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ORIGINAL ARTICLE

Study of *Helicobacter pylori* Infection in Chronic Hepatitis (C) Infection Among Egyptian Patients

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Abstract

Objectives: **Our objective is to assess *H. pylori* infection in chronic hepatitis C Egyptian patients and its hepatic impact.**
Background: ***Helicobacter pylori* lives mainly on gastric mucosa and is one of the common infections worldwide.**
In liver cirrhosis it may lead to impaired liver functions due to fibrosis in chronic liver diseases as HCV.
Patients and methods: **This was a cross sectional prospective study included 40 of HCV infection and 20 health subjects as a control. It was conducted on in the Al-Azhar University Hospitals during the period from February 1, 2020 to September 30, 2021.**
After approval of local ethics committee, all patients included in the study were informed about the procedure before starting.
Patients were subjected to complete history and clinical examination; complete investigations needed and endoscopic examination.
Results: **Our study revealed a non-significance for age, gender, BMI, with predominance antidiabetic drug use. There was significant increase in WBCs, Hb decrease, decrease platelets, ESR. In addition there was a elevations in liver functions, total and direct bilirubin with significant reduction in serum albumin. Also, a significant elevation in triglycerides (TG), LDL and HDL without significant change in cholesterol level was present.**
HCV infection present significantly whether by ELISA or qualitative PCR and *H. Pylori* also present significantly with significant elevation of α-FP in our cases. U/S revealed significant presence of cirrhotic changes with or without splenomegally.
Conclusion: **From our study we can concluded a positive association between *H. pylori* infection and chronic hepatitis C and the resulting cirrhosis.**

Keywords: Chronic liver disease, CLD, *Helicobacter pylori*, HP, LC, Liver cirrhosis

1. Introduction

Helicobacter pylori (*H. Pylori*), is a micro-aerophile, a gram −ve bacillus, resistant to the activity of the gastric juice. The bacteria may take the vegetative form (spiral form) or sporulation form. *H. Pylori* lives mainly on the surface of epithelial cells of mucous membranes of the prepyloric part of the stomach. The cilia presented on the bacteria allow it to move into the intercellular spaces and adhere to the surface of the cell. Infection with these bacteria is one of the most common infections in the world. In highly developed countries, 50 % of the population is infected, whereas in the developing countries the proportion reaches as much as 90 %.[1]

H. Pylori infection causes local (limited to gastric mucous membrane) and general increase of pro-inflammatory cytokines; interleukin-1 (IL-1), IL-2, IL-4, IL-6, IL-8, IL-10, IL-17, interferon-β and tumor necrosis factor-α.[2]

H. Pylori may influence extra-gastric organs, exacerbating cardiovascular diseases, metabolic diseases and disturbing liver functions especially in patients with liver cirrhosis (LC).[3]

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H. Pylori infection in the group of patients with LC may influence exacerbating of inflammatory lesion in the stomach, which could directly and indirectly lead to impairment of liver functions. This is especially dangerous in patients with advanced liver injury; studies on this group of patients point to high importance of cytopathological effect of *H. Pylori* on hepatocytes.[4]

H. Pylori infection causes multiorgan injuries, including chronic injury of the pancreas which indirectly influences the functions of the liver.[5]

H. Pylori infection is significantly more frequent among patients with post inflammatory LC related to HCV or HBV infection than in patients with alcoholic LC or primary biliary cirrhosis, also the incidence of esophageal varices correlate with the incidence of *H. Pylori* infection.[6]

This study aimed to assess *H. pylori* infection in chronic hepatitis C Egyptian patients and its hepatic impact.

2. Patients and methods

This was a cross sectional prospective study included 40 of confirmed HCV infection and twenty subjects of the matched age and sex. It was conducted on in the Al-Azhar University Hospitals during the period from February 1, 2020 to September 30, 2021.

Patients with chronic liver disease; or received treatment of *H. Pylori* in the previous two months, those on medication known to cause hepatic affection, history of alcohol consumption, those with cardiovascular, renal diseases, malignancy or those with insufficient clinical and or laboratory data.

After approval of local ethics committee, all patients included in the study or their relatives were informed well about the procedure and had an informed written consent before carrying the procedure.

All patients were subjected to complete history and clinical examination; complete laboratory "CBC, Renal function tests, liver function tests, lipid profile," radiologic examination by ultrasonography, serological "HBsAg, HCV, ANA-titre, PCR for HCV, α-feto protein, qualitative detection of *H.Pylori* antigen in stool" and endoscopic investigations.

