


Original Research Article

# A study of changes in stomach wall at sites other than the ulcer in chronic duodenal ulcer patients

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## Abstract

**Background:** A duodenal ulcer is usually caused by an infection with a germ (bacterium) called *Helicobacter pylori* (*H. pylori*). A 4- to 8-week course of acid-suppressing medication will allow the ulcer to heal.

**Aim:** To study changes in stomach wall at sites other than the ulcer in chronic duodenal ulcer patients.

**Materials and methods:** This study was conducted in 150 patients in the department of surgery in Government General Hospital/ Government Medical College, Medical College, Nizamabad, during the period of July 2014 to May 2016.

**Results:** In the present study, out of 150 patients in this study, 90 were males and 60 were females. Age ranged from 23 to 82 years old. In endoscopy findings, chronic gastritis of antrum was found to be 138 (92%), gastric ulcer was detected in 7% cases i.e. in 11 patients. The pangastritis rate was 65% and the inflammation was caused due to erythematous gastritis. In histopathology findings, chronic gastritis of antrum was found in 143 cases which constituted 95%. The pangastritis rate was 85%, common gastritis of antrum was atrophic type which constituted 80% of the patients, common gastritis of fundus was superficial type which was in 62% of patients, and intestinal metaplasia of stomach was seen in 5% of patients. From biopsy, helicobacter Pyroli in gastric antrum infection rate was 85%, Helicobacter Pyroli in gastric fundus infection rate was 40% and Helicobacter Pyroli in corpus of stomach infection rate was 5%.

**Conclusion:** Chronic gastritis is caused by presence of *Helicobacter pylori*. The most common site for *Helicobacter pylori* is the gastric antrum followed by fundus. In Chronic duodenal ulcers cases, the

most common pathological abnormality is chronic superficial atrophic gastritis of antrum followed by pangastritis.

## Key words

Stomach wall, Changes, Duodenal ulcer, Chronic.

## Introduction

A duodenal ulcer is usually caused by an infection with a germ (bacterium) called *Helicobacter pylori* (*H. pylori*). A 4- to 8-week course of acid-suppressing medication will allow the ulcer to heal [1]. In addition, a one-week course of two antibiotics plus an acid-suppressing medicine will usually clear the *H. pylori* infection. This usually prevents the ulcer from coming back. Anti-inflammatory medicines used to treat conditions such as arthritis sometimes cause duodenal ulcers [2]. If you need to continue with the anti-inflammatory medicine then you may need to take long-term acid-suppressing medication. Your stomach normally produces acid to help with the digestion of food and to kill germs (bacteria). This acid is corrosive so some cells on the inside lining of the stomach and the first part of the small intestine (the duodenum) produce a natural mucous barrier [3]. This protects the lining of the stomach and duodenum. There is normally a balance between the amount of acid that you make and the mucous defence barrier. An ulcer may develop if there is an alteration in this balance, allowing the acid to damage the lining of the stomach or duodenum. Infection with *H. pylori* is the cause in about 19 in 20 cases of duodenal ulcer. More than a quarter of people in the UK become infected with *H. pylori* at some stage in their lives. Once you are infected, unless treated, the infection usually stays for the rest of your life [4]. In many people it causes no problems and a number of these bacteria just live harmlessly in the lining of the stomach and duodenum. However, in some people this bacterium causes an inflammation in the lining of the stomach or duodenum [5]. This causes the defence mucous barrier to be disrupted (and in some cases the amount of acid to be increased) which allows the acid to cause inflammation and ulcers. Studies

have been conducted which eradicated of helicobacter pylori, which finally heals the peptic ulcer diseases and reduces recurrence rates of ulcers.

## Materials and methods

This study was conducted in the Department of Surgery in Nizam's Medical College, Hyderabad during the period of July 2014 to May 2016. 150 patients were selected who were having symptoms of chronic duodenal ulcers for at least 1 year and who had undergone examination of upper gastro-intestinal endoscopy. Gastric fundus, antrum and body biopsy was taken and it was investigated in histopathology laboratory and rapid urease test was done separately.

## Results

Out of 150 patients in this study, 90 were males and 60 were females. Age ranged from 23 to 82 years old. The highest number of patients was in the age range of 40-50 years (**Table – 1**).

**Table - 1:** Demographic distribution.

Sex Distribution	Males	Females
Total 150	90 (60%)	60 (40%)
Age distribution		
Age in years	No of patients	Percentage (%)
0-10	0	0
10-20	0	0
20-30	7	5
30-40	38	25
40-50	72	48
50-60	27	18
>60	6	4

Chronic gastritis of antrum was found to be 138 (92%), gastric ulcer was detected in 7% cases i.e.

in 11 patients. The pangastritis rate was 65% and the inflammation was caused due to erythematous gastritis (**Table – 2**).

**Table - 2:** Endoscopic findings.

<b>Endoscopy Findings</b>	
Chronic Gastritis of antrum	138 (92%)
Gastric ulcer	11 (7%)
Pangastritis rate	65%

Chronic gastritis of antrum was found in 143 cases which constituted 95%. The pangastritis rate was 85%, Common gastritis of antrum was atrophic type which constituted 80% of the patients, common gastritis of fundus was superficial type which was in 62% of patients, intestinal metaplasia of stomach was seen in 5% of patients (**Table – 3**).

