Alcohol and dietary intake in the development of chronic pancreatitis and liver disease in alcoholism

Esteban Mezey, MD; Cynthia J Kolman, RD; Anna Mae Diehl, MD; Mack C Mitchell, MD; and H Franklin Herlong, MD

ABSTRACT Alcohol and dietary intake were determined in alcoholic patients with chronic pancreatitis and alcoholic liver disease. Patients with chronic pancreatitis, alcoholic hepatitis, and cirrhosis ingested ~50% of their calories as alcohol, and all had low mean intakes of protein, carbohydrate, and fat as compared with control subjects. Patients with severe alcoholic hepatitis had the lowest intake of nonalcohol calories and protein. Women with chronic pancreatitis had ingested alcohol for a shorter period of time than men whereas women with alcoholic hepatitis and cirrhosis had ingested less alcohol per kilogram body weight per day as compared with men. This study does not support the hypothesis that consumption of a high-protein and high-fat diet is a factor in the development of chronic pancreatitis in the alcoholic patient. The increased susceptibility of women as compared with men to alcoholic liver disease is established. Am J Clin Nutr 1988;48:148–51.

KEY WORDS Alcoholic hepatitis, cirrhosis, chronic pancreatitis, malnutrition, women, alcoholism

Introduction

Chronic alcoholism is a common cause of both liver disease and chronic pancreatitis. A toxic effect of alcohol is the principal etiologic factor of both these diseases in alcoholic patients. However, only a fraction of alcoholic patients develop either liver disease or chronic pancreatitis, suggesting that other factors, such as nutritional, hormonal, environmental or genetic, play a role in the pathogenesis.

Deficiencies of nutrients, mainly because of decreased dietary intake, are common in alcoholic liver disease (1). Furthermore, protein-calorie malnutrition was associated with both the severity and mortality of patients with alcoholic hepatitis (2). Chronic pancreatitis occurs in malnourished populations in tropical areas (3) and decreased exocrine pancreatic secretion in malnourished alcoholic patients is improved by increased dietary protein intake (4). However, others (3, 5) found that alcoholic patients with chronic pancreatitis have increased intakes of protein and fat. Ingestion of ethanol in combination with a high-protein and high-fat diet enhances concentrations of pancreatic enzymes (6). It was proposed that increases in enzyme concentration in exocrine pancreatic secretions result in protein precipitation in pancreatic ducts and contribute to the pathogenesis of pancreatitis (6). The risk of developing cirrhosis occurs at lower doses of ethanol ingestion in women than in men, suggesting that hormonal factors may be important (7). No definitive information is available on the relative risk of women as compared with men for the development of chronic pancreatitis. In one study, however, the duration of alcohol consumption was less in 8 women than in 111 men with chronic pancreatitis (8).

Our study determined whether alcohol intake, dietary intake, and nutritional state of alcoholic patients with chronic pancreatitis differs from that of patients with alcoholic hepatitis and cirrhosis.

Subjects and methods

One hundred and twenty nine alcoholic patients who were admitted to the hospital for pancreatitis or alcoholic liver disease were studied. Criteria for the diagnosis of chronic alcoholic pancreatitis included upper-abdominal pain, nausea or vomiting, elevation of serum amylase, and absence of cholelithiasis on abdominal sonography. The diagnosis was further confirmed by either one or more of the following: radiographic demonstration of intraabdominal calcification, sonography re

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vealing irregular echogenic pancreas or pseudocysts, endoscopic retrograde cholangiopancreatography showing typical pancreatic duct changes, or pancreatic exocrine insufficiency leading to steatorrhea. The diagnosis of alcoholic hepatitis was based on the clinical features of the disease (hepatomegaly, jaundice, fever, and leukocytosis) and a modest but persistent elevation of the serum aminotransferases (1.16–5.83 μkat/L; normal, < 0.58 μkat/L) with higher elevation of the serum aspartate aminotransferase than the serum alanine aminotransferase (9). The diagnosis of alcoholic hepatitis was confirmed by liver biopsy when possible (30 of 69 cases).

The patients with alcoholic hepatitis were divided into groups of moderate and severe illness based on the criteria documented by Maddrey et al. (10). By these criteria individuals with a discriminant function of 4.6 times prothrombin time (s) bilirubin (mg/dL) > 93 were classified as having severe alcoholic hepatitis. Those with a discriminant function < 93 were defined as having alcoholic hepatitis of moderate severity. The diagnosis of inactive cirrhosis was based on signs of portal hypertension (splenomegaly, ascites, or esophageal varices) in the absence of the clinical characteristics of alcoholic hepatitis described above. Liver biopsy done in 10 of 18 cases confirmed the diagnosis of inactive cirrhosis.

