Cerebral blood flow and cerebrovascular response to acetazolamide in patients with chronic alcoholism

M Oishi, Y Mochizuki, T Takasu

Abstract

Cerebral blood flow and cerebrovascular response to acetazolamide were studied in 12 patients with chronic alcoholism and 12 age matched healthy controls. Blood flows in the cerebral cortex, thalamus, and putamen were significantly lower in the chronic alcoholic group than in the healthy control group. The increase in blood flow caused by acetazolamide did not show any significant difference between the two groups. These findings suggest that the decreased cerebral blood flow in chronic alcoholism is due to decreased cerebral metabolism.

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Keywords: cerebral blood flow; alcoholism; acetazolamide

Cerebral blood flow has been reported to be decreased in patients with chronic alcoholism1–4 but the cerebrovascular response to acetazolamide in such patients has not yet been reported. To determine the effects of chronic alcoholism on regional cerebral blood flow and cerebrovascular response to acetazolamide, we performed a xenon CT study in patients with chronic alcoholism and compared cerebral blood flow and response to cerebrovascular acetazolamide between this group and healthy controls.

Materials and methods

We studied 12 men with chronic alcoholism (mean age 56 (SD 7)) and 12 age matched healthy men who did not drink alcohol (mean age 57 (SD 6)). All subjects gave their informed consent. The patients fulfilled the DSM-IV criteria of alcohol dependence5 and did not have any other relevant diseases (head trauma, depression, seizures, neurological complications of chronic alcoholism) or any significant abnormality on head CT and blood tests (hepatic function and standard blood biochemistry and haematology tests). No patients had Wernicke-Korsakoff syndrome and their average score for the mini mental state examination6 was 25. Mean height was 165 cm, mean weight 62.3 kg, and mean blood pressure 138/88 mm Hg in the chronic alcoholic group and these variables were not significantly different from the healthy control group. Any medications which may have affected cerebral blood flow or cerebral metabolism (including benzodiazepines) were discontinued two weeks before the study of cerebral blood flow. All patients drank alcohol the night before the cerebral blood flow examination but on the day of examination they did not drink alcohol until after the examination, which was performed in the afternoon.

Cerebral blood flow was measured by the stable xenon CT method.7 8 The mathematical modelling of Meyer et al9 and the procedure of Fatouros et al10 were used to generate paramet-

Mean (SD) of regional cerebral blood flows

<table>
<thead>
<tr>
<th></th>
<th>Regional blood flows (ml/100 g/min)</th>
<th>Absolute changes by acetazolamide (ml/100 g/min)</th>
<th>Increase rates by acetazolamide (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Alcoholic</td>
<td>Control</td>
<td>Alcoholic</td>
</tr>
<tr>
<td>Frontal cortex</td>
<td>52.8 (9.3)**</td>
<td>70.9 (9.8)</td>
<td>31.9 (6.6)</td>
</tr>
<tr>
<td>Temporal cortex</td>
<td>51.9 (9.2)**</td>
<td>68.4 (9.6)</td>
<td>32.9 (6.8)</td>
</tr>
<tr>
<td>Parietal cortex</td>
<td>51.1 (9.0)**</td>
<td>67.4 (9.5)</td>
<td>29.5 (6.6)</td>
</tr>
<tr>
<td>Occipital cortex</td>
<td>49.8 (8.8)**</td>
<td>66.6 (9.5)</td>
<td>27.7 (6.5)</td>
</tr>
<tr>
<td>Anterior cingulate cortex</td>
<td>52.2 (9.2)**</td>
<td>70.4 (9.7)</td>
<td>31.3 (6.1)</td>
</tr>
<tr>
<td>Frontal white matter</td>
<td>24.5 (7.4)</td>
<td>27.4 (7.5)</td>
<td>10.8 (2.8)</td>
</tr>
<tr>
<td>Occipital white matter</td>
<td>23.1 (7.0)</td>
<td>25.9 (7.2)</td>
<td>11.1 (2.9)</td>
</tr>
<tr>
<td>Thalamus</td>
<td>57.2 (9.4)**</td>
<td>71.5 (9.2)</td>
<td>32.3 (5.3)</td>
</tr>
<tr>
<td>Caudate nucleus</td>
<td>78.4 (9.5)</td>
<td>82.3 (9.2)</td>
<td>33.2 (5.4)</td>
</tr>
<tr>
<td>Putamen</td>
<td>72.4 (8.7)*</td>
<td>80.2 (8.4)</td>
<td>39.0 (5.6)</td>
</tr>
</tbody>
</table>

*P < 0.05; **P < 0.01 vs healthy control group.
Cerebrovascular response to acetazolamide was high in both (A) and (B). In the cerebral cortex, thalamus, and putamen were lower in (A) than in (B).

chronicalcoholism, (B) a healthy control. Before injection of acetazolamide, the blood flows were obtained with the procedure of Kawamura et al.11 Xenon CT examinations were performed before and 20 minutes after intravenous injection of 17 mg/kg acetazolamide.

Statistical analysis of the cerebral blood flows was performed by Mann-Whitney U test.

Results
The figure shows xenon CT examination before and after intravenous injection of acetazolamide. The table shows the mean (SD) of regional cerebral blood flow in patients with chronic alcoholism measured by single photon emission computed tomography. Acta Neurol Scand 1990;82:87–93.

Discussion
Cerebral blood flow has been reported to be decreased in patients with chronic alcoholism1–4 but only one study used the xenon CT method.12 The main advantage of this method is that it provides relatively high resolution and quantitative information on regional cerebral blood flow coupled with anatomy by CT.7 Hata et al5 reported that local cerebral blood flows were reduced throughout all grey matter in patients with chronic alcoholism without Wernicke-Korsakoff syndrome. However, the present study showed that the blood flow in the cerebral cortex, thalamus, and putamen were reduced but that in the caudate nucleus was normal. The normal blood flow in the caudate nucleus may be due to the few patients studied so far.

The mechanism of decreased cerebral blood flow in patients with chronic alcoholism has not yet been elucidated2–15 although it is thought to be due to decreased cerebral metabolism.2, 15 Because the effects of chronic alcoholism on metabolism are not uniformly distributed in the present study, the result cannot be explained by the possibility that the patients with chronic alcoholism are more sensitive to the anaesthetic effects of xenon. The present study showed that the cerebrovascular response to acetazolamide was normal in such patients. Acetazolamide is considered to dilate the cerebral arterioles by inhibiting the carbonic anhydrase in the red blood cells and increasing carbon dioxide in the arterioles.16 Although the possible influence of early withdrawal effects on cerebral blood flow cannot be excluded, the result of the present study suggests that the reduced blood flow is not due to vascular problems such as arteriosclerosis in patients with chronic alcoholism.


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