SPONTANEOUS INTRACEREBRAL HEMATOMAS (CH) constitute about a 10% of stroke cases. Since CH treatment is poor, prevention is at least as important as for cerebral infarction. Risk factors for CH occurrence have been scarcely studied. Furthermore, these studies were carried out before CT scan was available. It has been shown that many cases of CH can be misdiagnosed as cerebral infarction if CT scan is not used for diagnosis. These facts prompted us to study risk factors in a case-control study of our population of CT scan diagnosed cerebral hematomas.

Patients and Methods

This is a retrospective case-control study carried out on 73 CH patients consecutively admitted to the Department of Neurology. Inclusion criteria were: evidence of spontaneous CH on CT scan (isolated subarachnoid or intraventricular hemorrhage and traumatic CH were excluded); admission within the week following the development of stroke; absence of cerebral tumor, aneurysm or angioma (on CT scan or angiography). The control group was formed by 73 patients, paired in age and sex with cases, selected from neurological patients devoid of cerebrovascular or toxic-nutritional disease (excluding also dementia, loss of consciousness, epilepsy, parkinsonism and cerebellar syndrome patients). Data analyzed were the following: history of hypertension (with or without treatment), history of alcohol consumption (more than 80 grams per day at least in the previous year), history of liver disease or current evidence (clinical or pathological) of hepatopathy and evidence of coagulation disorder or previous anticoagulant treatment. Blood pressure (BP) levels were measured on admission and after 3 days. Blood glucose, cholesterol, triglyceride and hematocrit were measured within the first three days after admission. Electrocardiogram (EKG), performed in the week following admission, was considered to be abnormal when showing evidence of myocardial infarction, atrial fibrillation or flutter and ventricular hypertrophy. Statistical significance in differences between groups was studied by means of relative risk ratio for paired samples (including two-tailed McNemar test) when dealing with proportions, and Student's t test (for paired samples) for quantitative variables. In the group of cases a factorial analysis (by the method of main components with varimax rotation) was performed to determine the most evident relationships among those clinical variables previously identified as significant risk factors. With data from cases and controls a stepwise discriminant analysis was carried out to establish the relative weight of the different risk factors and their discriminant values. These tests were done on a IBM 370 computer with programs BMDP4M and 7M.

Results

In each of the groups (cases and controls) there were 52 males and 21 females, and the mean age was 58 years (range: 23 to 82). Results from qualitative data are set out in table 1. The most relevant factors were history of hypertension and alcoholism. Also significant were the presence of hepatic disease and EKG abnormalities. Quantitative data are summarized in table 2. It is evident that the significance of high blood pressure levels disappears when instead of admission BP the third day BP is analyzed. No patient was diagnosed of blood disorder and only one was on chronic anticoagulant therapy. The hepatic disorder was diagnosed on clinical grounds (by symptoms, signs and laboratory abnormalities) in 9 cases and by liver biopsy in 6. Eight were considered to be related to chronic alcoholism. Coagulation studies in these cases were either normal or showed moderate abnormalities (in no case platelet counts under 70,000/cc. or prothrombin time under 40%).

Results of factorial analysis are set out in table 3. In factor 1 positive variables are males, alcoholism, hematocrit and hepatic disease and negative variable is age. Factor 2 is defined by positivity of blood glucose, EKG abnormalities and age. Factor 3 is defined by positive variables hematocrit and hypertension. These three factors explain 58% of total variance. On a first discriminant analysis a correct classification of 63.2% was obtained with the variables blood glucose and hematocrit. The other variables did not add a significant weight. Since blood glucose could be elevated because of the brain lesion a new analysis was performed excluding that variable. In it a correct classifi-
TABLE 1 Results of Qualitative Factors in CH

<table>
<thead>
<tr>
<th>Factor</th>
<th>Cases</th>
<th>Controls</th>
<th>Relative Risk Ratio</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of hypertension</td>
<td>46.5</td>
<td>9.5</td>
<td>4.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Smoking</td>
<td>12.3</td>
<td>12.3</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Alcoholism</td>
<td>39.7</td>
<td>5.4</td>
<td>7.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hepatopathy</td>
<td>20.5</td>
<td>4.1</td>
<td>5</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>EKG abnormalities</td>
<td>35.6</td>
<td>12.3</td>
<td>2.8</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Discussion

