

Helicobacter Pylori Infection in Children with Upper Gastrointestinal Bleeding at Tanta University Hospital

Abstract

Background:In children, upper gastrointestinal bleeding (UGIB) is a potentially dangerous and fatal clinical disease. The fact that the majority of risk factors for infection are strongly associated with substandard living circumstances provides evidence in favour of the oral-oral or fecal-oral pathways of H. pylori transmission. The purpose of this study was to determine the prevalence of H. pylori infection in kids who had been diagnosed with UGIB and to compare the results of the endoscopy with the infection.

Methods:This prospective, case-control cross-sectional study was carried out on 70 children aged <18 years old, both sexes, with upper gastrointestinal tract (GIT) bleeding in the form of hematemesis, melena, or both and 30 healthy children as control. Gastrointestinal endoscopy was performed on each patient, and stool samples were tested for the H. pylori antigen. Results: Patients with H. pylori infection were positively correlated with age, and older patients had a considerably higher presentation rate of H. pylori infection.

Low socio-economic standard level, large family members, family history and epigastric abdominal pain were significantly high in h. pylori positive group. The history of umbilical catheterization, percentage of splenomegaly and hepatosplenomegaly was significantly higher in variceal group. Mean hemoglobin level, total serum protein and serum albumin was significantly lower in variceal group.

Conclusions:26 (37%) cases with upper GI bleeding had H. pylori infection. H. pylori-infected patients were older, had larger family size, family history of H. pylori infection and lower standard of living than H. pylori-negative patients. They had lower serum hemoglobin level and presenting with abdominal pain(epigastric). H. pylori infected patients had endoscopic findings mostly gastritis (56%) and peptic ulcer (53%).

Keywords: Helicobacter Pylori, Children, Upper Gastrointestinal Bleeding, Tanta University Hospital

UNDER PEER REVIEW

Introduction:

Mucosal lesions and variceal hemorrhage are the usual causes of UGIB in children [1]. The common sources of UGIB in children include mucosal lesions and variceal hemorrhage [1].

Upper GI bleeding in infants and young children is often due to ulcers and stress erosion, but in older children it may be due to duodenal ulcers, esophagitis and esophageal varices [2].

6.4-10% of pediatric intensive care unit (ICU) admissions in Egypt were accounted for by UGIB [3]. Incidence of UGIB was observed in 6.4-10% of pediatric intensive care unit (ICU) admissions in Egypt [4].

Common signs and symptoms at presentation include hematemesis (73%), melena (21%), and coffee-ground emesis (6%); however, patients may also experience epigastric pain and abdominal tenderness [5].

Helicobacter pylori (*H. pylori*) is one of the most common chronic infections with a worldwide prevalence of about 50% in children [6]. *H. pylori* is a Gram negative spiral-shaped bacterium that is found in the gastric mucous layer or adherent to the epithelial lining of the stomach causing chronic gastritis [7].

It primarily occurs during childhood in impoverished nations [8]. The prevalence of *H. pylori* varies markedly between countries; about 50% of children are infected by 10 years of age in developing countries [9].

Poverty, poor hygiene, inadequate sanitation, household overcrowding, sharing beds, and ingesting contaminated food and water are contributing factors [10, 11]. These include lower socioeconomic status, bad hygiene, deficiency of sanitation, household crowding, bed sharing, and food- and water-borne transmission [10, 11].

There's a lack of studies concerning the link between *H. pylori* and UGIB in children [12].

This study aimed to assess the frequency of *H. pylori* infection in pediatric UGIB cases at

Tanta University Hospital's Pediatric Gastroenterology, Hepatology and Endoscopy Unit and to link it to endoscopic findings.

Patients and Methods:

This prospective, case-control cross-sectional study was carried out on 70 children aged <18 years old, both sexes, with upper GIT bleeding in the form of hematemesis, melena, or both and 30 healthy children as control. The study was conducted from January 2023 to June 2024 with permission from Tanta University Hospitals' Ethical Committee in Tanta, Egypt (approval code: 35669/8/22). The patients' family provided written informed permission.

Exclusion criteria were patients with bleeding originated from the nasal or oral cavities, bleeding tendency, lower GIT bleeding, patients on proton pump inhibitor or antacid or receiving anti-H. pylori treatment in the previous three months of this study, patients unfit for endoscopy.