Data were analyzed using corresponding statistical tests and P value < 0.05 was considered significant using SPSS version 16 soft ware (SPSS Inc, Chicago, ILL Company).

3. Results

The collected data from our patients of the study.

3.1. Sex

Twenty-eight of our cases were males (28/40, 70 %) and twelve were females (12/40, 30 %) while in control group eleven were males (11/20, 55 %) and nine were females (9/20, 45 %) and the statistical analysis revealed that males were more prominent in the group of cases ($P = 0.021$), while there were no significant difference between males and females in the control group or between both groups regarding gender (P = 0.215, 0.231 and 0.236, respectively) (Tables 1 and 2, Fig. 1).

3.2. Age

The age of cases ranged between 20 years and 75 years with a mean age of 59.25 ± 10.02 years while in control group it ranged between 38 years and 69 years with a mean age of 55.3 ± 8.65 years and the statistical analysis revealed a non-significant difference between both groups regarding age ($P = 0.121$), (Table 1, Figs. 2 and 3).

3.3. Alcohol and drug usage

Thirty-six of our cases were consuming some drugs (36/40, 90 %) while only four patients (10 %)

Table 1. Distribution of age and sex in cases and control groups in our study.

Variable	Cases ($n = 40$) No. (%)	Controls ($n = 20$) No. (%)	P Value
Sex			
Male	28 (70 %)	11 (55 %)	0.231
Female	12 (30 %)	9 (45 %)	0.236
P	0.021	0.215	
Drug usage:			
Yes	36 (90 %)	0 (0.0 %)	0.001 0.01
No	4 (10 %)	20 (100 %)	
P	0.01	0.001	
Age (Y):			
Range	20–75	38–69	0.121
Mean ± S.D.	59.25 ± 10.02	55.3 ± 8.65	
BMI (Kg/m^2):			
Range	19–38	18–33	0.270
Mean ± S.D.	25.55 ± 3.6	24.3 ± 4.3	

P is significant if ≤ 0.05.

Table 2. Drugs that most mostly used in cases of our study.

Variable	No. (%)	P Value
Insulin	19 (47.5 %)	
Oral hypoglycemic	8 (20 %)	
Antihypertensive	6 (15 %)	
Calcium channel blockers	1 (2.5 %)	0.021
β-Blockers	1 (2.5 %)	
Ator	4 (10 %)	
Premalor	2 (5 %	

P is significant if ≤ 0.05.

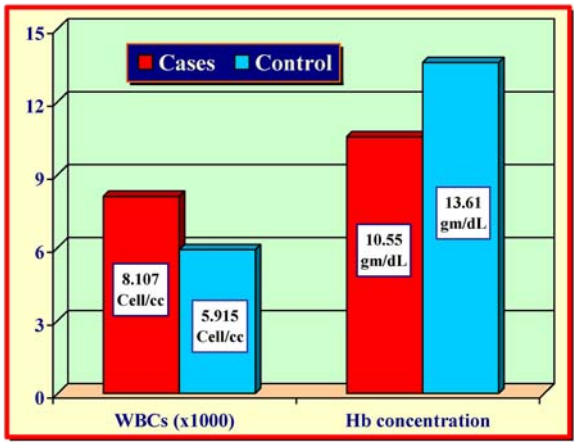

Fig. 1. WBCs and Hb concentration in cases of our study.

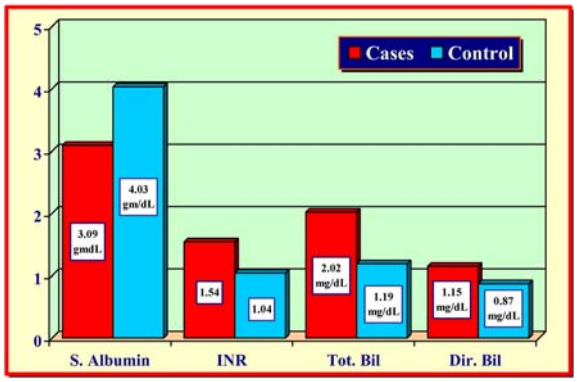

Fig. 2. Serum albumin, INR, total and direct bilirubin in both groups of the study.

