Gallbladder Motility in Patients with Irritable Bowel Disease

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Özet: *İRRİTABLE BARSAK HASTALIĞINDA SAFRA KESESİ MOTİLİTESİ*

İrritable barsak hastalığında otonom sinir sistemi ve kolesistokinin salınımındaki anormalliklerin rolü olduğu rapor edilmekle beraber, hastalıkda bozulan motilitenin gerçek nedeni bilinmemektedir. Öte yandan safra kesesi motilitesinin otonom sinir sistemi ve hormonal yolla kontrol edildiği bilinmektedir.

Bu çalışmada irritabl barsak hastalığında (n = 11) ve sağlıklı bireylerde (n=9) safra kesesi kontraksiyonları çalışılmıştır. Bazal safra kesesi volüm ölçümünü takiben irritabl barsak hastalığı olan bireylerle gönüllülere standart test yemeği verilmiştir. Ultrasonografik olarak 15 dakikalık aralarla 60 dakika boyunca ortalama safra kesesi volumleri izlenmiştir.

Açlık safra kesesi volümü irritabl barsak sendromu olan bireylerde kontrol grubuna göre farklılık göstermemiştir (22.97 ± 17.91 mL ve 20.25 ± 6.93 mL). Rezidüel ortalama safra kesesi volümü 15. dakika (13.41 ± 9.96 mL ve 15.45 ± 12.22 mL). 30. dakika (8.58 ± 8.90 mL ve 13.66 ± 10.03 mL), 45. dakika (9.78 ± 8.46 mL ve 11.41 ± 6.00 mL) ve 60. dakika (12.23 ± 9.01 mL ve 12.06 ± 7.99 mL) sonunda irritabl barsak hastalığı olan bireylerde, kontrol grubuna gör istatistiksel olarak anlamsız bulunmuştur. Ortalama safra kesesi volümleri, her iki grupta, yemeği takiben bir saat boyunca bazal değerlere göre anlamlı olarak küçüktür ($p < 0.05$).

Bu gözlemlere dayanarak, İrritabl barsak hastalığında safra kesesi kontrakatif fonksiyonlarında değişme olmadığı sonucuna varılmıştır.

Anahtar kelimeler : İrritable barsak hastalığı, Safra kesesi mütilitesi.

Summary: *The exact cause of altered motility in irritable bowel disease (IBD) is not clear, altough abnormalities of the autonomic nervous function and CK release have been reported. On the other hand, gallbladder function is regulated by autonomic nerves as well as hormonal control. Because of this reason, we studied gallbladder contractions in IBS patients (n=11) and healthy volunteers (n=9). After basal measurement, the volunteers and patients with IBD received standard test meal. Mean gallbladder volumes were scanned by using ultrasonography in 15 min intervals for 60 min in both groups.*

Fasting mean gallbladder volume shows no difference in IBD patients than in control ($p > 0.05$) (22.97 ± 17.91 mL vs. 20.25 ± 6.93 mL). Following a test meal the residual mean gallbladder volume at the end of 15th minute (13.41 ± 9.96 mL vs 15.45 ± 12.22 mL), at the end of 30th minute (8.58 ± 8.90 mL vs 13.66 ± 10.03 mL), at the end of 45th minute (9.78 ± 8.46 mL vs 11.41 ± 6.00 mL), and at the end of 60.th minute (12.23 ± 9.01 mL vs 12.06 ± 7.99 mL) shows no significant differences in IBD patients than in control subjects ($p > 0.05$).

Mean gallbladder volumes of both groups after meal intake were significantly lower during one hour period as compared to baseline value ($p < 0.05$).

Based on these observations. We conclude that patients with IBD have any abnormalities of gallbladder function.

Key words: Irritable bowel disease, Gallbladder motility.

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Peptides, released from the gastrointestinal mucosa into the blood after eating, act as hormones and affect gastric, small intestine, and colonic smooth muscle contractions. As in the enteric nervous system, a counterbalance between stimulating peptide such as motilin and cholecystokinin (CCK) and inhibiting peptides 51).

