
Short report

Cognitive deficits and their relationship to other neurological complications in chronic alcoholic patients

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SUMMARY In randomly selected chronic alcoholics hospitalised for the first time for detoxification a high prevalence (68%) of cognitive deficits was found. Peripheral neuronal damage was seen in 74%, autonomic neuronal damage in 24%. Cognitive deficits were not correlated with age, daily ethanol intake, duration of alcohol abuse or severity of liver damage. There was no correlation of peripheral, autonomic and central nervous system damage. Alcohol-induced damage of the nervous system is a common complication of chronic alcoholism, whose clinical importance often obscures possible concomitant liver damage.

Alcohol-induced brain damage and peripheral neuropathy have been recognised and extensively studied for a century. More recently, autonomic neuropathy has been found to be a relatively common and life-threatening complication of alcohol abuse. Moreover, in young alcoholic men there is a far greater prevalence of brain damage, evaluated by means of psychometric testing, than of liver damage. Despite this evidence and its importance for prognosis of chronic alcoholism, neurological complications are generally neglected, as compared to the great attention paid to the liver or the psychological or social problems caused by alcohol abuse. We studied the neurological complications, evaluated by means of neuropsychological and neurophysiological testing, in a group of chronic alcoholic patients. This report describes the prevalence and the clinical importance of alcohol-induced brain damage, evaluated by psychometric tests, and correlations with peripheral and autonomic nerve damage, with liver function and with other features of chronic alcoholic patients.

Subjects

Fifty patients, 41 men and nine women, aged 30–50 years (mean 42), hospitalised for alcoholic detoxification, were randomly selected for the study. All the subjects were informed of the aim of the study and gave their consent. For 14 patients (28%), alcohol abuse had been an important problem for less than 10 years, for 25 patients (50%) for 10–20 years and for 11 patients (22%) for more than 20 years. Average daily ethanol intakes were 100–150 g for 20 patients (40%), 150–300 g for 22 patients (44%) and more than 300 g for eight patients (16%). On the basis of biopsy, scintiscan or abdominal ultrasound and haematological parameters, 10 patients (20%) had no liver damage, 30 patients (60%) moderate and 10 patients (20%) severe liver damage. Patients suspected to have hepatic or Wernicke's encephalopathy were excluded from the study.

All the neuropsychological and neuropsychological tests were performed two to three weeks after alcohol withdrawal, while the patients, untreated with drugs, were still in hospital.

Methods

The neuropsychological performances of alcoholic patients were compared with those of randomly selected non-drinking controls, 16 men and four women, aged 28–53 (mean 41), hospitalised for minor surgical or orthopaedic disorders. The alcoholic patients and the controls did not significantly differ in age, sex distribution, education, social status or work activities. The battery of tests, in common clinical use, were administered in two 45-minute sessions.

The first session included the Konzentrations Verlaufs Test (KVT), to evaluate attention and concentration; Raven's Progressive Matrices 1938 (PM 38), to estimate intelligence factor "g"; and the Wechsler Memory Scale (WMS), to evaluate global Memory Quotient (MQ), short term verbal memory (STM verb), long term verbal mem-
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Cognitive deficits in patients with chronic alcoholic liver disease were assessed using the Wechsler Adult Intelligence Scale (WAIS) for evaluation of intellectual functions; the Rey Test (RT) and the Digit Symbol subtest of the Wechsler Memory Scale (WMS) for neuropsychological functions. Results showed significant cognitive impairments in patients with alcoholic liver disease compared to controls. The table below summarizes the mean scores and standard deviations for each test:

