**Serum Lipids, Platelets, and Fibrinolytic Activity in Cerebrovascular Disease**


**Summary** Fifty patients with occlusive cerebrovascular disease (ischemic thrombotic cerebrovascular disease — ITCBVD) were studied for clinical features, angiographic findings, serum lipids, platelet functions and fibrinolytic activity. Angiograms were abnormal in 24 of 36 cases. Two-thirds of these had an abnormality of the internal carotid artery in the neck; one-third had occlusion of the middle and/or anterior cerebral arteries. A statistically significant rise of serum triglycerides, pre-beta lipoproteins, platelet adhesiveness and aggregation, and a decrease in fibrinolytic activity were noticed in these patients as compared to age and sex matched controls. The correlation coefficient did not show any intercorrelation between the platelet adhesiveness and raised lipid fractions. These factors could be responsible for the atheroma resulting in large vessel occlusion.

ETIOLOGICAL factors responsible for many cases of cerebral infarction are not understood. It is uncertain what part is played by elevated blood lipids, enhanced platelet adhesiveness and aggregation, and decreased fibrinolytic activity in the pathogenesis of occlusive cerebrovascular disease. Varied alterations in lipid fractions have been reported from the U.S. and Japan, as well as from India. The role of the platelets in the pathogenesis of cerebrovascular disorders was discussed in a 1974 International Round Table Conference in Rome and it has been shown that platelet aggregation is increased in these disorders. Wu and Hoak have also found increased platelet aggregation in patients with transient ischemic attacks. Increase in platelet adhesiveness has been reported in cerebrovascular disease by earlier workers. The present study was undertaken to assess the status of lipids and two hematological factors of the blood coagulation system (platelet function and fibrinolytic activity) simultaneously in patients from Haryana (north India) who had occlusive cerebrovascular disease.

**Methods**

Fifty consecutive cases of ischemic thrombotic cerebrovascular disease (ITCBVD) were studied. All suddenly developed neurological deficits and satisfied currently accepted diagnostic criteria for stroke. The patients with recognized predisposing factors like hypertension, diabetes and syphilis were excluded. Patients with cerebral venous sinus thrombosis occurring during puerperium, patients with cerebral or subarachnoid hemorrhage or patients with any possible cardiac cause for embolism were also excluded. Clinical history and detailed physical examinations were recorded on special forms. The following studies were made immediately after the admission of the patient: from fasting A.M. blood samples hemoglobin, total and differential leukocyte count, also urinalysis, stool, E.S.R., blood and S.T.S., blood sugar, blood urea, complete C.S.F., serum triglycerides, serum phospholipids, serum free fatty acids, serum lipoproteins.

Age and Sex

There were 33 male and 17 female patients. The mean age was 55.5 years (SD ± 15.15). Maximum cases in either sex were in 6th and 7th decade. Clinical features are shown in table 1.

**Other Findings**

The carotid angiograms were abnormal in 24 of 36 patients (table 2). There were cerebral vessel abnormalities in 65.4% of group B and 69.1% (9 of 13) in group C. Mean levels of most lipid fractions were higher in groups A, B and C but the rise was statistically significant ($P < 0.05$) for serum triglycerides and pre-beta lipoproteins only. No rise in the mean level of serum cholesterol was observed in these patients. A statistically significant increase in the mean level of serum triglycerides, pre-beta lipoproteins and serum cholesterol was found in group D as compared to group E ($P < 0.05$).

Platelet adhesiveness and aggregation were increased in all groups of patients as compared to controls and this increase was statistically significant ($P < 0.05$). In group D and E the difference was not statistically significant ($P > 0.05$).

The correlation coefficient between platelet adhesiveness and each of the lipid fractions was calculated in order to assess the correlation between enhanced platelet adhesiveness and elevated lipid fractions. No statistically significant correlation was observed between...
rise in platelet adhesiveness and rise in levels of any of the lipid fractions (table 5). There was a decrease in the level of fibrinolytic activity as measured by whole blood clot lysis in agreement with most of the earlier workers.10-3l34 The statistically significant decrease was statistically significant ($P < 0.05$) (table 6).

