JGH Open Open Access An open access journal of gastroenterology and hepatology

JGHF Journal of Gastroenterology and Hepatology Foundation

doi:10.1002/jgh3.12165

ORIGINAL ARTICLE

Patterns of alcohol consumption and nutrition intake in patients with alcoholic liver disease and alcoholic pancreatitis in North Indian men

Karam R Singh,* Gaurav Muktesh,† Deepak Gunjan,‡ Rakesh Kochhar,† Virendra Singh,§ Ashim Das,¶ Pradeep Siddappa† and Kartar Singh†

*Unit of Gastroenterology, Regional Institute of Medical Sciences, Imphal, Departments of †Gastroenterology, §Hepatology, ¶Histopathology, Postgraduate Institute of Medical Education and Research, Chandigarh and ‡Department of Gastroenterology, All India Institute of Medical Sciences, Delhi, India

Key words
alcohol liver disease, alcoholic pancreatitis, body mass index, nutrition.

Accepted for publication 31 January 2019.

Correspondence
Rakesh Kochhar, Department of Gastroenterology, F block, Nehru Hospital, Postgraduate Institute of Medical Education and Research, Chandigarh 160012, India.
Email: dr_kochhar@hotmail.com

Declaration of conflict of interest: None to declare.
Author contribution: Karam R Singh contributed to the collection and assembly of data, analysis and interpretation of the data, and drafting of the article. Gaurav Muktesh contributed to the analysis of the data, drafting the article, critical revision of the article for important intellectual content, and editing the manuscript. Deepak Gunjan contributed to the analysis and interpretation of the data and drafting of the article. Rakesh Kochhar contributed to the patient's management, drafting of the article, and critical revision of the article for important intellectual content. Virendra Singh contributed to the drafting of the article and critical revision of the article for important intellectual content. Ashim Das contributed to the drafting of the article and critical revision of the article for important intellectual content. Pradeep Sidappa contributed to the patient's management, analysis and interpretation of the data, and drafting of the article. Kartar Singh contributed to the study design and critical revision of the article for important intellectual content.

Abstract

Background and Aim: Chronic alcoholism and nutrition play an important role in liver and pancreatic diseases. To compare drinking habits and nutritional data in patients with alcoholic liver disease (ALD) and alcoholic pancreatitis (ALP).
Methods: Clinical, anthropometric, dietary intake, laboratory, and imaging data were recorded in consecutive patients of ALD and ALP.
Results: In 150 patients of ALP (n = 76) and ALD (n = 74), the age of starting alcohol consumption (19.03 ± 3.78 *vs* 18.0 ± 2.59 years) and the mean amount of alcohol consumed per day (165.63 ± 87.99 *vs* 185.50 ± 113.54 g; P = 0.230) were similar. Patients with ALD consumed alcohol on a daily basis more frequently (90.5 *vs* 72.3%; P = 0.003) and had a longer duration of alcohol intake (21.6 + 0.2 *vs* 14.5 + 6.9 years; P < 0.0001) than patients in the ALP group. Binge drinking was more common in patients with ALP compared to patients with ALD (60.5 *vs* 20.3%); P < 0.0001). Patients with ALP had a lower body mass index (19.9 ± 3.49 *vs* 22.64 ± 4.88 kg/m^2; P = 0.001) and more frequent decrease in mid arm circumference (57.9 *vs* 44.6%; P = 0.042) and triceps skin fold thickness (67.1 *vs* 52.7%; P = 0.072) compared to patients with ALD.
Conclusion: There was no difference in the age of starting alcohol consumption and mean amount of alcohol consumption per day between the groups. Patients with ALD were more likely to be daily drinkers with a longer duration of alcohol intake. However, binge drinking and malnourishment was more common in the ALP group.

Introduction

Chronic alcoholism is a common cause of both liver diseases and chronic pancreatitis (CP). However, only a fraction of alcoholic patients develops either liver disease or CP. Approximately 30% develop cirrhosis,[1] whereas 2.5–3% develop acute or CP.[2] The factors that influence susceptibility to organ damage are poorly understood. The role of nutrition in both alcoholic liver disease (ALD) and alcoholic pancreatic disease remains controversial. For ALD, one study reported that protein calorie malnutrition is associated with an increase in mortality,[3] whereas another one

concluded that obesity and excess body weight have been associated with an increased risk of ALD.[4]