<table>
<thead>
<tr>
<th>Test</th>
<th>Controls (n = 20)</th>
<th>Alcoholics (n = 50)</th>
<th>Impaired Alcoholics (n = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM 38</td>
<td>41.1 ± 10.1</td>
<td>27.5 ± 11.5*</td>
<td>15 (30)</td>
</tr>
<tr>
<td>KVT</td>
<td>94.9 ± 12.9</td>
<td>80.8 ± 14.6*</td>
<td>11 (22)</td>
</tr>
<tr>
<td>UDFT</td>
<td>5.3 ± 1.5</td>
<td>4.1 ± 2.1*</td>
<td>14 (28)</td>
</tr>
<tr>
<td>RT</td>
<td>36.5 ± 9.5</td>
<td>25 ± 8.1†</td>
<td>14 (28)</td>
</tr>
<tr>
<td>WAIS subtests</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Block design</td>
<td>32 ± 8</td>
<td>20.7 ± 9.8†</td>
<td>12 (24)</td>
</tr>
<tr>
<td>Similarities</td>
<td>15.9 ± 3.3</td>
<td>11 ± 4.4*</td>
<td>18 (36)</td>
</tr>
<tr>
<td>Pictures</td>
<td>19.9 ± 4.9</td>
<td>15.9 ± 5.7*</td>
<td>8 (16)</td>
</tr>
<tr>
<td>Digit-symbol</td>
<td>42.6 ± 13.1</td>
<td>24 ± 10.9†</td>
<td>13 (26)</td>
</tr>
<tr>
<td>WMS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MQ</td>
<td>95.8 ± 15.5</td>
<td>80 ± 16†</td>
<td>10 (20)</td>
</tr>
<tr>
<td>STM Verb</td>
<td>10.2 ± 1.6</td>
<td>8.7 ± 2.1*</td>
<td>6 (12)</td>
</tr>
<tr>
<td>LTM</td>
<td>12.2 ± 4</td>
<td>8.9 ± 4.2*</td>
<td>7 (14)</td>
</tr>
<tr>
<td>STM Vis</td>
<td>11 ± 3</td>
<td>7.6 ± 4.4*</td>
<td>12 (24)</td>
</tr>
<tr>
<td>Learning</td>
<td>2.9 ± 1.7</td>
<td>1.8 ± 1.7*</td>
<td>8 (16)</td>
</tr>
</tbody>
</table>

Abbreviations are listed in the text.

* p < 0.05
† p < 0.001

Discussion

As expected, the alcoholic group scored lower than the control group in all the neuropsychological tests. This is in agreement with almost all previous reports with similar experimental designs, though there are some exceptions. Recent studies have suggested that deficits found at one interval after alcohol withdrawal may be significantly reduced after additional time has elapsed. In any case, much recovery occurs between two and three weeks after drinking ceases and we tested our patients after this interval.

The cognitive deficits observed indicate dysfunctions of reticulo-mesencephalic (KVT) and limbic structures (WMS), as well as of the right (UDFT, “block design”) and the left hemispheres (RT, “similarities”, “picture arrangement” and “digit-symbol substitution”) and the cerebral cortex as a whole (PM 38). These neuropsychological data agree with computed tomographic and neuropathological findings of widespread cerebral atrophy. We could not find any correlation between cognitive deficits and the duration of excessive drinking, daily ethanol intake or presence of liver damage. Though not logically to be expected, this agrees with previous studies. These data...
seem to indicate the existence of separate factors in
tolerance to prolonged toxic effects of alcohol on
neurons. Malnutrition, repeated head injuries, with-
drawal fits or concomitant liver dysfunction seem to
be secondary in the pathogenesis of cognitive
impairment.

As commonly observed in Italy, alcoholic men
outnumbered alcoholic women four to one. This
imbalance prevents accurate statistical analysis of
sex differences in alcohol-induced cognitive
impairment, though our men and women seem to
have similar cognitive deficits. The only significant
difference between sexes was the higher prevalence
of peripheral neuropathy and liver damage in the
alcoholic women (each in eight of nine).

Alcoholic-induced peripheral neuropathy was not
seen in approximately one third of our patients, and
autonomic neuropathy was not seen in three quar-
ters. The lack of significant association or correla-
tion of peripheral, autonomic and central nervous
system damage is particularly interesting. In other
words, for alcoholic patients the presence of peripheral
neuropathy is by no means predictive of
cognitive deficits or of autonomic neuropathy or vice
versa. This finding, in good agreement with clinical
experience, supports the hypothesis that ethanol can
damage the peripheral, autonomic and central nerv-
sous systems by different effects. Central neurons
might be more susceptible than peripheral and aut-
onamic ones to thiamine deficiency, in which con-
genital factors play an important role. Moreover,
alcoholism is associated with reduced cerebral blood
flow. Finally, though our patients were all nor-
motatives, their cerebral neurons might have suffered
from transient hypertensive episodes during either alcohol-induced stress reactions or occa-
sional alcohol withdrawal.

As a group, alcohol-induced neurological damage
assumes paramount clinical importance, often ob-
scuring possible concomitant liver disease.

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