Turk J Gastroenterol 2007; 18 (4): 221-224

The acute effect of oral ethanol intake on gastric myoelectrical activity in healthy volunteers

Sağlıklı gönüllülerde oral etanolün gastrik miyoelektrik aktivite üzerine akut etkisi

Arzu ÇELEBİ KOBAK, Serhat BOR

Department of Gastroenterology and Hepatology, Ege University, School of Medicine, İzmir

The acute effect of ethanol intake on the upper gastrointestinal system is not clearly known. Animal studies have shown that ethanol inhibited the antral muscle fiber contractions and slow wave activity. ***Background/aims:*** *To show the acute effect of oral ethanol intake on gastric myoelectrical activity in healthy volunteers.* ***Methods:*** *Fourteen healthy male volunteers were included in the study. They were given control solution with test meal and ethanol with test meal on two different days. Electrogastrography recordings were obtained in pre-/postprandial periods.* ***Results:*** *On the days of ethanol intake, blood alcohol level reached above 80 mg/dl in 10 volunteers. Ethanol intake did not show any significant effect on any of the electrogastrography parameters.* ***Conclusions:*** *Though reasonable blood alcohol level was achieved and six volunteers experienced upper gastrointestinal system complaints, ethanol had no effect on gastric myoelectrical activity.*

Key words: Ethanol, electrogastrography, gastric myoelectrical activity

Etanolün üst gastrointestinal sistem üzerindeki akut etkisi tam olarak bilinmemektedir. Hayvan çalışmalarında, etanolün antral kas kontraksiyonlarını ve yavaş dalga aktivitesini inhibe ettiği gösterilmiştir. ***Amaç:*** *Sağlıklı gönüllülerde, oral etanolün gastrik miyoelektrik aktivite üzerine akut etkisini göstermektir.* ***Metod:*** *On dört sağlıklı erkek gönüllü çalışmaya alınmıştır. Gönüllülere, bir gün, test yemeği ve kontrol solüsyonu, başka bir gün, test yemeği ve etanol verilmiş, pre ve postprandiyal dönemde elektrogastrografi kaydı alınmıştır.* ***Sonuç:*** *Etanol verilen günlerde, 10 gönüllüde, kan alkol düzeyi 80 mg/dl üzerine çıkmıştır. Etanol alımının elektrogastrografi parametreleri üzerine anlamlı etkisi gözlenmemiştir.* ***Yorum:*** *Yeterli kan alkol düzeyine erişilmiş olmasına ve altı gönüllüde üst gastrointestinal sistem yan etkileri ortaya çıkmasına rağmen, etanolün gastrik miyoelektrik aktivite etkilemediği görülmüştür.*

Anahtar kelimeler: Etanol, elektrogastrografi, gastrik miyoelektrik aktivite

INTRODUCTION

The upper gastrointestinal system (GIS) is the first body compartment exposed to the direct effect of alcohol and exposure carries a risk of adverse events. Alcohol intake causes some gastric symptoms like nausea, vomiting and epigastric pain. These symptoms can be manifestations of disturbed functions of either the central nervous system or of GI motility due to acute or chronic alcohol consumption.

Several studies about alcohol and gastric motility have shown that alcohol generally slowed down gastric motility and delayed gastric emptying (1-5). The mechanism of this alcohol-related gastroparesis is still not fully understood. The major component of alcohol, ethanol (EtOH), might induce a direct toxic effect on muscle and nerve cells, inhibit acetylcholine secretion at the neuromuscular junction or slow down gastric motility via some GIS hormones. Some animal studies demonstrated that EtOH induced depression of gastric myoelectrical activity (GMA) and decreased the amplitude of antral contractions (6-10).

There are limited data about the acute effect of EtOH intake on the human stomach. Based on the existing experimental studies, EtOH-induced acute GIS complaints might be related with its effect on GMA. Today, electrogastrography (EGG) is the only in vivo and non-invasive method of recording GMA by using electrodes placed on abdominal skin (11,12). The aim of our study was to show the probable acute effect of oral EtOH intake on GMA in healthy volunteers.