Control subjects were friends of some of the patients or hospital workers, were of the same socioeconomic status, and lived in the same neighborhoods as the patients. They were in the same decade of age and had no history or clinical evidence of pancreatic disease, liver disease, or other chronic medical illness.

The patients were questioned about their daily alcohol consumption and dietary intake for the time before the onset of symptoms that led to their most recent hospitalization. The control subjects were questioned about their alcohol consumption and dietary intake of the preceding day. The dietary ingestion of protein, carbohydrate, and fat was calculated with tables of food nutritional values by CJK, a registered dietician. Body weight was expressed as a percentage of ideal weight as determined from the height and sex using the 1983 Metropolitan Life Tables (11). Anthropometric measurements were performed by two observers using standard techniques (12). Triceps skinfold thickness (TSF) was measured in the middle of the right arm with a Lange caliper (Cambridge Scientific Industries, Cambridge, MD). Arm muscle circumference was calculated by subtracting TSF × π from measured midarm circumference. The anthropometric measurements are expressed as a percentage of normal values (12).

Basal energy expenditure (BEE) was calculated from the Harris-Benedict formulas (11) as follows: For males

\[
\text{BEE} = 66.5 + (13.7 \times \text{weight}) + (5 \times \text{height}) - (6.8 \times \text{age})
\]

For females

\[
\text{BEE} = 655 + (9.6 \times \text{weight}) + (1.7 \times \text{height}) - (4.7 \times \text{age})
\]

Differences among groups were assessed by one-way analysis of variance, by Duncan's new multiple range test, and by chi-square test (13). Sex differences in alcohol consumption were analyzed by unpaired Student's t test.

The study was approved by the Institutional Review Board of the Johns Hopkins Medical Institutions.

Results

Patients with chronic alcoholic pancreatitis had the lowest body weight (Table 1). The body weight of patients with alcoholic hepatitis and cirrhosis, although lower than that of control subjects were in the range of ideal body weight. These data need to be interpreted in light of the frequent presence of fluid retention in patients with alcoholic liver disease. Eleven of 18 (61%) patients with cirrhosis and 20 of 69 (29%) patients with alcoholic hepatitis had ascites at the time they were studied. The body weights in patients who did not have ascites were 99.9 ± 3.07%, 95.7 ± 3.10%, and 96.8 ± 2.9% of ideal body weight for those with moderate alcoholic hepatitis, severe alcoholic hepatitis, and cirrhosis, respectively. There was a similar reduction in TSF in patients with pancreatitis, alcoholic hepatitis, and cirrhosis. Arm muscle circumference was decreased in all patients but the decrease was less marked in patients with moderate alcoholic hepatitis.

Total calorie consumption was similar in the patients and control subjects (Table 2). However, alcohol accounted for ~50% of the calories in patients as compared with 15% in control subjects. The intake of protein, carbohydrate, and fat was decreased in the patients as compared with control subjects (p < 0.05). Patients with severe alcoholic hepatitis had the lowest protein in-

**TABLE 1**

Clinical characteristics of patients and control subjects*

<table>
<thead>
<tr>
<th>Patients with alcoholic hepatitis</th>
<th>Pancreatitis</th>
<th>Moderate</th>
<th>Severe</th>
<th>Cirrhosis</th>
<th>Control subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>42</td>
<td>50</td>
<td>19</td>
<td>18</td>
<td>28</td>
</tr>
<tr>
<td>Male:female ratio</td>
<td>25:17</td>
<td>32:18</td>
<td>15:4</td>
<td>8:10</td>
<td>19:9</td>
</tr>
<tr>
<td>Age</td>
<td>40.7 ± 1.6</td>
<td>41.8 ± 1.5</td>
<td>42.9 ± 2.2</td>
<td>47.7 ± 2.2</td>
<td>39.5 ± 1.8</td>
</tr>
<tr>
<td>Weight (% of standard)</td>
<td>92.4 ± 2.6†</td>
<td>103 ± 2.9</td>
<td>100.4 ± 5.0</td>
<td>104.4 ± 4.1</td>
<td>111.5 ± 4.3‡</td>
</tr>
<tr>
<td>TSF (% of standard)</td>
<td>71.5 ± 2.5</td>
<td>73.6 ± 4.8</td>
<td>71.8 ± 7.5</td>
<td>69.6 ± 5.3</td>
<td>92.2 ± 7.4‡</td>
</tr>
<tr>
<td>AMC (% of standard)</td>
<td>85.3 ± 2.6</td>
<td>90.7 ± 1.4§</td>
<td>86.0 ± 2.8</td>
<td>84.8 ± 3.3</td>
<td>108.0 ± 2.6§</td>
</tr>
</tbody>
</table>

