Scholars Journal of Applied Medical Sciences (SJAMS) **ISSN 2320-6691 (Online)**
Sch. J. App. Med. Sci., 2014; 2(6C):3042-3045 **ISSN 2347-954X (Print)**

(An International Publisher for Academic and Scientific Resources)
www.saspublishers.com **DOI:** 10.36347/sjams.2014.v02i06.040

Research Article

Prevalence of Intestinal Metaplasia in Infectious and Non Infectious Chronic Gastritis

Bashar A. Hassawi[1], Khesar Hussein[2], Zobayda Adnan[3]
[1]Assistant Professor, Department of Anatomy, College of Medicine, University of Duhok, Iraq
[2]Assistant Lecturer, Department of Anatomy, College of Medicine, University of Duhok, Iraq
[3]Assistant Lecturer, Department of Physiology, College of Medicine, University of Duhok, Iraq

***Corresponding author**
Bashar A. Hassawi
Email: b_hassawi@yahoo.com

Abstract: The objective of the study was to estimate the prevalence of intestinal metaplasia in dyspeptic patients in Duhok (Kurdistan region) and its association with *H. pylori* and dysplasia according to gender and age. It was a prospective observational study performed from Jan 2012 to Jul 2014 at Azadi Teaching Hospital Duhok City. A total number of 205 antral gastric biopsies were collected in Azadi GIT-Centre and examined histologically to detect intestinal metaplasia. These biopsies were stained by Giemsa to detect *H. pylori* organisms. *H. pylori* was found in 94 (46%) patients while intestinal metaplasia in general was found in 30 (15%) patients; (9.8% in association with H pylori) and (4.7% without *H. pylori*). Among intestinal metaplasia 6 cases show dysplasia (6/205= 3%). This study has documented mainly that the prevalence of intestinal metaplasia is significant among patients (In Duhok) with chronic gasritis and mainly in those patients who are positive for H Pylori with an active chronic gastritis. Intestinal metaplasia mostly affects patients above the age of 40 years with no significant gender difference.
Keywords: Intestinal metaplasia, Chronic gastritis, Duhok

INTRODUCTION

Chronic gastritis is defined as presence of chronic mucosal inflammatory changes leading eventually to mucosal atrophy and intestinal metaplasia. In Western world the prevalence of chronic gastritis in the later decades of life is higher than 50% [1]. After 1982 the etiology of gastritis and other different pathology of stomach (chronic non ulcer dyspepsia, peptic ulcer and even gastric carcinoma) has been changed completely when the Marshal B J and Warren J R discovered spiral and curved microoorganism called *Helicobnacter pylori* (*H. pylori*) [2].

This bacterium has ability to affect gastric mucosa and induces inflammation and eventually intestinal metaplasia which refers to replacement of gastric epithelium with columnar and goblet cells of intestinal variety [3]. The most common cause of intestinal metaplasia is H.pylori associated chronic gastritis [2, 4]. These metaplastic changes may become dysplastic and constitute a background for development of gastric carcinoma and that is why some considered it as a precancerous lesion; the risk for such tumor development is greatly increased in patients in whom mucosal inflammation progresses to multifocal mucosal atrophy and intestinal metaplasia [5, 6]. They thought that intestinal type gastric carcinoma "which presents in more than two thirds of gastric cancer" is to be arise from intestinal metaplasia of gastric epithelium, an assumption supported by electron microcopy and immunohistochemical studies [7].

Aims of study

- To detect the presence of intestinal metaplasia (IM) in gastric biopsies.
- To find out the association between H Pylori, Intestinal metaplasia and dysplasia.
- To assess the age and sex distribution of the IM in gastric biopsies

METHODOLOGY

During a period of two years a total 205 cases of endoscopic gastric biopsies were collected from Azadi teaching hospital This study was performed by collection of endoscopic antral mucosa biopsies from Jan/2012-Jul/2014, each biopsy contained at least 2 mucosal pieces that submitted to a conventional histological processing and stained with hematoxylin-eosin stain to asses the presence of intestinal metaplasia. These biopsies were stained by Giemsa to detect H.pylori organisms. The age of patients ranged from 11-90 years, (106) were males and (99) were females.

