SAS Journal of Medicine SAS J. Med., Volume-2; Issue-3 (May-Jun, 2016); p-49-54 Available online at http://sassociety.com/sasjm/

Original Research Article

# Prevalence of Helicobacter Pylori Infection and its Correlation with Complete Blood Count Parameters in Adult Males at Taif City, Saudi Arabia

Waleed Samy<sup>1,2</sup>, Gamal M. Elnemr<sup>2,3</sup>, Lotfi F. Issa<sup>4,5</sup>, Wael Sedik<sup>6,7</sup>
<sup>1</sup>Internal Medicine Department, Tanta Faculty of Medicine, Tanta University, Egypt
<sup>2</sup>Internal Medicine Department, College of Medicine, Taif University, KSA
<sup>3</sup>Medical and Radiological Researches Department, Nuclear Materials Authority, Egypt
<sup>4</sup>Public Health and Community Medicine Department, Faculty of Medicine, Al-Azhar University, Egypt
<sup>5</sup>Community Medicine Department, College of Medicine, Taif University, KSA
<sup>6</sup>Medical Biochemistry Department, Faculty of Medicine, Minia University, Egypt
<sup>7</sup>Medical Biochemistry Department, College of Medicine, Taif University, KSA

#### \*Corresponding author

Waleed Samy Email: <u>wsmohamed1@yahoo.com</u>

**Abstract:** Helicobacter pylori (H. pylori) infection is one of the most important human pathogens infecting more than 50% of the world's population. It is a major cause of various upper gastrointestinal disorders and can lead to intestinal and extra-intestinal manifestations. The aim of the study was to determine the prevalence of H. pylori infection and its correlation with complete blood count (CBC) in adult males at Taif city, KSA. A cross-sectional study was conducted on three hundred and nine adult male volunteers aging 20-25 years after their initial permission. CBC in addition to qualitative detection of IgG antibodies against H. pylori organisms was investigated. The three hundred and nine studied individuals were classified according to their hemoglobin concentration (Hb Conc.) and packed cell volume percent (PCV%) into 2 groups; group I that had normal Hb Conc. and PCV% (202 individuals "65.4 %") and group II that had increased Hb Conc. and/or PCV% (107 individuals "34.6 %"). Among participants there were 110 (35.6%) individuals with positive H. pylori antibody with 73 (36.1%) in group I compared to 37 (34.6%) in group II, with no statistical significant difference between both groups (P=0.8). The Prevalence of H. pylori in Taif city is relatively high when compared to other cities in the western region of Saudi Arabia. Hypoxia doesn't cause increase in H. pylori prevalence among Taif city residents with elevated Hb Conc. A confirmatory test to the positive H. pylori cases is needed. In addition, health education program and following sanitary measures are highly recommended for students and their community.

Keywords: Adult Male,CBC, H. pylori, Antibody, Taif City, Saudi Arabia.

#### **INTRODUCTION**

H. pylori are a helix shaped, microaerophilic, Gram-negative, flagellated bacteria. It is well known to be the most common human infection worldwide on the basis of the fact that approximately 50% of the world's populations harbor the organism in their upper gastrointestinal tract and that human beings are the main reservoir [1]. Infection with H. pylori is usually acquired in the early childhood and persists for life [2]. While over 80% of the infected individuals are asymptomatic [3], it induces gastric mucosal inflammation, which may result in a number of gastroduodenal diseases ranging from mild gastritis, atrophic gastritis, and peptic ulcer disease to malignant diseases such as gastric adenocarcinoma and Mucosa-Associated Lymphoid Tissue (MALT) Lymphoma. H. pylori has been classified as a class I carcinogen for gastric cancer since 1994 by the International Agency for Research on Cancer (IARC/OMS) [4]. The mode of H. pylori transmission is unknown, but it is thought to be mainly through the fecal-oral route.

Oral-oral, water-borne transmissions, or poorly disinfected endoscopes are other modes. Although immune cells that normally recognize and attack invading bacteria accumulate near sites of H. pylori infection, they are unable to reach the stomach lining[5]. In addition, H. pylori have developed ways of interfering with local immune responses, making them ineffective in eliminating this bacterium [6]. It has been shown that H. pylori-positive patients tend to have dyspepsia, but the relationship between H. pylori and dyspepsia remains controversial [7]. H. pylori bacterium is a spiral organism that lies in the interface between gastric epithelial cell surface and the overlying mucus gel. A variety of host and bacterial factors contribute to the pathogenesis of gastrointestinal diseases resulting from H. pylori infection. H. pylori organisms are intensely antigenic and secrete various factors like urease, catalase, mucinase, lipase, hemolysin, and alkaline phosphatase that decrease viscosity of the mucus. The production of catalase protects the bacteria against the toxic effects of reactive oxygen metabolites formed in neutrophils from hydrogen peroxide. The multiple polar flagella allow them to penetrate the mucous layer. Adherence of H. pylori to gastric epithelial cells and vacuolating cytotoxin were associated with degenerative changes in the epithelial cells [8].