Every patient underwent a comprehensive medical history, physical examination, and laboratory investigations, including a complete blood count (CBC), liver function tests (AST, ALT, and ALP), bilirubin (direct and total), total protein, and serum albumin levels, coagulation profile test (PTT, INR, and prothrombin time and concentration), and liver function tests (ALT, AST, and ALP) and detection of stool H. pylori antigen] and laboratory investigations [abdominal ultrasound].

Abdominal ultrasound for complete abdomino-pelvic examination. All patients were subjected to abdominal ultrasonographic examination by using (MENDALY 2016) to assess for organomegaly, portal hypertension or liver cirrhosis.

Gastrointestinal Endoscopy

Upper endoscopy was performed for all patients with upper GI bleeding after proper preparation and hemodynamic stabilization of the condition.

Endoscopic examination was performed for the following: Gross esophageal, gastric and duodenal examination, identification of *Helicobacter pylori* infection in stomach biopsy specimens following fixation and appropriate staining: The stomach mucosa was used for biopsy specimens, and the presence of *Helicobacter pylori* was determined by both stool antigen testing and histological inspection. It was suggested that patients fast for two hours before endoscopy for water, four hours for breastfeeding, and six hours for solids ^[13]. Sedation was recommended according to the age of the studied children. The endoscopic examination was carried out by using Pentax (EG-2731) esophagogastroduodenoscopy. The youngster was put on a height-adjustable stretcher. A breathing bag that was the appropriate size for the child's age was also provided, along with emergency medicine. Keep common auxiliary tools and equipment on available, such as clip systems or needles, which are haemostatic devices.

Post endoscopy

For the next fifteen to thirty minutes, either in the recovery room or on the ward, monitoring settings from the surgery were maintained, particularly for little newborns. The patient was positioned laterally. One hour following the conclusion of the endoscopy, drinks were permitted. If the patient's airway patency and cardiovascular function are stable and good, they will be released.

Analytical statistics

IBM Inc., Chicago, IL, USA used SPSS v26 for statistical analysis. The statistical variables were denoted by their mean and standard deviation (SD), and the unpaired Student's t-test was employed to compare them between the two groups. Frequency and percentage (%) were used to represent qualitative variables, and the Chi-square or Fisher's exact test were used for analysis as necessary. A statistically significant result was defined as a two-tailed P value less than 0.05.

Results:

There was no significant difference found between the study patients and controls in terms of demographics or other laboratory tests (Platelets, total leucocytic count, total and direct bilirubin, PT, PTT, INR, serum protein, and albumin). Patients' mean haemoglobin level was significantly lower than controls'. Patients' levels of ALT, AST, and ALP were much greater than those of controls. Table 1

Table 1: Demographic characteristics and laboratory data of the studied patients and controls

		Patients (n = 70)	Controls (n = 30)	Test of significance	P
Age (years)		10.607± 5.145	11.0±4.017	T=1.003	0.318
Sex	Male	32(45.7%)	13(43.3%)	X ² =0.48	0.826
	Female	38(54.3%)	17(56.7%)		
Residence	Urban	32(45.7%)	13(43.3%)	X ² =1.008	0.315
	Rural	38(54.3%)	17(56.7%)		
Socioeconomic standard	Low	40(57.1%)	14(46.7%)	X ² =0.928	0.335
	Moderate	30(42.9%)	16(53.3%)		
Number of family members	Up to 3	36(51.4%)	20(66.7%)	X ² =1.979	0.159
	More than 3	34(48.6%)	10(33.3%)		
Laboratory parameters					
Hb (g/dl)		9.420±1.080	11.023±0.679	8.955	<0.001*
Platelets (×10³/ul)		429.37±128.311	423.47±59.70	-0.314	0.754
TLC(×10³/ul)		5.136±0.799	5.097±0.627	-0.238	0.813
ALT (U/L)		49.251±41.884	21.633±6.316	-5.376	<0.001*
AST(U/L)		40.73±34.478	24.50±7.533	-3.736	<0.001*
ALP(IU/L)		72.643±28.976	40.000±4.152	-9.207	<0.001*
Total bilirubin (mg/dl)		0.589±0.222	0.539±0.229	-1.016	0.312
Direct bilirubin (mg/dl)		0.228±0.063	0.223±0.062	-0.347	0.730
Total serum Protein (g/dl)		9.134±0.659	9.127±0.663	-0.053	0.958
Albumin(g/dl)		3.848±0.430	3.716±0.396	-1.441	0.153
PT (sec)		12.400±1.220	12.445±0.472	0.268	0.790
PTT (sec)		28.757±4.318	29.200±2.483	0.645	0.521
INR		0.966±0.133	0.959±0.131	-0.253	0.803

Data are presented as mean ± SD or frequency (%). *Significant as P value ≤ 0.05, X²: Chi square, t: independent t test, Hb: hemoglobin, TLC: total leucocytic count, ALT: alanine transaminase, AST: aspartate aminotransferase, ALP: alkaline phosphatase, PT: prothrombin time, PTT: partial thromboplastin time, INR: international normalized ratio.