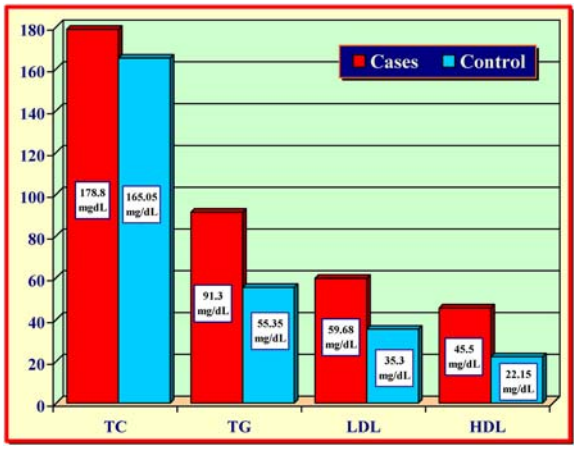

Fig. 3. Lipid profile in both groups of the study.

did not consume any drugs in addition any of the controls group consume any drugs (20/20, 100 %) and the statistical analysis revealed predominant drug usage in cases of our study ($P = 0.01$). Also, in our cases and controls no patient did consume alcohol (Table 1).

3.4. BMI (Kg/m^2)

The BMI of cases ranged between 19 and 38 kg/m^2 with a mean of 25.55 ± 3.6 kg/m^2 while in control group it ranged between 18 and 33 kg/m^2 with a mean of 24.3 ± 4.3 kg/m^2 and the statistical analysis revealed a non-significant difference between both groups regarding BMI ($P = 0.270$), (Table 1, Fig. 2).

3.5. Clinical assessment

3.5.1. Clinical examination

All cases of the study (100 %) showed positive general examination signs and all controls (100 %) are free of any general signs during examination denoting *H.Pylori* affection. During abdominal examination; thirty-three of cases establish positive signs (82.5 %), while 17.5 % of our cases did not show any abdominal signs during examination. In addition, all controls were fre of abdominal signs and the statistical analysis revealed significance of abdominal signs in affected patients by *H.Pylori* ($P = 0.001$) (Table 3).

3.6. Laboratory investigations

3.6.1. CBC "complete blood count"

The mean WBCs of cases was 8.107 ± 2.989 cell/cc and in controls it was 5.915 ± 1.814 cell/cc with significant increase in cases than controls ($P = 0.001$). Also, Hb concentration was 10.55 ± 0.96 gm/dl and in controls it was 13.61 ± 1.46 gm/dl with significant reduction of Hb concentration in cases than controls ($P = 0.022$). But There was no significant difference between both groups 'cases and controls' regarding platelets, ESR, cereatinine and FBS ($P = 0.270$, 0.935, 0.484 and 0.111, respectively (Table 4, Fig. 4).

3.6.2. Liver function tests

The mean AST of cases was 61.33 ± 20.31 U/dl and in controls it was 33.8 ± 7.46 U/dl with a significant

Table 3. Examination whether systemic or abdominal in cases and control groups in our study.

Variable	Cases ($n = 40$) No. (%)	Controls ($n = 20$) No. (%)	*P* Value
Systemic examination:			
Positive findings	40 (100 %)	0 (0.0 %)	0.001
Negative findings	0 (0.0 %)	20 (100 %)	0.001
P	0.001	0.001	
Abdominal examination:			
Positive findings	36 (90 %)	0 (0.0 %)	0.001
Negative findings	4 (10 %)	20 (100 %)	0.001
P	0.01	0.001	

P is significant if ≤ 0.05.

Table 4. Laboratory investigations 'CBC, ESR, FBS and creatinine' in both groups of our study.

Variable	Cases ($n = 40$)	Controls ($n = 20$)	P Value
WBCs ($x10^3$):			
Range	3–15	4.1–12	0.001
Mean ± S.D.	8.107 ± 2.989	5.915 ± 1.814	
Platelets ($x10^3$):			
Range	70–228	155–267	0.270
Mean ± S.D.	106.225 ± 32.037	206.75 ± 30.987	
Hb conc (gm/dl):			
Range	8–13	10–16	0.022
Mean ± S.D.	10.55 ± 0.96	13.61 ± 1.46	
ESR (mm):			
Range	15–45	18–37	0.935
Mean ± S.D.	26.63 ± 6.53	26.75 ± 5.08	
Creatinine (mg/dl):			
Range	0.6–1.4	0.48–0.85	0.484
Mean ± S.D.	1.02 ± 0.03	1.05 ± 0.13	
FBS (mg/dl):			
Range	70–215	70–120	0.111
Mean ± S.D.	103.2 ± 34.6	92.95 ± 14.38	

P is significant if ≤ 0.05.