H. pylori infection is widespread throughout the world. Its prevalence is highly variable in relation to geography, ethnicity, age, and socioeconomic factors. Studies point toward a relation between the low socioeconomic status and the high rate of H. pylori infection. Infection was more prevalent in developing countries, and incidence decreased in Western countries [9]. In developing countries, the prevalence of H. pylori ranges between 70%-90% [6], while in developed countries it is approximately 50%. However, detailed information on the prevalence of the bacteria in developing countries and on the factors that may influence the pattern of distribution remains scanty [10]. H. pylori prevalence in industrialized countries is slowly increasing during childhood, which continues through adolescence and early adulthood, with an abrupt increase around 50-60 years of age [11]. In nonindustrialized countries, H. pylori prevalence increases more rapidly during childhood and most adolescents and adults are infected. Thus, differences in H. pylori prevalence between industrialized and nonindustrialized countries are greater at younger ages and get smaller at older ages [12].

It has been reported that infection in Saudi Arabia is acquired at an early age and reaches up to 36.9% as age advances [13]. Differences in prevalence within populations are due to a variety of factors primarily relating to socioeconomic status and geographic origin [14]. H. pylori infection has been reported to be hyper-endemic in Saudi Arabia. Reports in the 1990s have shown a prevalence of 68-82.2% [15] in various age groups of patients including those with non-ulcer dyspepsia. However, reports in the 2000s have shown marked reduction in the prevalence to 35-55% [16]. There have been few reports from different parts of Saudi Arabia on H. pylori infection in patients with gastrointestinal diseases, but little information is available on the seroprevalence of H. pylori in healthy asymptomatic population. Taif city is located in the Western region of Saudi Arabia. It is a high altitude area that it is more than 2400 meters above the sea level [17]. So, hypoxic condition in Taif city is a suitable site for growth and multiplication of H. pylori which is a microaerophilic organism. Hence, the aim of this study was to determine the prevalence of H. pylori infection among adult males at Taif city in the age range 20-25 years (by positive serum antibody test) and to compare the prevalence of the disease in Taif city with other Saudi governorates.

#### **SUBJECTS and METHODS**

This is a cross-sectional study that was conducted on adult males at Taif City, Saudi Arabia. Three hundred and nine (309) healthy adult male volunteers aging 20-25 years were included in the study.

#### Inclusion criteria:

All persons involved in the study must be; 1. Born and living in Taif city.

2. In good health, with no symptoms referable to upper gastrointestinal tract.

#### Exclusion criteria:

Subjects were excluded if they had;

1. History of H. pylori infection or treatment.

2. History of peptic ulcer or frequent upper gastrointestinal tract symptoms.

3. History of regular use of antacids or antibiotics.

All volunteers were subjected to the laboratory analyses that were done as soon as blood samples drown and prepared without preservation or delay. Three milliliters (mL) of venous blood were collected from each volunteer with a 19 gauge vacutainer tubes for;

#### 1. Complete blood count analysis:

One mL of blood was collected in EDTA anticoagulation vacutainer tubes, mixed gently by hand inversion, analyzed by the Sysmex KX-21N automated hematology analyzer, using Sysmex hematology reagent. This analyzer was calibrated and controlled with a standard laboratory quality control method provided from Sysmex Corporation.

Subjects were classified into two groups based on their Hb Conc. and PCV% [18]:

a. Group I: with normal Hb Conc. and PCV% (i.e., with Hb Conc. 13-17 g/dL and PCV 40-50%).

b. Group II: with high Hb Conc. and/or PCV% (i.e., with Hb Conc. >17 g/dL or PCV >50%).