Controlled series studying risk factors for cerebral hematomas are few and with several insufficiencies. In none was CT scan considered essential for diagnosis and in some the hemorrhage group included subarachnoid and intraventricular hemorrhages. These series point out as main risk factors hypertension, chronic alcoholism, EKG abnormalities and funduscopic abnormalities. We have considered that it is important to base the diagnosis of CH on CT scan since up to a 24% cation of 65.7% was obtained with alcoholism and hypertension. The absence of significant relationship between hypertension and alcoholism, hypertension and hematocrit and alcoholism and hepatic disease was confirmed by use of chi-square and t tests.

No significant differences were found in risk factors when comparing lobar and basal ganglia hematomas (there were 41 basal ganglia hematomas, 28 lobar and 5 cerebellar).

TABLE 2 Results of Quantitative Factors in CH

<table>
<thead>
<tr>
<th>Factor</th>
<th>Cases mean (sd)</th>
<th>Controls mean (sd)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic BP on admission (mm Hg)</td>
<td>167 (31)</td>
<td>143 (23.5)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Diastolic BP on admission</td>
<td>98 (18)</td>
<td>85 (13)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Systolic BP on the third day</td>
<td>148 (27)</td>
<td>143 (23.5)</td>
<td></td>
</tr>
<tr>
<td>Diastolic BP on the third day</td>
<td>86 (15)</td>
<td>85 (13)</td>
<td></td>
</tr>
<tr>
<td>Blood glucose (mg/dl)</td>
<td>134 (56)</td>
<td>103 (30)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Blood triglycerides (mg/dl)</td>
<td>140 (59)</td>
<td>124 (71)</td>
<td></td>
</tr>
<tr>
<td>Blood cholesterol (mg/dl)</td>
<td>206 (50)</td>
<td>203 (39)</td>
<td></td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>47.5 (5)</td>
<td>44 (7)</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

TABLE 3 Results of Factorial Analysis

<table>
<thead>
<tr>
<th>Factor</th>
<th>Communality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>0.810</td>
</tr>
<tr>
<td>Alcoholism</td>
<td>0.701</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>0.607</td>
</tr>
<tr>
<td>Glycemia</td>
<td>0.0</td>
</tr>
<tr>
<td>EKG abnormalities</td>
<td>0.0</td>
</tr>
<tr>
<td>Hypertension</td>
<td>0.0</td>
</tr>
<tr>
<td>Age</td>
<td>0.432</td>
</tr>
<tr>
<td>Hepatic disease</td>
<td>0.482</td>
</tr>
<tr>
<td>VP</td>
<td>25%</td>
</tr>
</tbody>
</table>

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those obtained on admission and it has been shown that immediately after an acute stroke blood glucose levels tend to suffer a transient increase in many cases. So, we did not think that hyperglycemia should be considered as a definite risk factor. An abnormally high hematocrit, such as found in our CH cases had not been previously reported. A relationship between hypertension and hematocrit has been found to occur in some cases. In our study we could not find such correlation.

Some of the main risk factors for CH are different from risk factors for cerebral ischemia and infarction. Whereas hypertension is a strong factor in both entities, chronic alcoholism is not risk factor for cerebral infarction, except in some young adults. Smoking and diabetes are risk factors for infarction but not for CH. Other risk factors related to cerebral ischemia such as angina pectoris and peripheral vascular disease have not been found to be related to CH. Physiopathology is also different in CH and cerebral ischemia and infarction. CH is associated with lipohyalinosis and fibrinoid necrosis of small arteries, lesions usually related to chronic hypertension (and quite similar to those described in lacunar infarctions). Cerebral thrombotic infarction is often related to atheromatous lesions of greater vessels in which several systemic factors besides hypertension play a pathogenetic role.

Better control of hypertension has been associated with a reduced incidence of stroke, ischemic and hemorrhagic. The role of alcohol consumption reduction and control of hematocrit values on the incidence of CH seems worthy of study.

References
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