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Original Research Article

Impact of alcohol on gastric mucosa in a population with high prevalence of *Helicobacter pylori*

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ABSTRACT

Background: The purpose of the study was to see whether chronic alcohol abuse had any effect on the gastric mucosa in a population already affected by a high prevalence of *Helicobacter pylori*.
Methods: 35 males with a history of chronic alcohol abuse were compared with 35 males who were abstinent or social drinkers. All subjects had complaints of dyspepsia. All subjects underwent endoscopy and targeted biopsies were taken from three specific sites in the stomach, namely body, antrum and incisura. Biopsies were studied to look for changes of atrophic gastritis and intestinal metaplasia. The presence or absences of H. pylori on the tissue biopsy were also recorded.
Results: Atrophic gastritis were only assessable in 24 alcoholic patients and 21 non-alcoholic patients due to the inadequacy of the depth of the biopsy. AG were found to be equally distributed in both the groups. 23 (64.9%) patients in the alcoholic group and 19(54.5%) in the control group had AG (OR-1.54, p=0.47). Intestinal metaplasia was seen in 10 (28.5%) alcoholic group and 12 (34.2) in the control group (OR-0.65, p=0.45). Of the 42 subjects detected to have AG, 16 (38.1%) had IM. However, IM were always associated with AG. In addition, *H. pylori* were not seen to be different in the two groups. *H. pylori* were positive in 18 (51.4%) alcoholic and14 (40%) non-alcoholic patients (p=0.33).
Conclusions: Chronic alcohol abuse doesn't appear to have any major impact on the gastric mucosa in terms of producing premalignant lesions such as atrophic gastritis or intestinal metaplasia or enhancing the prevalence of *H. pylori*.

Keywords: Atrophic gastritis, Intestinal metaplasia, Gastric cancer

INTRODUCTION

The primary reason for conducting this study was to determine if alcohol abuse damaged the mucosa of the stomach in any way. In addition, it was to examine whether alcohol abuse affected the prevalence of *Helicobacter pylori (H. pylori)* colonization of the gastric mucosa. The evidence so far has been confusing. Some have claimed that alcohol does cause damage and others have claimed the opposite.[1,2] These studies have so far been conducted in areas with a low prevalence of *H. pylori* infection. We are reporting from a population with a high infection rate of *H. pylori*.[3] The parameters of mucosal damage that were studied were the presence of atrophic gastritis (AG) and intestinal metaplasia (IM). These are common signs of chronic mucosal damage and are also putative premalignant conditions.[4]

Alcohol abuse has a bearing on malignancy in the gastrointestinal tract. It is a definite risk factor for cancers of the oesophagus, colon, pancreas, liver and gall bladder as well as those of the pharynx and larynx.[5,6] Its risk

status for carcinoma stomach is still undefined. Bagnardi et al have concluded that heavy drinking increases the risk by an odd ratio of 1.4, but another meta-analysis has questioned the finding.[7,8]

H. pylori is widely reported to be a known risk factor for carcinoma stomach in certain populations.[9] India is known to have a high prevalence of *H. pylori* infection.[10] Yet the risks of developing cancer due to *H. pylori* in India are uncertain.[11] Carcinoma of stomach appear to have a high prevalence in North India, in comparison to the south.[12] Therefore, it was also pertinent to see if the association of alcohol abuse and *H. pylori* together produced more damage than either alone.

METHODS

This prospective cohort study was conducted at Pondicherry institute of medical sciences, during the period of October 2015 to March 2016. Subjects for this study were drawn from the patients who attended the medical gastroenterology outpatient department with symptoms of dyspepsia, and the patients who were admitted in the psychiatric ward for alcohol de addiction with complaints of dyspepsia.

Study population

Two groups of subjects were analysed in this study. This study group comprised of subjects between ages of 18 to 60 years with a history of dyspepsia. The study group consisted of chronic alcohol abusers, for the purpose of this study. Chronic alcoholic abusers were defined as adult male who consume a minimum 21 units of alcohol per week for a minimum for 2 years and the controls consisted of subjects who were either teetotallers or social drinkers. Social drinkers were defined as patients who consume less than 8 units of alcohol per month, and their last drink was at least one week prior to the study.