# 2. Detection of H. pylori antibodies:

Two mL of blood were collected in plain vacutainer tubes, without anticoagulation, left in the incubator at 37°C for 30 minutes to clot, centrifuged at 3000 rpm (revolutions per minute) for another 15 minutes, and then the supernatant sera were used, without preservation or delay, for the qualitative detection of antibodies against H. pylori organisms using a rapid one step chromatographic immunoassay method "from Abon Biopharm (Hangzhou) Co., Ltd, China" which utilizes a combination of H. pylori antigen coated particles and anti-human IgG to qualitatively and selectively detect H. pylori antibodies in the serum.

#### Statistical analyses:

Values were expressed as mean  $\pm$  SD. Statistical analysis of the results was performed based on the conventional standard statistical procedures using computed statistical analysis by SPSS, version 22.0 for Microsoft windows 7. Percentage was used to determine the prevalence rates of H. pylori. Unpaired-samples "t" test was applied to compare between parametric values; Pearson's correlation with correlation coefficient was applied for parametric results. The significant difference was considered at p<0.05.

## **Ethical considerations:**

Each studied subject was informed about the study objectives with stressing from our team on confidentiality of the collected data and sample results,

and also on getting a verbal consent to share in the study.

#### RESULTS

Table 1 revealed blood parameters of all studied subjects, where; red blood cells (RBCs) count was 5.7  $\pm$  3.38  $\times$  10<sup>6</sup>/µL, hemoglobin (Hb) concentration was  $16.7 \pm 0.95$  g/dL, packed cell volume (PCV) was 47.2 ± 2.4%, mean corpuscular volume (MCV) was 83.1 ± 3.8 fL, mean corpuscular hemoglobin (MCH) was 29.5 ± 1.8 pg, mean corpuscular hemoglobin concentration (MCHC) was  $35.4 \pm 1.2$  g/dL, red cell distribution width (RDW) was 13.2  $\pm$  0.8%, white blood cells (WBCs) count was 5.8  $\pm$  $1.7 \times 10^{3}/\mu$ L, neutrophils (Neut) count was  $3.1 \pm 1.4 \times$  $10^{3}/\mu$ L, lymphocytes (Lym) count was 2.5 ± 0.6 ×  $10^{3}/\mu$ L, content of the Mixture count (MXD = monocyte, eosinophils and basophils) was 0.3  $\pm$  0.2  $\times$  $10^{3}$ /µL, and platelet (Plt) count was 192.4 ± 69.6 ×  $10^{3}/\mu$ L.

| Table 1. Dioou parameters in an studied subjects |       |      |  |
|--|-------|------|--|
| Variables  | Mean  | SD   |  |
| RBCs (×10 <sup>6</sup> / $\mu$ L)                | 5.7   | 3.38 |  |
| Hb (g/dL)  | 16.7  | 0.95 |  |
| PCV (%)  | 47.2  | 2.4  |  |
| MCV (fL)   | 83.1  | 3.8  |  |
| MCH (pg)   | 29.5  | 1.8  |  |
| MCHC (g/dL)                                      | 35.4  | 1.2  |  |
| RDW (%)  | 13.2  | 0.8  |  |
| WBCs ( $\times 10^3/\mu$ L)                      | 5.8   | 1.7  |  |
| Neutrophils ( $\times 10^3/\mu$ L)               | 3.1   | 1.4  |  |
| Lymphocytes) ( $\times 10^3/\mu$ L)              | 2.5   | 0.6  |  |
| Mixture ( $\times 10^3/\mu$ L)                   | 0.3   | 0.2  |  |
| Platelets ( $\times 10^3/\mu$ L)                 | 192.4 | 69.6 |  |

| Table 1: Blood | parameters in a | ll studied | subjects |
|----------------|-----------------|------------|----------|
|                |                 |            |          |

Table 2 revealed comparison between blood parameters of both groups. There was a significant difference between both groups regarding; RBCs (p<0.0001), Hb Conc. (p<0.0001), PCV% (p<0.0001), MCV (p=0.048), MCH (p<0.0001), MCHC (p<0.0001), WBCs (P=0.0065), and Neut (P=0.0217), with no significant difference between both groups regarding RDW% (P=0.2312), Lym (P=0.1083), MXD (P=0.9999), and Plt (P=0.0673).