There was a positive relationship between age and h. pylori infected patients, so h. pylori infection was significantly presented in older aged patients. Low socio-economic standard level, large family members, family history and epigastric abdominal pain were significantly high in h. pylori positive group. **Table 2**

Table 2: Demographic characteristics of H. pylori status

		H. PYLORI status		Chi square test(X ²)	P
		Positive(n=26)	Negative(n=44)		
Age (Years)		14.654±2.741	8.216±4.727	T= -7.211	<0.001*
Sex	Male	11(42.3%)	21(47.7%)	0.193	0.660
	Female	15(57.7%)	23(52.3%)		
Residence	Urban	8(30.8%)	24(54.5%)	3.723	0.054
	Rural	18(69.2%)	20(45.5%)		
Socioeconomic standard	Low	19(73.1%)	21(47.7%)	4.288	0.038*
	Moderate	7(26.9%)	23(52.3%)		
Number of family members	Up to 3	6(23.1%)	30(68.2%)	13.310	<0.001*
	More than 3	20(76.9%)	14(31.8%)		
Clinical history	Drug Intake	10(38.5%)	14(31.8%)	0.320	0.572
	Previous GIT bleeding	10(38.5%)	20(45.5%)	0.326	0.568
	Previous GIT surgery	1(3.8%)	1(2.3%)	0.146	0.703
	Umbilical catheterization	1(3.8%)	5(11.4%)	1.179	0.278
	Family history of GIT bleeding	2(7.7%)	8(18.2%)	1.469	0.226
	Family history of H. pylori infection	19(73.1%)	17(38.6%)	7.760	0.005*
Symptoms and signs	Abdominal pain	25(96.2%)	41(93.2%)	0.268	0.605
	Epigastric	21(80.8%)	19(47.5%)	7.305	0.007*
	Other sites	5(19.2%)	21(52.5%)		
	Vomiting	23(88.5%)	32(72.7%)	2.403	0.121
	Abdominal tenderness	5(19.2%)	14(31.8%)	1.309	0.253
	Epigastric	3(60.0%)	7(50.0%)	0.148	0.701
	Other sites	2(40.0%)	7(50.0%)		
	Splenomegaly	2(7.7%)	7(15.9%)	0.985	0.321
	Hepatosplenomegaly	0(0.0%)	1(2.3%)	0.599	0.439
Type of bleeding	Hematemesis	8(30.79%)	22(50.0%)	3.371	0.185
	Melena	10(42.3%)	9(20.45%)		
	Both hematemesis and melena	8(53.8%)	13(29.54%)		

Data are presented as mean ± SD or frequency (%). * Significant as P value ≤0.05, X²: Chi square, t: independent t test, GIT: Gastrointestinal tract.

Mean Hb level was significantly lower in h. pylori positive group. There was no significant difference in other laboratory findings. Splenomegaly, liver cirrhosis and Hepatosplenomegaly weren't significantly associated with h. pylori positive group. Most of h. pylori cases were gastritis (34.61%), peptic ulcer (30.76%), duodenitis (11.53%) then GERD, esophagitis and esophageal varices represented the same percentage (7.69). **Table 3**

Table 3: Laboratory data and ultrasound and endoscopic findings of H. pylori status

	H. PYLORI		Independent t test	P
	Positive (n=26)	Negative (n=44)		
Hb (g/dl)	8.892±0.691	9.732±1.152	3.368	0.001*
Platelets (×103/ul)	423.85±136.043	438.55±124.204	0.776	0.440