Similarly, controversy exists with respect to pancreatitis patients. Curiously, a complex of pancreatic fibrosis and ductal calculi, clinically and histopathologically similar to alcohol-induced CP, occurs in various malnourished populations of Asia and Africa, which has been termed tropical pancreatitis.[5] The latest studies have, however, shown that malnutrition is an outcome of long-standing CP rather than an inciting event.[6] It has also been shown, on the other hand, that patients who develop alcoholic pancreatitis (ALP) consume a diet richer in protein and fat than that of controls.[7] Experimental animal studies have substantiated this claim.[8,9]

The type of alcohol, amount and duration of consumption, and its relation to the development of liver diseases are controversial. The net amount of alcohol consumed (independent of the form in which it is consumed) is the most important risk factor for the development of ALD.[10] The risk of developing cirrhosis increases with ingestion of >60–80 g/day or >20 g/day of alcohol for >10 years in men and women, respectively.[11] The relationship between the quantity of alcohol consumed and the development of liver disease is clearly not linear.[12,13] The type of alcohol consumed may influence the risk of developing liver disease. In one study, drinking beer or spirits was more likely to be associated with liver disease than drinking wine.[14] One of the studies comparing drinking history and patterns in patients with alcoholic use disorder and ALD concluded that lifetime alcohol intake was similar in both the groups.[15] The age of onset for alcohol drinking and duration of alcohol intake were also found to be similar in both the groups.[15]

In alcoholic pancreatic diseases, there is an association between attacks of pancreatitis and of larger-than-usual quantities of alcohol consumption.[16,17] However, a meta-analysis has shown decreased risk of pancreatitis in women consuming alcohol <40 g/day.[18] The type of beverage consumed appears to be unimportant in relation to the development of ALP. Wine, spirits, beer, and cider have all been incriminated in pathogenesis.[17] The drinking habits and the preferred alcoholic beverage of patients with ALP need to be documented and compared with those of a suitable control group before such factors can be accepted or dismissed as contributing to the development of the disease. One of the studies published recently found no significant difference in patterns of alcohol intake in patients with alcohol use disorder and ALP.[19] Unfortunately, very few studies have studied various aspects of alcohol intake, such as frequency, type of alcohol intake etc., to determine the risk of pancreatitis.[17] Moreover, even the studies comparing alcohol intake and nutrient intake amongst patients with ALD and ALP are few.[20] The aim of the present study was to make a direct comparison of drinking habits and nutrient intake between the ALD and the ALP patients.

Methods

This study was conducted on consecutive patients of ALD or ALP admitted to the department of Gastroenterology of Postgraduate Institute of Medical Education and Research, Chandigarh, India, during the period of January 2011 to July 2012. Acute ALP was diagnosed clinically when patients presented with a history of consumption of alcohol, with two of the following criteria[21]: (i) symptoms, such as epigastric pain, consistent with the disease; (ii) a serum amylase or lipase greater than three times the upper limit of normal; and (iii) radiological imaging consistent with the diagnosis, usually using computed tomography (CT) or magnetic resonance imaging (MRI). Pancreatitis was classified as acute unless there was CT, MRI, or endoscopic retrograde cholangiopancreatography (ERCP) findings of CP.[22] Written informed consent was obtained from all patients prior to enrolment in the study. The study was approved by the institutional ethics committee. Patients were included/excluded from the study after analysis and close scrutiny by a member of the treating team (a gastroenterologist).

Criteria for the diagnosis of chronic ALP included upper abdominal pain, nausea or vomiting, elevation of serum amylase, and absence of cholelithiasis on abdominal sonography. The diagnosis was further confirmed by one or more of the following: radiographic demonstration of pancreatic calcification, sonography demonstrating irregular echogenic pancreatic parenchyma, shrunken pancreas, ERCP showing typical pancreatic duct changes, or evidence of pancreatic exocrine insufficiency leading to steatorrhea.

The diagnosis of ALD was made by the documentation of excess alcohol intake (>80 g/day) and clinical, biochemical, and radiological evidence of liver disease. 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 (2–5 times of normal value) with higher elevation of the serum aspartate aminotransferase than the serum alanine aminotransferase (aspartate aminotransferase [AST]/alanine aminotransferase [ALT] > 1.5 and both AST and ALT <400, serum bilirubin >3 mg/dL).[23] The diagnosis of cirrhosis was based on signs of portal hypertension (splenomegaly, ascites, or esophageal varices), ultrasonographic evidence of changes in liver echotexture, irregularity of liver margins, or portal vein size >14 mm and in the absence of the clinical characteristics of alcoholic hepatitis described above.

