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Profile of gaster endoscopic biopsy results in patients at MRCCC siloam hospitals semanggi 2013 – 2017

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Abstract
The discovery of Helicobacter pylori (H. pylori) by Warren and Marshall in 1982 supported the increasing prevalence of H. pylori infection from year to year. H. pylori infection is associated with gastritis, peptic ulcer, gastric carcinoma, and MALT lymphoma. Often found in the gastric antrum, H. pylori colonise by producing urease to change the acid pH in the stomach (1.0-2.0) to the optimal pH to grow (6.7-7.0). The study found that patients who had the most gastric biopsy at MRCCC Siloam Hospitals Semanggi in 2014 were 92 patients. Of all data, most were found in female patients (59.3%) who had gastric endoscopic biopsies. Patients positively infected with H. pylori were 32 patients (8.6%), of whom 17 were WARD patients, and 15 outpatients were outpatients. Of the 32 positive patients, the majority suffered from non-atrophic active chronic gastritis, as many as ten patients. The results of this study are expected to be good information and knowledge for MRCCC Siloam Hospitals Semanggi and society.

Keywords: gaster, gastritis, infection, H pylori, MRCCC siloam hospitals semanggi

Introduction
The discovery of Helicobacter pylori (H. pylori) by Warren and Marshall in 1982 was preceded by hundreds of years of classified publications dealing with spiral bacteria, achlorhydria, gastritis, gastric urease, and antimicrobial therapy for peptic ulcers [1]. Helicobacter pylori (H. pylori) is a spiral-shaped, gram-negative bacterium that grows in the digestive system. H. pylori infection has a high prevalence and can infect half of the world's population [2]. H. pylorus is an essential factor in gastroduodenal disease. This infection is most commonly acquired in early childhood and causes chronic gastritis in both children and adults, and is a major cause of peptic ulcer disease in humans. The infection is also implicated in the etiopathogenesis of chronic gastritis, gastric atrophy, chronic diarrhoea, growth retardation in children, peptic ulcer disease (PUD), gastric carcinoma, and gastric mucosa-associated lymphoid tissue (MALT) lymphoma [1, 3]. It has been widely demonstrated that H. pylori play a significant role in several upper gastrointestinal diseases that have emerged as dyspepsia since their discovery in the early 1980s [4, 5].

Helicobacter pylori can be passed from person to person through direct contact with saliva, vomit, or faeces. The possible routes of transmission are feco-oral, oral-oral, and gastro-oral. Risk factors for H. pylori infection are associated with: living in crowded conditions – living in a house with many people, living without a clean water supply, living in a developing country, living with people infected with H. pylori. The highest risk factors were related to age and race/ethnicity. Nearly half of all African Americans are infected with the H. pylori bacteria [6].

H. pylori infection is a common problem in pediatric practice and is associated with poor socioeconomic conditions. H. pylori cause chronic inflammation (gastritis) by invading the stomach lining and thus can lead to ulcer formation.7 Most people infected with H. pylori have no symptoms. However, according to the Kyoto global consensus report, H. pylori infection is now considered an infectious disease, regardless of the presence or absence of symptoms.8 When infection causes ulcers, symptoms may include abdominal pain, excessive belching, feeling bloated, nausea or vomiting, appetite decreased, and sudden weight loss [7].

H. pylori infection can cause peptic ulcers, but the infection or the ulcer itself can cause more severe complications such as; internal bleeding. It can occur when a peptic ulcer ruptures through a blood vessel; obstruction, which can occur when the ulcer blocks food; perforation, which can occur when the ulcer penetrates the abdominal wall and peritonitis, which is an infection of the peritoneum, or lining of the abdominal cavity [8]. The problem that will be answered in this study is "what is the profile of the patient whose stomach biopsy was MRCCC Siloam Hospitals Semanggi?" intending to know the profile of patients undergoing gastric biopsy at MRCCC Siloam Hospitals Semanggi.

