

# Frequency of non alcoholic fatty liver in helicobacter pylori infected dyspeptic patients, is it a far reaching implication?

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## Research article

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# Abstract

**Background:** Fatty infiltration of the liver represents the intracytoplasmatic accumulation of triglycerides in the hepatocytes. We determined if *H. pylori* infection contributes to fatty liver infiltration and if it was related to gastric mucosal changes. As fatty liver disease is seen prevalent in non obese non alcohol consuming population of some asian countries. **Methods:** This cross-sectional study conducted between 22/11/2016 to 30/06/2018 in the Department of Medicine Aga Khan University Hospital. Eligible patients were enrolled after taking informed consent. Sociodemographic data were collected on age, gender, smoking and alcohol use, along with medical history related to dyslipidemia, hypertension, Type 2 diabetes mellitus, and ischemic heart disease. Fatty liver infiltrations were assessed using ultrasonography. *H.pylori* infection was detected by using carbon urea breath test (14C-UBT). **Results:** A total of 698 patients were enrolled during the study period with a mean age of  $44.01 \pm 16.03$  years and male patients were 373 (53%). A total of x patients were present with dyspepsia of the 299 (57%) of the patients were found to have positive *H. pylori* test. Of 299 patients with positive *H. pylori* test, 153 (20%) of the patients were diagnosed as having fatty liver infiltrations. *H. pylori* infection induced chronic active gastritis was associated with fatty liver infiltration in 62(71%) and absent in 200(64%) ( $P= 0.264$ ). **Conclusion:** The frequency of fatty liver infiltration in *Helicobacter Pylori* infected dyspeptic patients was 20%. Age and BMI are associated with fatty liver among dyspeptic *H. pylori* patients. **Keywords:** *H. pylori*, Fatty liver, BMI > 23; Type 2 diabetes. This is due to the incorporation of proinflammatory cytokines by the *helicobacter pylori* and its impact on gut microbiota leading to leaky gut and GUT-LIVER-AXIS interaction at different levels. Despite low BMI in our population, fatty liver is seen in connection with *helicobacter pylori* related gastric mucosal inflammation.

## Background

Fatty infiltration of the liver is a frequently reported finding on liver ultrasonography, a reflection of increased hepatic parenchymal echogenicity. Fatty change is the result of increased accumulation of small vacuoles of triglycerides around perinuclear location of liver parenchymal cells. It can be alcoholic or nonalcoholic in etiology in the background of obesity or metabolic syndrome, commonly known as non-alcoholic fatty liver disease (NAFLD). Epidemiological studies showed a NAFLD prevalence in general population of 9-37% worldwide [1]. This has given an ever-increasing prevalence of "Fatty Liver" in Asian countries, ranging from 12–24% in the general population [2]. Non-alcoholic fatty liver is strongly associated with several factors, notably type II diabetes, obesity, low HDL and high triglycerides. Premenopausal women possess a certain degree of safety which is lost in the post menopausal women. Male gender is more prone to get fatty infiltration of the liver even at a lean body mass, and the risk increases with the advancing age [3]. The situation is worse in South Asia, where there are the highest reported rates of overweight/obesity are seen (25% among men and 37% among women) [4-5]. In sharp contrast to other ethnic/racial groups, South Asians are more prone to show insulin resistance at a lower body mass index (BMI [ $\text{kg}/\text{m}^2$ ]); which is translated into their higher risk profile of metabolic disorders, they have a higher cardiometabolic disease prevalence [6-8]. This has prompted World Health Organization

(WHO) to establish new cutoffs for Asian population that are 18.5-<23.0, 23.0-27.5 and >27.5 kg/m<sup>2</sup> as normal, moderate and high respectively [9]. Susceptibility of weight gain related health risks and their deleterious effects are more pronounced in South Asians than most other ethnic/racial groups [6-8]. This has been taken into consideration by Indian health ministry by setting up more strict cutoffs of 23.0-25.0 and >25.0 kg/m<sup>2</sup> as overweight and obese respectively [10].

Significant metabolic impact posed by the non-alcoholic fatty liver disease (NAFLD) has spillover effects outside the liver. Our studied population with overweight and obesity have a basic lack of insight into the consequences of being overweight, with severe underestimation of metabolic disease burden. The group having a BMI between 23 and 27.5, have particularly common health-related consequences. Studies from Pakistan had shown a frequency of NAFLD of 14-15% however [11]. It is becoming a big health-related concern for South Asian population who are prone to insulin resistance, cardiovascular disease, and diabetes at a BMI lower than other ethnic/racial groups [12-13].

