

Irritable Bowel Syndrome and Chronic Gastritis, Hemorrhoid, Urolithiasis

Spastik Kolon ve Kronik Gastrit, Hemoroid, İdrar Taşı

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Abstract

Objective: Approximately 10-20% of the general population has irritable bowel syndrome (IBS), and IBS patients usually suffer from chronic gastritis (CG), hemorrhoids (H), and urolithiasis (U).

Material and Methods: We randomly chose consecutive patients with upper abdominal discomfort. All possible causes of IBS including celiac sprue, giardiasis, lactose intolerance, and cholelithiasis were investigated. U was diagnosed either by medical history or as a result of laboratory findings.

Results: IBS patients (51) and patients without IBS (70) were studied. CG was diagnosed in 78.4% (40) of IBS cases, whereas this ratio was 50.0% (35) in cases without IBS ($p<0.001$). Similarly, H was detected in 33.3% (17) of IBS cases, but it was only detected in 15.7% (11) of cases without IBS ($p<0.05$). Additionally, U was detected in 17.6% (9) of IBS cases and in 5.7% (4) of cases without IBS ($p<0.05$).

Conclusion: Relationships between IBS and CG, H, and U are significant. IBS is a cascade of many physiologic events that is initiated by infection, inflammation, and psychological disturbances like many stresses, and this eventually terminates with gut dysfunction. Gastric acid is probably not involved in the etiology of IBS, but psychological factors also seem to be important in CG. The significant association between CG and IBS also support this hypothesis. Therefore, we believe CG is one of terminating points of the physiologic cascade of events in IBS. Bearing these associations in mind will be helpful during prevention, treatment, and follow up of these disorders, especially in internal medicine, urology, and general surgery polyclinics and primary health centers.

Keywords: Irritable bowel syndrome, Chronic gastritis, Hemorrhoids, Urolithiasis

Özet

Amaç: Toplum genelinde spastik kolon sıklığı %10-20'dir ve bu hastalarda kronik gastrit, hemoroid ve idrar taşının da sık görüldüğü tahmin edilmektedir.

Gereç ve Yöntem: Üst karın şikayetleriyle ardı sıra başvuran hastalar çalışmaya alındı. Çöliyak hastalığı, giardiaz, laktoz intoleransı ve kolelitiazı da içerecek şekilde tüm muhtemel sebepler araştırıldı. İdrar taşı tanısı hastanın tıbbi hikayesi veya mevcut laboratuvar bulgularıyla konuldu.

Bulgular: Elli bir spastik kolon hastası ve 70 spastik kolonu olmayan hasta çalışıldı. Kronik gastrit, spastik kolona sahip hastaların %78.4'ünde tespit edilirken bu oran spastik kolonu olmayan grupta %50.0 olarak tespit edildi ($p<0.001$). Benzer şekilde, hemoroid oranı spastik kolon grubunda %33.3 iken, spastik kolonu olmayan grupta %15.7 olarak tespit edildi ($p<0.05$). Ayrıca, idrar taşı sıklığı spastik kolon grubunda %17.6 iken bu oran spastik kolonu olmayan grupta %5.7 olarak tespit edildi ($p<0.05$).

Sonuç: Sonuç olarak, spastik kolon anlamlı şekilde kronik gastrit, hemoroid ve idrar taşı ile ilişkili ve enfeksiyon, enflamasyon ve psikolojik problemler gibi birçok stres faktörünün tetiklemesiyle başlayıp genitoüriner sistem disfonksiyonuyla sonuçlanan bir olaylar şelalesidir. Kronik gastrit için de psikolojik faktörlerin önemli olduğu ve mide asiditesinin çok önemli bir sebep olmadığı düşünülmektedir ve spastik kolon ile kronik gastrit arasında tespit edilen bu anlamlı ilişki de bunu desteklemektedir. Kronik gastritin, bir fizyolojik olaylar şelalesi olan spastik kolonun son noktalarından birisi olduğunu düşünmekteyiz. Bu anlamlı birlikteliklerin akılda tutulması, bu hastalıkların önlenmesi, tedavisi ve takibinde, özellikle İç Hastalıkları, Üroloji ve Genel Cerrahi poliklinikleri ve birinci basamak sağlık kuruluşlarında klinisyenler açısından faydalı olabilir.

Anahtar Kelimeler: Spastik kolon, Kronik gastrit, Hemoroid, İdrar taşı

Introduction

When specifically asked, about one third of people report upper abdominal discomfort; most visits to primary health centers and internal medicine polyclinics are due to this complaint [1]. Irritable bowel syndrome (IBS), chronic gastritis (CG), gastroesophageal reflux disease (GERD) without esophagitis, duodenal and gastric ulcers, erosive gastritis and duodenitis, lactose intolerance, cholelithiasis, malignancy, giardiasis, celiac disease, and chronic pancreatitis are among the possible causes of this complaint. However, IBS and CG are probably the most commonly diagnosed illnesses among of all.