| Variables                         | Group I<br>(N=202) | Group II<br>(N=107) | P-value |
|-----------------------------------|--------------------|---------------------|---------|
| RBCs (×10 <sup>6</sup> / $\mu$ L) | 5.7±0.4            | 5.9±0.3             | 0.0001* |
| Hb (g/L)                          | 16.2±0.7           | 17.7±0.5            | 0.0001* |
| PCV (%)                           | 45.9±1.8           | 49.3±1.7            | 0.0001* |
| MCV (fL)                          | 82.8±4.1           | 83.7±3.2            | 0.048*  |
| MCH (pg)                          | 29.2±1.9           | 29.9±1.2            | 0.0001* |
| MCHC (g/dL)                       | 35.2±1.3           | 35.8±0.8            | 0.0001* |
| RDW (%)                           | 13.3±0.9           | 13.1±0.6            | 0.2312  |
| WBCs ( $\times 10^3/\mu$ L)       | 5.6±1.6            | 6.2±1.7             | 0.0065* |
| NEUT (×10 <sup>3</sup> /µL)       | 2.9±1.3            | 3.3±1.6             | 0.0217* |
| LYM (× $10^3/\mu$ L)              | 2.4±0.6            | 2.6±0.6             | 0.1083  |
| MXD (× $10^3/\mu$ L)              | 0.3±0.2            | 0.3±0.2             | 0.9999  |
| PLT (× $10^3/\mu$ L)              | $197.8 \pm 71.8$   | $182.7 \pm 64.5$    | 0.0673  |

Table 2: Comparison between blood parameters of both groups

<sup>\*</sup> Significant

Table 3 revealed prevalence of H. pylori in both groups. The H. pylori were positive in 110 cases with an overall prevalence of 35.6%. In group I; H. pylori was positive in 73 cases with prevalence of 36.1 % and in group II; H. pylori was positive in 37 cases with prevalence of 34.6%, without statistical significance between both groups (P=0.7854).

| Table 5: Frevalence of H. Fylori in groups I and H |               |      |               |      |       |         |
|--|---------------|------|---------------|------|-------|---------|
|  | H. Pylori     |      |               |      |       |         |
| Variables  | Sero-negative |      | Sero-positive |      | Total | P-value |
|  | No.           | %    | No.           | %    |       |         |
| Group I (Hb Conc. 13-17 g/dL)                      | 129           | 63.9 | 73            | 36.1 | 202   |         |
| Group II (Hb Conc. >17 g/dL)                       | 70            | 65.4 | 37            | 34.6 | 107   | 0.7854  |
| Total  | 199           | 64.4 | 110           | 35.6 | 309   |         |

Table 3: Prevalence of H. Pylori in groups I and II

Table 4 revealed non-significant negative correlation between H. pylori and RBC, Hb, PCV, MCV, MCH, MCHC, RDW and Neut with non –

significant positive correlation between H. pylori and WBCs, Lym, MXD, and Plt.

| Variables |                                    | r        | P-value |
|-----------|------------------------------------|----------|---------|
|           | RBCs ( $\times 10^6/\mu$ L)        | - 0.0217 | 0.7047  |
|           | Hb (g/dL)                          | - 0.0547 | 0.3383  |
|           | PCV (%)                            | - 0.0573 | 0.3152  |
|           | MCV (fL)                           | - 0.0445 | 0.4361  |
|           | MCH (pg)                           | - 0.038  | 0.5053  |
| H. Pylori | MCHC (g/dL)                        | - 0.0072 | 0.8998  |
|           | RDW (%)                            | - 0.0041 | 0.9425  |
|           | WBCs ( $\times 10^3/\mu$ L)        | 0.0683   | 0. 4843 |
|           | Neutrophils ( $\times 10^3/\mu$ L) | - 0.0186 | 0.849   |
|           | Lymphocytes ( $\times 10^3/\mu$ L) | 0.1325   | 0.1736  |
|           | Mixture ( $\times 10^3/\mu$ L)     | 0.5623   | 0.5648  |
|           | Platelets ( $\times 103/\mu$ L)    | 0.1333   | 0.1712  |

r (Correlation coefficient)

#### DISCUSSION

The study was conducted in Taif governorate in the Western region of Saudi Arabia. Taif city is a high altitude area that it is more than 2400 meters above the sea level [17]. Our study aimed to determine the seroprevalence of H. pylori in non-symptomatic young male subjects living in Taif city. Seroprevalence of H. pylori was positive in 110 subjects with overall prevalence of 35.6%. In group I, H. pylori seroprevalence was positive in 73 cases, with prevalence of 36.1% and in group II H. pylori was positive in 37 cases with prevalence of 34.6%, with no statistical significance difference between both groups (P=0.7854).