Literature Review
The stomach (stomach) is an intraperitoneal digestive organ located between the oesophagus and the duodenum. The stomach is located on the superior aspect of the abdominal region. The exact size, shape and position of the stomach can vary from person to person. All digested materials, including nutrients, must first be digested by the gastric organ, and thus, the stomach is the essential part of the digestive tract. The unique biological function of gastric acid secretion initiates the digestive process and acts as the first line of defence against food-borne microbes. The importance of the gastric microbiome and its association with H. pylori revolutionises our understanding of gastric diseases, especially prevention and a growing body of evidence points to the potential role of the gastric microbiome in several digestive mechanisms [9].

The stomach plays an essential role in the first stage of digestion by controlling the transmission of food, breaking it down to a semi-liquid consistency (mechanical digestion) and increasing secretions (chemical digestion). The stomach also acts as an endocrine and immunological organ. The stomach, which develops from the anterior part of the intestine, is located in the left upper quadrant of the abdomen (hypogastric). Forms a 'J'-shaped sac that extends from the oesophagus inferior part (gastro-oesophageal junction), a few centimetres below the diaphragm and to the left of the midline, ending at the gastroduodenal junction to the right of the midline. The size and position of the stomach changes according to the body position and quantity [1].

The concave superomedial portions of the stomach and the convex inferolateral portions are called the lesser and greater curvature. The ventral and dorsal parts of the stomach are called the anterior and posterior walls.1 Macroscopically, the stomach is divided into four parts: Cardia, fundus, body, and antrum (pyloric antrum). The body consists of about two-thirds of the stomach. The antrum (pyloric antrum) is the infundibular canal consisting of the distal one-third of the stomach, leading to the pylorus (pyloric ring), a curved bulge that separates the stomach from the duodenum.

The stomach wall consists of four layers: the muscular, submucosa, muscularis propria, and subserosa, lined by serosa. The surface of the gastric mucosa, especially the fundus and body, is rough, has longitudinal indentations ('rugae'), consisting of the mucosa and the muscularis mucosa with a core of submucosal tissue [10].

Histologically, the fundus and body are similar in appearance. All parts of the stomach display longitudinal folds (transverse in the area of the antrum) called rugae, which disappear as the stomach expands. These longitudinal folds involve the mucosal and submucosal layers. Rugae cause the stomach to expand when it is full of food and gastric fluids. In addition, the gastric epithelial layer penetrates the mucosa and forms a gastric well (foveolar gastric). The shallowest gastric well is in the fundus and the deepest in the pylorus. The microscopic structure of the gastric mucosa, which will be discussed in detail, is the microscopic structure of the gastric fundus because the other parts are variations of the gastric fundus. The gastric fundus mucosa consists of three layers: a) epithelium bordering the lumen; b) connective tissue beneath the epithelium called the lamina propria, and c) the muscular mucosal layer consisting of smooth muscle fibres.

Gastric epithelium: The lumen of the gastric fundus is lined with a simple cylindrical epithelium consisting of surface epithelial cells, which produces a thick mucus layer of jelly-like material that adheres to the gastric surface protects it from autodigestion. In addition, bicarbonate ions are trapped in the mucus layer so that at the mucus boundary with the surface epithelial cell membrane, a relatively neutral pH can be maintained, even though the lumen contents are low in pH (acidic).

Lamina propria stomach: Lamina propria stomach is composed of loose connective tissue that is highly vascular (contains many blood vessels). This connective tissue contains many plasma cells, lymphocytes, mast cells, fibroblasts, and many smooth muscle fibres. Most of the lamina propria is filled with gastric glands, which number about 15 million, which in the fundus are called fundus (oxyntic) glands which extend from the muscular mucosa to the bottom of the gastric wall.

Muscularis gastric mucosa: The smooth muscle cells that make up the muscular gastric mucosa are arranged in three layers. The three layers are the well-developed outer circular and outer longitudinal layers and the outermost circular and outermost layer. This third layer is not always present.

Collagen dense connective tissue is irregularly arranged in the gastric submucosa rich in vascular and lymphatic networks that supply and receive blood from the blood vessels of the lamina propria. The population of gastric submucosal cells is similar to that of actual connective tissue. The submucosal plexus is found in its usual location, near the muscular externa.

