Racial Differences in Platelet Survival Time in Patients with Symptomatic Coronary Atherosclerosis


Platelet survival times were studied in 40 patients (21 white and 19 black) with coronary artery disease and stable effort induced angina pectoris. The platelet survival times of 19 white controls (9.27 ± 0.49 days; mean ± SD) were not significantly different from those of 12 black controls (8.88 ± 0.81 days), and the platelet survival times for 21 white patients with coronary artery disease (8.46 ± 0.65 days) were lower than the times for both the white controls (p < 0.01) and the combined control group (p < 0.01). However, the difference between the mean platelet survival times of 19 black patients (9.22 ± 0.68) and the control groups was not significant, and the difference between the mean platelet survival times of the 21 white patients and the 19 black patients was significant (p < 0.01). Stepwise multiple linear regression analysis indicated that race was the most significant factor in predicting shortened platelet survival (r = 0.4783; p < 0.01). It is concluded that racial background should be considered in the interpretation of platelet studies and that reported racial differences in the rate and extent of atherosclerotic lesions may be related to racial differences in platelet consumption. (Arteriosclerosis 3:138–140, March/April 1983)

The platelet survival time has been reported to be shortened in 50% to 65% of patients with symptomatic coronary atherosclerosis.1–4 We performed platelet survival studies as part of the initial evaluation of the effects of antiplatelet drugs in patients with stable effort angina pectoris and found fewer than one-third with shortened platelet survival times. This unexpected low incidence is explained by the finding that platelet survival times were decreased less often in our black than in our white patients.

Methods

Platelet Survival Testing

One unit of autologous platelets was collected, labelled with 150 to 200 μC of Na251CrO4, washed, and reinfused into the subject according to the method of Aster as modified by Harker.5 Blood samples were collected into disodium edatate anticoagulant daily for 7 to 9 days, and the platelets were extracted by differential centrifugation. Platelet radioactivity was measured in a well-type gamma counter, and a weighted platelet survival time was determined using a computer program based on a linear-exponential model of platelet disappearance.6 Platelet survival times were studied in 31 control subjects. All were believed to be in good health and were taking no medication. Twelve controls were black (10 males; 2 females) and 19 white (16 males; 2 females). Smoking history and serum cholesterol and triglyceride values were recorded.

Forty outpatients volunteered for the study. All patients met the criteria of the New York Heart Association for stable effort angina pectoris. All had treadmill exercise tests which produced ischemic ST segment response, and coronary arteriograms which showed greater than 50% luminal diameter narrowing of one or more coronary arteries. No patients were taking drugs known to affect platelet function, except for sublingual nitroglycerin for symptomatic control of stable angina pectoris. Beta-adrenoceptor blocking drugs were discontinued before the platelet survival test.

The following data were recorded: age, smoking history, fasting serum cholesterol and triglyceride levels, presence of Q waves on electrocardiogram, and the number of coronary arteries involved by greater than 5% luminal diameter narrowing. The extent of coronary artery involvement was also quan-
titated by means of a coronary angiography scoring system. Statistical analysis was performed using Student's two-tailed t test and stepwise multiple linear regression analysis.

Results

The platelet survival times of 19 white controls (9.27 ± 0.49 days) was not significantly different from those of 12 black controls (8.88 ± 0.81 days) (Table 1). The mean platelet survival time for the combined control group (9.12 ± 0.62 days) was similar to that reported for normal subjects. No statistical difference existed in age, race, smoking history, or serum cholesterol and triglyceride concentrations.

The platelet survival times for the 21 white patients with coronary artery disease (8.46 ± 0.65 days) were lower than the times for both white controls (p < 0.001) and the combined control group (p < 0.01). Ten of 21 white patients (48%) had platelet survival times shorter than 2 SD below the means of both the white and the combined control groups; however, the platelet survival times of the 19 black patients with coronary artery disease (9.22 ± 0.68 days) was not significantly different from either the black controls alone or the combined control group. None of the 19 black patients had a platelet survival time shorter than 2 SD below the mean of either of these two control groups (Table 1).

The difference between the mean platelet survival times of the 21 white patients and the 19 black patients was significant (p < 0.01) (Table 1). The black patients as a group had a significantly higher level of serum cholesterol than the white patients (p < 0.05). No significant differences in age, smoking history, serum triglycerides, presence of Q waves on electrocardiogram, or angiographic findings between the white vs the black groups of patients were found (Table 2). The mean score for coronary artery involvement for the white group was 8.06 ± 3.47 and for the black group was 8.22 ± 3.44 (p not significant). Ranking of variables by stepwise multiple regression analysis demonstrated that race was the most significant factor in predicting shortened platelet survival (R = 0.488873; p < 0.01).