**Discussion**

The patients in this study were a selected group in which the known predisposing causes of CBVD, such as diabetes, hypertension, and cardiac causes of embolism, were not operative. Abnormalities in large cerebral vessels had been found by angiography in two-thirds of the patients. Sixty-six percent of the abnormalities were in the internal carotid artery (extracranial) and 20.8% were in the middle cerebral artery (extracranial portion). The statistically significant ($P < 0.05$) rise observed in serum triglycerides and pre-beta lipoprotein fractions was in agreement with most of the reported studies.3-4 Lack of rise in serum cholesterol levels in such patients was also similar to previous reports.1-3-8-10 These lipid abnormalities in all subgroups were identical. Platelet adhesiveness and aggregation were increased in all groups of patients and the rise was statistically significant ($P < 0.05$). This finding is similar to previous reports.5-10-18 The correlation coefficient did not reveal any intercorrelation between platelet adhesiveness or the raised lipid fractions. This suggests that platelet abnormalities may be one of the factors in the pathogenesis of atheroma, independent of the lipid changes. A statistically significant decrease

### Table 3 Comparison of Serum Lipids in Various Groups

<table>
<thead>
<tr>
<th></th>
<th>Serum triglycerides mg%</th>
<th>Serum phospholipids mg%</th>
<th>Serum fatty acids (o/o/L)</th>
<th>Serum cholesterol mg%</th>
<th>Serum lipoproteins Beta</th>
<th>Pre-beta</th>
<th>Alpha</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Group A</strong></td>
<td>135 ± 42.02*</td>
<td>191 ± 57.17*</td>
<td>65.8 ± 340.30*</td>
<td>189.04 ± 34.67*</td>
<td>58.7 ± 11.65*</td>
<td>28.04 ± 7.00*</td>
<td>16.45 ± 7.0*</td>
</tr>
<tr>
<td><strong>Controls</strong></td>
<td>92 ± 18.71</td>
<td>190 ± 56.22</td>
<td>574 ± 166.41</td>
<td>185.42 ± 36.58*</td>
<td>57.77 ± 11.51</td>
<td>19.36 ± 3.71</td>
<td>19.35 ± 2.17</td>
</tr>
<tr>
<td><strong>p value</strong></td>
<td>&lt;0.05</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td><strong>Group B</strong></td>
<td>135 ± 56.23</td>
<td>184 ± 50.06</td>
<td>701 ± 339.07</td>
<td>194 ± 36.31</td>
<td>54.80 ± 11.27</td>
<td>29.32 ± 7.78</td>
<td>16.64 ± 6.95</td>
</tr>
<tr>
<td><strong>Controls</strong></td>
<td>104 ± 14.96</td>
<td>199 ± 31.59</td>
<td>562 ± 199.95</td>
<td>189 ± 41.13</td>
<td>58.83 ± 2.54</td>
<td>22.03 ± 5.05</td>
<td>19.93 ± 2.67</td>
</tr>
<tr>
<td><strong>p value</strong></td>
<td>&lt;0.05</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
<td>&lt;0.01</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td><strong>Group C</strong></td>
<td>126 ± 43.69</td>
<td>199 ± 65.08</td>
<td>561 ± 283.86</td>
<td>179 ± 20.02</td>
<td>52.78 ± 13.06</td>
<td>27.26 ± 5.47</td>
<td>17.26 ± 5.58</td>
</tr>
<tr>
<td><strong>Controls</strong></td>
<td>99 ± 21.65</td>
<td>210 ± 35.23</td>
<td>540 ± 160.87</td>
<td>192 ± 30.94</td>
<td>58.84 ± 5.0</td>
<td>21.65 ± 4.17</td>
<td>19.0 ± 2.81</td>
</tr>
<tr>
<td><strong>p value</strong></td>
<td>&lt;0.05</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
<td>&lt;0.01</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td><strong>Group D</strong></td>
<td>139 ± 45.33</td>
<td>157 ± 60.64</td>
<td>561 ± 313.62</td>
<td>190 ± 30.72</td>
<td>53.75 ± 11.54</td>
<td>28.41 ± 6.2</td>
<td>17.16 ± 7.42</td>
</tr>
<tr>
<td><strong>Group E</strong></td>
<td>97 ± 30.66</td>
<td>209 ± 53.60</td>
<td>679 ± 336.24</td>
<td>170 ± 25.09</td>
<td>57.37 ± 3.03</td>
<td>20.33 ± 2.05</td>
<td>20.12 ± 2.66</td>
</tr>
<tr>
<td>p value</td>
<td>&gt;0.05</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