Muscularis externa gastric smooth muscle cells consist of three layers. The innermost oblique layer is not visible except in the cardia, and the circular middle layer is visible in all stomach parts and very clearly in the pylorus because this layer forms the pyloric sphincter. The outer longitudinal layer is most pronounced in the cardia and body of the stomach but is less developed in the pylorus. The myenteric plexus is located between the middle circular layer and the outer longitudinal layer. The entire stomach is covered by serosa, which consists of thin subserous loose connective tissue and is covered by a stratified squamous epithelium that looks wet and slippery on the outside. This serosa functions as a lubricant so that there is no friction in the stomach [11].

On closer inspection, the mucosa is composed of polygonal layers centred on the gland openings (foveolae or orifices) and lined by tiny, narrow grooves (areas gastrique) [12]. Under the microscope, the mucosal surface of the body/fundus is different from that of the antrum, showing the appearance of 'Morocco leather, where the mucosa of the antrum has a rougher leaf-like pattern [13]. Under endoscopic magnification with narrow-band imaging (NBI), the body/fundus mucosa shows a honeycomb appearance, and each chamber is depicted with an exemplary subepithelial capillary network. In contrast, the antral mucosa shows a coarser mesh or knot appearance with capillary blood vessels. These findings are used as the basis for the initial diagnosis of gastric neoplasms [14].

Gastric Hormones: Hormones regulate several critical physiological functions in the stomach, including secretion and motility. Abnormal production of some of these hormones is associated with the development of various diseases of the stomach. As the stomach expands, its intraluminal pressure remains relatively constant due to the hormone ghrelin, which causes hunger and causes receptive relaxation of the external muscular smooth muscle fibres. The stomach gradually excretes small amounts of its contents through the pyloric valve into the duodenum. The stomach makes food liquid and continues digestion through gastric acid and various enzymes, namely pepsin, renin, gastric lipase, and paracrine hormones.

Another primary hormone produced by the stomach is gastrin. Gastrin is secreted by G cells, which are mainly located in the antrum of the stomach. It has a well-known function in regulating gastric acid secretion. Gastrin secretion is inhibited by somatostatin, which is secreted by

D cells in the stomach and intestines. Recent evidence suggests that gastrin also regulates other critical cellular pathways in the stomach, including cell proliferation, migration, invasion, angiogenesis and apoptosis and recent *in vitro* studies have shown that gastrin plays a crucial role in maintaining gastric stem cells [15].

Gastric microbiome: The stomach secretes gastric acid, composed chiefly of proteolytic enzymes and hydrochloric acid, thereby creating the necessary environment for protein denaturation and nutrient absorption. Stomach acid also limits the number of microorganisms that enter the small intestine and reduces the risk of pathogenic infection. When the human gastric lumen has a pH of 1.0-2.0, the mucus layer causes an increase in pH of up to 6.0-7.0 at the mucosal surface. Reduction of gastric acid secretion increases the risk of bacterial overgrowth. Also, it affects the composition of intestinal or oral microorganisms, including organisms that cause disease and which have nitrosating abilities that are generally not cultured from a normal and healthy stomach [16]. Prolonged mucosal inflammation, associated with H. pylori, causes several structural changes, including loss of glands in the gastric mucosa and replacement of normal cells by metaplastic cells. In gastritis associated with H. pylori, atrophic changes previously occur in the angled transitional mucosa, then involve the distal stomach (antrum-restricted atrophic gastritis) before spreading to the proximal oxyntic mucosa (multifocal atrophic gastritis) or atrophic pan-gastritis. The density of H. pylori increases with the onset of gastritis so that H. pylori can overpower other bacteria [17].

Helicobacter pylori infection is the most common infection worldwide and is associated with uncomplicated dyspepsia, heartburn and peptic ulcer disease, most commonly leading to upper gastrointestinal bleeding and, finally, to severe complications of gastric malignancy. 90% of duodenal ulcers and 70% of gastric ulcers are associated with Helicobacter pylori infection.16 Helicobacter pylori is a spiral-shaped gram-negative, microaerophilic bacterium that infects the epithelial lining of the stomach. The genus Helicobacter is grouped in the subdivision of Proteobacteria, order Campylobacterales, family Helicobacteraceae, and this family also includes the genera Wolinella, Flexispira, Sulfurimonas, Thiomicrospira, and Thiovulum. The genus Helicobacter comprises more than 20 recognised species, with many species awaiting formal recognition. Members of the genus Helicobacter are all microaerophilic organisms and, in most cases, are catalase and oxidase-positive, and many but not all species are also urease positive [18].

