

Histopathological Examination of Dyspeptic Patients With and Without Hiatal Hernia

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ABSTRACT

Background: Hiatal hernia with dyspeptic complaints is a severe disease which disturbs the quality of life. Pathological findings in endoscopy are an important factor in the treatment of these patients. **Objective:** To investigate the histopathological differences related to hiatal hernia in dyspeptic patients. **Material and Methods:** A retrospective study was conducted of 116 patients with dyspeptic complaints. The patients were divided into two groups according to the presence of hiatal hernia. Biochemical tests and endoscopic biopsy results of these groups were compared. **Results:** The mean age of our study population was 52.4 years where, 50% were male and 50% were female. Hiatal hernia was found in 44.8% of the patients and 22.4% of the patients had intestinal metaplasia. The rate of intestinal metaplasia was found to be statistically lower in the hiatal hernia group ($p=0.011$). **Conclusion:** Intestinal metaplasia is less frequently found in patients with hiatal hernias in our study; it may be attributed to the fact that these patients had earlier dyspeptic complaints and therefore, earlier medical treatment and earlier initiation of a protective gastric diet.

Key words: Hiatal hernia, intestinal metaplasia, dyspeptic

Hiatal hernia (HH) has a high prevalence in economically developed Western countries and low prevalence in economically underdeveloped countries such as Africa. HH is more common in older people and is more common in females than in males [1,2]. HH is a protrusion of the stomach into the mediastinum through a defect in the esophageal hiatus of the diaphragm. HH is not a part of any disease but it is a common anatomic disorder [3]. Although, the reason of the HH is often unknown; extraction of stomach due to esophageal contraction and high intra-abdominal pressure or due to deficit of frenoesophageal junction can be determined as a reason [4]. There are generally two types of hiatal hernia; sliding and paraesophageal. HH is commonly asymptomatic and is often diagnosed incidentally when evaluating other dyspeptic problems [4]. The symptoms associated with a hiatal hernia generally are related to

esophageal compression and distortion leading to some degree of esophageal obstruction. Dysphagia and regurgitation are common symptoms and those ruin the quality of life [5]. Symptoms related to regurgitation, such as recurring aspiration pneumonia, recurring sinus infections, recurring and persistent cough, asthma, and changes in voice quality and strength may also occur [5,6]. Upper gastrointestinal (GI) endoscopy should be an early step within these symptoms. Upper GI contrast radiography is also very helpful in establishing the diagnosis and extent of a hiatal hernia [7].

Gastric intestinal metaplasia is a precancerous lesion associated with an increased risk of dysplasia and cancer [8]. Intestinal metaplasia (IM) occurs with the replacement of surface, foveolar, and/or glandular epithelium in the oxyntic or antral mucosa by intestinal epithelium [8].

Helicobacter pylori infection and related genomics have been associated with the development of gastric intestinal metaplasia including host genetic factors, environmental factors, rheumatologic disorders, diet and intestinal microbiology [9]. It is known that intestinal type of gastric adenocarcinoma is highly co-related with intestinal metaplasia [10]. The aim of this study was to investigate the histopathological differences related with hiatal hernia in dyspeptic patients.

MATERIALS & METHODS

Current retrospective analysis was performed on hospital data of adult patients who were treated at a University Hospital of Turkey between March 2018 and June 2018. Patients with dyspeptic complaints including epigastric pain, early satiety, postprandial distress, were eligible for enrollment in the study. Patients who had taken H. pylori treatment six month before the study were excluded. Results of blood samples including hematologic and biochemical tests, endoscopic biopsy specimens, and extensive radiologic procedures, were obtained from hospital data. Patients routinely underwent upper GI endoscopy and to identify both microorganism and intestinal metaplasia, gastric biopsy materials, from study patients, were examined. Endoscopic procedures were performed by a gastroenterology doctor at the study hospital. Gastric biopsy samples were tested for urease activity by using a commercial hp fast test (GI Supply® • Camp Hill, PA, USA) which has a urea containing medium and a pH reagent. Intestinal metaplasia and atrophic gastritis were evaluated by an expertise pathologist.

Obtained results were compared with dyspeptic control population wherein, the controls were also required to be medical treatment free for at least 6 months from the time of study entry. Antrum biopsies of dyspeptic subjects without hiatal hernia were examined for both H.pylori and premalignant lesions of the gastric mucosa by the same method. Prognostic values of H.pylori and other clinicopathologic factors were also evaluated. According to the endoscopic and radiologic results patients were divided into two groups i.e. hiatal hernia group and without hiatal hernia group. These two groups were compared in terms of blood tests results and biopsy results.

Statistical analysis was performed and two-sided P values of 0.05 or less were considered to indicate statistical significance. All statistical analyses were performed with the use of SAS software, version 9.3 (SAS Institute).

RESULTS

A total number of 116 patients with dyspeptic complaints who underwent endoscopy were included in the study. The mean age of our study population was 52.4±12.2 years and 50% were male and 50% were female. Hiatal hernia was found in 52 (44.8%) of the patients and 22.4% of the patients had intestinal metaplasia. Rest of the patients (64 patients; 55.2%) had no hiatal hernia. The patients in our study were classified into 2 groups: patients with hiatal hernia (n=52) and patients without hiatal hernia (n=64) according to endoscopic results. There was no statistically significant difference in mean age and biochemical tests results except ALP levels between two groups. There was also no statistically difference in gender and H. Pylori infection between two groups (Table 1).