Moreover, excessive straining, feeling of incomplete evacuation, repeated toilet visits due to urgent evacuation or early filling sensation, flatulence, periods of diarrhea and/or constipation, frequency, urgency, reduced feeling of well being, and disturbed social life caused by both gastrointestinal and urinary tract symptoms are often reported by IBS patients. In addition to these complaints, it seems that IBS patients usually suffer from CG, hemorrhoids (H), and urolithiasis (U). In this study, we sought to determine whether there are significant relationships between IBS and CG, H, and U.

Materials and Methods

We randomly took consecutive patients with upper abdominal discomfort that had visited the internal medicine polyclinic of the Dumlupınar University between August and December 2005. Medical histories including alcohol consumption and history of U were obtained, and a questionnaire for IBS was performed. IBS was diagnosed according to Rome II criteria in the absence of red flag symptoms, which are not typical for IBS, such as pain or diarrhea that often awakens/interferes with sleep, weight loss, fever, or an abnormal physical examination. Patients with regular and moderate alcohol intake (20 g/day or higher) were detected. An upper gastrointestinal endoscopy, including sample biopsies from the distal esophageal, gastric and duodenal regions according to macroscopic appearances and symptoms of cases, was performed. Additionally, duodenal contents were aspirated to search for trophozoites of *Giardia lamblia*. The following were performed for each patient: routine hematologic and biochemical tests, abdominal ultrasonography, fresh fecal sample examination, rectosigmoidoscopy for cases that had ever been symptom-

atic for H, urinalysis, and an abdominal X-ray in supine position. Additionally, an intravenous pyelography was performed in cases suspected of presenting with U as a result of the urinalysis and abdominal X-ray. U was diagnosed either by medical history or as a result of current laboratory findings. A test for lactose intolerance was also performed. Lactose (50 g, orally) causes diarrhea with abdominal bloating and discomfort within 30 min and a rise in blood glucose of < 20 mg/dL in cases with lactose intolerance. CG was diagnosed histologically. Infiltration of monocytes into the gastric mucosa is hallmark of CG. Additionally, microscopic examination shows stereotypical changes in the epithelium such as degeneration, focal intestinal metaplasia, dysplasia, and glandular atrophy [2]. All histologic samples were evaluated by the same pathologist who was blind about the subclassifications of the patients. Because of the highly variable clinical severity of celiac disease and high sensitivity and specificity of the endomysial antibody (EMA), EMA was used as a screening test for celiac disease; jejunal biopsy was planned for only EMA positive cases to see absence of villi and elongated crypts. A detailed history for GERD was taken, and a distal esophageal biopsy was taken from all symptomatics that were macroscopically abnormal to rule out esophagitis, Barrett's metaplasia, and/or any malignant transformation. Thinning of the squamous mucosal layer and basilar cell hyperplasia was considered esophagitis. Changes in the columnar mucosa are features of Barrett's metaplasia. Results were compared between IBS-positive and -negative patients. Comparison of proportions was used as the method of statistical analysis.

Results

Patients with IBS (51) and patients without IBS (70) were studied. The mean age of cases with and without IBS were 37 and 35 yrs, respectively, and the female/male ratio was 1.21 and 0.79, respectively. Thus, IBS was observed as a more common disorder among females than males (Table 1). CG was detected in 78.4% (40) of cases with IBS, whereas this ratio was 50.0% (35) among the cases without IBS ($p < 0.001$). Similarly, although the rate of H was 33.3% (17) in IBS cases, it was 15.7% (11) in cases without IBS ($p < 0.05$). Additionally, U was detected in 17.6% (9) of cases with IBS and in 5.7% (4) of cases without IBS ($p < 0.05$) (Table 2). Among the 13 cases with U, eight cases had been operated on before, but one of them still had U. Therefore, a total of six current U cases were detected. In total, eight cases of gastric and duodenal ulcers were detected, all of which were histologically benign. In five of the eight cases with gastric or duodenal ulcers, nonsteroidal anti-inflammatory drugs (NSAIDs) were detected as the underlying etiology. In total, 17 cases of giardiasis were diagnosed via fresh fecal samples and examination of duodenal fluid. Duodenal contents alone were diagnostic in three cases with giardiasis. Additionally, cysts, but not trophozoites, could be detected in the fresh fecal samples. Although there were social drinkers among the study cases, there was no patient with regular or moderate alcohol intake. Additionally, we did not detect any IBS case with EMA positivity. We therefore did not need to take any jejunal biopsies. In addition to these