TLC($\times 10^3$/ul)	5.208 \pm 0.620	5.093 \pm 0.891	-0.631	0.530
ALT (U/L)	26.215 \pm 28.507	40.66 \pm 35.6	1.761	0.083
AST(U/L)	32.19 \pm 30.620	45.77 \pm 35.948	1.611	0.112
ALP(IU/L)	61.923 \pm 25.771	74.43 \pm 26.88	1.910	0.06
Total bilirubin (mg/dl)	0.49 \pm 0.2	0.59 \pm 0.22	1.899	0.075
Direct bilirubin (mg/dl)	0.23 \pm 0.06	0.24 \pm 0.06	0.673	0.548
Total serum Protein (g/dl)	9.26 \pm 0.65	8.99 \pm 0.63	1.712	0.09
Albumin(g/dl)	3.926 \pm 0.356	3.803 \pm 0.466	-1.165	0.248
PT (sec)	12.057 \pm 1.329	12.602 \pm 1.118	1.754	0.086
PTT (sec)	29.00 \pm 5.878	28.614 \pm 3.126	-0.310	0.758
INR	0.97 \pm 0.11	0.99 \pm 0.15	0.591	0.501
Ultrasound findings				
No abnormal findings	24(92.3%)	36(81.8%)	1.683	0.776
Splenomegaly	2(47.69%)	7(15.90%)		
Hepatosplenomegaly	0(0.0%)	1(2.3%)		
Cirrhotic liver	1(3.8%)	3(6.8%)		
Endoscopic findings				
Esophageal varices	2(7.7%)	8(18.2%)	19.002	0.001*
GERD	2(7.7%)	7(15.90%)		
Gastritis	9(34.61%)	7(15.90%)		
Peptic ulcer	8(30.76%)	7(15.90%)		
Esophagitis	2(7.69%)	9(20.45%)		
Mallory Weiss tear	0(0.0%)	3(6.81%)		
Duodenitis	3(11.53%)	3(6.81%)		

Data are presented as mean \pm SD or frequency (%). * Significant as P value \leq 0.05, X^2 : Chi square, t: independent t test, Hb: hemoglobin, TLC: total leucocytic count, ALT: alanine transaminase, AST: aspartate aminotransferase, ALP: alkaline phosphatase, PT: prothrombin time, PTT: partial thromboplastin time, INR: international normalized ratio.

Demographic data was insignificantly different between both groups. The history of umbilical catheterization, percentage of splenomegaly and hepatosplenomegaly was significantly higher in variceal group. **Table 4**

Table 4: Demographic characteristics of variceal and non-variceal patients

		Variceal (n =10)	Non-variceal (n=60)	Chi square test (X^2)	P
Age (Years)		12.3 \pm 3.95	10.32 \pm 5.29	T=1.126	0.264
Sex	Male	4(40.0%)	28(46.7%)	0.154	0.695
	Female	6(60.0%)	32(53.3%)		
Clinical history	Drug Intake	3(30.0%)	21(35.0%)	0.095	0.758
	Foreign body ingestion	0(0.0%)	2(3.3%)	0.343	0.558
	Previous GIT bleeding	6(60.0%)	24(40.0%)	1.400	0.237
	Previous GIT surgery	0(0.0%)	2(3.3%)	0.343	0.558
	Systemic infection	2(20.0%)	8(13.3%)	0.311	0.557
	Umbilical catheterization	5(50.0%)	1(1.7%)	25.551	<0.001*
	Family history of GIT bleeding	1(10.0%)	9(15.0%)	0.175	0.676
Symptoms and signs	Family history of H. pylori infection	3(30.0%)	33(55.0%)	2.145	0.143
	Abdominal pain	10(100.0%)	56(93.3%)	0.707	0.528
	Epigastric	6(60.0%)	34(60.7%)	0.002	0.966
	Other sites	4(40.0%)	22(39.3%)		

	Vomiting	10(100.0%)	45(75.0%)	3.182	0.074
	Abdominal tenderness	5(50.0%)	14(23.3%)	3.082	0.079
	Epigastric	2(40.0%)	8(57.1%)	0.434	0.510
	Other sites	3(60.0%)	6(42.9%)		
	Splenomegaly	9(90.0%)	0(0.0%)	61.967	<0.001*
	Hepatosplenomegaly	1(10.0%)	0(0.0%)	6.087	0.014*
Type of bleeding	Hematemesis	8(30.79%)	22(50.0%)	3.371	0.185
	Melena	10(42.3%)	9(20.45%)		
	Both hematemesis and melena	8(53.8%)	13(29.54%)		

Data are presented as mean \pm SD or frequency (%). * Significant as P value \leq 0.05, X²: Chi square, t: independent t test, GIT: Gastrointestinal tract.