Patients who were found to have a liver pathology and alcoholic hepatitis, cirrhosis, varices, signs of portal hypertension and any definite disease found on endoscopy were excluded and woman and adult males less than 18 years of age were excluded from the study.

All patients enrolled in the study had chronic dyspepsia. Dyspepsia for the purpose of this study was defined as upper gastro intestinal symptoms as burping, belching, early satiety and post cibal discomfort. People with abdominal pain of moderate to severe intensity were excluded from this study. Each patient gave written consent after having been informed about the nature of the study and the procedure that was going to be performed.

Sample size

The sample size; n=35 for each group, was calculated using Epi info software with 80 % power (Z=0.84), 95% confidence interval (z=1.96), r=1 (equal number of cases and controls), assumed odds ratio (OR=4) and the proportion exposed in the control group is 20%.

Endoscopy and histopathological examination of tissues

Prior to endoscopy, baseline data about the duration, severity of dyspepsia, the amount and duration of alcohol consumption were noted. Prior use of proton pump inhibitors and antibiotics were also recorded. The endoscopy was done by a single endoscopist who was unaware of the group to which the subject belonged.

Single biopsies were taken from body, incisura and the antrum of the stomach. No attempt was made to identify the disease tissue using narrow band imaging prior to taking the biopsies. Biopsies were collected in formalin and processed for histological examination. They were studied by single pathologist, who was also blinded to the group to which the subjects belonged. Special preferences were given to the presence of *H. pylori*, atrophic gastritis and gastric metaplasia. Although *H. pylori* were graded mild, moderate and severe it was not used in the analysis in this study.

Statistical analysis

Data was entered in individual proforma. The data was transcribed to the computer using SPSS version 20.0. The association of atrophic gastritis with *H. pylori* infection were determined using Chi Square. Independent ODDs ratio and its significance was estimated in the model I including alcoholism, H2 Blocker, and recent antibiotic use.

RESULTS

The subjects were all male and the ages were comparable. Alcoholic mean and SD 40.74$\pm$5, non-alcoholic mean and SD 41.85$\pm$12.6. Regarding symptoms, the majority of patients in both the groups complained of a burning type of abdominal pain. 81% of patients complained of abdominal discomfort, 17% had abdominal pain, 11% had bloating. The mean duration of symptoms was 3 months in both groups. Distribution of symptoms is depicted in (Table 1).

In the alcohol group, the mean alcohol consumption per week was 120 units. The alcohol most commonly consumed was brandy. The mean duration of alcohol consumption is 9.5$\pm$5 yeara. 4 patients in the control group were social drinkers with a monthly consumption of 7 units.

Endoscopy was normal in 26 patients (13 in each group), 14 had mild erythematous pan gastritis (7 in each group). 9 had oesophageal candidiasis (6 in alcohol group and 3 in non-alcohol group) and 7 had mild erythematous antral gastritis. (3 in alcohol and 4 in non-alcohol group), 6 had mild erosive duodenitis (4 in alcohol group and 2 in non-

alcohol group). Other findings such as pre pyloric ulcer, Lax Les, achalasia, focal duodenal mucosa, esophagitis, and duodenal lymphangiectasia are labelled as miscellaneous (Table 1).

Table 1: Endoscopy Findings of patients.

Findings	Alcoholic	Non alcoholic	Total
Normal	13	13	26
Oesophageal candidiasis	6	3	9
Mild erythematous pan gastritis	7	7	14
Mild erythematous antral gastritis	3	4	7
Mild erosive duodenitis	4	2	6
Miscellaneous	2	6	8
Total	35	35	70

Histological examination

Atrophic gastritis & Intestinal metaplasia; out of 24 subjects in alcoholic group, 23 patients were found to have atrophic gastritis. Out of 21 subjects in non-alcoholic group 19 were found to have atrophic gastritis, (Table 2). The differences between the 2 groups were not significant (p=0.6).

Table 2: Atrophic gastritis in alcoholic and non-alcoholic patients.

Atrophic gastritis	Alcoholic	Non alcoholic	Total
Negative	1	2	3
Positive	23	19	42
Total	24	21	45

Interstitial metaplasia (IM) could be assessed in all patients, IM were seen in 10 (28.5%) alcoholic and 12 (34.2%) patients in control group. The difference again is not statistically significant (OR-0.65, p=0.45) (Table 3).