Table 1. Comparison of upper abdominal discomfort cases with and without irritable bowel syndrome

| | IBS* positive | IBS negative |
|--|---------------------------|---------------------------|
| Number and gender distribution | 51 (28 females) | 70 (31 females) |
| Female ratio | 54.9% | 44.2% |
| Mean age, standard deviation, and range (year) | 37.68 \pm 13.15 (19-74) | 35.15 \pm 13.36 (15-78) |

*Irritable bowel syndrome

Table 2. Comparison of upper abdominal discomfort cases with and without irritable bowel syndrome according to chronic gastritis, hemorrhoids, and urolithiasis

| | Cases with CG† | Cases with H‡ | Cases with U§ |
|--|--------------------|---------------|---------------|
| Patients with IBS* (51 in number) | 40 (78.4%) | 17 (33.3%) | 9 (17.6%) |
| Patients without IBS (70 in number) | 35 (50.0%) | 11 (15.7%) | 4 (5.7%) |
| <i>p</i> -value | <0.001 | <0.05 | <0.05 |
| *Irritable bowel syndrome | †Chronic gastritis | ‡Hemorrhoids | §Urolithiasis |

pathologies, we detected GERD without esophagitis, esophagitis, lactose intolerance, and cholelithiasis, but not malignancy, erosive gastritis or duodenitis, Barrett's metaplasia, or chronic pancreatitis, which is probably due to the small number of cases in our study (Table 3).

Discussion

Approximately 10-20% of general population has IBS [3]. Additionally, as was also shown in this study, IBS is more common among female than males for unexplained reasons. Psychological factors seem to precede the onset or exacerbation of gut symptoms, and many potentially psychiatric disorders such as anxiety, depression, and sleep disorders frequently coexist with IBS [4]. For example, thresholds for sensations of initial filling, evacuation, urgent evacuation, and utmost tolerance, recorded via a rectal balloon, significantly decreased by focusing the examiners' attention on gastrointestinal stimuli by reading pictures of malignant gastrointestinal disorders in IBS cases; however, no remarkable change of these thresholds was observed in the nonpatient group [5]. Thus, although IBS is described as a physical - not psychological - disorder according to Rome II guidelines, psychological factors may be crucial for initiation of the physical disorder.

Although the underlying causes of pathophysiologic changes remain unclear, low-grade mucosal inflammation and abnormal intestinal motility are accepted mechanisms that alter gut functions and generate symptoms [6]. According to the Rome II criteria, IBS is not a disease; instead it is a functional disorder, and it is actually characterized as a brain-gut dysfunction. However, based on our experience, we think IBS is a more complex condition than this. Parallel to the high incidence of CG found in our study, Chadwick and colleagues studied the role of inflammation in 77 IBS cases. Colonic biopsies were taken for conventional histology and immunohistology. Normal histology was recorded for 38 of the 77 IBS cases, 31 demonstrated microscopic inflammation, and 8 fulfilled the criteria for lymphocytic colitis. However, in the group with "normal" histology, immunohistology revealed increased intraepithelial lymphocytes as well as increased CD3+ and CD25+ cells in the lamina propria, which is evidence of immune activation. These features were even more evident in the microscopic inflammation group, who additionally revealed increased neutrophil, mast cell, and natural killer cells. All of these immunopathological abnormalities were most evident in the lymphocytic colitis group, who also demonstrated HLA-DR staining in crypts and increased CD8+ cells in the lamina propria [7]. A direct link between immune activation and symptoms was provided by the work of Barbara et al. [8], who demonstrated not only an increased prevalence of mast cell degranulation in colon, but also a direct correlation between the proximity of mast cells to neuronal elements and pain severity in IBS. In addition to these findings, there is some evidence for extension of the inflammatory process beyond the mucosa. Tornblom and colleagues addressed this issue in ten patients with severe IBS by examining full-thickness jejunal biopsies obtained via laparoscopy [9]. They detected a low-grade infiltration of lymphocytes in the myenteric plexus in nine cases, four of whom had an associated increase in intraepithelial lymphocytes and six demonstrated evidence of neuronal degeneration. Nine patients had hypertrophy of the longitudinal muscles, and seven had abnormalities in the number and size of interstitial cells of Cajal. The finding of intraepithelial lymphocytosis was consistent with the reports of Chadwick and colleagues in the colon and of Wahnschaffe and colleagues in the duodenum [10]. Therefore, IBS is a cascade of physiologic events that is initiated by infection, inflammation, psychological disturbances like many stresses and terminates with gut dysfunction.