* ± SEM. TSF, triceps skinfold; AMC, Arm muscle circumference.
† Significantly different from all other groups at p < 0.05.
‡ Significantly different from all other groups at p < 0.1.
§ Significantly different from control subjects and patients with pancreatitis and cirrhosis at p < 0.05.
TABLE 2
Dietary and alcohol intake of patients and control subjects*

<table>
<thead>
<tr>
<th>Patients with alcoholic hepatitis</th>
<th>Protein (g/d)</th>
<th>Carbohydrate (g/d)</th>
<th>Fat (g/d)</th>
<th>Alcohol (g/d)</th>
<th>Nonalcohol calories (g/d)</th>
<th>Total calories (g/d)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Men</td>
<td>Women</td>
<td>Men</td>
<td>Women</td>
<td>Men</td>
<td>Women</td>
</tr>
<tr>
<td>Protein (g/d)</td>
<td>57.1 ± 4.8</td>
<td>53.2 ± 3.3</td>
<td>40.0 ± 3.2</td>
<td>56.9 ± 5.0</td>
<td>76.2 ± 4.8†</td>
<td>44.7 ± 3.7†</td>
</tr>
<tr>
<td>(% calories)</td>
<td>9.1 ± 0.6</td>
<td>8.2 ± 0.4</td>
<td>7.1 ± 0.5</td>
<td>8.8 ± 1.4</td>
<td>13.3 ± 1.0‡</td>
<td>7.6 ± 1.0</td>
</tr>
<tr>
<td>Carbohydrate (g/d)</td>
<td>139.2 ± 10.0</td>
<td>137.6 ± 9.2</td>
<td>115.0 ± 8.3</td>
<td>147.3 ± 24.3</td>
<td>221.2 ± 13.7‡</td>
<td>136.6 ± 11.9‡</td>
</tr>
<tr>
<td>(% calories)</td>
<td>22.1 ± 1.3</td>
<td>21.0 ± 1.1</td>
<td>20.6 ± 2.3</td>
<td>23.0 ± 2.4</td>
<td>38.6 ± 2.0</td>
<td>26.2 ± 2.0</td>
</tr>
<tr>
<td>Fat (g/d)</td>
<td>53.8 ± 4.2</td>
<td>57.4 ± 4.0</td>
<td>50.6 ± 6.9</td>
<td>60.6 ± 9.2</td>
<td>85.6 ± 3.7†</td>
<td>57.4 ± 4.0</td>
</tr>
<tr>
<td>(% calories)</td>
<td>19.3 ± 1.3</td>
<td>19.8 ± 1.2</td>
<td>20.4 ± 1.9</td>
<td>21.2 ± 2.6</td>
<td>33.6 ± 2.0</td>
<td>26.2 ± 2.0</td>
</tr>
<tr>
<td>Alcohol (g/d)</td>
<td>177.8 ± 14.5</td>
<td>191.6 ± 12.3</td>
<td>165.8 ± 17.8</td>
<td>178.1 ± 23.4</td>
<td>47.7 ± 11.6‡</td>
<td>31.8 ± 11.6‡</td>
</tr>
<tr>
<td>(% calories)</td>
<td>49.5 ± 2.6</td>
<td>51.0 ± 2.2</td>
<td>51.9 ± 3.2</td>
<td>47.0 ± 4.4</td>
<td>14.5 ± 2.8‡</td>
<td>10.8 ± 2.8‡</td>
</tr>
<tr>
<td>Nonalcohol calories</td>
<td>1269 ± 97</td>
<td>1276 ± 80</td>
<td>1075 ± 83</td>
<td>1364 ± 151</td>
<td>1965 ± 84‡</td>
<td>1519 ± 84‡</td>
</tr>
<tr>
<td>(% of BEE)</td>
<td>96.7 ± 8.4</td>
<td>87.8 ± 5.3</td>
<td>67.1 ± 5.6†</td>
<td>92.8 ± 8</td>
<td>124.0 ± 4.9‡</td>
<td>92.8 ± 4.9‡</td>
</tr>
<tr>
<td>Total calories</td>
<td>2514 ± 142</td>
<td>2627 ± 113</td>
<td>2203 ± 134</td>
<td>2572 ± 151</td>
<td>2298 ± 115</td>
<td>2157 ± 115</td>
</tr>
<tr>
<td>(% of BEE)</td>
<td>185.3 ± 12.5</td>
<td>179.7 ± 8.6</td>
<td>149.6 ± 11.3</td>
<td>183.2 ± 13.8</td>
<td>143.4 ± 5.7</td>
<td>128.4 ± 5.7</td>
</tr>
</tbody>
</table>

* X ± SEM.
† Significantly different from all other groups at p < 0.05.
‡ Significantly different from all other groups at p < 0.01.
§ Significantly different from pancreatitis, moderate alcoholic hepatitis, and cirrhosis at p < 0.05.

take and the lowest intake of nonalcohol calories expressed per BEE. The mean daily amount of alcohol ingested was similar in patients with pancreatitis, alcoholic hepatitis, and cirrhosis (Table 2).

