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Original Article

Analysis of Helicobacter pylori gastritis according to Sydney classification.

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Abstract:

Introduction: Since H. Pylori has been isolated in stomach and duodenum, there were individual methodologies to explain the grading according to colonization and density of microorganism. In 1990 Sydney system of classification was proposed at the 9th world congress of gastroenterology in Sydney Australia, based on topographical, morphological and etiological findings. This classification revised in 1994 and updated by experts in Houston Texas. H. Pylori is major cause of chronic gastritis results in gastric and peptic ulcer. It also causes MALT lymphoma and malignancy. For histopathological examination four specimens, two from antrum and two from corpus are recommended.

Objective: To evaluate the Sydney system of classification and grading for H. Pylori in local population.

Methodology: 287 cases of biopsies received at the department of pathology Muhammad Medical College, Mirpurkhas Sindh Pakistan, during the period of January 2017 to December 2018 were revised. Sections stained with Hematoxylin and Eosin; Alcian blue and periodic acid Schiff to define H. Pylori and associated pathological changes. The microscopical findings classified according to upgraded Sydney pattern.

Results: Out of 287 cases of gastric biopsies 23 (8.0%) were positive for H. Pylori. chronic inflammation present in 168 (58.5%). Neutrophilic activity found in 58 (18.1%). Atrophy without metaplasia observed in 31 (10.8%). Intestinal metaplasia present in 1 (0.3%) cases.

Conclusion: Sydney system of grading is best to evaluate chronic gastritis and reliable indicator of H. Pylori microorganism.

Keywords: Chronic gastritis, Gastric carcinoma. H. Pylori.

Introduction:

Helicobacter pylori is a major etiological factor in pathogenesis of the chronic gastritis.[1] These are spiral to curved bacilli present in the biopsy specimens of stomach.[2] Infection with H. Pylori is widespread in developing countries, more than 68% of adults are infected. The source of infection is contaminated water, food, poverty, household overcrowdings, poor sanitation and orofecal route infections.[3] It could be possible to isolate H. Pylori bacilli on culture by inoculate the plate of chocolate agar or campylobacter medium. For culturing the bacilli, fresh biopsy specimens are required. The specimen is collected in a bottle containing 0.5 ml of sterile physiological saline.[4] H. Pylori bacilli are catalase and oxidase positive, also show strong urease activity.[5] These microorganisms cause antral gastritis, increase the acid production which result in peptic ulcer. Patchy mucosal atrophy progress to involve gastric body and fundus, which condition is known as multifocal atrophic gastritis.[6] In 1990 Sydney system of classification and grading was proposed at the 9th world congress of gastroenterology in Sydney Australia, based on combination of topographical, morphological and etiological findings to evaluate gastritis. In 1994 Sydney sys-

tem was revised and updated by experts in Houston Texas.[7,8] To study the biopsy specimens anatomical site has importance to detect H. Pylori. Four specimens, two from antrum and two corpus are recommended for correct H. Pylori status.[9] Normally gastric mucosa contains individual scattered mononuclear cells in the lamina propria. Due to infection with H. Pylori there is increase in number of inflammatory cells, mostly mononuclear cells are increased in number. There is presence of T–Lymphocytes, B–Lymphocytes, plasma cells and eosinophils. Neutrophilic activity is associated with tissue damage. In glandular atrophy, there is loss of glandular tissue, thinning of mucosa[10] and it could be resulted in intestinal metaplasia. The intestinal metaplasia is recognized morphologically by presence of goblet cells.[11]

Table 1: Sydney system of grading [(12)].

Feature	Definition	Grading Guidelines
Chronic inflammation	Increased lymphocytes and plasma cells in the lamina propria.	Mild, moderate, or severe increase in density
Activity	Neutrophilic infiltrates of the lamina propria, pits, or surface epithelium	Less than one third of pits and surface infiltrated=mild; one third to two thirds= moderate; more than two thirds= severe.
Atrophy	Loss of specialized glands from either antrum or corpus	Mild, moderate, or severe loss.
Intestinal metaplasia	Intestinal metaplasia of the epithelium	Less than one third of mucosa involved= mild; one third to two thirds= moderate; more than two thirds = severe.
Helicobacter Pylori	H. Pylori density	Scattered organisms covering less than one third of the surface= mild colonization: large clusters or a continuous layer over two thirds of surface= severe; intermediate numbers= moderate colonization.

Methodology:

This retrospective study performed at department of pathology Muhammad Medical college Mirpurkhas Sindh Pakistan. 287 cases of biopsies received during the period of January 2017 to December 2018 were revised. Endoscopic biopsies from antrum and corpus fixed in buffered neutral formalin were processed and stained with hematoxylin and Eosin (H&E). Modified Giemsa stain employed to reveal H. Pylori microorganisms. Alcian blue (AB, PH2.5) and periodic acid Schiff (PAS) stain also used to demonstrate intestinal metaplasia. During microscopical examination all the features observed and classified to upgrade Sydney pattern. H. Pylori colonization graded according to involvement of gastric mucosa. Neutrophilic activity noted in the mucosa, crypts and lamina propria. Atrophic changes graded by loss of gastric glands and replacement of intestinal type of epithelium. Dysplastic changes also observed carefully.

