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Research Article

FACTORS ASSOCIATED WITH GASTRO-DUODENAL DISEASE IN PATIENTS UNDERGOING UPPER GI ENDOSCOPY AT THE ST. DOMINIC HOSPITAL, AKWATIA

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A B S T R A C T

Background: Symptoms associated with gastro-duodenal disease are among the commonest complaints for which patients seek medical attention, with the annual prevalence of dyspepsia approximating 25%. Limited data exists regarding its associated risk factors despite accumulating evidence indicating that gastro-duodenal disease is common in Ghana. The aim of this study was to document the risk factors associated with gastro-duodenal disease of patients undergoing gastroscopy at the district hospital in Ghana.

Methods and Material: The study used a cross-sectional design to consecutively recruit patients referred to the Endoscopy Unit of the St. Dominic Hospital (SDH) with symptoms of gastro-duodenal disease during the study period. The study questionnaire was administered to study participants. Helicobacter pylori(H. pylori) infection was confirmed by rapid-urease examination at endoscopy.

Results: A total of 429 patients who undergone gastroscopy during the study period were included, of those, 187(43.6%) were males and the median age of 46(33, 62). About half (53.2%) of the patients had a history of NSAIDs use and 50.6% tested positive to H. pylori. Gastric ulcer had strong statistically significant associations with cigarette smoking (cOR=7.16, p= 0.012) NSAIDs use(cOR=3.74, p= 0.001) and combination of H. pylori and NSAID use(cOR= 4.79, p= 0.01) however duodenal ulcer and gastric cancer did not achieve the requisite significance level with H. pylori

Conclusion: H. pylori alone was not a major risk factor for gastro-duodenal disease. However, NSAIDs-related gastric ulceration has been shown to be common in H. pylori infected patients. It highlights the need for awareness of the adverse gastro-duodenal effects of NSAIDs.

INTRODUCTION

Symptoms associated with gastro-duodenal diseases are among the commonest complaints for which patients seek medical attention, with the annual prevalence of dyspepsia approximating 25%.[1] Gastro-duodenal disease are leading causes of morbidity and mortality globally. Peptic ulcer disease (PUD), gastro-duodenitis and gastric cancers affect millions of people worldwide.[2] A study conducted at the premier Teaching Hospital in Ghana, reviewed 6977 upper gastro-intestinal (GI) endoscopies between January 1995 and December 2002. Chronic duodenal ulcer (DU) 19.6%, gastritis 12.7%, duodenitis 10.2%, oesophagitis 7.5%, gastric ulcer 3.5% and gastric cancer 2.3% were the most frequent pathologic diagnoses.[3] Adam Gyedu *et al*. in their study in Kumasi, Ghana, reported gastro-duodenitis 27.4%, gastric ulcer 4.7%, duodenal ulcer 3.0% and gastric cancer 2.1% as common pathologic findings among patients with dyspepsia who undergone upper GI endoscopy.[4] A recent study conducted in a district hospital in Ghana among patient with dyspepsia, gastritis 44.3%, duodenitis 10.4%, duodenal ulcer 21.7%, gastric ulcer 18.9% and gastric cancer 10.4% were the common diagnosis made after endoscopy.[5] H pylori and the use of NSAIDs or aspirin are the main risk factors of both gastric and duodenal ulcers.[6,7-9] However, only a few people with H pylori infection or taking NSAIDs or aspirin develop peptic ulcer disease, suggesting that individual susceptibility to bacterial virulence and drug toxicity is essential to the initiation of mucosal damage. Limited data exists regarding the use of NSAIDs and inter-relationship with other important factors such as *H. pylori* in addition to smoking and alcohol

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consumption, despite accumulating evidence indicating that gastro-duodenal disease is common in Ghana especially in the rural areas. The aim of this study was to document the risk factors associated with gastro-duodenal diseases of patients undergoing upper GI endoscopy at the district hospital in Ghana. Identifying the risk factors will help managed and educate patients to prevent gastro-duodenal disease or its recurrence.

MATERIALS AND METHODS

Ethical Approval

A formal approval of this study was obtained from the Ethical and Protocol Committee of the University of Ghana School of Medicine and Dentistry. This study was conducted in accordance with the Helsinki Declaration

Participants Recruitment

The study used a cross-sectional design to consecutively recruit medical in-patients and clinic outpatients referred to the Endoscopy Unit of the St. Dominic Hospital (SDH) with symptoms of gastro-duodenal disease from 14th January, 2018 to 26th June, 2019.

