Sirolimus eluting stent aborted recurrent distal left main in-stent restenosis involving bifurcation

C H Lee, P A Lemos and P W Serruys

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Evidence for association between hepatitis C virus seropositivity and coronary artery disease

C Vassalle, S Masini, F Bianchi, G C Zucchelli

METHODS

The enrolment of patients was conducted according to previously described criteria. Our study population included 491 patients (92 females, 399 males), with a mean (SEM) age of 66 (0.5) years and with angiographic documentation of CAD (> 50% stenosis). A control group of 195 patients (8 females, 115 males) with a mean age of 61 (11.6) years was also recruited; these patients were hospitalised at the same institution for reasons other than suspected CAD (that is, valvar heart disease, cardiomypathy, and hypertensive heart disease) and had angiographically documented normal coronary arteries. At the time of blood sampling, all subjects gave a complete history which included cardiovascular risk factors such as smoking habits, hypertension, diabetes and dyslipidaemia. Each patient was found to be negative for hepatitis B, as evaluated by anti-hepatitis B surface antigen positivity. Patient exclusion criteria were severe liver damage and cirrhosis, acute or chronic inflammatory disease, immunological disease, and history or presence of neoplastic disease. In addition, patients with a stenosis < 50%, a stenosis on a minor vessel, or with atypical chest pain were excluded from the study.

Venous blood samples were collected under standardised conditions after an overnight fast and centrifuged within 15 minutes (3000 g for 10 minutes). Serum samples were immediately analysed for measurements of antibodies against HCV (AXSYM System, Abbott Laboratories, Illinois, USA). Intra-run and inter-run coefficient of variation (%CV) results were always < 8%. Specificity, calculated in 4383 blood donors, was found to be 98.84%. Data were expressed as the mean (SEM). Statistical analyses were performed using unpaired t tests, \( \chi^2 \) tests, and univariate and multivariate logistic regression (Statview statistical package, version 5.0.1, SAS Institute). A probability value of \( p \leq 0.05 \) was considered significant.

RESULTS

The percentage of HCV seropositivity was 2% in control subjects and 6.3% in the CAD group (\( \chi^2 = 5.3, p < 0.05 \)), increasing with the number of vessels affected (4.5% for one vessel disease, 6.6% for two vessel disease, and 8.4% for three vessel disease, \( p < 0.05 \)). Results of the univariate logistic regression analysis showed that in addition to other conventional atherogenic risk factors (age, sex, smoking habit, hypertension, diabetes, and dyslipidaemia), HCV seropositivity was found to be associated with the presence of CAD with an odds ratio of 3.2 (95% confidence interval (CI) 1.1 to 9.2, \( p < 0.05 \)) (table 1). After adjustment for other confounding risk factors, the multivariate logistic regression analysis

<table>
<thead>
<tr>
<th>Table 1 Predictors for coronary artery disease</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Odds ratio (95% CI)</strong></td>
</tr>
<tr>
<td><strong>Unadjusted</strong></td>
</tr>
<tr>
<td><strong>Adjusted</strong></td>
</tr>
<tr>
<td>Age</td>
</tr>
<tr>
<td>HCV seropositivity</td>
</tr>
<tr>
<td>Sex</td>
</tr>
<tr>
<td>Smoking habit</td>
</tr>
<tr>
<td>Hypertension</td>
</tr>
<tr>
<td>Diabetes</td>
</tr>
<tr>
<td>Dyslipidaemia</td>
</tr>
</tbody>
</table>

*Odds ratio derived from logistic regression analysis including all the variables listed in the table.

Abbreviations: CAD, coronary artery disease; HCV, hepatitis C virus
showed that HCV seropositivity still represented an independent predictor for CAD with an odds ratio of 4.2 (95% CI 1.4 to 13.0, p < 0.05) (table 1).

DISCUSSION
Several studies have suggested that some infectious agents may cause cellular and molecular changes that contribute to the pathogenesis of atherosclerosis. The data obtained indicate the identification of viral genomes in the atherosclerotic plaques and also pro-atherogenic effects of viral infection in cells relevant to atherogenesis (smooth muscle cells, monocyte macrophages, T cells, endothelial cells). Experimental models have also shown promotion and acceleration of atherosclerosis by infectious agents. Recent results indicate that infection by several different pathogens can confer high risk in both early and advanced atherogenesis (the first considered as the development of new plaques and the latter defined by the development/progression ratio of carotid stenosis). However, very little data about the possible relation between HCV infection and its associated putative pathogenic processes and atherosclerosis are available. Very recent data have indicated that seropositivity for HCV may have a role in the pathogenesis of carotid atherosclerosis. Our findings suggest that HCV seropositivity might be considered, in the clinical setting, as one of the risk factors affecting the onset and development of CAD. Further studies are needed at this point to verify the potential additive effect of HCV infection with respect to the presence of other pathogens. This study might be relevant for adding new predictive and prognostic factors to the CAD multifactorial entity.

REFERENCES