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

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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 alcoholism 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.

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 cerebralcortex, thalamus, and putamen were lower in (A) than in (B). However, the present study showed that the blood flow in the cerebral cortex, thalamus, and putamen 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 elucidated although it is thought to be due to decreased cerebral metabolism. 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 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 arteries by inhibiting the carbonic anhydrase in the red blood cells and increasing carbon dioxide in the arterioles. 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.

Results

The figure shows xenon CT examination before and after intravenous injection of acetazolamide. The table shows the mean (SD) of the blood flows. Blood flows in the cerebral cortex, thalamus, and putamen were significantly lower in the chronic alcoholic group than in the healthy control group. The absolute changes and the increased rates of blood flows did not show any significant differences between the two groups.

Discussion

Cerebral blood flow has been reported to be decreased in patients with chronic alcoholism but only one study used the xenon CT method. 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. Hata et al 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.

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