Introduction

Silent myocardial ischaemia has been defined as objective evidence of (transient) ischaemia in the absence of chest pain or an anginal equivalent in a patient with coronary artery disease [1]. Cohn classifies persons with silent myocardial ischaemia into three categories:

Type I: Asymptomatic individuals with silent myocardial ischaemia

Type II: Patients with prior myocardial infarction and objective evidence of ischaemia in the absence of chest pain or an anginal equivalent

Type III: Patients with coronary artery disease who have both silent and painful episodes of ischaemia

Although it is generally accepted that silent myocardial ischaemia occurs in a considerable proportion of persons with ischaemic heart disease, the precise mechanisms involved remain to be elucidated [2, 3]. Both an increase in myocardial oxygen demand and abnormalities of coronary vasomotor tone may play a role in the occurrence of silent ischaemia. As angioplasty studies have shown that angina is the last in a chain of events to occur after occlusion of a coronary artery (occlusion of vessel → abnormalities of ventricular function → ischaemic ECG changes → chest discomfort), silent myocardial ischaemia may be the result of failure to reach the pain threshold or a higher threshold for pain stimuli. A generalized defective perception of pain stimuli has also been mentioned as a contributing factor to silent myocardial ischaemia. Silent myocardial ischaemia in type II and III patients is usually detected by ST-segment changes on ambulatory ECG monitoring or during ECG exercise testing. As asymptomatic persons suffering from coronary heart disease (type I) are not likely to undergo either exercise testing or ambulatory ECG monitoring, silent myocardial ischaemia usually goes unnoticed in these persons and reliable prevalence data are lacking.

Nevertheless, some insight into the magnitude of silent myocardial ischaemia in the population at large can be obtained from findings in population-based studies. Thirty years of follow-up in the Framingham Heart Study indicate that up to approximately 30% of initial myocardial infarctions (as evidenced by ECG) go unrecognized [5]. The percentages for men and women are 27 and 34%, respectively. About half of these “unrecognized” myocardial infarctions are truly “silent.” The other half reported symptoms attributed to other causes (e.g., alleged gallstone disease, peptic ulcer, etc.) that may in fact have been symptoms of myocardial infarction.

Silent myocardial infarction in the Rotterdam Study

As the Framingham Heart Study results indicate that silent myocardial infarction is an indicator of increased risk (both all-cause and cardiovascular disease mortality for survivors of an unrecognized infarction are comparable to that for survivors of a myocardial infarction that was recognized as such) and appears to be more prominent in elderly persons, we set out to investigate the prevalence of silent myocardial infarction, and factors related to the prevalence of silent myocardial infarction, in the Rotterdam Study.

The present preliminary results are based on information from the first 3,224 participants of the Rotterdam Study (mean age 69.8 years, SD 8.7, 61% women) in whom ECGs were obtained.

Table 1: Results of comparing selected characteristics of persons who sustained non-silent myocardial infarction.

<table>
<thead>
<tr>
<th></th>
<th>Silent MI</th>
<th>Non-silent MI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>134</td>
<td>99</td>
<td></td>
</tr>
<tr>
<td>Women (%)</td>
<td>55.2</td>
<td>27.2</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Age (years)</td>
<td>74.1 (9.0)</td>
<td>71.5 (8.1)</td>
<td>0.021</td>
</tr>
<tr>
<td>Pre-load glucose (mmol/l)</td>
<td>7.6 (3.2)</td>
<td>7.2 (2.7)</td>
<td>0.394</td>
</tr>
<tr>
<td>Post-load glucose (mmol/l)</td>
<td>7.7 (3.7)</td>
<td>6.4 (2.3)</td>
<td>0.003</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>146.5 (23.1)</td>
<td>135.5 (21.1)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>75.0 (14.2)</td>
<td>71.3 (11.5)</td>
<td>0.034</td>
</tr>
</tbody>
</table>

Values and numbers, percentages or means with standard deviation in parenthesis.

mm Hg increase in systolic and diastolic BP was associated with an increased risk of 23% (CI 8–42) and 33% (CI 6–67), respectively.

**Conclusion**

In conclusion, it appears that a substantial proportion of non-fatal myocardial infarction in the elderly remains unrecognized. "Silent" survivors of a myocardial infarction are more likely to be women, and to have an elevated blood pressure and post-load glucose level compared to those in whom the infarction was recognized. Yet, they have a risk to suffer from subsequent acute and chronic cardiovascular disease that is similar to patients who had their myocardial infarction recognized.

**Discussion**

Question:

If you think that autonomic dysfunction may be an important variable in the genesis of silent myocardial infarction, did you check for orthostatic hypotension in your cohort — because this might be a common denominator in the mechanism of the problem? And did you define this as a drop in systolic blood pressure of more than 20 mm Hg on standing blood pressure?

Answer:

Yes, we have done these measurements and data on orthostatic hypotension are available on all participants. Unfortunately we have not yet looked at the association with silent MI.

In fact, the complete database of the Rotterdam Study has only recently become available. But it is a very good suggestion which we will certainly follow.

**References**