Serum albumin, total serum protein, and mean haemoglobin level were all much lower in the variceal group. The variceal group had considerably higher levels of ALT, AST, and ALP.

Other laboratory tests (platelets, total leucocytic count, total and direct bilirubin, PT, PTT, and INR) did not show any significant differences between the two groups. Variceal patients had Hepato-splenomegaly, Splenomegaly and liver cirrhosis in 10% ,90% and 40% respectively. **Table 5**

Table 5: Laboratory data and ultrasound findings of variceal and non-variceal patients

		Variceal (n=10)	Non-Variceal (n=60)	Independent t test	P
Hb (g/dl)		8.920 \pm 0.953	9.75 \pm 1.19	3.368	0.001*
Platelets ($\times 10^3$/ul)		452.100 \pm 160.231	425.580 \pm 123.433	0.776	0.440
TLC($\times 10^3$/ul)		5.180 \pm 1.134	5.128 \pm 0.741	-0.631	0.530
ALT (U/L)		118.400 \pm 31.903	37.727 \pm 30.836	1.761	0.083
AST(U/L)		106.70 \pm 42.669	29.73 \pm 15.870	1.611	0.112
ALP(IU/L)		132.00 \pm 22.010	62.750 \pm 14.538	1.910	0.06
Total bilirubin (mg/dl)		0.6100 \pm 0.242	0.585 \pm 0.221	1.899	0.075
Direct bilirubin (mg/dl)		0.225 \pm 0.0677	0.228 \pm 0.0639	0.673	0.548
Total serum Protein (g/dl)		8.65 \pm 0.58	9.14 \pm 0.665	1.712	0.09
Albumin(g/dl)		3.46 \pm 0.64	3.833 \pm 0.369	-1.165	0.248
PT (sec)		12.300 \pm 1.251	12.416 \pm 1.225	1.754	0.086
PTT (sec)		27.200 \pm 4.939	29.017 \pm 4.196	-0.310	0.758
INR		0.956 \pm 0.142	0.968 \pm 0.1335	0.591	0.501
H. pylori status	Positive	2(20.0%)	24(40.0%)	1.469	0.226
Ultrasound findings					
No abnormal findings		0(0.0%)	60(100.0%)	70.00	<0.001*
Splenomegaly		9(90.0%)	0(0.0%)		
Hepatosplenomegaly		1(10.0%)	0(0.0%)		
Cirrhotic liver		4(40.0%)	0(0.0%)		

Data are presented as mean \pm SD or frequency (%). * Significant as P value \leq 0.05, X²: Chi square, t: independent t test, Hb: hemoglobin, TLC: total leucocytic count, ALT: alanine transaminase, AST: aspartate aminotransferase, ALP: alkaline phosphatase, PT: prothrombin time, PTT: partial thromboplastin time, INR: international normalized ratio.

Discussion

Intraluminal bleeding from the oesophagus to the ligament of Treitz within the gastrointestinal system can result in UGIB, a medical emergency. Thanks to updated recommendations, appropriate resuscitation, and improved endoscopic diagnostic and treatment methods, UGB care has undergone tremendous improvement in recent years [14].

The current investigation found that 26 (37.14%) individuals who experienced upper gastrointestinal bleeding had a pathologically confirmed *H. pylori* infection, as shown by a positive stool antigen test. This finding is higher than that of previous studies, but it is comparable to that of Gimiga et al. [15] who observed in other underdeveloped nations.

The most prevalent endoscopic findings in patients with upper GI bleeding in our study were gastritis (16 cases, 22.8%), peptic ulcer (15 cases, 21.4%) and esophagitis (11 cases, 15.7%). Moreover, esophageal varices were found in 10 cases (14.2%), GERD in 9 cases (12.85%) and duodenitis in 6 cases (8.5%). EL-Mazary et al. [4] discovered that the primary causes of bleeding in a sample of Egyptian children with upper gastrointestinal bleeding, aged between 4 and 14 years, were gastritis and peptic ulcer disease, which is in line with our findings. Peptic ulcers were discovered by Cleveland et al. [16] in 24% of children who had upper GI haemorrhage. Children with upper gastrointestinal haemorrhage had a 14.5% ratio of oesophagitis, according to Gimiga et al. [15]. On the other hand, other endoscopic findings were described in other research. According to Rafey et al. [17], oesophagitis accounted for 40% of endoscopic findings.