CCK is the major hormone that modulates postprandial gallbladder contraction (2) and CCK acts as a neurotransmitter to stimulate postganglionic inhibitory neurons and also acts directly on the smooth muscle of gallbladder and sphincter of Oddi and indirectly through cholinergic nerves on the smooth muscle of the intestine (3). Motilin is an other regulatory peptide of the gut which causes contractions of duodenal, ileal, colonic and gallbladder smooth muscle in vitro (3). Motilin has been shown to be about as potent as CCK in stimulation of gallbladder contraction, and its effect is seen with serum levels within the physiologic range, motilin may participate in the control of gallbladder emptying (3).

Cramping abdominal pain of colonic origin and an altered bowel habit are the hallmark of the irritable bowel disease (IBD) (1). Colonic motor activity is abnormally increased in these patients with colonic pain, eg, after meals, after administration of CCK or cholinergic drugs, or after emotional stress. Altered small bowel motility may also occur and appears to be corelated with symptoms (4). The exact cause of altered motility in IBD is not clear, although abnormalities of the autonomic nervous function (5) and CCK release (6) have been reported.

Information on gallbladder motility in IBD is very limited and variable results have been reported. Pathogenesis of disease may be related to the hyperactivity of CCK or colonic hypersensitivity to this peptide. Because of this reason, we postulated that the more dramatic increase will be observed in tonic and/or phasic activity of gallbladder which promotes emptying during the postprandial period in patients with IBD. For this purpose, we investigated meal-induced gallbladder emptying in patients with IBD and compared to healthy volunteers.

MATERIAL AND METHODS

Nine healthy volunteers (age 36 ± 16) and 11 patients with IBD (age 32 ± 12) agreed to participate in the study after the protocol and the test procedures had been explained them. All patients with IBD had abdominal pain as a prominent symptom and no one had history of biliary colic, jaundice, or gastrointestinal surgery. All patients had normal findings on clinical examination, rigid sigmoidoscopy, and laboratory evaluation, which included stool examination for ova and parasites, and occult blood, complete hemogram, ESR and liver function tests. Barium series of gastrointestinal tractus were also done to rule out any organic pathology. Barium series of colon were spastic in all cases. Al medications were stopped at least 48 h prior to study.

Gallbladder volumes were measured by using ultrasonography. Scans were performed at 8 am after 12 hour fasting period. After basal measurement the volunteers and patients with IBD received ensure (250 cal/250 mL , protein 16.7%, fat 30%, carbonhydrate 53.3%). Gallbladder volumes were scanned 15 min intervals for 60 min for each subjects after receiving Ensure. Gallbladder volume and emptying were measured using by ultrasonography (7) which is a 3,5 or 5 MHz transducer real time ultrasound scans (General Eletrics RTX 200). Subjects were scanned supine in the right anterior oblique position by radiologist trained in ultrasonography. The gallbladder was visualised in the longitudinal and transverse planes, and measurement of maximal lenght, width, and height were taken in duplicate. The volume of the gallbladder was subsequently calculated using the ellipsoid method (volume = 0.52 x lenght x width x height) (8).

The results are expressed as mean ± SEM unless otherwise stated. For statistical analysis, the Wilcoxon signed rank or Mann Whitney U test was used where appropriate and difference were considered significant at $p < 0.05$.

RESULTS

Fasting mean gallbladder volume shows no difference in IBD patients than in control (22.97 ± 17.91 mL vs 20.25 ± 6.93 mL). Gallbladder contraction were evaluated after standart test meal initiated prompt contraction in both groups. The residual mean gallbladder volume at the end of 15th minute (13.41 ± 9.96 mL vs 15.45 ± 12.22

mL) at the end of 30th minute (8.58 ± 8.90 mL vs 13.66 ± 10.03 mL), at the end of 45th minute (9.78 ± 8.46 mL vs 11.41 ± 6.00mL), and at the end of 60. th minute (12.23 ± 9.01 mL vs 12.06 ± 7.99 mL) shows no significant differences in IBD patients than in control subjects (Table I).