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 dietary ingestion of proteins, carbohydrates, and fats was calculated with tables of nutritional value of food using the recall method. History of smoking was recorded. Body weight was expressed as a percentage of ideal weight as determined for the height and gender. Anthropometric measurements were performed using standard techniques. Triceps skin fold thickness (TSFT) was measured in the middle of the right arm with a Lange caliper. Mid arm circumference (MAC) was measured in sitting position with the arm hanging freely from the side. The anthropometric measurements were expressed as a percentage of normal values. For calculating dry body weight in patients with ALD (which was used for calculating body mass index [BMI]) with ascites, subtracting a percentage of weight based upon severity of ascites (mild, 5%; moderate, 10%; severe, 15%) was carried out, with an additional 5% subtracted if bilateral pedal edema was present .[24]

A MAC of less than or equal to 22 cm and TSFT<10 mm were considered to be a significant decrease.[25,26] Binge drinking was defined as >5 standard drinks (>50 g) on one occasion.[27] The stringent recording of dietary intake, alcohol intake, and anthropometric measurements was performed by the investigator

(a gastroenterologist) in the presence of a dietician using standard diet charts.

The type of alcohol consumed was noted in terms of spirit, beer or wine, and country-made alcohol. The duration of alcohol intake in years was also recorded. In round figures, 30 mL of spirit, 360 mL of beer, and 120 mL of wine were considered a standard drink containing about 10 g of ethanol. The mean alcohol content of various local liquors available in north India was considered to be 16 g in 30 mL. Exclusion criteria were as follows: BMI >40 kg/m^2, HBsAg, anti-hepatitis C virus or -human immunodeficiency virus positivity, diabetes mellitus, and presence of cholelithiasis.

Statistical analyses. Statistical analysis of data was performed using SPSS software (Version 23.0, IBM Corp., Armonk, NY, USA). Mean ± SD was calculated for the normally distributed variable. Categorical variables were compared using χ^2 test, and the quantitative variable was compared using an independent sample *t*-test and Mann–Whitney test depending on the distribution of data. A *P*-value of <0.05 was considered statistically significant.

Results

A total of 150 consecutive cases of ALP and/or ALD were enrolled. Seventy-four patients had ALD, which included 25 (16%) cases of alcoholic hepatitis and 49 (32.7%) cases of cirrhosis of liver. Seventy-six patients had ALP, comprising 46 (30.7%) patients of acute ALP and 30 (20%) of chronic ALP. All the subjects in this study were males. The alcoholic drinks consumed included spirits (whiskey, brandy, rum, vodka, country liquor), beer, and rarely wine.

The minimum age at the time of starting alcohol consumption was 13 years, and the maximum was 39 years, with a mean of 19 ± 3 years (Table 1). ALD patients were older, with a history of longer duration of consumption of alcohol. Most of them were daily alcohol consumers who often started consuming alcohol from the morning. On the other hand, ALP patients demonstrated more binge drinking with friends and often at meal time. However, there was no difference in age of onset and amount of alcohol consumption between the two groups (Table 1).

There was no difference in type of alcohol used in both the groups of patients. Smoking was, however, reported in 70 of 150 (46.7%) patients, showing no statistically significant difference between ALP and ALD patients (52.6 *vs* 40.5%, $P = 0.138$) (Table 1).

Nutritional parameters. The observed mean BMI for all the patients was 21 ± 4 kg/m^2. Patients with ALD had higher mean weight and BMI compared with patients with ALP (Table 2). Patients with ALP had more significantly decreased MAC and triceps fold thickness compared to ALD (Table 2). In our study, the mean daily calorie, protein, and fat intake was 1643 ± 392.4 kcal/day, 47 ± 14.6 g/day, and 40.9 ± 1 g/day, respectively, in the entire cohort. However, there was no difference in the mean calorie intake, fat intake, protein intake, and carbohydrate intake between alcoholic liver disease (ALD) and ALP groups.

Laboratory parameters. Anemia was seen in 129 (86%) patients, present in 72 (55.8%) with ALD and 57 (44%) with ALP patients ($P = 0.01$). Macrocytic anemia was found in 17 of 74 (22.97%) patients with ALD and 5 of 76 (6.57%) with ALP ($P = 0.001$). Serum folate levels were deficient (normal range: 5.4–1 8.0 ng/mL) in 14 (18.91%) patients with ALD and 3 (3.94%) patients with ALP ($P = 0.001$), whereas serum vitamin B_{12} levels were deficient in 19 (25.67%) patients with ALD and 5 (6.57%) patients with ALP ($P < 0.001$).

