Prevalence of coronary atherosclerosis in asymptomatic population

See page 45 for the article to which this Editorial refers

Atherosclerosis and especially coronary atherosclerosis is the leading cause of mortality and morbidity in the industrial world. Major advances in medical, interventional and surgical therapy, together with effective secondary prevention, has resulted in extended life expectancy and an improvement in the quality of life of most patients with clinical coronary artery disease. Despite these achievements, the prevalence of coronary artery disease seems to remain high. However, the exact data on the prevalence of coronary atherosclerosis or clinical coronary artery disease are extremely diverse (Table 1).

Autopsy studies in humans, who died from a non-cardiac cause, revealed manifest coronary artery disease in 4-5%\(^4\) and initial morphological (clinically silent) signs of aortic and coronary atherosclerosis in 50–100% of the young population below 35 years\(^9\). Clinical and/or ECG signs of coronary artery disease were found in 3–9% of the general population in north India\(^4\). Non-invasive tests are well designed for screening due to their non-invasive nature; however, their low specificity and sensitivity in asymptomatic populations leads to extremely variable results: the prevalence varies between 2·5 and 31·7\(^2\,7\,8\). The gold standard for the detection of significant coronary atherosclerosis is still coronary arteriography. Due to its invasive nature, it is not possible to use it for screening entirely asymptomatic (healthy) populations. Thus, only data from patients with other forms of cardiac diseases and without symptoms of coronary artery disease are known: the prevalence of significant coronary atherosclerosis among valvular/congenital heart disease patients is 4·5\(^1\), among mitral valve prolapse patients 13·5\(^5\) and among heavy smokers with emphysema referred for lung volume reduction surgery 15\%^6\.

Enbergs et al. in this issue\(^10\) present a relatively large group of 331 patients referred for catheter ablation therapy of cardiac arrhythmias. They underwent routine coronary arteriography as part of a diagnostic catheterization procedure. Despite the relatively high proportion of patients with chest pain unrelated to arrhythmias (21% of the group) and the higher then expected use of ‘coronary’ drugs, the prevalence of significant coronary artery disease (defined as at least one vessel with >50% stenosis) was low (7·3%) and was only slightly higher than among valvular/congenital heart disease. Furthermore, among patients without chest pain the prevalence was only 3·8% and the severity of lesions was very mild. Thus, this paper confirmed the low prevalence of obstructive coronary atherosclerosis in asymptomatic humans and provides a good scientific basis for the avoidance of routine coronary arteriography in this particular arrhythmia population.

Table 1 Studies on the prevalence of coronary or carotid atherosclerosis

<table>
<thead>
<tr>
<th>Authors</th>
<th>Study population</th>
<th>Methods</th>
<th>Atherosclerosis prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gensini GG, 1972(^1)</td>
<td>Valvular or congenital heart disease without symptoms of CAD (n=278)</td>
<td>CAG</td>
<td>4·5% (males 6%, females 3·4%)</td>
</tr>
<tr>
<td>Froelicher VV, 1976(^2)</td>
<td>Asymptomatic males (n=1390)</td>
<td>Exercise testing + CAG in positives</td>
<td>2·5%</td>
</tr>
<tr>
<td>Diamond GA, 1979(^3)</td>
<td>Asymptomatic humans, death unrelated to CAD General population in north India (n=3575)</td>
<td>Autopsy</td>
<td>4·5% (males 6·4%, females 2·6%)</td>
</tr>
<tr>
<td>Singh RB, 1997(^4)</td>
<td>General population in north India (n=3575)</td>
<td>Clinical history and ECG</td>
<td>9·0% (urban) 3·3% (rural)</td>
</tr>
<tr>
<td>Leung DY, 1997(^5)</td>
<td>Mitral valve prolapse patients (n=96)</td>
<td>CAG</td>
<td>13·5%</td>
</tr>
<tr>
<td>Thurnheer R, 1997(^6)</td>
<td>Heavy smokers with emphysema before lung volume surgery (n=41)</td>
<td>CAG</td>
<td>15%</td>
</tr>
<tr>
<td>Piolte L, 1998(^7)</td>
<td>Asymptomatic adults (n=4334)</td>
<td>Exercise treadmill testing</td>
<td>15% abnormal test 2·7% referred to CAG</td>
</tr>
<tr>
<td>Micieli G, 1998(^8)</td>
<td>Asymptomatic males (n=441)</td>
<td>Carotid sonography</td>
<td>31·7%</td>
</tr>
<tr>
<td>Strong JP, 1999(^9)</td>
<td>Autopsies, age 15–34 years, external cause of death (n=2876)</td>
<td>Autopsy, included intimal lesions and fatty streaks</td>
<td>Aorta: 100% RCA: &gt;50%</td>
</tr>
<tr>
<td>Enbergs A, 1999(^10)</td>
<td>Patients before radiofrequency ablation therapy (n=331)</td>
<td>CAG</td>
<td>7·3% (males 10·4%, females 3·8%)</td>
</tr>
</tbody>
</table>

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For an approximate screening of atherosclerosis, non-invasive methods may have limited value: the relationship between ultrasonographically detected carotid atherosclerosis and the presence of coronary atherosclerosis is an example. The weak correlation between the extent of coronary atherosclerosis and clinical symptoms of coronary artery disease remains another logistic problem. There is little consensus on the practical question: should an entirely asymptomatic patient with proven significant coronary atherosclerosis be treated by all available means including revascularization? Clearly, improving the possibility of establishing a diagnosis of coronary atherosclerosis in asymptomatic humans presents a new dilemma: full treatment or watchful waiting?

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References


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Blockade of the renin angiotensin system in heart failure: the potential place of angiotensin II receptor blockers

See page 53 for the article to which this Editorial refers

Neurohormonal activation is one of the hallmarks of chronic heart failure and inhibition of the neurohormonal systems, in particular the sympathetic nervous system and the renin angiotensin system, have become important targets in chronic heart failure. Indeed, the favourable effect of angiotensin converting enzyme (ACE) inhibitors in chronic heart failure has largely been attributed to blockade of angiotensin II production, the target hormone of the renin angiotensin system. For several years, however, it has been known that production or formation of angiotensin II may also occur via other, non-ACE pathways, such as the enzyme chymase[1]. This so-called escape from suppression of the renin angiotensin system during ACE inhibition is often encountered in circumstances of increased activity, such as chronic heart failure, but the clinical relevance of this escape is largely unknown.

In the present issue Roig et al.[2] report on 70 patients treated with full-dose ACE inhibitors, in whom plasma angiotensin II levels were examined. They found that 50% of patients had increased angiotensin II levels, despite high-dose enalapril or captopril. More importantly, they showed that these patients not only had higher plasma neurohormones, but that their prognosis was also significantly worse compared to those in whom angiotensin II remained suppressed.

Activation of the renin angiotensin system, and increased angiotensin II levels, are known to correlate

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