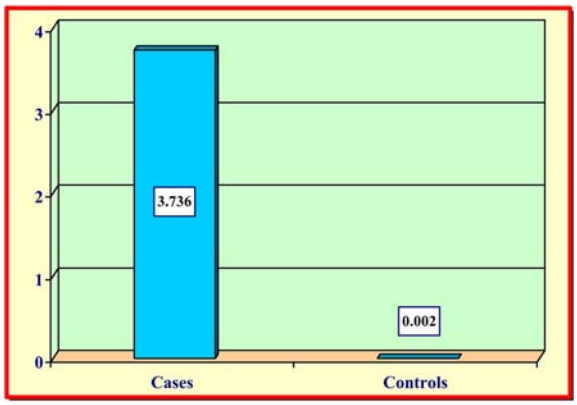

Fig. 4. PCR mean values in both groups of the study.

increase in cases ($P = 0.021$); the mean ALT of cases was 62.13 ± 21.03 and in controls was 32 ± 6.4 U/dl with significant increase in cases ($P = 0.021$); in addition the mean γGT of cases was 71.4 ± 17.83 mg/dl while in controls it was 36.55 ± 21.78 mg/dl with a significant increase in cases than controls ($P = 0.001$), (Table 5, Fig. 5). The mean Alkaline phosphate (ALP) enzyme in cases was 145.03 ± 36.89 U/dl, while in controls it was 60.0 ± 18.8 U/dl with a significant increase in cases than controls ($P = 0.001$) (Table 5).

The mean serum albumin of cases was 3.09 ± 0.43 gm/dl while in controls it was 4.03 ± 0.45 gm/dl with a significant reduction in cases than controls (0.021); the mean INR in cases was 1.54 ± 0.19 and in controls it was 1.04 ± 0.07 with marked increase in cases than controls ($P = 0.021$); also, the mean total bilirubin in cases was 2.02 ± 0.54 mg/dl and in controls it was 1.19 ± 0.16 mg/dl with significant increase of total

Table 5. Liver function tests in both groups of the study.

Variable	Cases ($n = 40$)	Controls ($n = 20$)	P Value
AST (IU/dl):			
Range	13–95	20–52	0.021
Mean ± S.D.	61.33 ± 20.31	33.8 ± 7.46	
ALT (IU/dl):			
Range	17–97	22–45	0.021
Mean ± S.D.	62.13 ± 21.03	32 ± 6.4	
AlP (IU/dl):			
Range	71–190	27–90	0.001
Mean ± S.D.	145.03 ± 36.89	60.6 ± 18.8	
γGT(mg/dl):			
Range	40–110	13–93	0.001
Mean ± S.D.	71.4 ± 17.83	36.55 ± 21.78	
Serum albumin (gm/dl):			
Range	2.3–4.5	3–5	0.021
Mean ± S.D.	3.09 ± 0.43	4.03 ± 0.45	
INR (%):			
Range	1.2–1.9	1–1.25	0.021
Mean ± S.D.	1.54 ± 0.19	1.04 ± 0.07	
Total bilirubin (mg/dl):			
Range	0.7–3.5	1–1.5	0.031
Mean ± S.D.	2.02 ± 0.54	1.19 ± 0.16	
Direct bilirubin (mg/dl):			
Range	0.3–2.6	0.71–0.92	0.002
Mean ± S.D.	1.15 ± 0.53	0.87 ± 0.06	

P is significant if ≤ 0.05.

bilirubin in cases than controls ($P = 0.031$) in addition the mean direct bilirubin in cases was 1.15 ± 0.53 mg/dl and in controls it was 0.87 ± 0.06 mg/dl with marked increase in cases than control ($P = 0.002$) (Table 5 Fig. 6).

3.6.3. Lipid profile

Regarding lipid profile; the mean total cholesterol (TC) of cases was 178.8 ± 31.64 mg/dl and in controls it was 165.05 ± 30.73 mg/dl with a nonsignificant

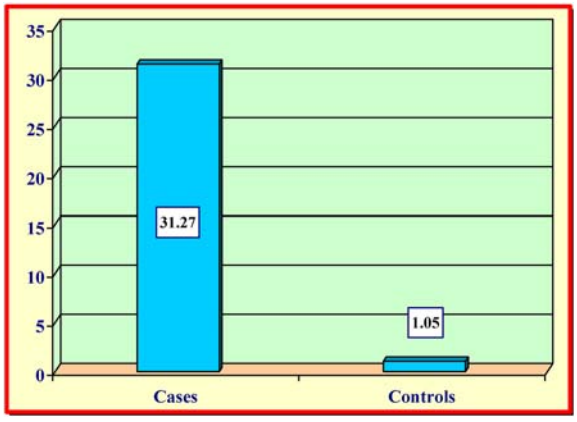

Fig. 5. AFP mean values in both groups of the study.