*Significant.*

### Table 4 Comparison of Platelet Abnormalities in Various Groups

<table>
<thead>
<tr>
<th>Group</th>
<th>Platelet adhesiveness (%)</th>
<th>Platelet aggregation time (in seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A Patients</td>
<td>66 ± 5.76*</td>
<td>19 ± 3.54*</td>
</tr>
<tr>
<td>Controls</td>
<td>49 ± 4.25</td>
<td>43 ± 8.74</td>
</tr>
<tr>
<td>B Patients</td>
<td>57 ± 5.74</td>
<td>19 ± 3.02</td>
</tr>
<tr>
<td>Controls</td>
<td>48 ± 3.86</td>
<td>44 ± 8.83</td>
</tr>
<tr>
<td>C Patients</td>
<td>67 ± 5.06</td>
<td>19 ± 3.53</td>
</tr>
<tr>
<td>Controls</td>
<td>50 ± 4.41</td>
<td>44 ± 8.25</td>
</tr>
<tr>
<td>D Patients</td>
<td>67 ± 6.04</td>
<td>19 ± 3.0</td>
</tr>
<tr>
<td>E</td>
<td>69 ± 5.98</td>
<td>18 ± 3.60</td>
</tr>
</tbody>
</table>

*Significant.*
TABLE 5 Intercorrelation of Platelet Adhesiveness and Lipid Fraction in Various Groups of Patients

<table>
<thead>
<tr>
<th>Platelet adhesiveness</th>
<th>Triglycerides</th>
<th>Phospholipids</th>
<th>Free fatty acids</th>
<th>Cholesterol</th>
<th>Pre-beta lipoprotein</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>0.0642</td>
<td>0.2222</td>
<td>0.0076</td>
<td>0.2186</td>
<td>0.3545</td>
</tr>
<tr>
<td>Group B</td>
<td>0.2915</td>
<td>0.0860</td>
<td>0.1509</td>
<td>0.0040</td>
<td>0.2765</td>
</tr>
<tr>
<td>Group C</td>
<td>0.1043</td>
<td>0.2245</td>
<td>0.1844</td>
<td>0.2972</td>
<td>0.3192</td>
</tr>
<tr>
<td>Group D</td>
<td>0.0439</td>
<td>0.1509</td>
<td>0.0542</td>
<td>0.1508</td>
<td>0.1902</td>
</tr>
</tbody>
</table>

None of these values was statistically significant (p > 0.05).

Group A: Patients as a whole.
Group B: Patients above 40 years of age.
Group C: Patients below 40 years of age.
Group D: Patients with abnormal angiograms.

in the level of fibrinolytic activity was seen in patients as compared to controls (P < 0.05). This was due to decrease in plasminogen activator or plasmin. Menon and Pilgeram have described decreased level of plasminogen activator associated with ITCBVD. Saroop and Chandrasekar, however, found an imbalance between fibrinogen and fibrinolytic activity in favor of the former. This study, therefore, indicates that there is an increase in serum triglycerides, pre-beta lipoproteins, platelet adhesiveness and aggregation and a decrease in fibrinolytic activity in ITCBVD. These changes may be causative or associated with the cause of atheroma and alteration of blood coagulability.

References

Serum lipids, platelets, and fibrinolytic activity in cerebrovascular disease.
B C Bansal, C Prakash, R K Arya, S K Gulati and S C Mittal

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