*Helicobacter pylori* (*H. pylori*) are Gram-negative, ubiquitous bacteria that colonize gastric mucosal epithelium of humans [14]. The percentage of *H. pylori* infection in the previous investigation conducted on dyspeptic patients in the Southern region of Pakistan showed 54% of seroprevalence [15]. Low-grade inflammatory response initiated by the *H. pylori*, induces specific mechanisms which express virulence peptides that resemble host antigens. Proinflammatory cytokine release is the main driver of host immune response with protean manifestation in the form of elevated tumour necrosis factor-alpha (TNF- $\alpha$ ) and other cytokines [16]. *H. pylori* becomes a horseman of this whole equation and develop an environment for its prolonged stay causing a chronic inflammatory state. This study aimed at exploring the association between *H. pylori* infection and fatty liver infiltration among patients with dyspepsia. Furthermore, the magnitude of gastric mucosal changes in these patients.

To our knowledge, ours is the first research study describing fatty infiltration of the liver in our dyspeptic patients with *H. pylori* infection population who are at a risk for obesity, type 2 diabetes, and cardiovascular diseases.

## Methods

### *Study population*

The study was approved by the institutional ethics board of Aga Khan University Hospital on 21st of November 2016, informed consent was taken. The study was conducted among adults who attended the clinic for symptoms of dyspepsia in the form of abdominal discomfort or pain, fullness and bloating and underwent an endoscopic examination of upper gastrointestinal tract from 22nd November 2016 to 30th June 2018. There were 325 (47%) females and 373 (53%) males with their age ranged from 18 to 90 year with a mean age of 44  $\pm$  16 years. The age range of male was 18-85 year and female 18-90 year. A history, physical examination, and baseline tests were done. Data was collected regarding anthropometric, complete blood count, fasting blood, glycated hemoglobin A1C (HbA1c), serum cholesterol, triglyceride

and result of *H. pylori* test. Body mass index (BMI) was calculated as weight in kilogram divided by the square of height in millimeter. The waist was measured between the iliac crest and rib cage. Modified criteria for South Asians was used for BMI calculation [17]. BMI was defined as “Healthy” to “23” and “Overweight” when greater than 23. There were 204(29%) patients with BMI up to 23 while 494(71%) had a BMI greater than 23. The fasting plasma glucose (FPG) level of 7.0 mmol/ l was the criteria used for Type 2 diabetes mellitus diagnosis [18]. The criteria used for labeling Hypertension was a blood pressure > than 140/90 mm of Hg as defined by the JNC 7 (Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure) [19]. Dyslipidaemia was defined as a disorder of lipid metabolism reflected in abnormal levels of cholesterol or triglycerides in the blood. Patient’s inclusion criteria included adults 18 and older with dyspepsia for more than six months. Celiac disease, inflammatory bowel disease, pregnant and lactating females, and those not giving consent or willing to participate in

the study were excluded. Written and verbal information about the study was received by all the participants. Patients with dyspeptic symptoms were investigated for *H. pylori* infection using either gastroscopy with biopsy and histology examination or <sup>14</sup>C-urea breath test (<sup>14</sup>C-UBT) [20]. *H. pylori* infection was detected in 399 (57%). The mean age of the two groups of patients was similar.

### ***Diagnosis of H. pylori infection***

*H. pylori* infection is diagnosed with the help carbon urea breath test (14C-UBT) commonly known as a urea breath test. For this, patients were asked to fast for six hours and wash their mouths before UBT. Patients are asked to take the test in sitting position and swallow a capsule containing 14C-urea (Helicap, Noster System AB Stockholm, Sweden) with water. This is followed by the collection of breath samples in a cartridge system (Heliprobe Breath Card, Noster System AB Stockholm, Sweden) after 10 min. The patients exhaled breath into the cartridge system which changed color to yellow from orange. Breath card was inserted into a  $\beta$ -scintillation counter (Heliprobe-analyser, Noster System AB Stockholm, Sweden) and activity was measured for 250 s. Results were expressed both as counts per minute (HCPM) and graded as (0: not infected, CPM < 25; 1: equivocal, CPM 25-50; 2: infected, CPM > 50) [21]. UBT is considered negative when grades 0 & 1 are the test result, so *H. pylori*-negative is the consideration given to test result.