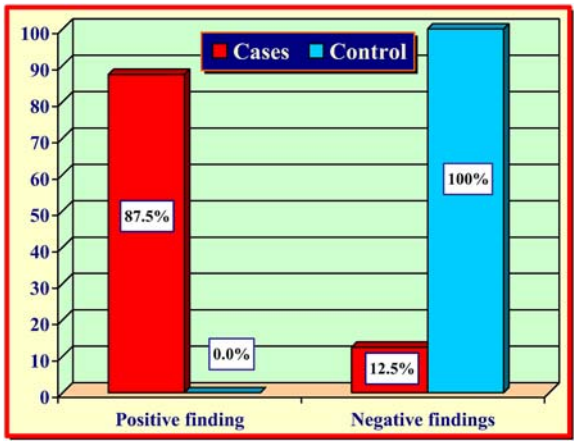

Fig. 6. Findings of abdominal U/S in both groups of our study.

Table 6. Lipid profile in cases and control groups of our study.

Variable	Cases ($n = 40$)	Controls ($n = 20$)	*P* Value
Total cholesterol (mg/dl):			
Range	53–240	110–220	0.113
Mean ± S.D.	178.8 ± 31.64	165.05 ± 30.73	
Triglycerides (mg/dl):			
Range	50–130	35–93	0.001
Mean ± S.D.	91.3 ± 19.84	55.35 ± 16.85	
LDL (mg/dl):			
Range	28–140	21–53	0.021
Mean ± S.D.	59.68 ± 24.15	35.3 ± 8.24	
HDL (mg/dl):			
Range	6–97	7–41	0.020
Mean ± S.D.	45.5 ± 21.2	22.15 ± 10.42	

P is significant if ≤ 0.05.

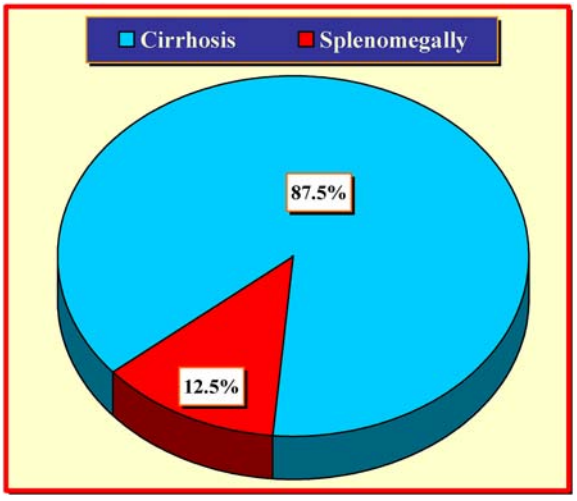

Fig. 7. The most common findings of abdominal U/S in both groups of our study.

difference between both groups ($P = 0.113$); the mean TG of cases was 91.3 ± 19.84 mg/dl and in controls was 55.35 ± 16.85 mg/dl with significant increase in cases ($P = 0.001$); in addition the mean LDL of cases was 59.68 ± 24.15 mg/dl while in controls it was 35.3 ± 8.24 mg/dl with a significant increase in cases than controls ($P = 0.021$); also, the mean HDL in cases was 45.5 ± 21.2 mg/dl and in controls it was 22.15 ± 10.42 mg/dl with significant increase in cases than controls ($P = 0.020$), (Table 6, Fig. 7).

3.6.4. Serological test

All cases and controls of the study (100 %) were negative for ANA-titre examination without significance between both groups ($P = 1.0$); also, both groups were negative for HBsAg without significant difference between both groups ($P = 1.0$) showed positive general examination signs and all controls (100 %) and the same thigh found for *H.Pylori* Ag testing as both groups were negative ($P = 1.0$) BUT all cases of the study were positive for HCV serology and all controls were negative to HCV serology with significant predominance of HCV infection in cases ($P = 0.001$) ($P = 0.001$) (Table 7).

3.7. Qualitative serological tests

3.7.1. PCR for HCV

When performing the PCR test for qualitative detection of HCV the values ranged between 0.47 and 50 with a mean of 3.736 ± 7.65 for cases while for controls its range was 0–0.03 with a mean value of 0.002 ± 0.006 with a significance increase in the PCR value in cases than controls ($P = 0.004$), (Table 8, Fig. 8).