Helicobacter species can be divided into two main lineages: Helicobacter gaster and enterohepatic (nongastric). Helicobacter species in the stomach have adapted to the hostile conditions found on the gastric mucosal surface, and nowadays, mammalian stomachs can be infected by members of the Helicobacter genus. All known species of Helicobacter gaster are urease positive and are highly mobile through flagella. The main feature of H. pylori is microaerophilic, with optimal growth at 2 to 5% O2 levels and the need for an additional 5 to 10% CO2 and high humidity. It does not require H2, although it is not detrimental to growth. Many laboratories use standard microaerobic conditions of 85% N2, 10% CO2, and 5% O2 for H. pylori culture. Growth occurs at 34 to 40°C, with an optimum of 37°C. Although its natural habitat is acidic gastric mucosa, H. pylori are considered a neutralophile. Bacteria will survive briefly at pH 4, but growth occurs only in a relatively narrow pH range of 5.5 to 8.0, with optimal growth at neutral pH [19].

H. pylori have many characteristics in common with campylobacter. These bacteria have many flagella at one pole and move actively. Culture sensitivity may be decreased due to previous therapy, contamination with other mucosal bacteria, and other factors. H. pylori grow in 3-6 days if incubated at 37ºC in a microaerophilic atmosphere, as does C. jejuni. Medium for primary isolation included Skirrow's medium with vancomycin, polymyxin B, trimethoprim, chocolate medium, and other selective media to which antibiotics were added (e.g., vancomycin, nalidixic acid, amphotericin). Colonies appear translucent and 1-2 mm in diameter. H. pylori are oxidase-positive, and catalase-positive has a characteristic morphology, is motile, and is a strong urease producer [20]. Helicobacter pylori infection occurs globally, but the prevalence differs between countries and populations in the same country18. The prevalence of infection is associated with age, socioeconomic class, and country of origin19. Prevalence rates ranging from 20-50% are reported in the adult population in developed countries, but prevalence is much higher in developing countries, with prevalence as high as 90% in some countries. The prevalence is higher in areas with low socioeconomic conditions and poor sanitation conditions than rural areas in contrast to urban areas. Family socioeconomic status during childhood appears to be a significant marker of infection. It is believed that infection occurs mainly in childhood, and the acquisition rate has decreased with improved sanitary conditions and possibly antibiotic use among children in developed countries [21, 22]. In many countries, the incidence of H. pylori infection has decreased concerning increasing standards of living. Nevertheless, the prevalence of this bacterium is still everywhere, especially in the East. It is a significant cause of chronic gastritis and a principal etiologic agent for gastric cancer and peptic ulcer disease. In most areas, the primary mechanism of spread is the intrafamilial transmission. Prevalence remains high in most developing countries and is generally related to socioeconomic status and hygiene levels. Global and regional prevalence of H. pylori has not been systematically reported to date. H. pylori are found in water, stomachs of animals such as cats and sheep, and in the milk of goats, sheep, and cows. Helicobacter pylori have been found in human saliva and dental plaque. The primary mode of transmission is person to person and group, which is mainly found in families. Usually, infection occurs when these bacteria are in fluids, food, or even if the equipment is contaminated with bacteria and inhaled by humans. It can occur through oral-oral, feco-oral, and gastro-oral (through gastric secretions, vomit, and endoscopes that are not correctly disinfected). The infection can also be transmitted from water [23].

H. pylori grow optimally at pH 6.7-7.0 and will die or not grow at pH in the gastric lumen. Gastric mucus is relatively impermeable to acids and has a strong buffering capacity. The mucus pH is low on the luminal side of the stomach (1.0-2.0), while on the epithelial side, the mucus pH is around 7.4. H. pylori are found in the interior of the mucus layer near the epithelial surface, where the physiological pH is present. H. pylori also produce proteases that alter the properties of mucus and further decrease the acidity of the

stomach to diffuse through the mucus. H. pylori have potent urease activity, which produces ammonia and further increases its acid-buffering capacity. H. pylori are relatively motile, even in mucus, and can reach the surface of the epithelial lining. H. pylori are present on the surface of gastric-type epithelial cells but not on the intestinal-type surface.

