, in adult smokers, and in patients with coronary artery disease (CAD). The brachial artery is suitable for the evaluation of endothelial dysfunction, as its status correlates closely with atherosclerotic changes that occur in the coronary and carotid arteries. The brachial artery is
Flow-mediated vasodilation (FMV) in CAD and non-CAD patients

Figure 3.

Flow-mediated vasodilation (FMV) in 1-, 2-, 3-vessel disease

Figure 4.

Endothelium-dependent and -independent vasodilation in single- and multi-vessel disease

Figure 2.

References


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