Statistical methods include chi-square and t-statistics were calculated to measure the association between different parameters.

RESULTS

A total 205 biopies from dyspeptic patients (Vary 11-90 years; mean age 43.7) were examined and showed; gastritis (n = 164), peptic ulcer (n = 30), erosion (n =10) and normal (n = 1) consecutively (Table 1).

H. pylori was found in 94 (46%) patients (11-75 years; mean age 43.3) (Fig. 1). Intestinal metaplasia in general was found in 30 (15%) patients (Fig. 2). Intestinal metaplasia that is associated with *H. pylori* was seen in 20 (9.8%) patients, while intestinal metaplasia without *H. pylori* was seen in 10 (4.9%) patients (Table 2). Among intestinal metaplasia 6 cases showed dysplasia (6/205= 3%) (Fig. 3).

From 30 intestinal metaplasia positive cases 13 were males & 17 were females. The prevalence of intestinal metaplasia according to gender was 7.20% in male & 7.80% in female and by application of Chi-square test this difference was insignificant (p>0.05).

20 biopsies out of total 30 biopsies of intestinal metaplasia (66.6%) were present in patients above the age of 40 years. The peak age distribution was between 40-59 years that included (33.33%) of cases.

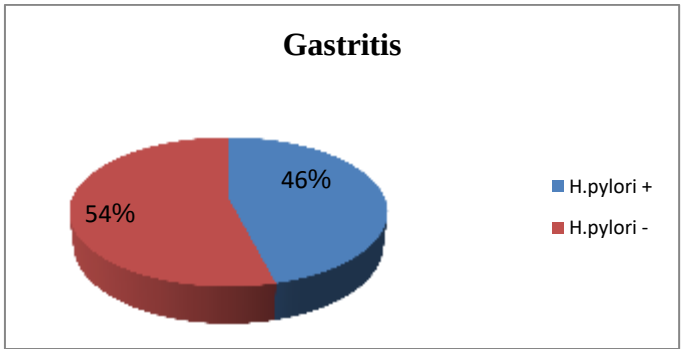

Fig. 1: Percentage of H.pylori + among total 205 cases

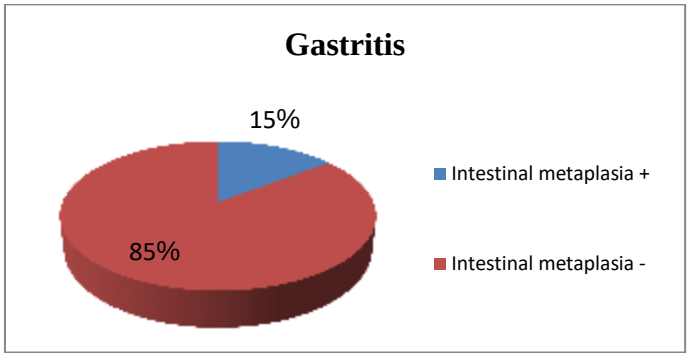

Fig. 2: Percentage of IM + among total 205 cases

Table 1: *Characterstics of patients according to histological diagnosis* (n=205)

Diagnoses	Patients (n)	Patients(n) IM+ (% n)	Patients(n) HP+ (% n)
Normal	1	0 (0%)	-
Erosion	10	2 (20%)	2 (20%)
Gastitis	164	24(14.6%)	82 (50%)
Gastric ulcer	30	4 (13.3%)	10 (33.3%)
Total	205	30(15%)	94(46%)

IM+= Intestinal metaplasia positive, HP+= H Pylori positive

Table 2: Number of cases and percent of IM+ in association with HP (n=30)

