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Pattern of Alcohol Consumption and its Effect on Gastrointestinal Symptoms in Inflammatory Bowel Disease

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Abstract

Alcohol consumption is a potential trigger for flare in Inflammatory Bowel Disease (IBD) flare because of alcohol's pro-oxidant effects and its deleterious effects on gut barrier function. The association with alcohol consumption and IBD flare is unclear. To test this hypothesis, we evaluated the pattern of alcohol consumption and its self-reported effect on gastrointestinal (GI) symptoms in patients with IBD. We recruited 129 consecutive patients: 52 patients with Crohn's Disease, 38 patients with Ulcerative Colitis, and 39 patients with Irritable Bowel Syndrome (IBS). All participants completed a validated questionnaire on disease activity, the CDAI or UCAI respectively, validated questionnaires to quantify alcohol consumption by NIAAA criteria, and two structured questionnaires we designed to access patients' perception of the effect of alcohol on their GI symptoms and on overall GI symptom severity. The pattern of current, light, moderate, and heavy alcohol consumption in inactive IBD was similar to the general US population. Specifically, 56 of 90 (62%) of inactive IBD patients were current drinkers, compared to 61% in the general US population. Of current drinkers, 75% of IBD (N=42), and 43% of IBS (N=9) reported a worsening of GI symptoms with alcohol consumption (p=0.01); however, overall GI symptom severity did not differ when compared to quantity of alcohol consumed. Patients with inactive IBD drink alcohol in quantities similar to the general population. Current drinkers with inactive IBD are more likely to report worsening of GI symptoms with alcohol than current drinkers with IBS.

Keywords

Crohn's Disease; Ulcerative Colitis; Alcohol Consumption

Introduction

A therapeutic goal in inflammatory bowel disease (IBD) is to prevent episodes of symptomatic flare and prolong periods of asymptomatic remission. Identifying potentially modifiable factors for disease flare is important to limit the use of steroids, avoid

hospitalizations, and improving quality of life. Numerous studies have examined whether environmental factors such as diet modify disease activity in IBD. Studies have shown a link between diet and the development of IBD (Geerling et al., 2000; Reif et al., 1997; Sakamoto et al., 2005), but thus far there is no conclusive evidence that diet can modulate disease activity.

The best established enviromental factor that can affect IBD is cigarette smoking. Cigarette smoking has been shown worsen disease activity in CD (Cottone et al., 1994), and is associated with an increased need for steroids and a higher risk for surgery (Cosnes et al., 2001). However, in UC smoking reduces the risk of colectomy and decreases the extent of disease Green et al., 1998). The exact mechanism of the effect of smoking in IBD is not known, but it does highlight the important role social factors can play in the disease.

Another important social factor is alcohol consumption. Alcohol consumption has been associated with a wide variety of deleterious health effects such as liver disease and cardiomyopathy (Klatsky, 2007). In addition, acute and chronic alcohol consumption have been shown to modify the immune system and could therefore play an important role in IBD. Acutely, alcohol consumption has been shown to inhibit the immune system by decreasing T cell activity and interlukin (IL)-12 levels in healthy controls (Mandrekar et al., 2004). Alcohol can also increases monocyte production of anti-inflammatory cytokines such as IL-10 (Norkina et al., 2007). Chronically, alcohol increases liver Kupffer cell activity and is associated with increased generation of proinflammatory mediators such as TNF-α, IL-1, and IL-6 (Khoruts et al., 1991). Alcohol has also been shown to acutely disrupt gut barrier function, and can increase intestinal permeability in human subjects (Keshavarzian et al., 1994) to which patients with IBD are particularly susceptible (Wyatt et al., 1997).

Even though alcohol has a variety of effects on the immune system, alcohol consumption in IBD has rarely been examined in previous studies. Accordingly, the aims of this current study was to: (1) determine the rate and pattern of alcohol consumption in a cohort of patients with inactive IBD by NIAAA criteria; (2) evaluate the self-reported effect of alcohol consumption on IBD patients GI symptoms and (3) determine if there is any correlation with quantity of alcohol consumed and overall severity of GI symptoms.

