Gastroduodenal Morphology and Related Symptoms in Chronic Alcoholics

T. Hauge, J. Persson and Å. Nilsson

Department of Internal Medicine, Central Hospital Karlstad, Sweden; Department of Internal Medicine, Lund University Hospital, Sweden

(Received 17 January 1997; In final form 24 February 1997)

Twenty-four chronic alcoholics admitted to hospital for detoxification after a drinking spree were examined by upper gastrointestinal endoscopy. Biopsy specimens were taken from corpus/fundus, antrum and duodenum for tissue histology (eosin stain). From the duodenum villus index and ultrastructure (scanning electron microscopy, SEM) were also performed. As a control group 12 subjectively healthy non-alcoholics referred to upper gastrointestinal endoscopy mainly for dyspepsia were chosen.

Gastrointestinal symptoms were common in alcoholics (88%). Endoscopic and histological gastroduodenitis were not more common in the alcohol group. There was no correlation between gastrointestinal symptoms and endoscopic or histological gastroduodenitis in both groups. In the duodenum, 50% of the alcoholics and 82% in the control group had alterations by scanning electron microscopy. Ten of the 11 alcoholics with an abnormal ultrastructure had diarrhoea. In the control group dyspepsia (ulcus suspect) was correlated to a pathological SEM.

Keywords: Alcoholics, Gastrointestinal symptoms, Upper gastrointestinal endoscopy, Tissue pathology, Villus index, Scanning electron microscopy, SEM

INTRODUCTION

Gastrointestinal symptoms like abdominal pain/dyspepsia, diarrhoea and nausea are common among individuals with chronic alcohol ingestion. Morphological alterations and functional disturbances in the upper gastrointestinal tract can also be related to alcohol abuse [1–3].

The acute alcohol induced gastritis, or better subepithelial hemorrhages, which disappears within a few days is well known [4,5]. Similar acute findings are found in the duodenum [1,6,7].

The chronic effects on the gastroduodenal morphology have not been extensively studied. A proven correlation between alcohol and chronic gastritis has not been found [4,8]. In the small intestine the effects of chronic alcohol abuse on the morphology are conflicting. Usually duodenal and jejunal biopsies have showed normal histology, but a reduction in villus height in relation to
crypt depth has been observed [2,9-12]. It is therefore possible that chronic alcohol consumption in man induces slight histological changes. Studies on the ultrastructure in the small intestine have showed pronounced ultrastructural alterations [3,10,13]. Only a few studies have been performed on the duodenal mucosa by gastrointestinal endoscopy.

The aim of the present study was to investigate the effects of chronic alcohol ingestion on the gastroduodenal mucosa by endoscopy, light microscopy and ultrastructure (scanning electron microscopy, SEM). The morphological findings were also compared to gastrointestinal symptoms.

MATERIAL AND METHODS

Twenty-four chronic alcoholics (5 women and 19 men) admitted to hospital for detoxification after a drinking spree were examined with upper gastrointestinal endoscopy as soon as possible. Mean average age was 46 years (range 33–59). The endoscopical examination was performed 4 days (range 2–7) after last alcohol intake. Mean alcohol consumption was 338 g pure alcohol/day (range 102–680) in the weeks before admission. The alcoholics were questioned about past medical history, alcohol consumption, present symptoms with particular regard to diarrhoea, nausea and abdominal pain and physically examined directly after admission. Diarrhoea was defined as abnormal, frequent, loose stools. Patients with manifest alcohol related somatic diseases, peptic ulcers, malnutrition or use of drugs known to affect intestinal integrity, were excluded. The smoking incidence in the alcohol group was 100%. None were treated with gastric acid inhibitors at the time for examination.

The control group consisted of individuals referred for upper endoscopy mainly for dyspepsia. They were 12 individuals (2 women, 10 men) with mean average age 42 years (range 24–72). These were subjectively healthy, had no or a very little alcohol consumption and had no gross pathological changes by the upper endoscopy. Seven controls had been treated with gastric acid inhibitors.

The biopsy specimens were performed with an Olympus FB-24K forceps by one and the same endoscopist (TH). Biopsies were taken from corpus/fundus, antrum and the distal part of the descending duodenum for light microscopy. From the duodenum two biopsies were also taken for scanning electron microscopy (SEM). For the light microscopy a haematoxylin/eosin stain was performed. An experienced pathologist examined all biopsy specimens having no information about the patients. The villus index was defined as the ratio between height of the villi plus the depth of the crypts (from the bottom of the crypts up to the tip of the villi) over the depth of the crypts [10]. Thus, a flat mucosa will give a mucosal index of 1.0, and a normal mucosa could give an index up to 6.

For the electron microscopy (SEM) the biopsies were immediately fixed in 4% glutaraldehyde solution in 0.1 M phosphate buffer (pH 7.2), rinsed carefully with 0.9% NaCl, dehydrated in rising concentrations of ethanol, dried and coated with gold in a combined system with a sputter (Edwards S 150 A sputter coating unit) and a modified vacuum unit (Edwards vacuum coating unit, 140 d E12–E14). Finally it was examined in a Cambridge Stereoscan 360 scanning electron microscope. Each specimen was studied at 3 different magnifications assessing the structural architecture of the small intestine (×100, ×1000, ×10000).