Table 3: Intestinal metaplasia in alcoholic and non-alcoholic patients.

Intestinal metaplasia	Alcoholic	Non alcoholic	Total
Negative	25	23	48
Positive	10	12	22
Total	35	35	70

Out of the 42 subjects detected to have AG, 16 (38.1%) had IM and 26 (61.9%) had no IM. However intestinal metaplasia was always associated with atrophic gastritis (Table 4).

H. pylori infection

Prevalence of *H. pylori* by histological examination was 18 (51.4%) alcoholic and 14 (40%) non-alcoholic patients. The difference in prevalence in the 2 groups were not significant (p=0.33) (Table 5).

Table 4: Atrophic gastritis and intestinal metaplasia.

Atrophic gastritis	Intestinal metaplasia		
	Negative	**Positive**	**Total**
Negative	3	0	3
Positive	26	16	42
Total	29	16	45

Table 5: *H. pylori* in alcoholic and non-alcoholic patients.

H. Pylori	Alcoholic	Non alcoholic	Total
Negative	17	21	38
Positive	18	14	32
Total	35	35	70

Due to the absence of muscularis mucosa of the biopsies in 25 subjects (11 in alcoholic group and 14 in non-alcoholic group) AG could be assessed in only 45 subjects (24 in alcoholic group and 21 in non-alcoholic group). Out of 35 alcoholic subjects, 18 were *H. pylori* positive and 17 were *H. pylori* be negative. 14 and 10 could be assessed for atrophic gastritis respectively. Out of this number all 14 who had a combination of alcoholism and *H. pylori* positive had atrophic gastritis, while 9 out of 10 who were alcoholic but *H. pylori* negative had atrophic gastritis (p=1.0, Pearson chi square=0.60) (Table 6). Therefore, the combination of alcohol and *H. pylori* does not make atrophic gastritis more likely.

Sub group analysis of intestinal metaplasia in alcoholics with and without *H. pylori* showed that group with alcoholism with *H. pylori* positive and alcoholism with *H. pylori* negative; 5/18 (28%) and 5/17 (30%) respectively, were fairly similar. Therefore, alcoholism does not appear to cause more atrophic gastritis or intestinal metaplasia in the combination of *H. pylori.*

Relationship of age to atrophic gastritis and intestinal metaplasia

The mean age and SD of patients with atrophic gastritis in the group who were alcoholic were 40.6±5, and was no different from that of the control group 43.2±11.8. Similarly, the mean age and SD of patients with intestinal metaplasia in the group who were alcoholics were 44.8±8.9, and was no different from that of the control group 42.5±11.6).

Table 6: Intestinal metaplasia, alcoholics and non-alcoholics with *H. pylori*.

Parameters			Intestinal metaplasia		Total	P value
			Negative N (%)	Positive N (%)		
Alcoholic	H. *pylori*	Negative	12 (70)	5(30)	17	1.00
		Positive	13 (72)	5(28)	18	
	Total		25 (100)	10 (100)	35	
Non-alcoholic	H. *pylori*	Negative	14 (67)	7(33)	21	1.00
		Positive	9 (64)	5(28)	14	
	Total		23 (100)	12 (100)	35	
Total	H. *pylori*	Negative	26 (69)	12(31)	38	0.96
		Positive	22 (69)	10(31)	32	

Table 7: Similar studies showing varying results.

Author	Country/year	Population	Findings
Brenner	Germany/2001	1410	Alcohol consumption decreases *H. Pylori*
Hauge	Sweden/1997	22	Alcohol consumption is associated with increase in *H. Pylori*
Hauge, Pressen	Sweden/1994	24	Alcohol abuse is not associated with *H. Pylori*
Uppal	Japan/1991	18	Alcohol abuse associated with increase in *H. Pylori*
Li Zhang	China/2009	139	Alcohol abuse associated with increase in *H. Pylori*
Lei Gao	Germany/2009	9444	Alcohol abuse decreases *H. Pylori*
Ohkuma	Japan/2000	163	*H. Pylori* increases AG, IM
Brenner	Germany/1999	1785	Alcohol abuse decreases *H. Pylori*
Ozasa	Japan/1999	62	Alcohol not associated with AG, *H. Pylori* causes AG
Parl ff	Germany/1979	72	Alcohol associated with chronic gastritis
Buzas G. M.	Hungary/1997	114	Alcohol abuse decreases *H. Pylori*
Paunio M.	Finland/1994	451	Alcohol abuse associated with increase *H. Pylori*