### ***Histological analysis***

Gastric biopsies obtained at upper gastrointestinal endoscopy are fixed with formalin and embedded in paraffin were stained with eosin and hematoxylin stains for histological assessment and examination. The extent and degree of inflammatory activity were referred to as acute and chronic inflammation and scored as per the updated Sydney classification system [22]. The gastric mucosal infiltration by mononuclear cells and polymorphonuclear leucocytes, intestinal metaplasia and atrophy were graded as follows: 0, none; 1, mild; 2, moderate; 3, marked [22]. Inflammatory activity is said to be chronic, defined by increased infiltration of lymphocytes and plasma cells in the lamina propria and is graded into mild,

moderate or marked based on the density of cellular infiltrate. The activity of chronic gastric inflammation commonly reported as chronic active gastritis said to be present when neutrophilic infiltration of the lamina propria, pits or surface epithelium is encountered and graded as 0, nil; mild, <1/3 of pits and surface infiltrated; moderate, 1/3-2/3; and marked, >2/3. Gastritis as a whole, is the score calculated by total sum of grade of gastritis (mild, 1; moderate, 2; marked, 3 infiltrations with lymphocytes and plasma cells) and activity of gastritis (mild, 1; moderate, 2; marked, 3 infiltrations with neutrophilic leukocytes) either in the corpus or antrum[22].

### ***Diagnosis of fatty liver disease (FLD)***

Abdominal ultrasonography is the routine testing evaluation carried out for fatty liver infiltration. It routinely evaluates.g., liver, gallbladder, pancreas, kidneys, spleen, and abdominal aorta. Main determinants of fatty liver were described as a) an increase in the brightness of liver, b) an increase in the hepato-renal echo contrast pattern, c) existence of vascular blurring in the hepatic parenchyma, d) deep attenuation of hepatic echo, e) borderline blurring existing between liver and gallbladder, or right kidney and f) existence of focal hypoechoic lesion. For labeling a liver as fatty liver, ultrasonographic findings satisfy both a) and b) in addition to at least one of the findings between c) to f) [23]. The diagnosis was double-checked by the ultrasonographer and gastroenterologists.

### ***Statistical Analysis***

Statistical analysis was performed with SPSS 17.0 for Windows (SPSS, Chicago, IL). Continuous data are presented as mean  $\pm$  standard error of the mean (SE). Categorical data are presented as numbers. Independent samples *t*-test or Mann–Whitney test was used for comparison of continuous variables. Chi-square test or Fisher's exact test was used to compare categorical variables. Significance was set at  $P < 0.05$ .

## **Results**

A total of 698 patients were enrolled during the study period with a mean age of  $44.01 \pm 16.03$  years and male patients were 373 (53%). The baseline characteristics of the two study groups are similar. The *H. pylori* infection increased with age and plateaued in the 3<sup>rd</sup> to the 6<sup>th</sup> decade (Table 1). BMI was greater than 23 in the majority of patients. *H. pylori* infection was positive in 399 (57%). Majority of patients had mild chronic active gastritis demonstrated on histological examination of the gastric biopsy specimen (Table 1). The comorbidity factors associated with *H. pylori* infection included dyslipidemia 15%, type 2 diabetes in 14%, hypertension in 13% and ischemic heart disease in 12% (Table 1). Fatty change of the liver was documented in 22% (Table 1).

### ***Association of age with liver fatty infiltration in association with H. pylori***

The fatty change was present in 88 out of 399 (22.1%) *H. pylori* positive patients compared to 65/299 (21.7%) *H. pylori* negative patients ( $p=0.920$ ). In the age group of 31-50 years, the change was

present; in 31/399 (8.0%) of *H. pylori* positive patients compared to 15/299 (5.0%) in *H. pylori* negative patients and the difference again was not statistically significant ( $p=0.147$ ) (Table 2).

**Association of BMI with liver fatty infiltration in association with *H. pylori*** BMI >23 was seen in 319/399 (79.9%) of *H. pylori* positive patients compared with 175/299 (58.5%) *H. pylori* negative patients ( $p=0.000$ ). However, this relation could not be found with fatty change in the liver. In patients with a BMI of up to 23, fatty change was seen in 4/80 (5%) patients with positive *H. pylori* compared to 10/124 (8%) *H. pylori* negative patients ( $p=0.572$ ). In patients with BMI > 23, the fatty change was seen in 84/319 (26.3%) patients who were positive for *H. pylori* compared to 55/175 (31.5%) in *H. pylori* negative patients ( $p=0.572$ ).

(Table 2).

#### **Association of comorbid with liver fatty infiltration in association with *H. pylori* infection**

Dyslipidaemia, type 2 diabetes, hypertension and ischemic heart disease were associated with liver fatty infiltration irrespective of *H. pylori* infection status, with p-value of < 0.001 in each case (Table 2).