H. pylori exhibit a narrow range of organs and target organs, but infection is usually lifelong. It shows a solid adaptation to its natural habitat, the mucus layer that lines the stomach's epithelial cells. As a result, H. pylori lacks the biosynthetic pathways generally found in other bacteria, such as most enteric bacteria. It has been concluded from comparative genomic and metabolic studies that H. pylori has a metabolic route with very few redundancies and lacks biosynthetic pathways for some amino acids. As a result, H. pylori can be grown only in a chemically determined medium with arginine, histidine, isoleucine, leucine, methionine, phenylalanine, valine, and some strains also require alanine and serine. H. pylori are urease, catalase, and oxidase-positive, characteristics often used to identify H. pylori. H. pylori can catabolise glucose, and both genomic and biochemical information indicates that other sugars cannot be catabolised by H. pylori [14].

Research Method

This study is descriptive and uses a descriptive-analytic method to determine the profile of patients undergoing gastric biopsies at MRCCC Siloam Hospitals Semanggi from 2013 – 2017. This study was conducted at MRCCC Siloam Hospitals Semanggi, Plaza Semanggi, Jl. Garrison Dalam No.2-3, RT.5/RW. 6. Karet Semanggi, Setia Budi South Jakarta, DKI Jakarta 12930 from September 2018 to early November 2018. The population in this study were patients who performed biopsies at MRCCC Siloam Hospitals Semanggi in the 2013 – 2017 period, with 665 populations. The samples in this study were patients with gastric biopsies at MRCCC Siloam Hospitals Semanggi in 2013 – 2017, as many as 371 samples. The instrument of this research is the Anatomical Pathology form. Data were analysed using the Statistical Package for Social Sciences (SPSS) version 24 Program.

Result and Discussion

During the period 2013 – 2017, patients who underwent gastric endoscopic biopsy in 2017 were 68 samples (18.3%), in 2016 a total of 77 samples (20.8%), in 2015 a total of 71 samples (19.1%), in 2014 a total of 92 samples (24.8%), and in 2013 there were 63 samples (17%).

Table 1: Frequency Distribution by Year (n=371)

Variable	Frequency	%	Valid %	Cumulative %
2017	68	18.3	18.3	18.3
2016	77	20.8	20.8	39.1
2015	71	19.1	19.1	58.2
2014	92	24.8	24.8	83.0
2013	63	17.0	17.0	100.0
Total	371	100.0	100.0	

Patients who performed gastric endoscopic biopsy at MRCCC Siloam Hospitals Semanggi who were less than 30 years old were 51 patients (13.7%), between 30-39 years were 102 patients (27.5%), between 40-49 years were 117 patients (31.5%), between 50 – 59 years as many as 60 patients (16.2%), and over the age of 59 years as many as 41 (11.1%).

Table 2: Frequency Distribution by Age

Variable	Frequency	%	Valid %	Cumulative %
<30	51	13.7	13.7	13.7
30-39	102	27.5	27.5	41.2
40-49	117	31.5	31.5	72.8
50-59	60	16.2	16.2	88.9
>59	41	11.1	11.1	100.0
Total	371	100.0	100.0	

Patients who performed gastric endoscopic biopsy in 2013 – 2017 were 151 (40.7%) male patients and 220 (59.3%) female patients.

Table 3: Frequency distribution by gender (n = 371)

Variable	Frequency	%	Valid %	Cumulative %
Male	151	40.7	40.7	40.7
Female	220	59.3	59.3	100.0
Total	371	100.0	100.0	

Based on the abnormality location, during 2013 – 2017, endoscopic biopsies of the stomach were performed on the antrum in 249 (67.1%) patients, fundus in 62 (16.7%) patients, and fundus and antrum in 60 (16.2%) patients.

