Detect The Most Important Changes That Occur in The Stomach Tissue of Patients Infected With H. Pylori

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KEYWORDS
H. pylori; stomach. Necrosis; ulcer.

ABSTRACT
The current study aimed to detect the most important changes that occur in the stomach tissue of patients infected with H. pylori. Samples were collected from the Endoscope Unit for the period from 12/14/2022 to 1/30/2024 at Azadi Teaching Hospital and Al-Watan Al-Ahli Hospital in Kirkuk Governorate, from both sexes for the age group (14-57) years. Stool samples were taken from the same patients those undergoing laparoscopy for the purpose of investigating the presence of Helicobacter pylori. Tissue biopsy samples were taken by the gastroenterologist using laparoscopic forceps, and (88) samples were obtained from these patients, with two tissue biopsies from each patient. The histological results of people infected with Helicobacter pylori bacteria showed many histological changes, including infiltration of inflammatory cells, which was accompanied by multiple tissue necrosis, and damage to some gastric glands as a result of degeneration of glandular epithelial cells with thickening of the basal membrane, in addition to necrosis and damage to most of the gastric glands. Narrowing of the lumen of some glands, sometimes their complete atrophy, metaplasia of the epithelial cells of the gastric glands and the epithelial tissue lining the antral area, and cell shedding occur. It is concluded from the current study that H. pylori has led to many tissue lesions in the stomach of patients, the most important of which is stomach ulceration.

1. Introduction

H. pylori are Gram-negative bacilli that move using unipolar sheathed flagella. These bacteria are characterized by the phenomenon of polymorphism, a curved or spiral shape found in tissue biopsy samples and in the cultures in which these bacteria were first isolated. This shape is characterized by the presence of one to three curves when viewed inside the body of the organism (in vivo). [1]. After the year 2000, the pattern of spread of Helicobacter pylori infection witnessed significant changes thanks to developments in health infrastructure and eradication techniques. During this period, infection rates in European countries declined significantly, while rates continued at their original pace in Asian countries. On the Asian continent, some interesting trends have emerged; Pakistan and India recorded the highest prevalence rates of Helicobacter pylori, reaching rates of 81% and 63.5% respectively. In West Asia, Turkey was characterized by the highest prevalence rate, reaching 77.2% [2]. Many old and recent studies address possible routes of transmission of H. pylori between individuals. These transmissions occur either horizontally, which is the transmission of infection within family members, or vertically, which is the transmission of infection through contact with individuals outside the family or through exposure to environmental pollutants, undercooked foods, and the use of contaminated water that is not suitable for drinking. Common methods that have been studied in studies include ancient and modern by researchers [3-7]. Infection with the bacteria known as H. pylori causes gastritis, whether acute or chronic. Acute gastritis occurs as a result of the colonization of these bacteria in the mucus layer of the stomach wall. When bacteria are exposed to this environment, the immune system responds by amassing large amounts of immune white cells. This acute inflammatory response leads to a reduction in the level of stomach acidity (Hypochlohydira) as a result of the acute inflammatory response [8]. H. pylori bacteria are classified in the first category of carcinogens according to the World Health Organization (WHO) classification. Studies have shown the existence of two mechanisms that stimulate these bacteria to contribute to the development of stomach cancer [9-10]. The current study aimed to identify the most

important changes that occur in the stomach tissue of patients infected with H. pylori.

2. Materials and Methods

Samples Sample collection

Samples were collected from the Endoscope Unit for the period from 12/14/2022 to 1/30/2024 at Azadi Teaching Hospital and Al-Watan Al-Ahli Hospital in Kirkuk Governorate, from both sexes for the age group (14-57) years. Stool samples were taken from the same patients Those undergoing laparoscopy for the purpose of investigating the presence of Helicobacter pylori, as well as blood samples for the purpose of performing a complete blood count (CBC) analysis.

H. pylori detection tests

Invasive Method tests

Which relies on upper gastrointestinal endoscopy to take gastric biopsies for histopathological tests, bacterial culture test, rapid urease test, and the polymerase chain reaction (PCR) method, but one of the disadvantages of these methods is that they require endoscopic examination to obtain a biopsy sample and are therefore difficult to use in epidemiological studies.

Non-invasive tests

It does not require upper gastrointestinal endoscopy and relies on external samples such as breath (Urea breath test), blood (serological tests), stool antigen test (SAT), and saliva and urine samples to detect IgM, IgG, and IgA antibodies (Megraud et al.,2014; Khoder et al 2019).