3.7.2. Alpha feto-protein

When performing the alpha feto-protein the values ranged between 0.47 and 50 with a mean of

Table 7. Serological findings in both groups in our study.

Variable	Cases ($n = 0$) No. (%)	Controls ($n = 20$) No. (%)	*P* Value
ANA titre:			
Positive findings	0 (0.0 %)	0 (0.0 %)	1.0
Negative findings	40 (100 %)	20 (100 %)	
P	0.001	0.001	
HBsAg:			
Positive findings	0 (0.0 %)	0 (0.0 %)	1.0
Negative findings	40 (100 %)	20 (100 %)	
P	0.001	0.001	
HCV:			
Positive findings	40 (100 %)	0 (0.0 %)	0.001
Negative findings	0 (0.0 %)	20 (100 %)	
P	0.001	0.001	
H.Pylori Ag:			
Positive findings	0 (0.0 %)	0 (0.0 %)	1.0
Negative findings	40 (100 %)	20 (100 %)	
P	0.001	0.001	

P is significant if ≤ 0.05.

Table 8. Serological findings in both groups in our study.

Variable	Cases ($n = 40$) No. (%)	Controls ($n = 20$) No. (%)	*P* Value
Abdominal U/S:			
Positive findings	35 (87.5 %)	0 (0.0 %)	0.001
Negative findings	5 (12.5 %)	20 (100 %)	
P	0.01	0.001	
Upper GIT endoscopy:			
Positive findings	34 (85 %)	6 (30 %)	0.021
Negative findings	6 (15 %)	14 (70 %)	
P	0.01	0.031	

P is significant if ≤ 0.05.

31.27 ± 1.5 for cases while for controls its range was 0–4 with a mean value of 1.05 ± 1.07 with a significance increase in alpha feto-protein levels in cases than controls ($P = 0.002$), (Table 8, Fig. 9).

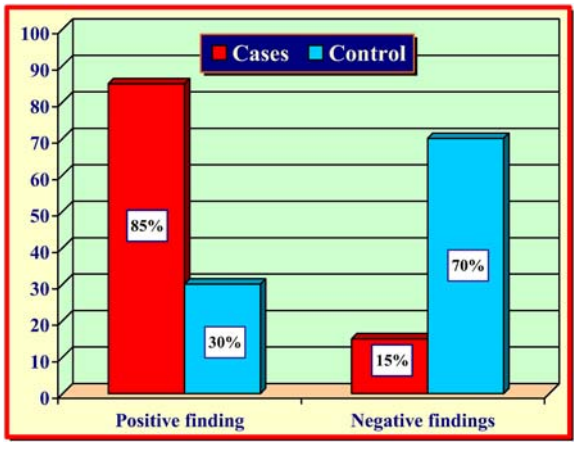

Fig. 8. Upper GIT endoscopic findings in both groups of our study.

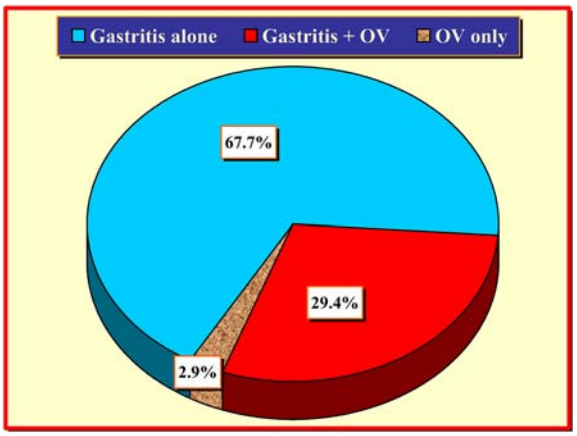

Fig. 9. The most common upper GIT endoscopic findings in both groups of our study.