On the other hand, gastric acid is probably not involved in the etiology of IBS; however, psychological factors seem to also be crucial for the development of CG. Our results indicate a statistically significant relationship between CG and IBS. The well-known importance of psychological factors as triggering events of IBS also supports this idea. Clearly, diet is implicated as regards to predisposition to constipation, colorectal cancers, and diverticular disease; however, a meaningful dietary role in CG is doubtful. Some dietary habits may be the triggering factor for CG, but this relationship is not always seen even in the same patients. The most important etiologic association of CG is chronic infection by bacillus *Helicobacter pylori* (*H. pylori*). *H. pylori* is linked to CG, peptic ulcers, gastric carcinoma, and mucosa-associated

Table 3. Comparison of upper abdominal discomfort cases with and without IBS according to other underlying etiologies

| | Cases with CG† | Cases with H‡ | Cases with U§ |
|-------------------------------------|----------------|---------------|---------------|
| GERD† without esophagitis | 5 (9.8%) | 4 (5.7%) | > 0.05 |
| Esophagitis | 1 (1.9%) | 2 (2.8%) | > 0.05 |
| Gastric ulcer | 1 (1.9%) | 1 (1.4%) | > 0.05 |
| Duodenal ulcer | 2 (3.9%) | 4 (5.7%) | > 0.05 |
| Lactose intolerance | 41 (80.3%) | 52 (74.2%) | > 0.05 |
| Cholelithiasis | 7 (13.7%) | 8 (11.4%) | > 0.05 |
| Malignancy | 0 (0%) | 0 (0%) | > 0.05 |
| Giardiasis | 7 (13.7%) | 10 (14.2%) | > 0.05 |
| Celiac disease | 0 (0%) | 0 (0%) | > 0.05 |
| Erosive gastritis and/or duodenitis | 0 (0%) | 0 (0%) | > 0.05 |
| Barrett's metaplasia | 0 (0%) | 0 (0%) | > 0.05 |
| Chronic pancreatitis | 0 (0%) | 0 (0%) | > 0.05 |

*Irritable bowel syndrome †Gastroesophageal reflux disease

lymphoid tissue (MALT)-lymphoma [11], and it is recognized as a class I gastric carcinogen [12]. Although *H. pylori* infects over 50% of the population worldwide, only a small subset of infected individuals experience *H. pylori*-associated disorders. The debate has been further intensified as some studies have suggested the possibility that *H. pylori* infection may be beneficial in some humans. This hypothesis is based on increased incidence of GERD, Barrett's esophagus, and adenocarcinoma of esophagus following *H. pylori* eradication in some countries. Recent studies have shown that *H. pylori* infection protects against GERD and esophageal carcinoma. A current hypothesis about this issue is that there is a nearly symbiotic and balanced relationship between the bacterium and the human body. The colonization by bacteria may either be beneficial or of low biological cost to the host. Thus, the role of *H. pylori* in CG is obvious, but the answer to the question 'why every patient with CG does not need to visit a doctor?' is unknown. We believe that CG is one of the terminating points of the many physiologic events leading to IBS.

A meaningful dietary role in IBS is also doubtful. Many patients relate the onset of symptoms to intake of food and often incriminate specific food items, which may actually be a result of the significant association of IBS with CG. Although there is limited evidence for classical food allergy in IBS, Whorwell and colleagues suggested that testing for food intolerance utilizing IgG antibodies can lead to a successful dietary modification regime [13]. On the other hand, debate continues regarding the potential overlap between IBS and celiac sprue [14]. It is evident that the majority of celiacs present later in life, usually with vague and non-specific gastrointestinal symptoms. Celiac disease must, therefore, be considered in all new IBS patients, especially in areas of high prevalence of celiac disease and regardless of the nature of presenting symptoms [15]. Although we searched for

lactose intolerance in all study cases, there was not a significant difference between cases with and without IBS, and we were not able to diagnose any cases of celiac among the 121 study cases.

On the other hand, the relative risk of developing IBS was detected as 2.48 times higher in patients with urinary stone disease than in those without it. Urinary stone disease should be considered as an etiological factor during the management of IBS [16]. However, we actually believe that IBS is a cause of U because of its prolonged nature and frequently reported urinary and gynecological symptoms in IBS cases, but the basis for these associations is less clear. Additionally, it has been previously shown that besides bleeding, pain, soiling, and prolapse, many patients with grade 3-4 H have concomitant functional bowel symptoms, which are possibly associated with IBS [17]. Excessive straining, feeling of incomplete evacuation, repeated toilet visits, and periods of constipation are also found among the possible causes of H in IBS cases.

In conclusion, the relationships between IBS and CG, H, and U are significant. IBS is a cascade of many physiologic events that is initiated with infection, inflammation, and psychological disturbances like many stresses and eventually terminates with gut dysfunction. Gastric acid is probably not involved in the etiology of CG, but psychological factors also seem to be crucial for the development of CG. The significant association between CG and IBS also supports this idea. Therefore, we believe that CG is one of the terminating points of the physiologic cascade of events leading to IBS. Bearing these associations in mind will be helpful during prevention, treatment, and follow up of IBS patients, especially in internal medicine, urology, and general surgery polyclinics and in primary health centers.

Conflict interest statement The authors declare that they have no conflict of interest to the publication of this article.

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