Study site

SDH was founded in 1960 and has 339 beds and is the district hospital of Denkyembour district, Akwatia in Eastern region of Ghana. It is the main referral centre for other surrounding district hospitals. It offers a breadth of medical and surgical services including gastroenterology and endoscopy. The Endoscopy Unit is manned by a medical gastroenterologist with the support of trained nurses and auxiliary staff. It uses Olympus and video endoscopy equipment for endoscopic procedures. It runs endoscopy sessions twice per week and offers both upper and lower GI endoscopy services. Each session performs approximately 5 upper endoscopies and 1 lower GI endoscopy. Procedures performed are both diagnostic and interventional. The latter include variceal band ligation

Procedures

Study participant recruitment and data collection was performed at the Endoscopy Unit, SDH, between January 2018 and June 2019. Medical in-patients and clinic outpatients with symptoms of gastro-duodenal disease referred to the Endoscopy Unit, SDH were enrolled into the study. Study participants were consecutively recruited each week from endoscopy unit. All patients were given explanatory statements of the project and consented prior to endoscopy. Non-consenting patients were excluded from the analysis. Patients with previous H. pylori eradication treatment or proton-pump inhibitor (PPI)-use two weeks preceding endoscopic analysis and patients with oesophageal disease were also excluded from further study. Demographic data of patients taken included age, sex, occupation etc. Gastro-intestinal symptoms, medications such as NSAID use, alcohol intake and smoking history were taken. Reported gastrointestinal symptoms included dyspepsia as well as vomiting, anorexia, upper GI bleeding (melena/hematemesis), weight loss and dysphagia. Dyspepsia was defined as one or more of the following symptoms: post-prandial fullness, early satiation (inability to eat a normal sized meal) and epigastric pain/burning according to the Rome III criteria.[10] UGIE was performed using the Olympus CV-160 videoscope. Study participants were given the option of sedation with (intravenous midazolam 2mg) and/ or 10% lidocaine (xylocaine) throat spray. H. pylori infection was determined by the rapid-urease-campylobacter like-organism (CLO) test on gastric antral and body biopsies at UGIE (specificity 98%, sensitivity >93%; Cambridge Life Sciences Ltd, Cambridge, UK. Endoscopic findings per each participant were recorded in details.

Statistical Analysis

Data were analysed with STATA 15. Descriptive statistics were used to characterize patient demographic features. Continuous variables were presented as median (interquartile range) and categorical data were summarized using proportions. The Chi square and the Fishers exact test (where appropriate) of independence was performed to examine the association of different endoscopic findings with the presence of risk factors. A univariable logistic regression analysis was conducted to determine the factors that were associated with endoscopic findings. A p-value less than 0.05 was considered significant.

RESULTS

Socio-demographics characteristics and indications for endoscopy

Table 1 and 2 shows the socio-demographics and indications for endoscopy of the study population. A total of 429 patients who had upper GI endoscopy during the study period were included, of those, 187 (43.6%) were males giving a male to female ratio of 1:1.3. Their ages ranged from age 4 to 94 years, with a median age of 46 (33, 62).

Table 1 Baseline characteristics of study participants

Socio-demographic and clinical Presentations	Frequency (%)
Sex (n=429)	
Male	187 (43.6)
Female	242 (56.4)
Age (years)	
Overall	46 (33, 62)*
Males	45 (34, 60)*
Females	46.5 (31,62)*
Alcohol	32 (7.5)
Smoking	7 (1.6)
NSAIDs use	228 (53.2)
H. pylori infection	217 (50.6)

*median (IQR)

Table 2 Indications for endoscopy

Indications	Frequency (%)
Dyspepsia	371 (86.5)
UGIB	68 (15.9)
Vomiting	10 (2.3)
Weight Loss	11 (2.6)
Dysphagia	2 (0.5)
Others	3 (0.7)

*Multiple response analysis (total >429)

More than 80% (371/429, 86.5%) of patients had dyspepsia, the commonest clinical presentation encountered, Table 1. Other symptoms included vomiting (2.3%), haematemesis and/or melaena (15.9%), Dysphagia (0.5%), weight loss (2.6%). About half (53.2%) of the patients had a history of NSAIDs use and 50.6% tested positive to H. pylori. Only 7.5% and 1.6% had a history of alcohol and tobacco used respectively.