Statistical method: Wilcoxon’s rank sum test. The study was approved by the Local Ethical Committee and informed consent was obtained from all patients.

RESULTS

Symptoms: Twenty-one of 24 (88%) alcoholics had gastrointestinal symptoms like abdominal pain, diarrhoea and nausea. Abdominal pain/dyspepsia
was the most predominant symptom, fourteen patients (58%) had this symptom, twelve patients (50%) had diarrhoea by the examination. Among the controls 6 (50%) had abdominal pain/dyspepsia, none had diarrhoea.

**Endoscopy:** In the alcohol group only three had gastritis (12%) with subepithelial hemorrhages at the upper endoscopy. Twelve (50%) had a mild gastritis and nine (38%) alcoholics had a normal upper endoscopy. In the control group seven (58%) had a mild gastritis and five (42%) upper endoscopies were normal. The endoscopy did not demonstrate a difference between the two groups. A duodenitis was seen in two (8%) of the alcoholics and in none control patient by endoscopy.

**Morphology:** The histological examination in the alcohol group showed antral gastritis in six alcoholics (25%). Two (8%) had a corpus/fundus gastritis and 17 (71%) were normal. Helicobacter pylori was found in 7 (29%) according to culture. In the control group five (42%) had an antral gastritis and two (17%) had a corpus/fundus gastritis. Four of twelve (33%) were Helicobacter pylori positive. Six (50%) were histologically normal.

The histological examination demonstrated a duodenitis in eight (33%) alcoholics and in three (25%) of the controls.

There was no correlation between symptoms and findings by the upper endoscopy or the histological examination whether in alcoholics nor in the control group. For symptoms and histology, see also Table I.

In the duodenum the mucosal index was examined. The mean value in the alcohol group was 3.15 ± 0.15 SEM compared to 3.32 ± 0.20 SEM.

<table>
<thead>
<tr>
<th>Pat.</th>
<th>Symptom</th>
<th>Stomach</th>
<th>Duodenum (villus index)</th>
<th>Electronmicro (SEM)</th>
<th>Alcohol (g/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>no</td>
<td>normal</td>
<td>4.00</td>
<td>—</td>
<td>360</td>
</tr>
<tr>
<td>2</td>
<td>no</td>
<td>normal</td>
<td>3.75</td>
<td>normal</td>
<td>240</td>
</tr>
<tr>
<td>3</td>
<td>no</td>
<td>normal</td>
<td>3.81</td>
<td>normal</td>
<td>240</td>
</tr>
<tr>
<td>4</td>
<td>D</td>
<td>normal</td>
<td>2.75</td>
<td>normal</td>
<td>160</td>
</tr>
<tr>
<td>5</td>
<td>P</td>
<td>m.a.g.</td>
<td>3.45</td>
<td>normal</td>
<td>385</td>
</tr>
<tr>
<td>6</td>
<td>D, P, N</td>
<td>normal</td>
<td>2.57</td>
<td>path.</td>
<td>324</td>
</tr>
<tr>
<td>7</td>
<td>D, N</td>
<td>normal</td>
<td>2.13</td>
<td>path.</td>
<td>108</td>
</tr>
<tr>
<td>8</td>
<td>P, N</td>
<td>normal</td>
<td>3.40</td>
<td>normal</td>
<td>328</td>
</tr>
<tr>
<td>9</td>
<td>P</td>
<td>gastritis</td>
<td>3.17</td>
<td>normal</td>
<td>680</td>
</tr>
<tr>
<td>10</td>
<td>D, P</td>
<td>normal</td>
<td>2.40</td>
<td>path.</td>
<td>450</td>
</tr>
<tr>
<td>11</td>
<td>D, P</td>
<td>normal</td>
<td>1.44</td>
<td>path.</td>
<td>640</td>
</tr>
<tr>
<td>12</td>
<td>N</td>
<td>normal</td>
<td>4.10</td>
<td>normal</td>
<td>480</td>
</tr>
<tr>
<td>13</td>
<td>D</td>
<td>normal</td>
<td>3.38</td>
<td>path.</td>
<td>328</td>
</tr>
<tr>
<td>14</td>
<td>D</td>
<td>normal</td>
<td>3.45</td>
<td>normal/bact.</td>
<td>204</td>
</tr>
<tr>
<td>15</td>
<td>P, N</td>
<td>a.g.</td>
<td>4.09</td>
<td>—</td>
<td>570</td>
</tr>
<tr>
<td>16</td>
<td>D</td>
<td>normal</td>
<td>3.43</td>
<td>path.</td>
<td>353</td>
</tr>
<tr>
<td>17</td>
<td>D, P, N</td>
<td>m.a.g.</td>
<td>3.86</td>
<td>path.</td>
<td>480</td>
</tr>
<tr>
<td>18</td>
<td>P</td>
<td>carcinoma</td>
<td>2.29</td>
<td>(normal)</td>
<td>108</td>
</tr>
<tr>
<td>19</td>
<td>P</td>
<td>normal</td>
<td>3.55</td>
<td>normal/bact.</td>
<td>102</td>
</tr>
<tr>
<td>20</td>
<td>D, P</td>
<td>normal</td>
<td>3.07</td>
<td>path./bact.</td>
<td>125</td>
</tr>
<tr>
<td>21</td>
<td>P</td>
<td>normal</td>
<td>3.75</td>
<td>path.</td>
<td>360</td>
</tr>
<tr>
<td>22</td>
<td>D, P</td>
<td>normal</td>
<td>3.21</td>
<td>path.</td>
<td>370</td>
</tr>
<tr>
<td>23</td>
<td>P</td>
<td>gastritis</td>
<td>2.34</td>
<td>normal</td>
<td>360</td>
</tr>
<tr>
<td>24</td>
<td>D, P</td>
<td>a.g.</td>
<td>2.15</td>
<td>path.</td>
<td>350</td>
</tr>
</tbody>
</table>

