The aim of this study was to assess if heavy alcohol drinking influences blood flow velocity in cerebral arteries. Blood flow velocity (V mean) in the middle cerebral artery was measured by transcranial Doppler sonography (TCD) in heavy alcohol drinkers. Significantly decreased V mean was found in comparison with healthy volunteers.

INTRODUCTION

The aim of this study was to assess if heavy alcohol drinking influences blood flow velocity in cerebral arteries. It is well known that decreased cerebral blood flow velocity is present in patients with stroke. It can be explained by increased blood viscosity, which has been reported by Dafydd (1992). Increased viscosity results in a decrease in cerebral blood flow. Hypertension, heart disease and diabetes mellitus have been identified as risk factors in ischaemic brain infarction. Heavy current weekly alcohol consumption in men has been described as an independent risk factor for stroke (Gill et al., 1986; Hillbom and Juvela, 1996; Hillbom et al., 1999). Alcohol and stroke became the focus of epidemiological research about 15 years ago. Experimental studies have revealed both beneficial and untoward actions of alcohol with respect to the development of stroke, but the relationship between alcohol and stroke is still intriguing (Hillbom, 1987; Gorelick et al., 1989; Crews et al., 1998).

Transcranial Doppler sonography (TCD) is a non-invasive method which can measure cerebral blood flow velocity. That is why we have decided to use this method to study blood flow velocity in heavy alcohol drinkers, to see if there are changes, as in patients with stroke.

MATERIALS AND METHODS

The study comprised 53 patients (all men), who were heavy alcohol drinkers, mean age 45 years, hospitalized in the Department of Psychiatry at the Faculty Hospital in Košice. One patient had been drinking alcohol for 5 years, nine for <10 years and all other patients for >10 years. They were drinking hard spirits in combination with beer most frequently. One patient had been drinking alcohol for 5 years, nine for <10 years and all other patients for >10 years. They were drinking daily for some time before admission (i.e. not just for a few days) and were not binge drinkers. The reasons for hospitalization were delirium, changes in behaviour or decision to stop drinking. Patients stopped drinking from the day of admission, so they were not drinking at the time of the investigation. They did not use drugs which could influence blood flow velocity except for diazepam.

The middle cerebral artery (MCA) mean flow velocity (V mean) was determined by a 2 MHz pulsed Doppler probe (Trans-scan 3-D; EME, Germany) maintained in the temporal area by a headband with insonation through the middle temporal window at a depth of 50–55 mm during the first 2 days after admission to hospital. The pulsatility index [PI = (Vp – Vd)] was also determined.

Extracranial carotid arteries were examined by Doppler sonography and there was no evidence of internal or common carotid artery stenosis.

The results were compared with the blood flow velocity in the middle cerebral artery in 20 healthy volunteers (all men) of the same age with similar smoking habits. A total of 29% of the patients and 30% of the controls were non-smokers. To obtain detailed information about smoking habits in heavy alcohol drinkers is very difficult. Differences between these two groups were tested with Student’s t-test. P < 0.01 was selected as the point of minimal statistical significance.

RESULTS

The present study revealed a significant decrease in blood flow velocity in the middle cerebral artery in heavy alcohol drinkers. V mean (± SD) in patients was 51.9 ± 8.83 cm/s on the left side, compared to a corresponding value in the control group of 59.2 ± 9.1 cm/s (P < 0.01) and 51.9 ± 9.43 cm/s on the right side, compared to a corresponding value in the control group of 58.9 ± 9.2 cm/s (P < 0.01) (Table 1).

Two groups of patients were discerned. The first group (22 patients) with normal value of V mean and the second group (31 patients) in which blood flow velocity was significantly decreased (P < 0.001) in comparison with the 1st group and also with the controls (Fig. 1). This may have been related to an increased serum level of hepatic enzymes which was found in the second group (Table 2).

Table 1. Differences in blood flow velocity (V mean) in heavy alcohol drinkers and controls

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Patients (n = 53)</th>
<th>V mean (cm/s) (mean ± SD) Controls (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MCA&lt;sub&gt;sin&lt;/sub&gt;</td>
<td>51.9 ± 8.83**</td>
<td>59.1 ± 9.1</td>
</tr>
<tr>
<td>MCA&lt;sub&gt;dx&lt;/sub&gt;</td>
<td>51.9 ± 9.43**</td>
<td>58.9 ± 9.2</td>
</tr>
</tbody>
</table>

Blood flow velocity (V mean) in the left (MCA<sub>sin</sub>) and right (MCA<sub>dx</sub>) middle cerebral artery in heavy alcohol drinkers was significantly decreased (** P < 0.01 in Student’s t-test). n denotes numbers.