**Table - 3:** Histological findings.

<b>Histological Findings</b>	
Chronic Gastritis of antrum	143 (95%)
Pangastritis rate	85%
Common gastritis of antrum	Atrophic type (80%)
Common gastritis of fundus	Superficial type (62%)
Intestinal metaplasia of stomach	5%

**Table - 4:** Biopsy findings.

<b>Biopsy Findings</b>	
Helicobacter Pyroli in gastric antrum	85%
Helicobacter Pyroli in gastric fundus	40%
Helicobacter Pyroli in corpus of stomach	5%

Biopsy was taken from different parts of stomach and this showed that mostly it was superficial inflammation of mucosa and sub-mucosa which contained mostly of plasma cells and lymphocytes. Helicobacter Pyroli in gastric antrum infection rate was 85%, Helicobacter Pyroli in gastric fundus infection rate was 40% and Helicobacter Pyroli in corpus of stomach

infection rate was 5%. All of these showed active inflammation (**Table – 4**).

## **Discussion**

In the present study, out of 150 patients in this study, 90 were males and 60 were females. Age ranged from 23 to 82 years old. In endoscopy findings, chronic gastritis of antrum was found to be 138 (92%), gastric ulcer was detected in 7% cases i.e. in 11 patients. The pangastritis rate was 65% and the inflammation was caused due to erythematous gastritis. In histopathology findings, chronic gastritis of antrum was found in 143 cases which constituted 95%. The pangastritis rate was 85%, common gastritis of antrum was atrophic type which constituted 80% of the patients, common gastritis of fundus was superficial type which was in 62% of patients, and intestinal metaplasia of stomach was seen in 5% of patients. From biopsy, helicobacter Pyroli in gastric antrum infection rate was 85%, Helicobacter Pyroli in gastric fundus infection rate was 40% and Helicobacter Pyroli in corpus of stomach infection rate was 5%. All of these showed active inflammation. Various researchers have shown similar results which were similar to the present study. Jagmohan Mishra, et al [6]; conducted a study in which subjects were patients with chronic duodenal ulcer who underwent endoscopic examination, gastric biopsy and rapid urease test. Chronic gastritis of antrum, followed by erythematous pangastritis was the prominent feature both in endoscopy and histopathological examination. The Inflammatory change affected the mucosa and submucosa of the stomach wall. The prevalence rate of Helicobacter pylori was 84%, the antrum being the most common affected part (84%) followed by gastric fundus (41%). Chronic superficial atrophic gastritis of antrum, followed by pangastritis is the most common pathological abnormality in stomach wall in CDU cases. Gastric antrum is the most common site for H. pylori colonization followed by fundus. Presence of H. pylori in stomach wall is associated with active on chronic gastritis. Dr. M. Bhooma Reddy, Dr. Awais Ghori, conducted a prospective study which reported that chronic

gastritis of antrum, followed by erythematous pangastritis was the prominent feature both in endoscopy and histopathological examination. The inflammatory change affected the mucosa and submucosa of the stomach wall. The prevalence rate of *Helicobacter pylori* was 84%, the antrum being the most common affected part (84%) followed by gastric fundus (41%). Chronic superficial atrophic gastritis of antrum, followed by pangastritis is the most common pathological abnormality in stomach wall in CDU cases. Gastric antrum is the most common site for *H. pylori* colonization followed by fundus. Presence of *H. pylori* in stomach wall is associated with active on chronic gastritis. Vu C, et al. [8]; observed that chronic gastritis of antrum, followed by erythematous pangastritis was the prominent feature both in endoscopy and histopathological examination. The inflammatory change affected the mucosa and submucosa of the stomach wall. The prevalence rate of *Helicobacter pylori* was 84%, the antrum being the most common affected part (84%) followed by gastric fundus (41%). Chronic superficial atrophic gastritis of antrum, followed by pangastritis is the most common pathological abnormality in stomach wall in CDU cases. Gastric antrum is the most common site for *H. pylori* colonization followed by fundus. Presence of *H. pylori* in stomach wall is associated with active on chronic gastritis. Sirinthornpunya S [9] conducted a retrospective study which showed that five hundred and seventy patients with a mean age of 55.0 +/- 16.02 years with peptic diseases were studied. Endoscopic findings showed 106 GU patients (18.6%), 29 DU patients (5.1%), 3 combined GU and DU patients (0.5%) and 432 NUD patients (75.8%). The prevalence of *H. pylori* infection was 64% (365 of 570 patients). Prevalence of *H. pylori* infection were 61.3% of NUD cases, 68.9% of GU cases, 82.8% in DU cases and 100% in combined GU and DU cases. Nguyen TL, et al. [10]; reported that among the examined patients, 65.6% were infected with *H. pylori*. The prevalence of infection was significantly higher in those over 40 years of age than in those aged  $\leq 40$ . Chronic gastritis was present in all *H. pylori*-infected

individuals, 83.1% of whom had active gastritis, and 85.3% and 14.7% had atrophy and intestinal metaplasia, respectively. PU was present in 21% of infected patients, whereas its incidence was very low in non-infected individuals. The prevalence of PU was significantly higher in Hanoi than in Ho Chi Minh. The prevalence of *vacA m1*, which has been identified as an independent risk factor for PU in Vietnam, was significantly higher among *H. pylori* isolates from Hanoi than among those from Ho Chi Minh. This study by Nguyen TL et al concluded that *H. pylori* infection is common in Vietnam and is strongly associated with PU, active gastritis, atrophy and intestinal metaplasia. *vacA m1* is associated with an increased risk for PU and might contribute to the difference in the prevalence of PU and gastric cancer between Hanoi and Ho Chi Minh.

## Conclusion

Chronic gastritis is caused by presence of *Helicobacter pylori*. The most common site for *Helicobacter pylori* is the gastric antrum followed by fundus. In Chronic duodenal ulcers cases, the most common pathological abnormality is chronic superficial atrophic gastritis of antrum followed by pangastritis.

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