Table 1 Comparison patterns of alcohol consumption in alcoholic liver disease and alcoholic pancreatitis groups

	Alcoholic liver disease (*n* = 74)	Alcoholic pancreatitis (*n* = 76)	*P* value
Age (years) (mean + SD) (range)	45.7 + 10.2 (24–66)	37.8 + 9.2 (20–55)	<0.0001
Age at first alcohol use (years) (mean + SD) (range)	19.0 + 3.7 (13–39)	18.5 + 2.5 (5–35)	0.329
Duration of alcohol intake (years) (mean + SD)	21.6 + 9.2	14.5 + 6.9	<0.0001
Frequency (daily drinkers), *n* (%)	67 (90.5)	55 (72.3)	0.003
Pattern of drinking, *n* (%)			
Meal time drinking	20 (27)	29 (38.2)	0.146
Binge drinking	15 (20.3)	46 (60.5)	<0.0001
Drinking with friends	16 (21.6)	40 (52.6)	<0.001
Amount of alcohol consumed/day (g/day) (mean + SD)	185.59 + 113	165.63 + 88	0.230
Smoking	30 (40.5)	40 (52.6)	0.138

Table 2 Comparison of nutritional parameters in alcoholic liver disease and alcoholic pancreatitis groups

	Alcoholic liver disease (*n* = 74)	Alcoholic pancreatitis (*n* = 76)	*P* value
Weight (kg) (mean + SD)	65.95 + 14.2	58.12 + 10.9	<0.0001
BMI (kg/m^2) (mean + SD)	22.64 + 4.8	19.90 + 3.5	<0.0001
Decreased MAC (≤22 cm), *n* (%)	33 (44.6)	44 (57.9)	0.042
Decreased TSFT (<10 mm), *n* (%)	39 (52.7)	51 (67.1)	0.072
Total calorie intake/day (kcal/day) (mean + SD)	1629.3 + 465.4	1658.2 + 307.6	0.653
Total protein intake/day (g/day) (mean + SD)	47.3 + 15.3	46.6 + 14.0	0.762
Total fat intake/day (g/day) (mean + SD)	45.3 + 13.3	44.8 + 12.30	0.800
Total carbohydrate intake/day (g/day) (mean + SD)	251.9 + 87.1	252.6 + 69.92	0.957

BMI, body mass index; MAC, mid arm circumference; TSFT, triceps skin fold thickness.

Discussion

There has been a rapid change in the pattern and trends of alcohol use in India. The chief reason amongst them is that people are beginning to drink at younger ages. The percentage of the drinking population aged younger than 21 years has increased from 2% to more than 14% in the past 15 years.[28] The changing social norms, urbanization, increased availability, high-intensity mass marketing, and relaxation of overseas trade rules, along with poor level of awareness related to the harmful effects of alcohol, has contributed to increased alcohol use in our population.[28]

All the patients were males in our study. Because of the sociocultural conditions in India, females are rarely involved in alcohol drinking. The mean age of the total patient population was 42 ± 10 years. Interestingly, the youngest patient was 23 years. The patients of ALP (38 ± 9 years) were comparatively younger than those with ALD (46 ± 10 years), the difference being statistically significant. Previous studies have conflicting results.[29–32] Spicák *et al*., in their study, found that the mean age of alcoholic cirrhotic and ALP patients were 47.5 years and 37.8 years, respectively.[33] In another study from India, the mean age of patients with alcoholic liver cirrhosis was 52.4 years compared to 47.1 years in patients of alcoholic CP.[29] However, another study did not find any such difference.[30]

Alarmingly, the age at the time of starting alcohol consumption was as young as 13 years. The mean age of starting alcohol consumption was 19 years. However, no difference was noted in mean age of starting alcohol consumption between patients with ALD (19 ± 4 years) and ALP (18 ± 3 years). Veena *et al*., from South India, in their study, found that the mean age of starting alcohol was 22.8 years and 24.3 years amongst patients of alcoholic cirrhosis and CP, respectively.[29] First contact with alcohol before 15 years of age was reported in 25.8% of patients with pancreatitis and 8.8% of patients with cirrhosis in another study.[33] Thus, the age of starting alcohol consumption in our study is similar to other studies.

The duration of alcohol intake was longer in patients with ALD (21 ± 9 years) compared to that of ALP (14 ± 6 years). The maximum duration of alcohol consumption was 40 years. A study from South India has shown that the mean duration of alcohol consumption in both the groups was long, 29.5 ± 10.25 years in ALD and 21 ± 9.61 years in ALP.[29] Another study found the duration of alcohol intake to be 25 (10–52) years in alcoholic cirrhosis when compared to 20 (10–30) years in alcoholic CP.[31] Similar findings were noted by Noel *et al*.[32] In a study by Canha *et al*., although the duration of alcohol intake was longer in ALD patients compared to ALP patients, the difference was not statistically significant.[20] Thus, most studies have uniformly reported a lower duration of alcohol intake for patients presenting with pancreatitis compared to liver disease.