Histology processing

Stomach biopsies were collected from patients and fixed by using 10% formalin, processed by paraffin method, cut at six micrometers in thickness by using rotary microtome and stained with histological stains called Hematoxylin and Eosin (H&E) [11-13]. Sections were examined by using Optica Microscope (Italy).

3. Results and Discussion

Tissue biopsy samples were taken by the gastroenterologist using laparoscopic forceps, and (88) samples were obtained from these patients, with two tissue biopsies from each patient. Males were 38% (17) and females were 62% (27). Their ages ranged from (14-57) years. As for the control group, it included (44) healthy individuals and those negative for Helicobacter pylori.

The results of the current study show the normal appearance of the stomach antrum (Stomach Antrum) for the healthy control group of the first age group, as the normal histological structure of the mucous layer (Mucosa) is observed, which is damaged by the surface epithelium, which are columnar cells that secrete mucus that protects the stomach lining from acid. Hydrochloric acid and digestive enzymes. It is also noted that there are gastric glands in the lamina propria layer, which are arranged regularly within the tissue, as in the pictures (1).

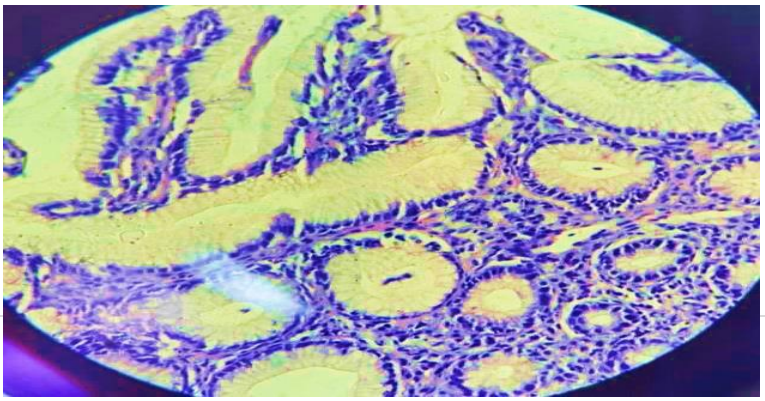
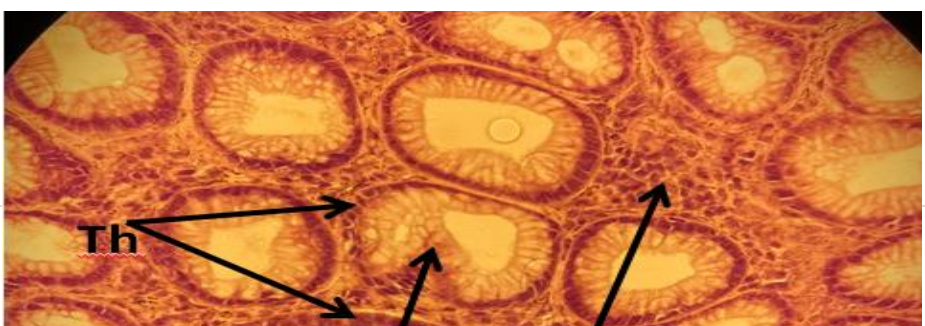


Figure (1): A cross-section of the stomach tissue of a healthy woman, showing the simple columnar

The results of the histological examination of a group of individuals infected with Helicobacter pylori in the first age group showed the appearance of many histological changes, including infiltration of inflammatory cells (In) and their sometimes accumulation in certain areas of the tissue, and damage to some gastric glands as a result of degeneration of glandular epithelial cells (D). With thickening of the basal membrane (Th), as in figure (2). In addition to necrosis (N) and damage to most of the gastric glands, as in figure (3). These changes were accompanied by narrowing of the lumen of some glands and sometimes their complete atrophy, as in the picture (4), and the metaplasia (M) of the epithelial cells of the gastric glands and the epithelial tissue lining the stomach antrum, as in figure (5). The infiltration of inflammatory cells was accompanied by multiple necrosis in the tissue as in figure (6). The results of the current study are consistent with [8], who, during his study of people infected with Helicobacter pylori bacteria, showed the occurrence of multiple tissue necrosis and peptic ulcers as a result of infection, and showed that the severity of tissue damage depends on the virulence of the infected strain and the genetic susceptibility of the host, in addition to environmental factors. While the figure (7) showed a general infiltration of inflammatory cells with degeneration of the glandular epithelial cells and thickening of their basement membrane, which caused narrowing of the lumen of some glands and disruption of the interface of others, in addition to the presence of multiple necrosis in different areas of the tissue. Infection with Helicobacter pylori led to necrosis of most gastric glands, in addition to degeneration of their epithelial cells, as in figure (8). The results of the current study agree with [14], who showed that non-infection with Helicobacter pylori bacteria is the cause. The main cause of abnormal histological changes in the mucosal surface epithelium (Mucosa), is represented by atrophic gastritis (AG), which was identified when the amount of gastric glands decreased or separated on a large scale when compared with the control group, and abnormal histological transformation of epithelial cells (Metaplasia). M) Damage to the mucous membrane with the infiltration of inflammatory cells, which causes the destruction of the protective layer of mucus and the occurrence of chronic inflammation. This leads to a reduction in acidic and enzymatic gastric secretions, which encourages bacteria to multiply significantly, weakens the digestive process and increases the susceptibility to the ulcer caused by the infection With Helicobacter pylori bacteria, chronic inflammation occurred in all members of the first group under study. In this respect, the results of the current study agreed with Dehesa et al., [15], who showed during his study that all people who had an infection with Helicobacter pylori bacteria were accompanied by the occurrence of chronic infectious inflammation, which It was detected through the general infiltration of inflammatory cells, which were lymphocytes and plasma cells in addition to neutrophils, which adds evidence to the prevailing belief that infection with Helicobacter pylori is the main cause of the tissue lesion. The study also showed an association with Helicobacter pylori with the occurrence of infectious metaplasia in (50%) of the cases that were studied, and this is consistent with the results of our current study, which shows the occurrence of abnormal changes to the histological structure in people infected with the bacteria when compared with the control group.