Table 4: Frequency distribution by location of abnormality (n = 371)

Variable	Frequency	%	Valid %	Cumulative %
Antrum	249	67.1	67.1	68.2
Fundus	62	16.7	16.7	83.8
Fundus + antrum	60	16.2	16.2	100.0
Total	371	100.0	100.0	

From 371 samples during 2013 – 2017, 32 (8.6%) patients were Helicobacter pylori-positive, and 339 (91.4%) patients were on gastric endoscopic biopsies.

Table 5: Frequency distribution by Helicobacter pylori (n = 371)

Variable	Frequency	%	Valid %	Cumulative %
Positive	32	8.6	8.6	8.6
Negative	220	91.4	91.4	100.0
Total	371	100.0	100.0	

During the period 2013 – 2017, 214 (57.7%) WARD patients (inpatient) and 157 (42.3%) Outpatient patients (outpatient) underwent gastric endoscopic biopsies.

Table 6: Distribution of WARD/Outpatient frequencies (n = 371)

Variable	Frequency	%	Valid %	Cumulative %
WARD	214	57.7	57.7	57.7
Outpatient	157	42.3	42.3	100.0
Total	371	100.0	100.0	

Based on table 7, in 2017, from 68 samples, patients who underwent gastric endoscopic biopsy were nine patients aged less than 30 years, 19 patients aged between 30 – 39 years, 17 patients aged 40 – 49 years, 17 patients aged 40 – 49 years, 12 patients aged 50 – 59 years, and 11 patients aged over 59 years. In 2016, 15 patients aged less than 30 years, 17 patients aged between 30 – 39 years, 26 patients aged between 40 – 49 years, 15 patients aged between 50 – 59 years, and four patients aged over 59 years from the total

sample a total of 77 patients. In 2015, from a total of 71 samples, 17 patients were aged less than 30 years, 19 patients aged between 30 – 39 years, 19 patients aged 40 – 49 years, six patients aged 50 – 59 years, and ten patients aged over 59 years. In 2014, from a total of 92 samples, seven patients were less than 30 years old, 25 patients were between 30 – 39 years old, 35 patients were 40 – 49 years old, 16 patients were 50 – 59 years old, and nine patients were over 59 years old. In 2013, with a total sample of 63 samples, three patients aged less than 30 years, 22 patients aged between 30-39 years, 20 patients aged 40-49 years, 11 patients aged 50-59 years, and seven patients aged over 59 years.

Table 7: Patient profile by Year and Age (n = 371)

Year	Age (Year)					Total
	<30	30 – 39	40 – 49	50 – 59	>59	
2017	9	19	17	12	11	68
2016	15	17	26	15	4	77
2015	17	19	19	6	10	71
2014	7	25	35	16	9	92
2013	3	22	20	11	7	63
Total	51	102	117	60	41	371

From table 8, regarding the distribution of gastric endoscopic biopsies by year and gender, it can be seen that in 2017, 34 male patients and 34 female patients, with a total sample of 68 people. In 2016, 40 male patients and 37 female patients, with a total sample of 77 people and in 2016, there were 24 male patients and 47 female patients, a total sample of 71 people. In 2014, a total sample of 92 patients, 31 male patients and 61 female patients, while in 2013, 63 patients, 22 male patients and 41 female patients.

Table 8: Patient profile by Year and Gender (n = 371)

Year	Gender		Total
	Male	Female	
2017	34	34	68
2016	40	37	77
2015	24	47	71
2014	31	61	92
2013	22	41	63
Total	151	220	371

Of the 68 samples in 2017 that performed gastric endoscopic biopsies, nine patients were positive for H. pylori infection, and 59 patients tested negative. In 2016, 6 patients were positive for H. pylori infection. Of the 71 patients in 2015, 5 patients were positively infected. In 2014, 5 of the 92 patients were also infected with H. pylori. In 2013, from a total sample of 63 patients, seven patients were infected with H. pylori.