Diagnoses	Patients(n) IM+ (% n)	Type of gasritis
IM+ with H.Pylori	20 (10%)	Active chronic gastritis
IM+ without H.Pylori	10 (5%)	Chronic gastritis only
Total	30 (15)	

IM+= Intestinal metaplasia positive, HP+= H Pylori positive

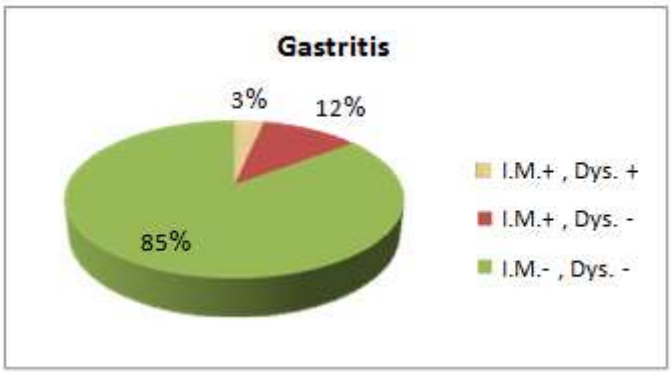

Fig. 3: Relationship between I.M. + and dysplasia among total 205 cases
I.M.+ = Intestinal metaplasia positive, I.M.- = Intestinal metaplasia negative, Dys.+ = Dysplasia positive, Dys.- = Dysplasia negative

DISCUSSION

H. pylori infection has been categorized as a type I carcinogen by The International Agency for Research on Cancer [8]. It has been considered as the primary cause of gastric cancer. The infection causes chronic inflammatory process in the gastric mucosa and over time, may result in the development of atrophy and IM [9, 10]. IM is frequently identified in distal gastric biopsies, especially in populations at high risk for gastric cancer. In countries such as those of eastern Asia, Eastern Europe, and Andean Latin America; the intestinal metaplasia (IM) of the gastric mucosa is a relatively frequent precancerous lesion. In the United States, majority of the population is at low risk for gastric cancer, but ethnic populations such as African Americans, Native Americans, and immigrants from Asia and Latin America are at risk [11, 12].

It has been reported that IM increases the risk of gastric cancer. But overall risk of gastric cancer in a patient with IM is extremely low when compared to that in an adenocarcinoma patient with Barrett's esophagus (BE) [13]. In the present study the prevalence of intestinal metaplasia in patients with dyspepsia was 15%, (7.20%) in males and (7.80)% in females, this rate is in agree with Carrilho C *et al*. [14] study and Hackelberger A *et al*. [15] rate in the gastric corpus. But it is significantly lower than Kim HJ *et al*. [16] study in Korea (42.5%) in males & (32.5%) in females and Zhang C *et al*. [17] study in China (39.9%). These high rates of IM in the above two studies are in keeping with the high prevalence of gastric carcinoma in these countries.

The prevalence of IM in the present study puts Iraqi population in the low prevalence area and this result had provided a confirmatory evidence to Al-Kanawy *et al*. study which reported that the prevalence of IM in Saudi Arabian was 15.2% (considered as a low prevalence area) [18].

In agree with previous studies, our study revealed no significant gender variation among patients with IM ($p>0.05$) and the peak age distribution of IM was between 40-49 years [19].

Giemsa stain was positive in 10 % (and negative in 5%) of biopsies with IM. This rate of *H. pylori* negative cases (5%) is probably because *H. pylori* colonization is usually absent in areas of IM [5].

CONCLUSIONS

- IM prevalent in patients with *H. pylori*.
- Searching for IM in each gastric biopsy is a major criterion for a severity of gastritis and progression of lesion toward neoplasia.
- Histopathological study still superior for tissue based test and serology to detect intestinal metaplasia.
- IM more common in patients older than 40.