Results:

Results according to Sydney pattern of gradings.

Table:2. Total number of cases and their involvement according to features of chronic gastritis. (n:287).

Features	Grading n=287	
	n	**%**
Chronic Inflammation	168	58.5
Neutrophilic Activity	52	18.1
Atrophy	31	10.8
Intestinal Metaplasia	12	4.1
H. Pylori	23	8.0
Dyspepsia	01	0.3

Table :3. Number and percentage of cases with mild grading.

Features	Grading, Mild (n=122)	
	n	%
Chronic Inflammation	56	33.3
Neutrophilic Activity	28	53.8
Atrophy	12	38.7
Intestinal Metaplasia	8	66.6
H. Pylori	18	78.2
Dysplasia	00	00

Table:4. Number and % of cases with moderate grading.

Features	Grading Moderate (n=125)	
	n	%
Chronic Inflammation	91	54.1
Neutrophilic Activity	16	30.7
Atrophy	10	32.2
Intestinal Metaplasia	03	25.0
H. Pylori	04	17.3
Dysplasia	01	100

Table:5. Number and percentage of cases with severe grading.

Features	Grading Severe (n=40)	
	n	%
Chronic Inflammation	21	12.5
Neutrophilic Activity	08	15.3
Atrophy	09	29.0
Intestinal Metaplasia	01	08.3
H. Pylori	01	04.3
Dysplasia	00	00

Out of 287 cases of gastric biopsies 23 (8.0%) were positive for H. Pylori. The microorganism graded as mild, moderate and severe according to its density and involvement of gastric mucosa. It was 78.2% mild. 17.3% moderate and 4.3% severe. Chronic inflammation was present in 168 (58.5 %) and graded 33.3% mild, 54.1% moderate and 12.5% severe. Neutrophilic activity found in 52 (18.1%) cases with 53.8% mild. 30.7% moderate and 15.3% severe. Atrophy without intestinal metaplasia observed in 31 (10.8%) cases with 38.7% mild. 32.2% moderate and 29.0 severe. Intestinal metaplasia was present in 12 (4.1%) cases and graded 66.6% mild. 25% moderate and 8.3% severe. Dysplastic changes were present in 1 (0.3%) cases and graded 100% moderate.

Discussion:

It is important to evaluate H. Pylori in chronic gastritis according to Sydney classification to conclude the level of colonization and density.[13,14] This methodology is helpful to irradiate the microorganism by proper treatment and deduce the prognosis.[15] H. Pylori causes MALT lymphoma and gastric carcinoma, by treatment of H. Pylori the mortality by gastric carcinoma has been reduced dramatically.[16] In most persons infection with H. Pylori is asymptomatic, the microorganism is well adopted to echogenic niche provided by gastric mucosa, urease enzyme produced by H. Pylori generates ammonia from endogenous urea which alters the gastric PH and help it to survive.[17] The cytotoxin associated gene A (cagA) and its variations are significant in disease outcome. Genetic predisposition of host also plays an important role.[18] In our study there are 58.5% cases of chronic gastritis, out of which 8.0% cases are involved by H. Pylori. Other causes of chronic gastritis could be non-H. Pylori and lymphocytic gastritis. [19] Endogenous or exogenous substances with toxic potential for gastric mucosa. Allergic eosinophilic gastritis, radiational and granulomatous reactive forms also cause chronic gastritis.[20] It is recommended to correlate histological findings with serological examination. The chromatography immunoassay is easy and urgent method to detect qualitative antibodies against H. Pylori .[21] Due to antibiotic therapy there is much improvement and decline in the incidence of intestinal metaplasia associated with H. Pylori infection but is still a cause of lethal malignancy.[22] Pathological findings according to upgraded Sydney classification are helpful to recognize the level of disease. Glandular atrophy is considered precancerous, topographical assessment and extent of atrophy could be reported to define well with Sydney classification, even the prognosis can be monitored by repeat biopsies after therapy.[23] Our findings of chronic gastritis revealed an agreement with many previous studies. Mild gastritis in 35.8% of patients. Moderate in 57.5% and severe in 14.8%.[24] In our study H. Pylori is present mild in 78.2% cases, moderate in 17.3% and severe in 4.3% which shows slight variation in correlation with other studies. Glandular atrophy is 12.3% and intestinal metaplasia in 15.8% cases[25] is also close to our study. Histological detection of H. Pylori is considered as first diagnostic method in chronic gastritis, its further confirmation is needed by serological examination.[26]

Conclusion:

Histopathological interpretation of chronic gastritis by upgraded Sydney classification is reliable indicator of H. Pylori infection. It has great value to define the grading of disease by well-established methodology to estimate treatment efficacy.

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