Materials and Methods

Study Design

This was a cross-sectional survey of IBD patients done by questionnaire. Patients were recruited from GI and IBD clinics in a metropolitan university practice in Chicago, IL (Rush University gastroenterology) and a private gastroenterology clinic in Macon, GA from July 2004 until 2006. During the one and a half year period, consecutive patients with the established diagnosis of CD and UC who were in clinical remission and patients with the diagnosis of IBS were invited to participate in the study. After signing an informed consent, all patients were given a set of questionnaires to complete. The questionnaire packet included a demographic data form, validated questionnaires on disease activity [Crohn's disease activity index (CDAI) or ulcerative colitis clinical activity index (UCAI), respectively] and alcohol consumption, as well as a structured questionnaires we developed regarding the effect of alcohol on the GI symptoms and overall severity of GI symptoms. All questionnaire packets were labeled by a sequential patient number to maintain patient confidentiality, and included a post-marked envelope for return to our GI office.

The study was approved by Rush University Investigational Review Board (IRB) (Chicago,IL) and Mercer University IRB (Macon, GA).

Inclusion Criteria and Exclusion Criteria

All IBD patients included in the study had biopsy proven CD or UC. IBS patients had to meet the Rome II criteria for IBS, and have a normal colonoscopy (except for hemorrhoids or diverticuli) and normal random colonic biopsies if the primary symptom was diarrhea. Patients were eligible to be included in the study if the clinician who saw them at the time of inclusion determined that they currently had inactive disease. The status of disease for IBD patients was then confirmed using IBD clinical indices – CDAI < 150 or UCAI < 4 for inactive disease. Patients were excluded from the study if they reported active disease in the three months prior to the enrollment.

Questionnaires

A. Demographic & Medical History Data Form—The demographic variables we collected included age, race, marital status, education, and occupation. In addition patients were asked to indicate different factors regarding the history of GI disease – length of duration of diagnosis, recent disease activity, and family history of GI medical conditions.

B. IBD disease activity indices—We used two validated questionnaires to access CD and UC, the CDAI and UCAI, that have been well described previously (Sandborn et al., 2002; D'Haens et al., 2007). These clinical questionnaires have IBD patients rate their current symptoms based on clinical questions – stool frequency, abdominal pain, general well being, etc.

C. Assessment of alcohol consumption—The National Institute of Alcohol Abuse and Alcoholism (NIAAA) has quantified alcohol consumption in the United Sates as part of the National Health Interview Survey (NHIS) since 1983 (Adams and Schoenborn, 2006). Pattern of alcohol consumption was determined by a validated questionnaire that divides alcohol consumption into a period specific normal week (PSNW). The PSNW has been found to be more accurate and has less underestimation of alcohol consumption than the quantity-frequency alcohol questionnaire (Romelsjo et al., 1995). The questionnaire asked patients to best describe the average amount of alcohol they consumed in a typical week over the last year. The volumes for a standard drink of 12 grams of alcohol were listed just below each type of beverage, to prompt subjects to answer more accurately. Accordingly, we grouped subjects into NIAAA categories as previously described – abstainers (less than 12 drinks per year), current drinkers (more than 12 drinks per year), light drinkers (3 or fewer drinks per week), moderate drinkers (3 to 14 drinks per week), and heavy drinkers (over 14 drinks per week). Binge drinkers were defined as over 5 drinks on one occasion. Subjects were also prompted to mark their "beverage of choice" or the main type of alcohol that they usually consumed.

D. Effect of alcohol consumption on GI symptoms—We developed a structured questionnaire to determine the perceived effects of alcohol consumption on GI symptoms. Subjects were asked if alcohol affected their gastrointestinal symptoms – diarrhea, abdominal pain, or bloating and were prompted to mark worse, the same, or better. Subjects then indicated how many drinks they believed it would take to make their symptoms worse, the same, or improved. In addition, if abstinent from alcohol, patients were asked if the reason for abstinence from alcohol was from worsening GI symptoms.

E. General Gastrointestinal Rating Severity Form—This structured questionnaire asked questions regarding the severity of gastrointestinal symptom based in four categories - overall GI symptoms, abdominal pain, diarrhea and/or constipation, gas/bloating, and heartburn. Subjects were prompted to rate their symptoms as either absent, mild, moderate,

or severe. This format is similar to other previously described questionnaires (Svedlund et al., 1988) accessing GI symptom severity.