Our results were more than that of Telmesani [19], who found that the prevalence of H. pylori in school children in Makkah city, Western Saudi Arabia using urea breath test was positive in 27.4% (it was positive in 45/103 "43.7%" in intermediate school students and 41/211 "19.4%" in secondary school students). He concluded that the prevalence of H. pylori among the school children in Makkah, Saudi Arabia, is relatively low compared to developing countries. The prevalence was found to be higher among the younger age group. He explained that low rate of prevalence

might be related to a higher socioeconomic status relative to the developing countries. Katelaris *et al.;* [20] found that prevalence of H. pylori infection was 30% in a study of 197 Tibetan patients with dyspeptic symptoms from South India. In a community-based study from Italy involving 1033 patients with or without dyspeptic symptoms, nearly three-fourths of the patients were found to be normal on endoscopy. The overall prevalence of H. pylori infection was 58% in this study with the prevalence of H. pylori infection being 93% in patients with peptic ulcers. High rates of H. pylori infection at high altitudes in comparison to coastal areas was also reported from Peru [21].

Poddar and Yachha [22] found that H. pylori infection rates in adults are even higher and increase with age. Also, Mishra *et al.*; [23] indicated that earlier report showed 80-90% infection rates by the age of 20 years. This is much higher than our results which may be due to higher socioeconomic standard in Saudi Arabia. In the United States, a 20% infection rate among adolescents is being reported [24] and recently an overall prevalence of 36% was reported, suggesting rapidly improving socioeconomic conditions [25]. High prevalence of H. pylori was reported by many authors previously. It was reported that 40% of the Saudi population in the age group of 5-10 years and 70% of people  $\geq$ 20 years of age had H. pylori [26] which is largely higher than the results of our study.

Prevalence of H. pylori was about 35.6% in our study with non-significant correlation between prevalence of H. pylori with blood parameters. These results are higher than the study done in Makkah area (low altitude) which found H. pylori prevalence of 27.4% [19], but is in agreement with Senra et al.; [27] who studied prevalence of H. pylori in the healthy population of Ubrique and Grazalema (mountain location) and in Barbate (coastal location) and found positive titers of 30% in the coastal population and 54% in the mountain location. He concluded that living in mountain locations involves a greater ecological risk for H. pylori infection (p<0.05). Also, Khan and Ghazi [16] found in their study that H. pylori seropositivity between male age groups 20-50 years was 37%-55%. Lastly, like our results, Ahmed et al.; [28] concluded that high altitude did not affect the prevalence of duodenal ulcer or the frequency of H. pylori because his results were comparable with those from the low altitude areas of the Kingdom of Saudi Arabia and other low land developing countries.

## CONCLUSION

Hypoxia in Taif city does not cause increase in H. pylori prevalence among Taif residents. However, prevalence in Taif city is relatively high when compared to other cities in the western region of Saudi Arabia.

# RECOMMINDATION

A confirmatory test to the positive H. pylori cases such as urea breath test or stool antigen test is mandatory to identify active disease and treat it to avoid complications. Also, serious measures should be taken immediately to advocate the implementation of sanitary conditions and health education against the transmission of H. pylori to block the infection process for students and their communities.

#### STUDY LIMITATION

The subjects involved in this study were volunteers and not randomly selected. The number of subjects involved was relatively small. Females were not included in this study and carried out only on males due to difficulty of male investigators to take samples from female subjects.

#### REFRENCES

- 1. Atherton J, Blaser M; Coadaptation of Helicobacter pylori and humans: ancient history, modern implications. The Journal of Clinical Investigation 2009; 119(9):2475-87.
- Kusters J, M van Vliet A, Kuipers E; Pathogenesis of Helicobacter pylori infection. Clinical Microbiology Reviews 2006; 19(3):449-90.