Table 9: Patient profile by Year and H. pylori (n = 371)

Year	*Helicobacter pylori*		Total
	Positive	Negative	
2017	9	59	68
2016	6	71	77
2015	5	66	71
2014	5	87	92
2013	7	56	63
Total	32	339	371

From table 10, it can be seen that three patients who were positively infected were aged less than 30 years, eight positive patients were aged 30-39 years, seven positive patients were aged 40-49 years, eight positively infected patients were aged 50-59 years, and six patients who were positively infected were more than 59 years old.

Table 10: Patient profile by Age and H, pylori (n = 371)

Age	*Helicobacter pylori*		Total
	Positive	Negative	
<30	3	48	68
30-39	8	94	77
40-49	7	110	71
50-59	8	52	92
>59	6	35	63
Total	32	339	371

Based on gender and H. pylori infection, out of 151 male patients, 18 patients were positively infected with H. pylori and out of a total of 220 female patients, 14 were positively infected.

Table 11: Patient profile by Sex and H. pylori (n = 371)

Gender	*Helicobacter pylori*		Total
	Positive	Negative	
Male	18	133	151
Female	14	206	220
Total	32	339	371

From table 12, it can be seen that 17 out of 214 patients in WARD (inpatient) were positive for H. pylori infection, and 15 out of 157 Outpatient (outpatient) patients were infected with H. pylori.

Table 12: Patient profile based on WARD/Outpatient and H. pylori (n = 371)

WARD/*Outpatient*	*Helicobacter pylori*		Total
	Positive	Negative	
WARD	17	192	214
Outpatient	15	242	157
Total	32	339	371

From table 13, Helicobacter pylori were most commonly found in antrum biopsies as many as 18 out of a total of 253 patients.

Table 13: Patient profile by location of the abnormality and H. pylori (n = 371)

Abnormalities Location	*Helicobacter pylori*		Total
	Positive	Negative	
Antrum	20	229	249
Fundus	3	59	62
Fundus + antrum	9	51	60
Total	32	339	371

Table 14 shows that positive for H. pylori infection, as many as 20 patients had active atrophic chronic gastritis, 11 patients had active chronic gastritis, non-atrophic, and one patient had chronic non-active atrophic gastritis. Meanwhile, none of the patients suffering from chronic non-active non-atrophic gastritis was positive for H. pylori infection. These patients were infected with H. pylori with a total of 32 positive patients.

Table 14: Patient Profile by H. Pylori and Conclusions (n = 371)

Conclusion	*Helicobacter pylori*		Total
	Positive	Negative	
Active chronic gastritis, atrophic	20	12	32
Chronic active, non-atrophic gastritis	11	54	65
Chronic inactive, atrophic gastritis	1	88	89
Chronic non-active, non-atrophic gastritis	0	185	185
Total	32	339	371

From table 15, the most frequent diagnosis experienced by gastric endoscopic biopsy patients at MRCCC Siloam Hospitals Semanggi is chronic non-active non-atrophic gastritis as many as 185 patients, the highest occurred in 2014 as many as 60 patients. In addition, the diagnosis that often occurs is chronic non-active atrophic gastritis as many as 89 patients, and the highest occurred in 2016 as many as 24 patients, chronic active non-atrophic gastritis as many as 65 patients, the highest in 2016 which was 17 patients and the least was Chronic active non-atrophic gastritis as many as 32 patients, the highest occurred in 2017 as many as 17 patients.

Table 15: Patient Profile by Year and Conclusion (n = 371)

Conclusion	Year					Total
	2017	2016	2015	2014	2013	
Active chronic gastritis, atrophic	17	7	4	4	0	32
Chronic active, non-atrophic gastritis	7	17	14	14	13	65
Chronic inactive, atrophic gastritis	23	24	22	14	6	89
Chronic non-active, non-atrophic gastritis	21	29	31	60	44	185
Total	68	77	71	92	63	371

In table 16, the most cases of patients aged less than 30 years, 30-39 years, 40-49 years, 50-59 years, and less than 59 years were non-atrophic chronic non-active gastritis with 18, 31 patients respectively. 62, 35, 19 patients.