Statistical Analysis

We used the Pearson's chi-square test of association to compare proportional differences between groups. We used a p-value of 0.05 for all tests and data was analyzed using, SPSS version 12.0.1 (SPSS, Inc. Chicago, Ill).

Results

Patient Characteristics

A total of 129 subjects completed the study: 52 with CD, 38 with UC, and 39 with IBS. Subject characteristics are listed in Table 1. Mean age was 42, 39, and 53 for CD, UC, and IBS respectively. The mean age was significantly older patients in our IBS subjects. All groups had a predominance of female subjects ranging from 62% in CD to 82% in IBS. There were no other statistical differences in gender, marital status, mean years of GI diagnosis, mean years of education, and family history of GI disease between the three groups.

Alcohol Consumption

The quantity of alcohol consumption by the NIAAA criteria for light, moderate, and heavy drinkers is also listed in Table 1. Patients who abstained from alcohol were 46% of CD, 42% of UC, and 48% of IBS. The rate of current drinkers was 54% in CD, 61% in UC, and 49% in IBS. There was no statistical differences between the rates of abstainers, current drinkers, light, moderate, or heavy drinkers between the three groups.

After quantifying alcohol consumption in our cohort by the NIAAA criteria, a comparison was made with the general US population and is listed in Table 2. Overall pattern and quantity of alcohol consumption was very similar between these two populations. Specifically, the rate of abstainers, the number of current drinkers, and the number of binge or heavy drinkers were comparable. There were slightly more moderate drinkers, 29% vs. 14%, in our IBD cohort.

The Effect of Alcohol on GI symptoms

All patients completed the questionnaire on the perceived effect of alcohol on their GI symptoms. Most of the abstainers (n=51) could not recall the effect of alcohol on their GI symptoms, except those who quit specifically to avoid alcohol. The profile of patients that completed the effect of alcohol on their GI symptoms is listed in Table 3. Current drinkers with inactive IBD reported a statistically significant worsening of GI symptoms with alcohol when compared to the current drinkers with IBS (p= 0.01). To represent the data graphically subjects that ranked their symptoms the same or improved were grouped together and are shown in Figure 1.

We also assessed the severity of GI symptoms in relationship to the quantity of alcohol consumed in Table 4. There was no statistical difference in overall severity of GI symptoms when compared with quantity of alcohol consumed. Similarly was no difference between the severity of other self reported GI symptoms (abdominal pain, diarrhea/constipation, or gas/bloating) and the quantity of alcohol consumed per week in IBD or IBS subjects. The severity of GI symptoms did differ significantly between disease groups, with IBS patients reporting more severe GI symptoms than IBD (p=0.02).

No correlation was observed between the type of alcoholic beverage consumed and GI symptoms in IBD or IBS subjects. However, those with IBD who had heartburn or other upper GI symptoms used significantly less amount of alcohol compared to IBD subjects who did not have these symptoms (Figure 2). This association was not found in subjects with IBS.

Discussion

The concept that the environment may alter disease activity has been an area of controversy in IBD. Previous clinical studies in diet (Jowett et al., 2004), stress (Mawdsley et al., 2006), and sleep (Keefer et al., 2006) have found a possible relationship, but clinical studies have been disappointing. Currently there is no direct evidence that day to day environmental factors alter the course of disease in IBD, except for one prevalent social habit, cigarette smoking. Alcohol consumption is another prevalent social habit that can also modify immune activity. Alcohol has been previously examined through a prospective detailed diet history in 191 patients with inactive UC over a one year period (Jowett et al., 2004). In that cohort, patients that had a relapse or disease flare over that year consumed 14 grams of alcohol daily compared to 10 grams daily in non-relapsers. In CD, the acute effects of five alcoholic drinks in inactive CD showed that those drinks associated with increased plasma glucose levels but not increased ethanol were associated with worsening GI symptoms (Hey et al., 2007).