4. Discussion

(HCV infection is one of the most serious global health problems. The incidence of HCV infection is increasing, with over 185 million people infected worldwide. Moreover, approximately 370,000 HCV-infected individuals die of liver-related causes each year, HCV-related liver disease can progress in an insidious manner over several decades. The advanced forms of the disease are LC and hepatocellular carcinoma (HCC). Approximately 20–30 % of subjects chronically infected with HCV are estimated to develop LC 15–25 years later. A recent systematic review found that, in HCV-infected patients with compensated LC, 2.8 %, 11.7 % develop hepatic decompensation, 1.8–8.3 % develop HCC, and 2.7–6.7 % die or undergo liver transplantation each year.[7–9]

Helicobacter pylori (*H. pylori*), is a Gram-negative spiralshaped bacteria that colonizes the gastric mucosa and can induce chronic gastritis, gastric ulcers, and gastric malignancy. Worldwide, about 50 % of the population is infected with *H. pylori*. Both developing and developed countries have a high incidence of *H. pylori* infection.[10]

Egypt has the highest prevalence of HCV in the world, estimated nationally at 14.7 %. The overall prevalence of *H. pylori* is high in developing countries and lower in developed ones. The overall reasons for these variations involve socioeconomic differences between populations. In Egypt, the prevalence is around 90 % in adults.[9,11]

In Egypt, infection with (HCV) is the most frequent reason of CLD. Thrombocytopenia develops in HCV patients due to a variety of causes, which could be categorized into those that cause reduced output, splenic sequester, and raised destruction.[12,13]

Most of cirrhotic patients are immunodeficient and all host systems are comprimized, for example, the acute phase response, macrophage, neutrophils, and lymphocyte function, so these patients are more prone to infections (e.g., *H. Pylori*) and it is seen that there is association between infections and the cirrhosis related complications, such as hepatic encephalopathy, variceal bleeding, peptic ulcer, and portal hypertensive gastropathy.[8]

LC and *H. Pylori* infection are two common diseases in Egypt and reducing the incidence of complications in cirrhotic subjects is an important step in current gastroenterology practice, early diagnosis and effective prevention of complications and its contributing factors will help in reducing mortality and morbidity in these patients, so considering the role of *H. Pylori* infection in cirrhosis related complications.[8]

This prospective cross sectional comparative clinical study carried out on 40 patients of confirmed HCV infection and twenty healthy subjects of the matched age and sex at Al-Azhar University Hospitals. And all cases were examined for the presence or absence of Helicobacter Pylori in both groups.

Demographic data revealed that the age of patients and controls was in the matched age worldwide without significance of any gender or BMI, with predominance of drug usage in our cases especially antidiabetics, antihyperlipidemic and β-blockers.

Attallah and his colleagues, (2022), revealed in their study that the age of cases was significantly increased in cases with HCV chronic infection with *H. Pylori* which disagree with our results but without significant difference regarding gender which was agreed with our results.[14]

Mohamed and his colleagues, (2020), in their study revealed that there was nonsignificant difference between cases and controls regarding Age, sex, and the age was in the matched age of the disease which run in lines with our finding in this study while there was a nonsignificant difference between both groups regarding diabetes mellitus, which disagree with our results.[8]

Mohammed and his coworkers, (2021), found in their study that the age of cases of HCV with *H. Pylori* was 54.8 years without predominance of any gender, which run in lines with our findings.[13]

Abdel-Razik and his colleagues, (2020), revealed in their study a nonsignificant difference between cases and control regarding Age, sex, and BMI, which run in lines with our results.[15]

Zaki and coworkers, (2016), found in their study that there was nonsignificant difference between both groups of the study regarding age and sex, which was in agree with our study.[11]

Laboratory investigations in our cases revealed significant increase in WBCs and significant decrease in Hb concentration in cases than controls. Also, there was decrease in platelets, ESR, elevation of blood sugar but in a nonsignificant manner.

Mohamed and his colleagues, (2020), in their study revealed that there was a significant decrease of platelets in cases than controls which run in lines with our finding.[8]

Zaki and coworkers, (2016), found in their study that there was a significant reduction of platelets count in group of cases, which was in agree with our study.[11]

Mohammed and his coworkers, (2021), found in their study that WBCs and Hb concentration didn't show significant change in cases of HCV with *H. Pylori* while platelets significantly reduced which disagree with what we found in our study.[13]

Chen and his colleagues, (2020), found in their study that there was significant increase in WBCs in cases of the study, which agreed with our findings in our results.[16]

Attallah and his colleagues, (2022), revealed in their study the significant reduction in platelets which agreed with our results.[14]

Abdel-Razik and his colleagues, (2020), revealed in their study a nonsignificant difference between cases and control regarding Hb concentration and platelets with significant increase in WBCs, which run in lines with our results.[15]

In our study, there were significant elevations in liver function tests as elevations of AST, ALT, GGT, ALP with significant elevation of INR, in addition to significant elevation of total and direct bilirubin with significant reduction in serum albumin.