Daily alcohol intake was similar in men and women with pancreatitis but the duration of ingestion was shorter in women (Table 3). By contrast women with alcoholic hepatitis and cirrhosis reported ingestion of less ethanol than men with these diseases but the duration of ingestion was similar. Four women but only one man with alcoholic hepatitis and three women but no men with cirrhosis had ingested < 80 g alcohol/d. The lowest daily alcohol ingestion of 20 g was in a woman with cirrhosis. The difference in daily ingestion of alcohol in women with alcoholic hepatitis and cirrhosis as compared with men was significant when expressed per kilogram of body weight (p < 0.05).

The types of alcoholic beverage ingested by the patients and control subjects are shown in Table 4. Except for a higher ingestion of spirits in patients with pancreatitis, no other differences in beverage choice were evident. Smoking was very common in both patients and control subjects. The percentage of smokers for the patients ranged between 60 and 89% in the various patient groups as compared with 75% in the control subjects.

Discussion

This study shows that patients with chronic alcoholic pancreatitis as well as those with alcoholic liver disease have similar low dietary intake of protein, carbohydrate, and fat. These findings differ from previous reports in which high-protein and high-fat intake was observed in patients with chronic alcoholic pancreatitis in Europe (3) and in Mexico (5) but confirm observations made in the

TABLE 3
Amount and duration of alcohol intake in males and females with alcoholic pancreatitis and liver disease*

<table>
<thead>
<tr>
<th>Alcohol intake</th>
<th>Amount</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disease</td>
<td>Men g/d</td>
<td>Women g/d</td>
</tr>
<tr>
<td>Pancreatitis</td>
<td>180.0 ± 18.8</td>
<td>174.7 ± 23.4</td>
</tr>
<tr>
<td>Alcoholic hepatitis</td>
<td>193.2 ± 12.2</td>
<td>134.2 ± 15.2</td>
</tr>
<tr>
<td>Moderate</td>
<td>215.1 ± 14.8</td>
<td>143.1 ± 15.4</td>
</tr>
<tr>
<td>Severe</td>
<td>185.3 ± 19.1</td>
<td>94.0 ± 19.5</td>
</tr>
<tr>
<td>Cirrhosis</td>
<td>218.5 ± 29.7</td>
<td>135.5 ± 18.4</td>
</tr>
<tr>
<td>Control</td>
<td>69.8 ± 15.5</td>
<td>15.1 ± 6.6§</td>
</tr>
</tbody>
</table>

* X ± SEM.
† Significantly different as compared with men at p < 0.01.
§ Significantly different as compared with men at p < 0.05.
‡ Significantly different from all other groups of the same sex at p < 0.01.
United States (14) and in Australia (15). The amount of daily alcohol consumption in patients with pancreatitis reported in this study is comparable with that found in other reports. Furthermore, women with chronic pancreatitis had consumed alcohol for a shorter period than men although there was no sex difference in the total daily amount of alcohol consumed. A shorter duration of alcohol ingestion was previously demonstrated in a small group of 8 women compared with 111 men with chronic alcoholic pancreatitis (8).

Patients with severe alcoholic hepatitis had the lowest intake of protein and nonalcohol calories expressed as a percent of BEE. Mendenhall et al (2) also found that patients with severe alcoholic hepatitis had the lowest intake of nonalcohol calories. Furthermore, they demonstrated that protein-calorie malnutrition was associated with severity of alcoholic hepatitis and with mortality (2). In this study total daily alcohol consumption as a percent of total calories was comparable in patients with moderate and severe alcoholic hepatitis and with cirrhosis.

This study demonstrates the occurrence of alcoholic liver disease in women at a lower daily alcohol consumption even after intake is corrected for body weight. Previous studies found a lower total consumption of alcohol in women as compared with men with cirrhosis, which when expressed per kilogram of body weight was no longer significant (7). A shorter duration of alcohol ingestion found in women as compared with men with cirrhosis by others (7, 16) was not confirmed in this study.

In conclusion, patients with severe alcoholic hepatitis had the lowest consumption of protein and nonalcohol calories as a percentage of basal energy expenditure. Alcoholic liver disease occurs in women at a lower daily alcohol ingestion. This study does not support the suggestion that high dietary intake of protein and fat are factors in the development of chronic pancreatitis in the alcoholic patient.

References