Table 1 - Demographic and laboratory characteristics of Hiatal Hernia and without Hiatal Hernia Group

Variable	Hernia Group (n=52)	Without Hernia Group (n=64)	P value
Age (years)	50.2±15.4	54.3±12.3	0.1
Gender (n%)			
Male	31(59.6%)	27(42.2%)	0.06
Female	21(40.4%)	37(57.8%)	
Hemoglobin[g/dL]	13.1±2.0	12.2±2.6	0.05
Hematocrit [%]	39.8±5.7	37.9±7.7	0.15
WBCx10 ³ mL	7.6±2.1	7.4±2.7	0.66
Platelets x10 ³ mL	244.3±70.4	259.4±66.0	0.24
ALT[U/L]	26.5±21.7	21.8±16.9	0.21
AST[U/L]	24.1±10.8	21.1±8.7	0.12
ALP	85.5±30.2	126.9±79.6	0.018
GGT	25.0±22.5	27.5±28.4	0.69
Total Bilirubin	0.6±0.4	0.7±1.3	0.68
Direct Bilirubin	0.2±0.1	0.2±0.2	0.16
H.Pylori (n,%)			
Positive	26(50%)	35(54.6%)	0.61
Negative	26(50%)	29(45.4%)	

Data expressed as mean±SD, categorical variables are reported as number (percentage). WBC- white blood cell ; ALT- alanine aminotransferase; AST- aspartate aminotransferase; ALP- Alkaline Phosphatase; GGT- gamma glutamyl transferase; H.Pylori- Helicobacter Pylori

According to pathology results intestinal metaplasia was detected in 26 patients of the total study population (22.4 %). Intestinal metaplasia was found in 11.5% of the patients in hiatal hernia group and also was found in 31.3 % of patients without hiatal hernia group. The rate of intestinal metaplasia was found to be statistically lower in the hiatal hernia group (P=0.011) (Table 2).

Table 2 - The Presence of Intestinal Metaplasia and Helicobacter Pylori in Patients with or Without Hiatal Hernia

Tested Parameters	Hiatal Hernia		P Value
	Positive (n=52)	Negative (n=64)	
Intestinal Metaplasia (n,%)			
Positive	6(11.5%)	20(31.2%)	0.011
Negative	46(88.5%)	44(68.8%)	
H.Pylori (n, %)			
Positive	26(50%)	35(54.6%)	0.61
Negative	26(50%)	29(45.4%)	

DISCUSSION

Hiatal hernia occurs when the normal anatomic relationship of the diaphragmatic hiatus is impaired by the gastroesophageal junction. It is a very common pathological condition that can cause gastroesophageal reflux. These patients may have stomach ulcers, hemorrhages and obstructions. Nevertheless, HH occurs in 54% to 94% of patients with reflux esophagitis, a rate strikingly higher than that in the healthy population [11].

Although the etiology of a HH remains unclear, it is well known that especially in gastroesophageal reflux patients with HH causes Barrett's Esophagus which is a precursor to esophageal adenocarcinoma [12]. It was determined what kind of lesions lead to gastroesophageal reflux and HH in proximal region but also the pathology of the hiatal hernia in the distal region, i.e. in the antrum, has not been investigated much.

Intestinal Metaplasia (IM) is a common pathologic finding in patients with gastric ulcers and having chronic gastritis lead to significant loss of parietal cell mass. This oxyntic atrophy may be associated with IM. In previous studies, IM was considered as a pre-cancerous lesion; however, several studies have investigated the effect of IM on gastric ulcer healing and H. pylori eradication [13].

Throughout Europe, Asia, and Latin America ranging from 10 months to 19 years of follow-up, 10 observational studies, a systematic review, showed the risk of gastric cancer to be 4- to 11-fold higher with incomplete metaplasia compared to without incomplete metaplasia [15]. Atrophic gastritis, intestinal metaplasia, mild-moderate dysplasia, and severe dysplasia were associated with annual incidences of gastric cancer of 0.1%, 0.25%, 0.6%, and 6.0%, respectively [16]. The incidence of gastric cancer associated with IM ranges from 0% to 10% in systematic reviews, with the variable range attributable to various sample sizes and follow-up periods [17,18].

We have investigated the relationship of HH with IM that is important in patients with the development of gastric cancer; we found that IM was less statistically significant in patients with hiatal hernia.

There were several limitations of the study. First, small sample size may have been caused a bias against control group. Second, due to retrospective nature of the study, the results may not have generalised the entire study population. On the other hand, this small study was the first in English literature to show a negative association between hiatal hernia and intestinal metaplasia.

CONCLUSION

Although the mechanisms of IM formation are not fully understood yet, IM was less frequently found in patients with hiatal hernias in our study; it may be attributed to the fact that these patients had earlier dyspeptic complaints and therefore earlier medical treatment and earlier initiation of a protective gastric diet.

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