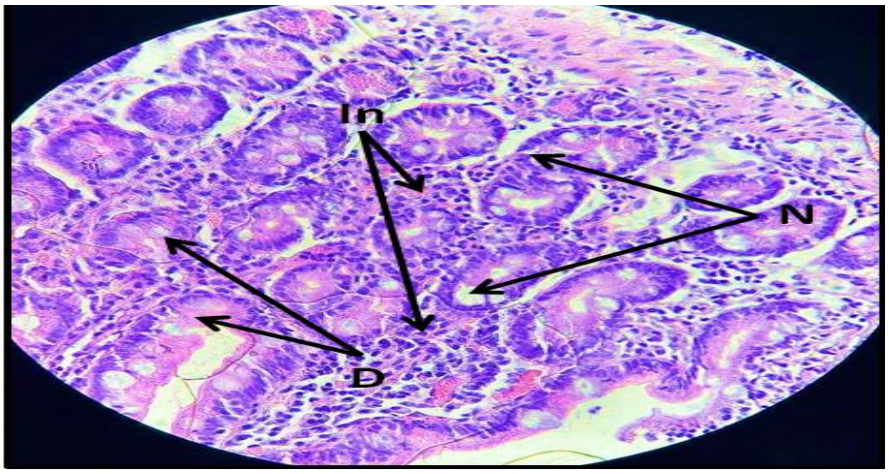


Figure (3): shows a cross-section of the stomach tissue of an infected woman, showing infiltration of inflammatory cells (In), degeneration of epithelial cells (D) of most of the gastric glands, narrowing of their lumen, and necrosis (N) (H&EX400).

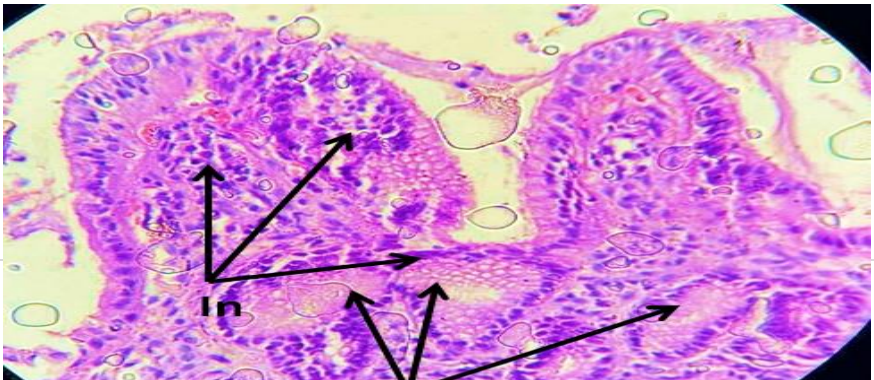


Figure (5): A cross-section of the stomach tissue of an injured man showing necrosis of most of the gastric glands (N), narrowing of the lumen, and infiltration of inflammatory cells (In) (H&EX400).