In the current study, *H. pylori* infection was found in 56% of cases diagnosed endoscopically as gastritis, in 53% of cases with peptic ulcer, in 20% of cases with esophageal varices and in 50% of cases with duodenitis.

Similarly, most children in a previous study done in Saudi Arabia with *H. pylori* infection had peptic disease^[18]. A lengthier research conducted by Hasosah et al. [19] revealed that 55% of children with stomach ulcers had an *H. pylori* infection.

The findings of our study indicated that the following factors were associated with a higher likelihood of *H. pylori* infection: older age, greater family size, presence of anaemia, melena (including both haematemesis and melena), and gut discomfort (particularly epigastric). The endoscopic results of duodenitis, gastritis, and stomach ulcers were strongly linked to a higher incidence of *H. pylori* infection.

Based on the current study's results, 10 individuals (14.28%) had esophageal varices identified.

Similar findings were reported among children with upper GI bleeding from Iran (16%) and Sudan (16%)^[20, 21]. However, a higher incidence of esophageal varices was reported in some developing countries (in India 31% and in Iraq 39%)^[22, 23].

Moreover, in our study, ultrasonic features of liver cirrhosis were found in four (40%) cases and splenomegaly in 9 (90%) cases out of the ten variceal patients. One of the main consequences of portal hypertension, which is linked to increased portal input and increased outflow resistance, might be the existence of esophageal varices, which can result in splenomegaly. This is in line with a research by Ma et al. [24], which found that 40% to 85% of patients with liver cirrhosis also had portal hypertension, the primary cause of gastro-oesophageal varices. One of the major side effects of cirrhosis and portal vein hypertension is variceal haemorrhage.

Assessment of laboratory investigations may play an important role in the prediction of the severity of GI bleeding. Generally, the most important laboratory finding in our study was the significantly lower serum Hb concentration in the studied patients (9.4 ± 1.08 g/dl) than controls (11.02 ± 0.67). Acute or chronic blood loss as well as alimentary bleeding (haematemesis, melena, or both) may be to blame for this. This is especially noticeable in patients who have *H. pylori* infection and variceal haemorrhage. Comparably, Hb was much lower in children with severe upper GI bleeding group compared to moderate upper GI

bleeding group, as reported by Di Girolamo et al. [25]. According to El-Hodhod et al. [26], anaemia was identified in 60% of children, with 31% having moderate anaemia and 9% having severe anaemia. The range of Hb was 3.9 to 15 g/dl, with a mean of 10.5 ± 2.21 g/dl.

The significantly lower serum Hb level observed in the studied *H. pylori* infected children compared to those non-infected is consistent with a recent Egyptian study done in our Hepatology and Gastro-enterology Unit on 70 Egyptian children^[27]. Lower levels of Hb with indices of iron deficiency anemia (IDA) (low serum ferritin and high soluble transferrin receptor, STFR) were significantly evident in *H. pylori* positive patients. Improvement of iron status following anti *H. pylori* therapy was clearly noted in those responders.

There is a correlation between *H. pylori* infection and IDA, according to cross-sectional and epidemiological research done on children and adolescents [28, 29].

Occult blood loss resulting from stomach inflammation and ulceration, as well as enhanced iron absorption and utilisation by *H. pylori* bacteria, are potential pathogenic pathways by which *H. pylori* may contribute to anaemia in the individuals under study. Furthermore, anaemia most likely developed as a result of reduced iron absorption brought on by hypo- or achlorhydria, chronic gastritis, and ascorbic acid insufficiency [30].

Limitations:

There was a rather limited sample size. There was just one centre for the study. the potential for confounding factors to appear in the results.

Conclusion

26 (37%) of the 70 individuals who experienced upper gastrointestinal haemorrhage also had *H. pylori* infection. In comparison to *H. pylori*-negative individuals, *H. pylori*-positive patients were older, had bigger families, a family history of *H. pylori* infection, and a worse quality of living. Their serum haemoglobin level was decreased, and they were experiencing epigastric discomfort in their abdomen. Gastritis was the most frequent cause of upper

gastrointestinal haemorrhage, followed by duodenitis, GERD, oesophagitis, peptic ulcer, and esophageal varices. *H. pylori* infected patients had endoscopic findings mostly gastritis (56%) and peptic ulcer (53%). Variceal patients had ultrasonographic features of liver cirrhosis (40%) and splenomegaly (90%). They had lower hemoglobin and higher liver enzyme levels than non-variceal patients.

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