Serum Lipids, Platelets, and Fibrinolytic Activity in Cerebrovascular Disease


SUMMARY Fifty patients with occlusive cerebrovascular disease (ischemic thrombotic cerebrovascular disease — ITCBV D) 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. Increased 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.

From the Medical College, Rohtak (Haryana), India.

Dr. Bansal is Associate Professor of Medicine, Dr. Prakash is Professor and Head of Department of Medicine, Dr. Gulati and Dr. Mittal are Registrars of Medicine. Dr. Arya is Professor of Pathology, Medical College, Jammu.

Reprint requests to Dr. Bansal, 15/8FM, Medical College, Rohtak (Haryana) India.
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 and euglobulin clot lysis time in all the groups and this 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. These findings were in accordance with those of earlier workers.10-3134 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.5,18 Lack of rise in serum cholesterol levels in such patients was also similar to previous reports.1,8,7,8,10 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.5,18-20

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
LIPIDS, PLATELETS AND LYSIS IN CBVD/Bansal et al. 139

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>0.0360</td>
<td>0.1509</td>
<td>0.0690</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>0.1598</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.

Table 6  Fibrinolytic Activity in Various Groups

<table>
<thead>
<tr>
<th>Groups</th>
<th>Whole blood clot lysis time (hrs)</th>
<th>Bunglubolin clot lysis time (hrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A Patients</td>
<td>36 ± 38.2*</td>
<td>10 ± 5.9*</td>
</tr>
<tr>
<td>Controls</td>
<td>22 ± 16.8</td>
<td>6 ± 3.8</td>
</tr>
<tr>
<td>p value</td>
<td>&lt;0.05</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>B Patients</td>
<td>27 ± 50.16</td>
<td>10 ± 6.1</td>
</tr>
<tr>
<td>Controls</td>
<td>24 ± 29</td>
<td>6.5 ± 4.5</td>
</tr>
<tr>
<td>p value</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>C Patients</td>
<td>33 ± 24.4</td>
<td>10 ± 5.9</td>
</tr>
<tr>
<td>Controls</td>
<td>18 ± 10.5</td>
<td>6 ± 3.8</td>
</tr>
<tr>
<td>p value</td>
<td>&lt;0.05</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>D Patients</td>
<td>30 ± 29</td>
<td>9 ± 5.8</td>
</tr>
<tr>
<td>E Patients</td>
<td>33 ± 22.8</td>
<td>9 ± 3.7</td>
</tr>
<tr>
<td>p value</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

* p < 0.05

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

Stroke. 1978;9:137-139
doi: 10.1161/01.STR.9.2.137

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/9/2/137

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org/subscriptions/