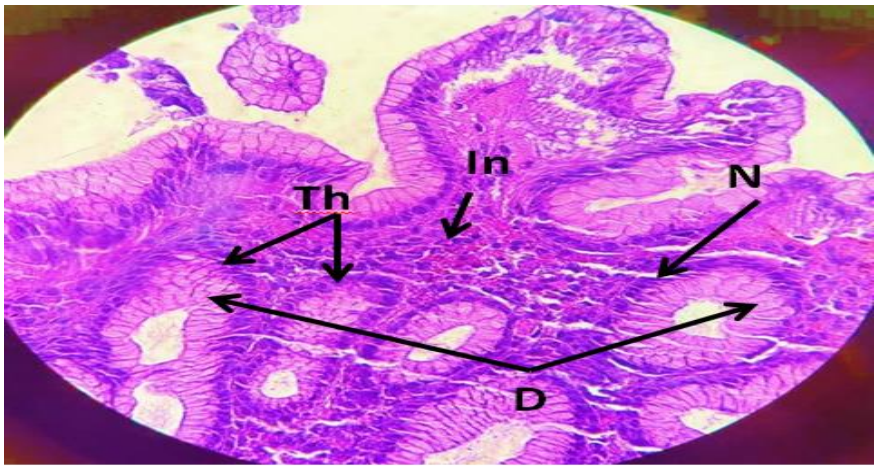


Figure (6): A cross-section of a man's stomach tissue showing inflammatory cell infiltration (In), thickening of the basement membrane (Th) and degeneration of epithelial cells (D) of some necrotic glands (N) (H&EX400).

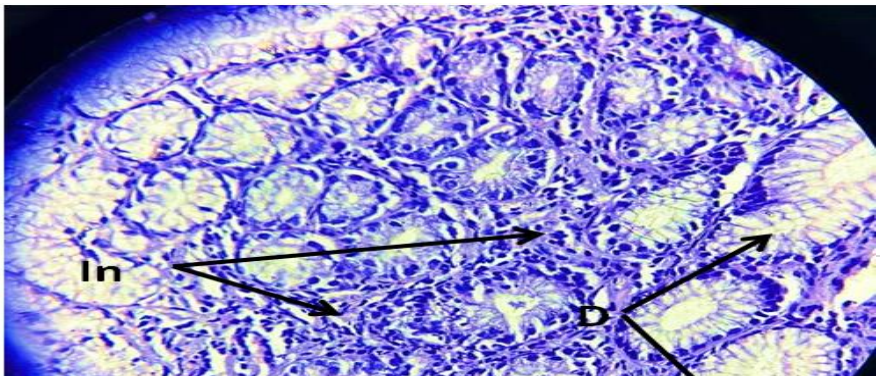


Figure (7): A cross-section of an infected woman's stomach tissue showing infiltration and accumulation of inflammatory cells (In) with degeneration of epithelial cells of some gastric glands (H&EX400).

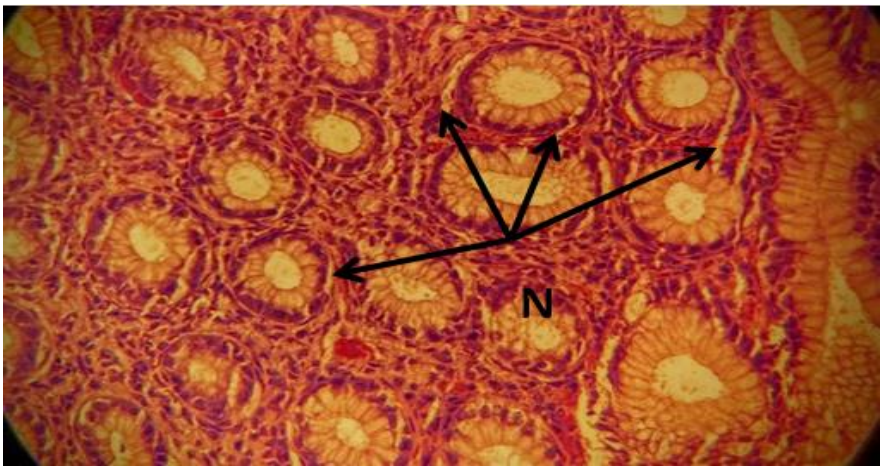


Figure (7): A cross-section of the stomach tissue of an infected woman showing multiple necrosis of most of the gastric glands (N) (H&EX400).

4. Conclusions

The results of the current study, after conducting a complete blood analysis of all four individuals in the study groups using the CBC machine, which counts blood cells, showed a significant decrease in the red blood cell count, hemoglobin concentration, and the size of agglutinated blood cells, as well as the platelet count for the same age groups compared with the same age groups for the groups. Proper control, which demonstrated a strong association between Helicobacter pylori infection and anemia represented by a decrease in the number of red blood cells, hemoglobin concentration, and platelet count.

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