Duration of alcohol consumption and intestinal metaplasia

As the majority of patients who could be assessed for atrophic gastritis in the alcoholic group was over 95%, it was not possible to determine whether the length of drinking was related to the presence of atrophic gastritis, however with intestinal metaplasia this analysis could be made. Those with intestinal metaplasia had a mean drinking period of 11±8 years and those without intestinal metaplasia had mean drinking period was 9.28±4.5 years. This shows that intestinal metaplasia does not seem to be affected by the duration of drinking.

DISCUSSION

The primary reason for conducting this study was to determine if alcohol abuse damaged the mucosa of the stomach in any way and whether it affected *H. pylori* colonization of the gastric mucosa and if the combination of alcohol abuse and *H. pylori* made any damage worse. *H. pylori is* associated with atrophic gastritis and intestinal metaplasia which are putative premalignant conditions.[13]

The fact that alcohol is commonly used in India is an important reason to study its impact on the gastric mucosa. According to one report, 20 to 38 percent of males and 10 percent of the female population consume alcohol.[14] A rural study has reported 4.6 % of alcohol imbiber drinkers are dependent on alcohol and 6% were drinking alcohol at hazardous levels.[15]

Alcohol abuse is a definite risk factor for cancers of the oesophagus, colon, pancreas, liver and gall bladder as well as those of the pharynx and larynx.[5,6] Its risk status for carcinoma stomach is still undefined. Bagnardi et al have concluded that heavy drinking increases the risk by an odds ratio of 1.4, but another meta-analysis has questioned the finding.[7,8] Alcohol is also the third-largest risk factor for all human disease.[16] The duration of drinking may explain the varying results found in other studies.

H. pylori are widely reported to be a known risk factor for carcinoma stomach in certain populations.[9] India is known to have a high prevalence of *H. pylori* infection.[10] Yet the risks of developing cancer due to *H. pylori* in India are uncertain.[11] Carcinoma stomach varies in prevalence across the subcontinent of India. It appears to

have a high prevalence in the North East, a low level in the North of India and a middling level of prevalence in the South. Epidemiological review of gastric cancer in India.[12]

Our results show that there is no difference in the incidence of atrophic gastritis or gastric metaplasia in those who abused alcohol versus teetotallers. However, it was interesting to note that there was a trend towards finding gastric metaplasia more commonly in those with a longer duration of drinking. This difference however was not statistically significant. A larger cohort may clarify this issue more clearly. Duration of drinking may explain the varying results found in other studies.

Again, there were no difference in the incidence of *H. pylori* in those who used alcohol as against those who did not. This issue has been investigated by others and the results are varied. Three studies from Germany using large numbers have shown a decrease in *H. pylori* and those from China and Japan have shown an increase in those abusing alcohol (Table 7).[17-20] The sample size in this study was too small to resolve this issue. However, as there was no difference in the calculated prevalence, in this small study it is unlikely that alcohol abuse has a major impact on those on *H. pylori* in a population with a high prevalence of *H. pylori*. As regards to the third question, the incidence of gastric metaplasia was equal in those alcoholics with *H. pylori* compared to alcoholics without *H. pylori.*

Therefore, there did not seem to be a big difference in the damage to the mucosa in the presence or absence of H.*pylori*. Current study was the first to be conducted in a population of high prevalence of *H. pylori*. Despite the high prevalence of *H. pylori*, alcohol abuse did not make any remarkable changes in the gastric mucosa. The only indication of damage is that gastric metaplasia which correlates with gastric cancer of all the premalignant lesions tends to occur more often in those with a longer history of drinking.

In general, we want to emphasize from the findings in our study that the impact of alcohol seems to be minimal on the gastric mucosa even in populations of high prevalence of *H. pylori*. This fits in with the data that alcohol is not risk factor for Gastric Cancer.

CONCLUSION

Chronic alcohol abuse doesn't appear to have any major impact on the gastric mucosa in terms of producing premalignant lesions such as atrophic gastritis and intestinal metaplasia or enhancing the prevalence of *H. pylori*.

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