#### **Association of the grade of gastritis with fatty infiltration with *H. pylori* infection**

Moderate gastritis was associated with fatty liver change irrespective of *H. pylori* status. (Table 2).

## **Discussion**

This study demonstrated that BMI >23 was associated with *H. pylori* infection. However, this study could not demonstrate the association of fatty change in the liver with *H. pylori* infection though there was an association of moderate to severe gastritis with the fatty change in the liver irrespective of *H. pylori* status.

Pathogenesis of fatty liver infiltration has been linked to an increase in free fatty acids. It occurred at an early age in the presence of the *H. pylori* infection whereas in the absence of *H. pylori* infection, fatty infiltration of the liver was demonstrated in the 5<sup>th</sup> decade of life (Table 2). This is in keeping with the local prevalence of the *H. pylori* infection [15] that showed the seroprevalence of the *H. pylori* increased with age [15]. Fatty infiltration of the liver was not related to the gender of the patients as was the seroprevalence of *H. pylori* infection that was not associated with the gender distribution [15]. BMI greater than '23' with *pylori* infection were associated with a higher incidence of the liver fatty infiltration in 84 (96%) as compared to the 55 (85%) in patients without *H. pylori* infection (Table 2). This is in agreement with a previous study showing a high BMI was associated with an increased risk of the *H. pylori* infection [24].

In this study, dyslipidemia and type 2 diabetes were associated with *H. pylori* infection and a significant liver fatty infiltration (Table 2). Both dyslipidemia and type 2 diabetes are individually known to be associated with fatty liver infiltration. *H. pylori* infection was significantly associated with dyslipidemia

(Table 2). This is consistent with the results of an earlier study that showed *H. pylori* infection was significantly associated with higher total cholesterol level (coefficient = 2.114,  $P < 0.001$ ), higher LDL-C level (coefficient = 3.339,  $P < 0.001$ ) and lower HDL-C level coefficient =  $-1.237$ ,  $P < 0.001$ ) [25]. A higher prevalence of *H. pylori* in patients with type 2 diabetes has also been described. The prevalence of *H. pylori* infection was greater and described as 61.7% and 58.5 %, among type 2 diabetes and nondiabetics, respectively [26]. In our study, the moderate grade of gastritis and chronic active gastritis was associated with fatty liver infiltration with and without the *H. pylori* infection. *H. pylori* induces up regulation of several cytokines such as TNF- $\alpha$ , C-reactive protein and interleukin (IL)-1 $\beta$ , due to chronic low-grade inflammatory activity, which may influence pancreatic  $\beta$  cell secretion and brings about a state where insulin action is altered. The milieu of autocrine and paracrine effects induced by *H. pylori*-induced gastritis have some indirect effects in the form of alteration of gastric hormones secretion, including gastrin, somatostatin, leptin, and ghrelin which could affect glucose homeostasis, insulin action and sensitivity.

*H. pylori* are the culprit of peptic ulcer disease, several gastric and extragastric manifestation other than gastritis. Above all, the gastric malignancies are the most fearsome of all. DNA of gastric mucosal cells are vulnerable to the oxidative damage induced by the reach and free oxygen species by the *H. pylori* infection generated inflammation and accumulation of these toxic free radicals. *H. pylori* induced infiltration of the gastric mucosa and activation of neutrophils and macrophages leads to the generation of large amounts of reactive oxygen species (ROS) produces inflammation [28, 29]. The association of *H. pylori* infection with severe ongoing oxidative stress modulate many processes in gastric mucosa. Whether due to resistance to its action or deficient levels, alteration in the leptin action, is a co-producer of steatosis. There is a strong role of TNF- $\alpha$  in the early stages of fatty liver as evidenced by several animal studies and data from clinical models as well. More than 50% of the patients have derangement of liver enzymes and found to have simple steatosis as the cause for it. This is because of the asymptomatic course of fatty liver disease, most of them discovered incidentally on the workup of hepatomegaly and/or abnormal liver biochemical profile [30]. In the nonalcoholic variant, the predominant elevation of liver biochemical tests is seen in alanine transaminase levels [31]. In conclusion, in our population, there was a strong association seen in 30-50 years age group which is an early onset of fatty liver infiltration in *H. pylori*-infected dyspeptic patients. A BMI > than 23, dyslipidemia and type 2 diabetes in the context of *H. pylori* infection have a greater predisposition to fatty liver. *H. pylori* bring about an intense milieu of proinflammatory cytokines and it is supposed to be having a close interaction with gut microbiota, further prospective studies in this regards need to be done to understand this situation more clearly.