Mean gallbladder volumes of both groups after meal intake were significantly lower during one hour period as compared to baseline value ($p < 0.05 - 0.005$) (Figure -1).

DISCUSSION

The IBS is a motor disorder consisting of altered bowel habits, abdominal pain, and the absence of detectable organic pathology (4). The failure of laboratory studies to show any morphologic, histologic, microbiologic, or biochemical abnormalities in patients with IBD supports the concept that IBD is primarily a disorder of gastrointestinal motility (4). Altered bowel habits seen in IBD can be explained on the basis of altered motility, which, in turn, may be a response to emotional states, to meal, to neurohumoral agents and to gastrointestinal hormones such as CCK, glucagon and vasointestinal peptide (VIP) (4).

The exact cause of altered motility in IBD patients is not clear, although abnormalities of CCK release have been reported in patients with IBD (5,6). Exogenous CCK infusion produces an increase in colonic motility that is associated with abdominal pain; and suggesting that CCK, a gastrointestinal hormone released by meals, may account for some of the postprandial symptoms in patients with IBD have normal gallbladder function as compared with healthy volunteers. Fasting and residuel mean gallbladder volume for one hour period were all normal in IBD. The results of recent studies which reported that fasting and residuel gallbladder volume in IBD patients had any differences as compared with healthy subjects are similar to our findings (9,10), but others are not (8,11). These variable results may due to numerous factors; i) Not all patients with IBD have demostrable colonic myoelectric abnormality; only 40% have slow basic electric rhythm in colonic smooth muscle (12, 13), and this abnormality may be paroxysmal (14). ii) Gallbladder motility also varies and may show hypo or hypercontractility, depending upon the severity and type of symptoms in IBD (15). iii) Patients with IBD show a delayed postprandial release of CCK into the plasma (6) and the delayed release of CCK prevent maximal contraction of the gallbladder in IBD patients for one hour period in this study. iv) Motilin which has been shown to be about as potent as CCK in stimulation of gallbladder contraction (3), to be dereased in constipation predominant patients (16).

Further studies are also needed to confirm whether gallbladder contraction varies among different groups of IBD patients with different symptom syndromes.

Table I :Values of gallbladder volume and statistical comparison of the patients with irritabale bowel disease and healthy subjects (control).

Time	IBD	Control	P
Baseline	22.97 ± 17.91 mL	20.25 ± 6.93 mL	NS*
15th minute	13.41 ± 9.96 mL	15.45 ± 12.22 mL	NS
30th minute	8.58 ± 8.90 mL	13.66 ± 10.03 mL	NS
45th minute	9.78 ± 8.46 mL	11.41 ± 6.00 mL	NS
60th minute	12.23 ± 9.01 mL	12.06 ± 7.99 mL	NS

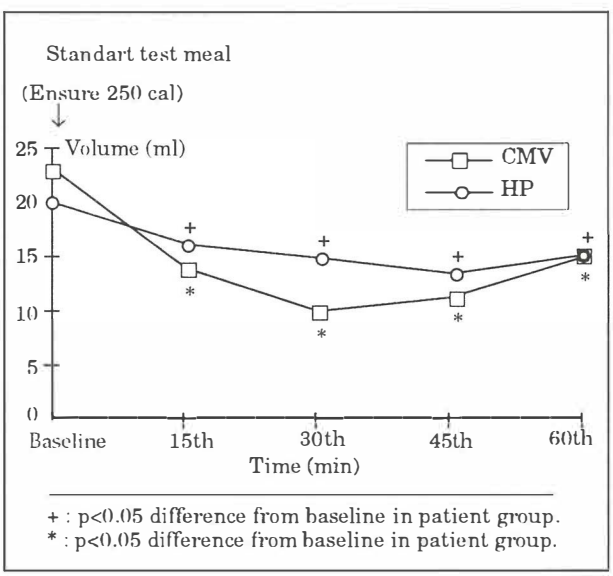

Figure 1 : Values of gallbladder volumes in baseline and after administration of standart test meal in patients with irritable bowel disease and healthy subjects.

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