Table 2. Clinical Characteristics of 40 Patients with Symptomatic Coronary Artery Disease

<table>
<thead>
<tr>
<th>Clinical characteristic</th>
<th>White (n = 21)</th>
<th>Black (n = 19)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Platelet survival time</td>
<td>8.46 ± 0.65</td>
<td>9.22 ± 0.68*</td>
</tr>
<tr>
<td>Age (yrs)</td>
<td>53.5 ± 7.6</td>
<td>54.5 ± 8.0</td>
</tr>
<tr>
<td>Serum cholesterol (mg/dl)</td>
<td>214.6 ± 28.9</td>
<td>245.8 ± 51.5†</td>
</tr>
<tr>
<td>Serum triglycerides (mg/dl)</td>
<td>192.4 ± 90.1</td>
<td>158.4 ± 89.5</td>
</tr>
<tr>
<td>No. of diseased vessels</td>
<td>19 ± 0.7</td>
<td>19 ± 0.8</td>
</tr>
<tr>
<td>(&gt;50% narrowing)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking history</td>
<td>11.2 ± 14.4</td>
<td>14.6 ± 13.1</td>
</tr>
<tr>
<td>(cigarettes per day)</td>
<td>38.9 ± 20.7</td>
<td>23.8 ± 17.9</td>
</tr>
</tbody>
</table>

Values are means ± sd.

*p < 0.001.
†p < 0.05.

Discussion

Platelet survival time is reported to be shortened in groups of patients with coronary atherosclerosis and, to a lesser extent, in patients with other atherosclerotic conditions. We observed similar findings in a group of white patients with symptomatic coronary artery disease. However, for a group of black patients with symptomatic coronary artery disease, the platelet survival times were not significantly different from those of our controls.

Cigarette smoking and cigarette smoking by fatty acids in platelets have all been associated with the shortening of platelet survival time. None of these factors appears to explain the difference in platelet survival times found in our patients. Interestingly, our group of black patients had a significantly higher level of total serum cholesterol than our white patients (p < 0.05) although their platelet survival times were longer. However, we did not measure plasma concentration of high density and low density lipoprotein cholesterol. Racial differences in lipoprotein concentration may be a factor in explaining the reduced rate of coronary heart disease in blacks.

Platelet survival time is inversely proportional to the rate of platelet consumption, so that the shortened times seen in patients with atherosclerosis are believed to reflect increased platelet consumption. In addition, substances released by platelets as they are utilized (e.g., platelet factor 4) may be elevated in patients with coronary artery disease; however, evidence for elevated platelet factor 4 in coronary
heart disease has been contested.15 Evidence of increased platelet consumption in atherosclerotic conditions may reflect intrinsic abnormalities of platelet function, abnormalities of the blood vessel wall, or the presence of circulating factors capable of altering the function of otherwise normal platelets or blood vessels. An increasing array of evidence, both from animal and human studies, suggests that platelets may play a key role in the development of atherosclerosis.16 Our platelet survival data suggest that those black patients who develop symptomatic coronary artery disease are less likely to show evidence of platelet consumption than white patients.

Coronary artery disease is remarkably rare in black African populations17,18 and is less common in blacks than whites in the United States.19,20 In addition, racial differences in the extent of atherosclerotic lesions have been noted in extensive postmortem studies of patients from 16 cities worldwide.21 In three locales (New Orleans, Puerto Rico, and Sao Paulo) data from white and black subjects were compared and atherosclerosis was found to be more extensive in whites than in blacks on the basis of percentage of arterial surface involved by raised atherosclerotic plaques. Among New Orleans whites in the 55- to 64-year age groups, 52% of the surface of the abdominal aorta, and 30% of the surface of the coronary arteries was involved by atherosclerotic plaques. Among New Orleans blacks in the same age group, the numbers were 34% and 24% respectively.22 We could find no difference between white and black patients in the extent of coronary artery involvement by angiography. However, the coronary artery scoring techniques are crude and, furthermore, may not necessarily represent the extent of involvement throughout the entire arterial system.

When the amount of arterial surface involved by fatty streaks was measured in younger age groups, either no racial difference was seen, or blacks were shown to have slightly more extensive involvement.22 This last point may be important because platelet-derived material can be detected in raised atherosclerotic plaques, but not in fatty streaks.23 Thus, one might infer that white blacks deposit as much lipid as whites on their arterial intimal surface, the process of turning these fatty streaks into raised atherosclerotic plaques — a process which seems to involve platelets— is less extensive in blacks.

Our platelet survival data support the idea that racial differences in the rate and extent of atherosclerotic lesions may be related to racial differences in platelet consumption. If so, this might be due either to true inherent differences, or to other factors such as differences in diet, lifestyle, or lipoprotein distribution. Thus, racial background should be considered in the interpretation of platelet survival studies.

References


Index Terms: platelets • platelet survival • atherosclerosis • coronary artery disease
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