- 3. Blaser M; Who are we? Indigenous microbes and the ecology of human diseases. EMBO Reports 2006; 7(10):956-60.
- 4. Khalifa M, Sharaf R, Aziz R; Helicobacter pylori: a poor man's gut pathogen? Gut Pathogens 2010; 2:2-12.
- 5. Bakri MM; Prevalence of Helicobacter pylori infection and the incidence of ureA and clarithromycin resistance gene 23S rRNA genotypes status in Saudi Arabia. Saudi journal of biological sciences 2013; 20(1):75-8.
- 6. Atherton JC; The pathogenesis of Helicobacter pylori-induced gastro-duodenal diseases. Annual Review of Pathology 2006; 1:63-96.
- Waleed M, Iqbal S, Nabeel A, Basil A; Prevalence of Helicobacter pylori infection among new outpatients with dyspepsia in Kuwait. BMC Gastroenterology 2010; 10: 14.
- 8. Aroori S; Helicobacter pylori. Gastroenterol Today 2001; 5,131-3.
- Yamaoka, Yoshio; Helicobacter pylori: Molecular Genetics and Cellular Biology. Caister Academic Pr 2008; ISBN 1-904455-31-X.
- 10. Kamal E, Bani H, Shadi M; Prevalence of Helicobacter pylori in Northern Jordan. Saudi Medical Journal 2001; 22(10):843-7.
- Torres J, Perez-Perez G, Goodman KJ, Atherton JC, Gold BD, Harris PR, *et al.*; A comprehensive review of the natural history of Helicobacter pylori infection in children. Arch Med Res 2000; 31:431-9.
- 12. Bardhan PK; Epidemiological features of Helicobacter pylori infection in developing countries. Clin Infect Dis 1997; 25:973-8.
- Hanafi MI, Mohamed AM; Helicobacter pylori infection: seroprevalence and predictors among healthy individuals in Al Madinah, Saudi Arabia. J Egypt Public Health Assoc. 2013; 88(1):40-5.
- Abdulaziz A. Bin Saeed; Glimpse of the Epidemiological Research on Helicobacter Pylori in Saudi Arabia. Saudi J Gastroenterol, 2009; 15(2): 85.
- 15. Almadi M, Aljebreen A, Tounesi F, Abdo A; Helicobacter pylori prevalence among medical students in a high endemic area. Saudi Med J 2007; 28:896-8.
- Khan M, Ghazi H; Helicobacter pylori infection in asymptomatic subjects in Makkah, Saudi Arabia. J Pak Med Assoc 2007; 57:114-7.
- 17. Alsulaimani AA, Alzahrani AK; Prevalence of congenital anomalies at high altitude area in Saudi Arabia. J of Med Res and Sci 2011; 1(3):44-51.
- Barbara Bain, Imelda Bates, Michael A Laffan, Mitchell Lewis; Reference ranges and normal values. In "Dacie and Lewis Practical Haematology", Elsevier Churchill Livingstone. 11<sup>th</sup> Edition, Chapter 2, 2012; 14.
- 19. Telmesani AM; Helicobacter pylori: prevalence and relationship with abdominal pain in school

children in Makkah City, western Saudi Arabia. Saudi Journal of Gastroenterology 2009; 15(2):100.

- 20. Katelaris PH, Tippett GH, Norbu P, Lowe DG, Brennan R, Farthing MJ; Dyspepsia, Helicobacter pylori, and peptic ulcer in a randomly selected population in India. Gut 1992; 33:1462-6.
- 21. Sharma PK, Suri TM, Venigalla PM, Garg SK, Mohammad G, Das P, *et al.*; Atrophic gastritis with high prevalence of Helicobacter pylori is a predominant feature in patients with dyspepsia in a high altitude area. Tropical Gastroenterology 2015; 35(4):246-51.
- 22. Poddar U, Yachha SK; Helicobacter pylori in children: an Indian perspective. Indian Pediatr 2007; 44:761-70.
- 23. Mishra S, Singh V, Rao GR, Dixit VK, Gulati AK, Nath G; Prevalence of Helicobacter pylori in asymptomatic subjects-A nested PCR based study. Infect Genet Evol 2008; 8:815-9.
- 24. Frenck RW Jr, Clemens J; Helicobacter in the developing world. Microbes Infect 2003; 5:705-13.
- Smith JG, Li W, Rosson RS; Prevalence, clinical and endoscopic predictors of Helicobacter pylori infection in an urban population. Conn Med 2009; 73:133-7.
- 26. Bakri MM; Prevalence of Helicobacter pylori infection and the incidence of ureA and clarithromycin resistance gene 23S rRNA genotypes status in Saudi Arabia. Saudi journal of biological sciences 2013; 20(1):75-8.
- 27. Senra-Varela A, Lopez-Saez JB, Gomez-Biondi V; Prevalence of Helicobacter pylori infection in two Spanish regions with different incidence of gastric cancer. European journal of epidemiology 1998; 14(5):49-4.
- Ahmed ME, Al-Knawy BA, Al-Wabel AH, Foli AK; Duodenal ulcer and Helicobacter pylori infection at high altitude: experience from southern Saudi Arabia. Canadian journal of gastroenterology = Journal canadien de gastroenterologie. 1996; 11(4):313-6.