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The PI was not significantly changed in either group (results not shown).

DISCUSSION

The present study was designed to examine blood flow velocity in heavy alcohol drinkers with regard to the possibility of association between alcohol abuse and ischaemic brain infarction. A significant decrease was revealed in blood flow velocity in the middle cerebral artery in heavy alcohol drinkers. But, at the same time, two groups of the patients were found, one with normal and a second with decreased, blood flow velocity. This difference was not dependent on age, amount of alcohol or period of drinking. An increased level of hepatic enzymes was found in the group with decreased blood flow velocity.

Reduced cerebral blood flow measured by single photon emission computed tomography in subjects with liver cirrhosis was observed by Melgaard et al. (1990) and Iwasa et al. (2000). A statistically significant decrease in blood flow in the middle cerebral artery has been described by Dillon et al. (1995) in patients with cirrhosis without clinically apparent encephalopathy, as well as by Larsen et al. (1995) who explained it on the basis of impaired autoregulation of cerebral blood flow.

Delirium, changes in behaviour or decision to stop drinking, but not liver problems, were the reasons for admission to the hospital. This is why liver biopsy was not performed, and, consequently, no classification of liver disease was made.

Strauss et al. (2000) described impaired autoregulation only in patients with hepatic encephalopathy and not in all patients with end-stage liver disease. Restoration of cerebral blood flow autoregulation was shown within 48 (24–120) h after spontaneous hepatic recovery or liver transplantation, but before complete alleviation of hepatic encephalopathy (Strauss et al., 1997). The authors did not consider impaired autoregulation to be of major pathophysiological importance in hepatic encephalopathy.

The aim of our study was to determine if heavy alcohol drinking influences cerebral blood flow velocity with regard to the possibility of association between alcohol abuse and ischaemic brain infarction. Low blood flow velocity in cerebral arteries which could be a risk factor for stroke was found. A liver lesion may be one of the reasons for impaired circulation. Differences in blood flow velocity in our patients were not dependent on psychological changes. Other factors must therefore be involved.

One such factor could be increased blood viscosity. Decreased blood flow velocity in patients with increased blood viscosity was described by Dormandy (Dormandy, 1983). Significant elevation of viscosity of both whole blood and plasma before stroke has been reported by Coull et al. (1991) and a significant decrease in erythrocyte deformability was observed on the first day after stroke (Mojžiš et al., 1999).

However, it is still unclear what causes decreased blood flow velocity. Blood and plasma viscosity, erythrocyte deformability and dehydration could be potential determinants and thus merit investigation.

Table 2. Liver enzymes (ukat/l) in heavy alcohol drinkers and controls

<table>
<thead>
<tr>
<th>Enzyme</th>
<th>First group (n = 22)</th>
<th>Second group (n = 31)</th>
<th>Control group</th>
<th>Normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td>AST</td>
<td>0.65 ± 0.013</td>
<td>5.4 ± 0.21</td>
<td>0.39 ± 0.01</td>
<td>0.5–1.5</td>
</tr>
<tr>
<td>ALT</td>
<td>0.5 ± 0.024</td>
<td>1.9 ± 0.11</td>
<td>0.4 ± 0.08</td>
<td>0.5–1.5</td>
</tr>
<tr>
<td>GGT</td>
<td>1.0 ± 0.09</td>
<td>5.2 ± 0.19</td>
<td>1.3 ± 0.1</td>
<td>1.5–2.5</td>
</tr>
</tbody>
</table>

Liver enzymes were increased in the second group (heavy alcohol drinkers with decreased blood flow velocity) and they were in the normal range in the first group (heavy alcohol drinkers without decreased blood flow velocity). n denotes numbers.

AST, aspartate aminotransferase; ALT, alanine aminotransferase; GGT, gamma-glutamyltransferase.

The PI was not significantly changed in either group (results not shown).

Fig. 1. Blood flow velocity ($V_{mean}$) in the left (MCA sin) and the right (MCA dx) middle cerebral artery in heavy alcohol drinkers (groups 1, 2) and controls. In the second group ($V_{mean}$) was significantly decreased. ($xxx = P < 0.001$).
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REFERENCES


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