Address for correspondence: Arzu ÇELEBİ KOBAK
Department of Gastroenterology and Hepatology, Ege University, School of Medicine, İzmir, Turkey
Phone: +90 232 365 11 11 • Fax: +90 232 365 11 11
E-mail: arzukobak@yahoo.com

Manuscript received: 07.12.2006 **Accepted:** 13.09.2007

Supported by: Ege University School of Medicine, Research Foundation (2000-002)

MATERIALS AND METHODS

Fourteen healthy male volunteers (medical students), with body mass indexes (BMI) between 20-25, were enrolled in the study. The main exclusion criteria were chronic alcohol abuse and existence of any systemic disease, operation or medication which might have an effect on GMA. The study was approved by the Human Ethical Committee of Ege University. All of the volunteers were informed and written consents were obtained before the study. Liver function tests, serum urea and creatinine, fasting blood glucose level, and whole blood counts were checked and abdominal ultrasonography was performed. Three volunteers were social drinkers (mean alcohol consumption: 23.3 (20-30) g/week) and they were asked to avoid alcohol for one week. Smoking was prohibited for eight hours before the procedure.

The aim in our study was to reach a blood alcohol level above 80 mg/dl, which is the upper limit of the legal blood alcohol level. We performed preliminary studies with different amounts of oral EtOH beginning from 0.5 g/kg body weight, the lowest amount used in human studies. We found that 0.8 g/kg oral EtOH would be best choice to reach the targeted blood alcohol level in our Turkish healthy male study population. The test solution consisted of 0.8 g/kg pure EtOH (99.9%, Riedel de-Haen, Germany) diluted to 40% in orange juice. EtOH was replaced with distilled water for control solution. The standard test meal was 500 kcal with 40% carbohydrate, 40% fat and 20% protein content and consisted of 75 g white bread, 90 g intermediate fatty white cheese, one spoonful of butter and one medium sized apple.

Cutaneous recording of GMA was performed by Polygram (Synectics Medical AB, Stockholm, Sweden) following an 8-hour fasting period. The gastric antrum was located and marked by ultrasonography and bipolar active surface electrodes were then placed along the antral axis. Skin was shaved when necessary. All the recordings were done at a frequency of 1Hz. The internal high-pass and low-pass filters were set at 1.8 and 16 cpm, respectively. Running spectral analysis method was used for EGG recordings. The collected data was digitalized and then analyzed with a computer program (ElectroGastroGram Version 6.30, Gastrosoft Inc., Synectics Medical AB, Stockholm, Sweden). Each tracing was visually inspected by a blinded observer to exclude the possible artefacts and artefacts were eliminated. The following EGG parameters were evaluated: mean dominant frequency (MnDF), normal gastric slow wave ratio, the percentage of tachygastria and bradygastria, power ratio (PR: mean dominant power (MnDP) after test meal / fasting), and dominant frequency instability coefficient (DFIC).

GMA was recorded by EGG on two different days, 60 minutes preprandially and 60 minutes postprandially, for each volunteer. The following meal was taken by the volunteer 10 minutes after preprandial recording: on the first day: control solution with standard test meal and on the second day: 0.8 g/kg pure EtOH diluted to 40% with orange juice and standard test meal.

On the days of EtOH intake, venous blood samples were taken at 15, 30, 45 and 60 minutes during postprandial recording for detection of blood alcohol level. Blood alcohol level was determined with enzymatic spectrophotometric method (Ethanol, Boehringer Mannheim).

Mean frequency (MnDF) of gastric slow waves between 2.4-3.7 cpm was defined as normogastria, 3.7-9 cpm as tachygastria and 0.5-2.4 cpm as bradygastria. Normogastria ratio ≥70% was accepted as normal. PR (MnDP after test meal / fasting) value <1 was accepted as abnormal because MnDP was expected to increase after a meal (11,13).

First and second day postprandial EGG parameters were compared (MS Excel 2000). Paired t-test was used for statistical evaluation of the results. PR value <1 was accepted as abnormal. The number of subjects with PR value lower than 1.0 on the first and the second day were also compared (Fisher's exact test).

RESULTS

Ages of the healthy volunteers were between 22-25 years and their mean BMI was 22.3±1.3. Blood alcohol level reached above 80 mg/dl in 10 subjects after EtOH with standard meal (mean concentration: 86.1±16.6) during the 60-minute period.