3.15 (± 0.15 SEM) 338 (102–680)

D = diarrhoea, P = abdominal pain, N = nausea, m.a.g. = mild antrum gastritis, a.g. = antrum gastritis.
in the control group, what means a slightly, not
significant, reduction \((p = \text{NS})\).

Ultrastructure (SEM): Eleven of 22 alco-
holics (50\%) had pathological scanning electron
microscopy: decreased glycocalyx layer and ir-
regularities in the microvilli. Two biopsies could
not be examined. There was a good correlation
between these findings and diarrhoea (10 of 11
had diarrhoea) in the alcohol group and four of
these 11 alcoholics (36\%) had a histological du-
odenitis. The alcoholics with pathological scanning
electron microscopy did not have a higher alco-
hol consumption than the other. By dividing the
alcohol group into pathological and normal
ultrastructure, there was no correlation between
villusindex and ultrastructural findings.

Controls: Nine of eleven (82\%) had a patho-
logical SEM, one of the controls could not be
examined. There was a correlation between
ultrastructure and dyspepsia (ulcus suspect).
Seven of 11 (64\%) controls were referred to
upper gastrointestinal endoscopy for dyspepsia.
Of the two controls with previous dyspepsia one
had received pharmacological treatment for ulcus
ventriculi, one was operated for a hiatus hernia
and came for a one year follow up endoscopy.
The two controls with normal SEM in the duode-
num could not remember any episodes of dys-
pepsia (ulcus suspect).

**DISCUSSION**

The present study shows that gastrointestinal
symptoms like abdominal pain/dyspepsia and diar-
rhoea are common in alcoholics. Gastroduodenitis
was not more frequent among excessive alcoholics
compared to a control group. Half of the alcohol
group and 82\% of the control group had electron
microscopical alterations, and there was a corre-
lation between these findings and symptoms.

A slightly, not statistical significant, reduced
mucosal index in the duodenum compared to
controls was also observed. The mean value of
the mucosal index \((3.15 \pm 0.15 \text{ SEM})\) was almost

the same as found in biopsies taken by a Watson
capsula \((3.18 \pm 0.21 \text{ SEM})\) from the upper jeju-
num in another study [10]. This finding support
the few previous studies of alcohol effects on the
duodenal mucosa [2].

Fifty percent of the alcoholics had pathological
ultrastructure by scanning electron microscopy.
In a previous study, 70\% had SEM alterations
after chronic alcohol intake. It is possible that
alcohol effects on the duodenum differs from the
jejenum. The ultrastructural changes correlated
to diarrhoea (10 of 11 had diarrhoea), accepted
as one of the main consequences of acute and
chronic high alcohol intake [14]. The frequency
of SEM alterations was surprisingly higher in
the control group. Comparable studies from a
normal population does not exist, but control
biopsies from healthy individuals have showed
normal ultrastructure [8].

Our control group was a small group mainly
with dyspepsia (ulcus suspect). Therefore the con-
trols are not identical with a perfectly healthy
control group. On the other hand, it would be
difficult to recruit individuals free from symp-
toms for a upper gastrointestinal endoscopy.

The poor correlation between endoscopic and
histological findings is well known from other
studies [2,15].

In conclusion, the chronic alcohol effects on
the gastroduodenal mucosa are in accordance
with previous studies. Alcohol causes slight
changes in mucosal index in the duodenum, and
more pronounced ultrastructural alterations.
Diarrhoea had a correlation to ultrastructural
alterations in the alcohol group. Dyspepsia had
the same correlation in the control group. These
observation requires further investigation.

**Acknowledgements**

Thanks to R. Stenling M.D. Ph.D. Department
of Pathology, Umeå University Hospital, Sweden.

Financial support was given by the Swedish
Medical Society and by the Centre of Public
Health Research, Karlstad, Sweden.
GASTRODUODENAL MORPHOLOGY AND RELATED SYMPTOMS

References

Submit your manuscripts at
http://www.hindawi.com