## Declarations

**Ethical approval and consent to participate:** This study was approved by the Ethical Review Committee of Aga Khan University Hospital, Karachi, Pakistan, on 21st of November 2016, ethical approval number granted was 4532-Med-ERC-16.

All procedures performed in this study were in accordance with the ethical standards of our institutional research committee (Ethical Review Committee, Aga Khan University Hospital, Karachi, bearing reference number 4532-MED-ERC-6) and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent was obtained from all individual participants included in the study.

**Consent for publication:** Authors obtained consent for publication by using the data collection form approved by the Ethical Review Committee of our University Hospital.

**Availability of data and materials:** Data set used during the current study are available from the corresponding author on reasonable request.

**Competing interest:** Authors declare that they have no competing interests.

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#### **Authors contributions:**

Basit Siddiqui has made contributions to conception and design, interpretation of data, drafting the manuscript and revising it critically for important intellectual content. Rabeea Azmat has made contributions to conception and design, interpretation of data, drafting the manuscript and revising it critically for important intellectual content. Javed Yakoob has made a contribution to conception, acquisition, and interpretation of data and drafting the manuscript. Zaigham Abbas has made contributions to critically analyzing the data and manuscript. Shiyam Sundar Tikmani has made a contribution in acquisition and interpretation of data and final revision. Zain Mushtaq has made a contribution in data collection. Saad Bin Zafar has made a contribution in data collection. Muhammad Tahir Khan has made contributions to design, drafting of the manuscript and revising it critically for important content.

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## Tables

Table 1. Clinical details of the patients studied

Age (year)	18-30	159(23)
	31-50	224(32)
	51-65	244(35)
	65 and >	71(10)
Gender	Male	373(53)
	Female	325(47)
BMI	Upto 23	204(29)
	23 and >	494(71)
Dyslipidemia	Positive	105(15)
	Negative	593(85)
Type 2 Diabetes	Positive	100(14)
	Negative	598(86)
Hypertension	Positive	89(13)
	Negative	609(87)
Ischemic Heart Disease	Positive	81(12)
	Negative	617(88)
Fatty infiltration liver	Positive	153(22)
	Negative	545(78)
Histology		
Helicobacter pylori	Positive	399(57)
	Negative	299(43)
Gastritis	Mild	368(53)
	Moderate	330(47)
Grade	Chronic active gastritis	488(70)
	Chronic inflammation	201(30)

Table 2 . Association of patients clinical details with liver fatty infiltration in association with and without *H. pylori*

		Helicobacter pylori positive (n=399)			Helicobacter pylori negative (n=299)		
		Fatty infiltration liver			Fatty infiltration liver		
		Positive	Negative	P value	Positive	Negative	P value
Age (year)	18-30	5(6)	73(23)	< 0.001	3(5)	78(33)	< 0.001
	31-50	31(35)	127(41)		15(23)	51(22)	
	51-65	37(42)	88(28)		40(61)	79(34)	
Sex	65 and >	15(17)	23(7)	0.218	7(11)	26(11)	0.854
	Male	41(47)	168(54)		35(54)	129(45)	
	Female	47(53)	143(46)		30(46)	105(76)	
BMI	Upto 23	4(4)	76(24)	< 0.001	10(15)	114(49)	< 0.001
	23 and >	84(96)	235(76)		55(85)	120(51)	
Hypertension	Positive	36(41)	27(9)	< 0.001	15(23)	11(5)	< 0.001
	Negative	52(59)	284(91)		50(77)	223(95)	
Type 2 Diabetes	Positive	46(52)	27(9)	< 0.001	15(23)	12(5)	< 0.001
	Negative	42(48)	284(91)		50(77)	222(95)	
Dyslipidemia	Positive	47(53)	26(8)	< 0.001	16(25)	16(7)	< 0.001
	Negative	41(47)	285(92)		49(75)	218(93)	
Ischemic Heart Disease	Positive	33(37)	19(6)	< 0.001	16(25)	13(6)	< 0.001
	Negative	55(63)	292(94)		49(75)	221(94)	
Histology							
Gastritis	Mild	39(44)	181(58)	0.021	21(32)	127(54)	0.002
	Moderate	49(56)	130(42)		44(68)	107(46)	
Grade	Chronic active	62(71)	200(64)	0.264	55(85)	171(73)	0.055
	gastritis						
	Chronic inflammation	26(29)	111(36)		10(15)	63(27)	