Table 16: Patient profile by Age and Conclusion (n = 371)

Conclusion	Age					Total
	<30	30-39	40-49	50-59	>59	
Active chronic gastritis, atrophic	8	7	8	6	3	32
Chronic active, non-atrophic gastritis	12	16	23	7	7	65
Chronic inactive, atrophic gastritis	13	28	24	12	12	89
Chronic non-active, non-atrophic gastritis	18	31	62	35	19	185
Total	51	102	117	60	41	371

The study results with the presentation of the table above show that during the period 2013 – 2017 at MRCCC Siloam Hospitals Semanggi, patients who performed gastric endoscopic biopsies by year were the most in 2014 as many as 92 (24.8%). Based on age, the most at the age of 40-49 years as many as 117 (31.5%) patients. Based on gender, the highest number was in women, namely 220 (59.3%). There were 220 (59.3%) female patients who performed gastric biopsy compared to 151 (40.7%) male patients. In the abnormality location, it was found that the most occurred in the antrum area as many as 249 (67.1%) patients.

A total of 32 (8.6%) patients were positively infected with Helicobacter pylori. The remaining 339 (91.4%) patients were declared negative. 214 (57.7%) who performed gastric endoscopic biopsy were WARD patients (inpatient), whereas 157 (42.3%) were outpatients. Based on table 7, a comparison of years and age, it was found that most patients who performed gastric endoscopic biopsy in 2014 were aged 40-49 years, namely 35 patients. In table 8, a comparison of years and sex, it can be seen that in 2014 the most of the 61 patients were female.

In table 9, in the 2013-2017 period, patients positively infected with H. pylori were 32 patients, with the highest number in 2017 being nine people. In table 10, comparing age and H. pylori infection, the results showed that the most infections occurred in 2 age groups, namely 30 – 39 years and 50 – 59 years, each of 8 patients. In table 11, more male patients were infected, namely, 18 patients, compared to female patients, with only 14 patients.

Table 12 shows that most of the patients infected with H. pylori were hospitalised, namely 17 patients, while the other 15 were outpatients. It can also be seen from table 13, the comparison of the location of the disorder and H. pylori, it was found that H. pylori most often infects the antrum, namely as many as 24 patients. Data from table 14 found that most patients who were positively infected with H. pylori suffered from chronic active atrophic gastritis, as many as 20 patients. Table 15, which is a comparison between years and conclusions, the highest number is 60 patients in 2014 experiencing chronic non-active non-atrophic gastritis. Table 16 also shows that chronic non-atrophic non-active gastritis most often occurs in the 40-49 year age group. Based on the epidemiology of gastritis and H. pylori infection, both of which can occur from an early age, at MRCCC Siloam Hospitals Semanggi, the earliest cases of gastritis occurred in a male aged 14 years. However, the infection may increase with age because the ageing process makes the body more susceptible to infection [24, 25]. Risk factors and transmission of H. pylori are also significant and should be monitored, especially in people who live in slums, eat unhygienic food, or live with people infected with H. pylori. H. pylori are often found in the gastric antrum area because there are differences in histological structure compared to other parts [26].

Conclusion

Based on the study results, the following conclusions can be drawn: a) The stomach has a biological function that can protect the stomach from foreign microorganisms that enter the digestive system by secreting gastric acid; b) Helicobacter pylori has two types, gastric and intragastric and H. pylori more commonly infects the gastric mucosa

and epithelium because it is a good site for colonisation; c) H. pylori produces urease which can change the gastric pH from acidic to neutral pH which is the optimal means for growth; d) The results of gastric endoscopic biopsy at MRCCC Siloam Hospitals Semanggi showed that the most patients underwent biopsies in 2014 as many as 92 (24.8%) patients. Based on age, 40-49 years were the age group with the most gastric biopsies, with 117 samples (31.5%). Most of the patients were female, 220 (59.3%) patients; e) Based on the location and infection of H. pylori, the biopsy was most often performed in the antrum, as many as 253 (68.2%) patients. The most positive found in the antrum is 20 patients. The patient's biopsy results also showed that 8.6% were positive for H. pylori infection, and another 91.4% were negative. H. pylori are the most common cause of gastritis, especially chronic active atrophic gastritis as many as 20 of 32 positive patients; and f) Most of the patients who underwent gastric biopsy had the most infections, namely chronic non-active non-atrophic gastritis, which was 185 patients.

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