Attallah and his colleagues, (2022), revealed in their study that liver enzymes (AST and ALT) showed significant increase in cases with HCV chronic infection with *H. Pylori* with significant decrease in serum albumin which agreed with our results but without significant elevation in the total bilirubin which disagreed with our results.[14]

Mohamed and his colleagues, (2020), in their study revealed that there was a significant elevation in AST, ALT and bilirubin in cases than controls with significant decrease in serum albumin in cases than controls which was in agree with our finding.[8]

Zaki and coworkers, (2016), found in their study that there was a significant elevation of ALT in group of cases which was agreed with our study.[11]

Mohammed and his coworkers, (2021), found in their study that laboratory investigations as liver function tests “AST, ALT, total and direct bilirubin, serum albumin” did not show significant change in cases of HCV with *H. Pylori* which conflicting with our findings.[13]

Chen and his colleagues, (2020), found in their study that liver enzymes and bilirubin were significantly elevated in NAFLD which run in lines with our finding while serum proteins "Albumin, globulin" did not show any significant change which conflicting with our results.[16]

Abdel-Razik and his colleagues, (2020), revealed in their study a non-significant difference between cases and control regarding liver function tests "AST, ALT, Total bilirubin, serum albumin and prothrombin time," which agreed with what we found in our results.[15]

Lipid profile in our study revealed significant elevation in TG, LDL, and HDL without significant change in cholesterol levels.

Chen and his colleagues, (2020), found in their study that significant elevation in the TC and significant decrease in HDL and LDL which conflicting with our results.[16]

In our study, serological tests significantly revealed HCV affection whether by ELISA or qualitative PCR and *H. Pylori* in our cases but without significance to prove HBV affection or ANA-titer. Also, revealed significant elevation of α-FP in our cases.

Attallah and his colleagues, (2022), revealed in their study the significant elevation of α-FP in cases which agreed with our results.[14]

Okushin and, (2018), in a large cross-sectional study in 2011, found positive correlations between *H. pylori* infection and other hepatic diseases such as chronic viral hepatitis, cirrhosis in cross-sectional studies and meta-analyses.[17]

Pogorzelska and his colleagues, (2017), found in their study the predominance of *H. Pylori* infections among patients with HCV infection which was agreed with our results.[6]

Zaki and coworkers, (2016), found in their study that there was a significant viral load for HCV and significant detection of *H. Pylori* antigen in stool of cases than controls which run in lines with our results (Zaki et al., *2016)*.[11]

Wang and his colleagues, (2016), in their meta-analysis study revealed that the significance of *H. Pylori* infection in patients with HCV infection, which run in lines with our results.[10]

Gutwerk and his colleagues, (2018), document in their study that the high serological *H. pylori* rate in younger patients, in particular those between 30 and 40 years, corresponds to other studies showing an elevation of *H. pylori* sero-prevalence in HCV patients; however, these data does not provide any evidence for potential interaction between either infection since *H. pylori* and HCV infections are acquired at different ages which conflicting with what we found in our study.[18]

Lee and his colleagues, (2018), found in their study that Anti-*H. pylori* antibody positivity was significantly and independently associated with cirrhosis in patients with HCV-related chronic hepatitis or cirrhosis in multivariate analyses.[19]

Abdel-Razik and his colleagues, (2020), revealed in their study a significant increase of α-FP in cases than control which run in lines with our results.[15]

Examination by Ultrasounds revealed to our cases revealed significant presence of cirrhotic changes, presence of splenomegally.

Attallah and his colleagues, (2022), revealed in their study the significant presence of fibrosis and cirrhosis in cases of *H. Pylori* with HCV, which agreed with our results.[14]

Upper GIT endoscopic examination in our study revealed a significant presence of gastritis, esophageal varices either in isolated forms or in combination.

Abdel-Razik and his colleagues, (2020), revealed in their study a non-significant difference between cases and control regarding endoiscopic finding during upper GIT endoscopic examination, which conflicting with our results.[15]

Pogorzelska and his colleagues, (2017), found in their study the predominance of gastritis in cases of LC accompanied with *H. Pylori*, which was agreed with our results but also with a non significant presence of esophageal varices, which disagree with our findings.[6]

4.1. Conclusion

From our study we can conclude that there was a positive association between *H. pylori* infection and CHC. In particular, we can also revealed strong correlations of *H. pylori* infection with HCV-related cirrhosis and HCV-related HCC.

Conflicts of interest

No conflict of interest.

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