Six subjects experienced upper GIS side effects, namely four with nausea without vomiting and two with mild vomiting, with a maximum two episodes of emesis after EtOH ingestion. In four of these six subjects, blood alcohol level exceeded 80 mg/dl. In addition, in six of eight subjects without any GIS side effects, blood alcohol level reached above 80 mg/dl. There were no statistically significant differences in PR, postprandial MnDF and normogastria ratios of the two groups with and without GIS side effects (Table 1).

Table 1. Postprandial EGG parameters of subjects with and without gastrointestinal adverse effects (nausea and vomiting) (p>0.05 for all comparisons)

GIS adverse effects	MnDF (cpm)	Normogastria (%)	Power ratio
Present (n=6)	3.05	87.8	2.3
Absent (n=8)	3.08	86	2.4

EGG: Electrogastrography. GIS: Gastrointestinal system. MnDF: Mean dominant frequency

Mean postprandial EGG parameters (% of tachygastria, bradygastria, MnDF, DFIC) and PR of all subjects after control solution with standard meal and ethanol with standard meal are listed in Table 2. There was no statistically significant difference between groups in any of the postprandial EGG parameters. None of the subjects on the first day and three of the subjects on the second day had PR <1 (p>0.05).

Table 2. Mean values of postprandial EGG parameters and PR after control solution with standard test meal and ethanol with standard test meal (p>0.05 for all comparisons)

Postprandial EGG parameters	Control solution	EtOH
MnDF (cpm)	3.0±0.1	2.9±0.1
Normogastria (%)	86.8±7.8	85.2±12.3
Bradygastria (%)	6.5±5.5	7.2±8.3
Tachygastria (%)	6.5±6.0	7.5±7.2
DFIC (%)	22.5±10	19.5±11.8
PR	4.4±3.9	5.4±5

EGG: Electrogastrography. EtOH: Ethanol. MnDF: Mean dominant frequency. DFIC: Dominant frequency instability coefficient. PR: Power ratio

DISCUSSION

Acute and chronic consumption of alcohol can cause some GIS side effects like nausea, vomiting and epigastric pain. These symptoms might be related with alcohol-induced disturbances of the central nervous system or gastric motility.

Central and peripheral neural effects of ethanol on the brain-gut axis and the linkage between the central nervous system and autonomous innervation of the GIS might play a key role in the development of EtOH-related disorders of the GI tract. Some animal studies indicated the effect of EtOH on the central portion of the brain-gut axis (14,15). Peripheral impairment of the brain-gut axis by EtOH might also affect gastric motility. Izbeki et al. (16) found that acute EtOH administration in rats activated inhibitory capsaicin-sensitive afferent neurons of the nervus vagus in the stomach, leading to delayed gastric emptying. Another study in mice showed that chronic administration of EtOH damaged the intramural neurons of the stomach, which might impair gastric motility (17).

Moreover, several experimental studies demonstrated that EtOH induced depression of GMA and decreased the amplitude of antral contractions (6-10). Knight et al. (6) investigated the effect of 6-8% intravenous and oral EtOH on antral contractions in dogs using dynamic scintigraphic technique. Oral EtOH decreased the amplitude of antral contractions in a dose-dependent manner (blood alcohol level between 120-174 mg/dl). Sanders et al. (7) investigated the effect of 0.1-1% EtOH on gastric muscles in vitro and found that EtOH decreased the power and frequency of phasic contractions of the stomach and furthermore, hyperpolarized the resting membrane potential and decreased the amplitude and duration of slow waves. These animal studies implicate that the mechanism of alcoholic gastroparesis might be due to the direct effect of EtOH on the muscle cell membrane, namely myoelectrical activity.

Pfaffenbach et al. (18) evaluated GMA and gastric emptying in dyspeptic chronic alcoholics. There was significant increase in postprandial normogastria ratio and decrease in postprandial bradygastria ratio when compared with the control group. However, when EGG parameters of two groups of alcoholics with delayed or normal gastric emptying were compared, no significant difference was found.

The only study addressing the acute effect of EtOH intake on GMA in man was performed by Riezzo et al. (19). Alcohol (0.5 g/kg body weight) administration produced tachygastria with a significant reduction in normal slow wave percentage. However, blood alcohol level was not measured in that study.

We investigated the acute effect of EtOH intake (0.8 g/kg, 99.9% pure ethanol diluted to 40%) on GMA in healthy volunteers using EGG. Though six volunteers experienced upper GIS side effects and blood alcohol level above 80 mg/dl was achieved in 10 subjects, EtOH showed no effect on any of the postprandial EGG parameters or PR.