The primary goal of the current study was to quantity of alcohol consumption in inactive IBD subjects by NIAAA criteria so the quantity could be compared to national averages. In our cohort, the quantity of alcohol consumed in patients with IBD was very similar to national averages. Specifically, the rate of abstainers, current drinkers, and binge drinkers were comparable. This is an important epidemiological observation that suggests that patients with inactive IBD consume alcohol in an equivalent proportion to the general population. Overall, IBD patients do not seem to avoid alcohol for concern over its effect on GI symptoms.

IBS patients were selected as a comparison group in this study with IBD for two reasons – 1) they have GI symptoms at baseline that could be altered by alcohol, and 2) they had normal colonoscopies without inflammation. Healthy controls were thought to be an inappropriate comparison group as most patients would not have baseline GI symptoms. Our questionnaire on the effect of alcohol on GI symptoms showed that 75% of current drinkers with either inactive UC or inactive CD reported worsening of their GI symptoms with alcohol compared to 43% of IBS, which was statistically different. However, overall GI symptoms did not differ by quantity of alcohol consumed when not directly about alcohol. This is a less sensitive means to access the effect of alcohol on GI symptoms, and it is not surprising that no difference was found between the category of alcohol consumption and the severity of overall GI symptoms.

There are several possible explanations for the finding that alcohol worsened GI symptoms more in IBD than IBS. One possible explanation is that alcohol can alter the immune system and increase the risk of disease flare as was shown in UC (Jowett et al., 2004). The main hypothesis as to how alcohol could affect the luminal immune system is by increasing intestinal permeability and antigen exposure which has been shown previously in animal models (Tamai et al., 2000). Another possible explanation is that alcoholic beverages cause an osmotic diarrhea due to a high sugar content as has also been previously shown in CD (Hey et al., 2007). A retrospective questionnaire study such as this one is not able to differentiate between these hypotheses, and further studies are needed to examine these findings.

There was a significant decrease in the severity of upper GI symptoms as quantity of alcohol consumed increased. This is not surprising as alcohol has been known to worsen GERD by decreasing LES tone and decreasing gastric motility (Castell et al., 2004), and is likely avoided by IBD patients with moderate to severe GERD. Overall GI severity was different among disease states, with our IBS patients reporting worse disease symptoms compared to IBD. This finding is also not surprising, as IBS subjects have been shown to report worse quality of life scores than IBD in previous studies (Pace et al., 2003), and is a common finding between these two disease states.

In summary, we found that inactive IBD patients consume alcohol in a similar quantity and pattern as the general US population. In addition, inactive IBD patients reported worsening of their GI symptoms with alcohol when compared to patients with IBS. This finding may be secondary to alcohol causing a leaky gut and increased luminal immune activity or could be secondary to the high sugar content of most alcoholic drinks. In the future, clinical studies are needed to examine the possible link between alcohol consumption and its effect on disease course and luminal immune activity in IBD.

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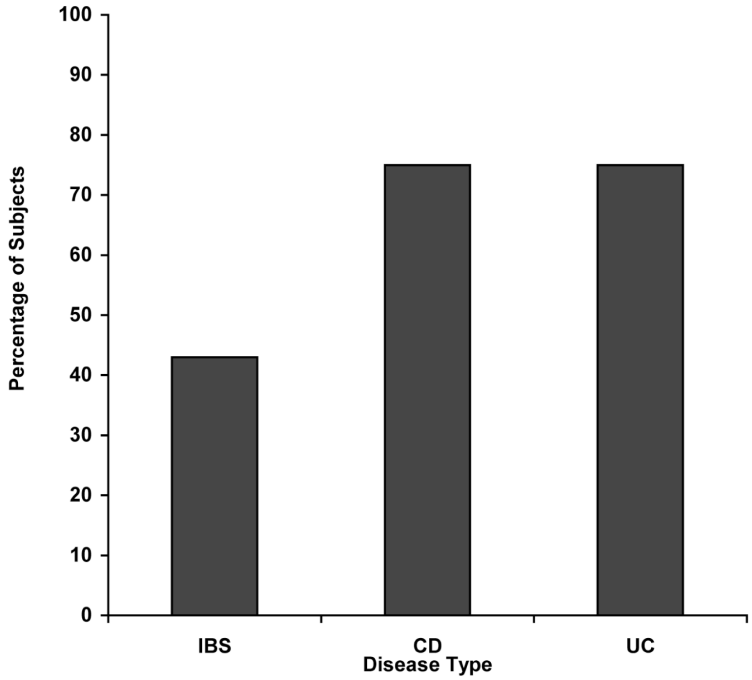