REFERENCES

1. Correa P, Piazuelo MB, Wilson KT; Pathology of gastric intestinal metaplasia: clinical implications. Am J Gastroenterol., 2010; 105(3): 493–498.
2. Marshal BJ, Warren JR; Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulcer. Lancet, 1984; 1(8390): 1311-1315.
3. Tang SJ, Wu R, Bhaijee F; Intestinal metaplasia of the stomach. Video Journal and Encyclopedia of GI Endoscopy, 2013; 1(1): 187-189.
4. Bashar H, Al-Mukhtar M; The Demonstration of *Helicobacter pylori* in the gastroduodenal endoscopic biopsies by using different stains. Tikrit Medical Journal, 2004; 10(2): 1-7.
5. Kumar V, Abbas AK, Fausto N; Robbinns and Cotran pathologic basis of disease. 7th edition, 2004: 813-823.
6. Kang KP, Lee HS, Kim N, Kang HM, Park YS, Lee DH *et al.*; Role of intestinal metaplasia subtyping in the risk of gastric

cancer in Korea. Journal of Gastroenterology & Hepatology, 2009; 24(1):140-148.
7. Rosi J; Rosi and Ackerman's Surrgical Pthology, 9th edition, 2011: 663.
8. International Agency for Research on Cancer, World Health Organization; Schistosomes, Liver Flukes and Helicobacter pylori. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 61, Lyon, France: 1994.
9. Correa P, Haenszel W, Cuello C, Tannenbaum S, Archer M; A model for gastric cancer epidemiology. Lancet, 1975; 2(7924): 58-60.
10. Filipe MI, Munoz N, Matko I, et al. Intestinal metaplasia types and the risk of gastric cancer: a cohort study in Slovenia. Int J Cancer, 1994; 57(3): 324–329.
11. Espey DK, Wu XC, Swan J, Wiggins C, Jim MA, Ward E *et al*.; Annual report to the nation on the status of cancer, 1975–2004, featuring cancer in American Indians and Alaska Natives. Cancer, 2007; 110(10): 2119–2152.
12. Wu X, Chen VW, Andrews PA, Ruiz B, Correa P; Incidence of esophageal and gastric cancers among Hispanics, non-Hispanic whites and non-Hispanic blacks in the United States: subsite and histology differences. Cancer Causes Control, 2007; 18(6): 585–593.
13. Fennerty MB; Gastric intestinal metaplasia on routine endoscopic biopsy. Gastroenterology, 2003; 125(2): 586–590.
14. Carillho C, Modcoicar P, Cunha L, Ismail M, Guissive A, Lorinzoni C *et al*.; Prevalence of *H. pylori* infection chronic gastritis and intestinal metaplasia in Mozambican dyspeptic patients. Virchows Arch., 2009; 454(2):153-160.
15. Hackelsberger A, Guther T, Schultze V, Labenz J, Roessner A, Malfertheiner P; Prevalence and pattern of *H. pylori* gastritis in gastric cardia. American Journal of gastroenterology. 1997; 92(12): 2220-2224.
16. Kim HJ, Choi BY, Byun TJ, Eun CS, Song Kim YS, Han DS; The prevalence of Atrophic gastritis and intestinal metaplasia according to gender, age and *H. pylori* infection in a rural population. Journal of preventive medicine and public health. 2008; 41(6): 373-379.
17. Zhang C, Yamada N, Wu YL, Wen M, Matsuhisa T, Matsukura N; *H. pylori* infection glandular atrophy and intestinal metaplasia in superficial gastritis, gastric erosion, erosive gastritis, gastric ulcer and early gastric cancer. World Journal of Gastroenterology, 2005; 11(6): 791-796.
18. Al-Kanawi B, Mordan N, Jamal A, Mirdad S, Abol Fotouh M, Ahemd ME *et al*.; *H. pylori* and intestinal metaplasia with its subtyping in gastric antrum in a Saudi population. Scandinavian Journal of Gastroenterology, 1999; 34(6): 562-565.
19. Khan AR; An age- and gender–specific analysis of *H. pylori* infection. Ann Saudi Med., 1998; 18: 6–8.