In the present study, 91% of patients with ALD and 73% with ALP consumed alcohol every day. However, no such difference was observed in another study.[31] So, it is difficult to conclude that more regular chronic intake of alcohol leads to ALD rather than ALP.

The pattern of alcohol intake was divided into two types: (i) those who started drinking from morning and (ii) those who started in the evening. A larger number (61%) of patients with ALD consumed alcohol from the morning than patients with ALP (30%). We also found that more patients of ALP (29 [38.26%]) consumed alcohol at dinner time compared to ALD (20 [27%]). Drinking outside the meal time has been reported to increases the morbidity of ALD by 2.7-fold.[34] Binge drinking was significantly greater in ALP (46 [60.5%]) patients than in those with ALD (15 [20.3%]). In a study from Iceland comparing ALP patients with those with alcohol use disorder, binge drinking was found to be much more common in alcohol use disorders than ALP, thereby suggesting that ALP may be an idiosyncratic reaction to alcohol intake.[19]

The mean amount of alcohol consumed per day by the total population was 178 ± 101 g. There was no significant difference between the two groups. Similar findings have been reported by other authors as well.[29,31] No difference in the type of alcohol used was observed between patients with ALD and ALP. Similar findings were observed in other studies.[29,31] In our study, smoking habits were not significantly different between both the groups. However, studies from Portugal and India found significantly higher smokers amongst ALP groups.[20,29]

The patients with ALP had significantly lower weight and lower BMI compared to the patients of ALD. Aparisi *et al*.[31] also reported similar findings, but Noel *et al*.[32] observed no difference between the two groups. Hence, we conclude that malnutrition is more common in the patients of ALP compared to the patients of ALD. In our study, patients with ALP had more significantly decreased MAC and triceps fold thickness compared to ALD. Mezey *et al*. observed a similar reduction of TSFT in patients with pancreatitis. They also found that MAC was decreased in all the patients, but the decrease was less marked in patients with alcoholic hepatitis.[35]

In our study, the mean daily calorie, protein, and fat intake were 1643 ± 392.4 kcal/day, 47 ± 14.6 g/day, and 40.9 ± 12 g/day, respectively. The daily calorie and protein intake were lower than the recommended daily requirement for Indians.[31] These observations are indicative of the presence of malnutrition in the whole study population. No difference in total calorie, protein, fat, and carbohydrate intake was seen between patients with ALP and ALD. In a study from France, ALP patients were found to consume a diet richer in saturated fats and animal proteins than alcoholic cirrhotic patients.[32] Wilson *et al*. reported that patients with ALP exhibited higher intakes of protein (109 ± 9 *vs* 88 ± 5 g/day), fat (148 ± 15 *vs* 115 ± 8 g/day), and carbohydrate (383 ± 39 *vs* 289 ± 25 g/day) in comparison to alcoholic cirrhotic patients.[36] A study from Portugal demonstrated that patients with ALD had a less abundant diet, whereas patients with ALP had a more abundant diet prior to onset of their illness.[20] To sum up, the daily calorie as well as protein, fat, and carbohydrate intake in Indian alcoholic patients as seen in our study were far less than that of the western population. Unlike other studies,[35] where the patients with ALP exhibited higher intake of proteins, fats, and carbohydrates than patients with ALD, we observed no such differences.

Our reported data highlights the alcohol consumption pattern, amount, and nutritional parameters in both alcoholic liver and pancreatic disease. Our data will help not just the management of patients but also the counseling of the patients regarding stopping alcohol consumption and nutritional advice. However, the present study has a few limitations. First, this was a single-

center study, and our findings need to be confirmed by large multicenter studies. Second, our institute is a tertiary referral center for patients with alcoholic liver and pancreatic diseases; thus, there is inherent referral bias. Following up on these patients prospectively over a longer period of time would have given more information regarding changes in alcohol consumption pattern and nutrition parameters after counseling.

In conclusion, while there was no difference in the age of starting alcohol consumption and total amount of alcohol consumed per day between patients of ALP and ALD, patients with ALD started consuming alcohol from the early morning itself. On the contrary, ALP patients were younger, did more binge drinking, consumed alcohol more with friends, and often in the evening. Our patients consumed lesser calories as well as proteins, fats, and carbohydrates compared to Western patients, and patients with ALP were more malnourished compared to the patients with ALD.

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