Figure 1.
Percentage of Subjects Reporting Worsening of Gl Symptoms with Alcohol Consumption

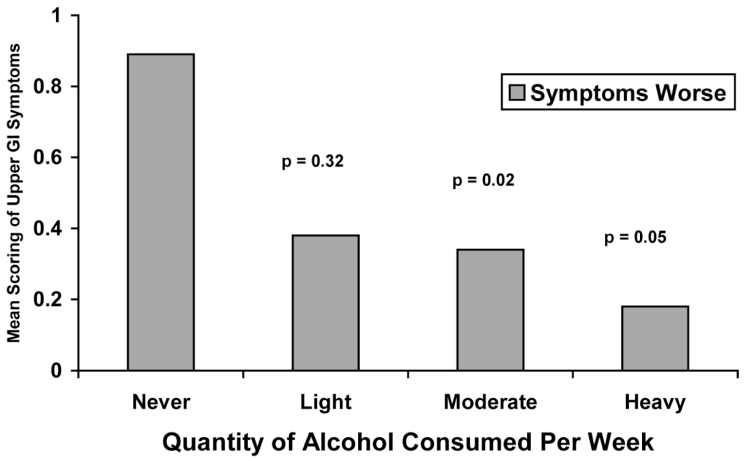

Figure 2.
Self-Reported Severity of Upper Gl Symptoms in UC and CD Quantity of Alcohol Consumption

Table 1

Patient Characteristics by Group

	CD (N=52)	UC (N=38)	IBS (N=39)	p-value
Mean Age	42.4	38.5	53.2	0.00
Gender, % female	62%, (32)	63%, (24)	82%,(32)	0.09
Marital Status, % Married	55%, (28)	47%, (18)	54%, (21)	0.07
Education, mean yrs	13.9	13.9	13.5	0.67
GI Diagnosis, mean yrs	12.7	9.5	8.9	0.11
Family History of GI Disease	50%	33%	62%	0.27
Abstainers	38%, (20)	37%, (14)	46%, (18)	0.82
Current Drinkers	62%, (32)	63%, (24)	54%, (21)	0.79
Light Drinkers	21%, (11)	26%, (10)	26%, (10)	0.61
Moderate Drinkers	33%, (17)	24%, (9)	18%, (7)	0.26
Heavy Drinkers	0%, (0)	3%, (1)	3%, (1)	0.50
Binge Drinkers	19%, (10)	16%, (6)	5%, (2)	0.15
Beer Drinkers	31%, (16)	29%, (11)	10%, (4)	0.54
Wine Drinkers	17%, (9)	08%, (3)	23%, (9)	0.19
Liquor Drinkers	12%, (6)	26%, (10)	18%, (7)	0.20

Table 2

Quantity of Alcohol Consumption between IBD and US Population

	CD	UC	IBS	US Population[1]
Abstainers	38%	37%	46%	39%
Current Drinkers	62%,	63%	54%	61%
Light Drinkers	21%,	26%	26%	43%
Moderate Drinkers	33%,	24%	18%	14%
Heavy Drinkers	0%	3%	3%	4%
Binge Drinkers	19%	16%	5%	15%

[1] Estimates calculated by the NIAAA from the 2004 NHIS

Table 3

Questionnaire about Alcohol and GI Symptoms

Disease	Abstainers	Drinkers	Same or Better*	Worse*
CD	20	32	8	24
UC	14	24	6	18
IBS	18	21	12	9

*Abstainers not included

Table 4

Category of Alcohol Consumption and Ranking of Overall GI symptoms as Severe or Moderate*

Disease	Light	Moderate	Heavy	p value
CD (N=49)	4%, (2)	16%, (8)	0%, (0)	0.72
UC (N=35)	6%, (2)	9%, (3)	3%, (1)	0.51
IBS (N=37)	27%, (10)	16%, (6)	3%, (1)	0.19

* 2 CD, 4 UC, and 4 IBS did not complete the questionnaire on overall GI symptoms