In our study, EtOH did not affect GMA. EtOH-induced upper GIS symptoms in our subjects also had no relation with gastric dysrhythmia. Gastric

motility is controlled by electrical slow wave activity. We could not demonstrate any change in GMA that might support alcohol-induced gastroparesis as reported in different studies. However, type of alcoholic beverage, route and dose of administration and achieved blood alcohol level might affect GMA and gastric motility differently. Moreover, the correlation of the different EGG parameters with gastric motility/emptying needs to be standardized. Further studies are necessary to show the effects of alcohol on GMA and gastric motility.

REFERENCES

1. Barboriak J, Meade RC. Effects of alcohol on gastric emptying in man. Am J Clin Nutr 1963; 23(9): 1151-3.
2. Brewster AC, Lankford HG. Ethanol and alimentary lipemia. Am J Clin Nutr 1966; 19 (4): 255-9.
3. Crean GP, Card WI, Beattie AD, et al. Ulcer-like dyspepsia. Scand J Gastroenterol Suppl. 1982; 79: 9-15.
4. Pihkanen TA. Neurological and physiological studies on distilled and brewed beverages. Ann Med Exp Biol Fenn 1957; 35 (Suppl): 9.
5. Mushambi MC, Bailey SM, Trotter TN. Effect of alcohol on gastric emptying in volunteers. Br J Anaesth 1993; 71: 674-6.
6. Knight LC, Maurer AH, Wikander R. Effect of ethyl alcohol on motor function in canine stomach. Am J Physiol 1992; 262 (2 pt 1): G 223-30.
7. Sanders KM, Bauer AJ. Ethyl alcohol interferes with excitation-contraction mechanisms of canine antral muscle. Am J Physiol 1982; 242(3): G222-30.
8. Reed JB, Bauer AJ, Sanders KM. Electrical basis for the effects of ethyl alcohol on canine corpus muscles. Eur J Pharmacol 1985; 119 (1-2): 31-7.
9. Berry RG, Sanders KM. Mechanical effects of ethyl alcohol on muscles of the proximal stomach. Proc West Pharmacol Soc 1984; 27: 115-7.
10. Sanders KM, Berry RG. Effects of ethyl alcohol on phasic and tonic contractions of the proximal stomach. J Pharmacol Exp Ther 1985; 235 (3): 858-63.
11. Chen JDZ, McCallum RW. Clinical applications of electrogastrography. Am J Gastroenterol 1993; 88: 1324-36.
12. Stern RM, Koch KL, Stewart WR. Electrogastrography: current issues in validation and methodology. Psychophysiology 1987; 24: 55-64.
13. Shimada Y, Watanabe M, Shibahara N, et al. Electrogastrographic power ratio in humans is not related to changes in antrum-skin distance but to antral motility. J Gastroenterol 1998; 33(3): 310-7.
14. Crankshaw DL, Briggs JE, Olszewski PK. Effects of intracerebroventricular ethanol on ingestive behavior and induction of c-Fos immunoreactivity in selected brain regions. Physiol Behav 2003; 79: 113-20.
15. Brzozowski T, Konturek PC, Konturek SJ. Central leptin and cholecystokinin in gastroprotection against ethanol-induced damage. Digestion 2000; 62: 126-42.
16. Izbeki F, Wittmann T, Jancso G, et al. Inhibition of gastric emptying and small intestinal transit by ethanol is mediated by capsaicin-sensitive afferent neurons. Naumyn Schmiedebergs Arch Pharmacol 2002; 365 (1): 17-21.
17. Jedrzejewska A, Staff-Zielinska E. Neuropathology of intramural autonomic innervation of mice stomach in chronic ethanol intoxication. Zentralbl Allg Pathol 1990; 136: 571-7.
18. Pfaffenbach B, Adamek RJ, Hagemann D, et al. Gastric emptying and antral myoelectrical activity in chronic alcoholics with dyspepsia. Hepatogastroenterology 1998; 45: 1165-71.
19. Riezzo G, Chiloiro M, Montanaro S. Protective effect of amtolmetin guacyl versus placebo diclofenac and misoprostol in healthy volunteers evaluated as gastric electrical activity in alcohol-induced stomach damage. Dig Dis Sci 2001; 46(8): 1797-804.