Endoscopic diagnosis

Table 3 shows endoscopic diagnosis of the participants. The major endoscopic diagnoses were gastritis which occurred in 321 (74.8%) patients followed by duodenitis (157/429, 36.6%), gastric ulcer (GU) (43/429, 10%), normal findings (90/429, 6.8%) duodenal ulcer (40/429, 9.3%) (9.3%) and gastric cancer (11/429, 2.6%)

Table 3 Endoscopic findings of the participants

OGD Findings	All patients (n=429)	95 % CI
All endoscopic abnormalities	399 (93.2)	90.4 - 95.2
Inflammatory conditions		
Gastritis	321 (74.8)	70.4 - 78.7
Duodenitis	157 (36.6)	32.0 - 41.1
Peptic Ulcer		
Gastric ulcer	43 (10.0)	7.5 - 13.3
Duodenal Ulcer	40 (9.3)	6.9 - 12.5
Malignancy		
Gastric Ca	11 (2.6)	1.4 - 4.6

*Multiple response analysis (total >429)

Factors associated with gastro-duodenal disease

There was a strong association between gastro-duodenitis and age ≥ 50 years (p = 0.001); GDs were also mor common in males (cOR =0.5, p = 0.003), cigarette smokers (cOR=0.1, p = 0.009) and NSAIDs use (cOR=0.59, p = 0.028) (Table 4). Conversely, GDs did not demonstrate a significant relationship with H. pylori and alcohol used (Table 4). Gastric ulcer had strong statistically significant associations with cigarette smokers (cOR=7.16, p= 0.012), age ≥ 50 years (cOR=4.06, p= <0.0001), NSAID use (cOR=3.74, p= 0.001) and combination of H. pylori and NSAID use (cOR= 4.79, p= 0.01) however duodenal and gastric cancer did not achieve the requisite significance level with H. pylori (Table 5 and 6). There was statistically significant association between duodenal ulcer and male sex (p=0.005), Age ≥ 50 years (p=<0.0001) and NSAID use (p= 0.028) (Table 5).

Table 4 Regression analysis: H. pylori infection, NSAID-use, Alcohol, Smoking, Age, Sex and Gastro-duodenitis

Variables	Gastro-duodenitis	
	cOR (95% CI)	p- value
Male Sex	0.50 (0.32 - 0.80)	0.003
Age ≥ 50 years	0.47 (0.29 - 0.74)	0.001
Alcohol	1.02 (0.43 - 2.43)	0.97
Smoking	0.10 (0.02 - 0.57)	0.009
H. pylori	1.02 (0.63 - 1.58)	0.99
NSAIDs	0.59 (0.37 -0.94)	0.028
H. pylori and NSAIDs	0.61 (0.34 - 1.09)	0.093

Table 5 Regression analysis: H. pylori infection, NSAIDs-use, Alcohol, Smoking, Age, Sex and Peptic ulcer disease

	Gastric ulcer		Duodenal Ulcer	
Variables	cOR (95% CI)	p- value	cOR (95% CI)	p- value
Male Sex	1.73 (0.92 - 3.3)	0.091	2.63 (1.33 - 5.19)	0.005
Age ≥ 50 years	4.06 (2.02 - 8.16)	< 0.0001	3.60 (1.77 - 7.29)	< 0.0001
Alcohol	1.31 (0.44 - 3.93)	0.629	2.46 (0.95 - 6.40)	0.064
Smoking	7.16 (1.55 - 33.14)	0.012	4.04 (0.76 - 21.54)	0.102
H. pylori	0.83 (0.44 -1.57)	0.574	1.52 (0.78 - 2.96)	0.213
NSAIDs	3.74 (1.74 - 8.0)	0.001	2.21 (1.09 - 4.46)	0.028
H. pylori and NSAIDs	4.79 (1.45 - 15.82)	0.01	1.59 (0.68 - 3.70)	0.285

Table 6 Regression analysis: H. pylori infection, NSAID-use, Alcohol, Smoking, Age Sex and Gastric cancer

	Gastric Ca	
Variables	cOR (95% CI)	p- value
Male Sex	6.07 (1.30 - 28.43)	**0.022**
Age ≥ 50 years	3.78 (0.99 - 14.44)	0.052
Alcohol	1	-
Smoking	1	-
H. pylori	0.55 (0.16 - 1.91)	0.346
NSAIDs	2.4 (0.63 - 9.17)	0.201
H. pylori and NSAIDs	1.47 (0.31 - 6.92)	0.625

DISCUSSION

In this study, there were no significant association between H. pylori infection and peptic ulcer disease. However, previous studies reveal a high incidence of H. pylori infection in patients with DU;[11] subsequent reviews confirmed that H. pylori is detectable in 80 - 95 percent of these patients.[11] While the association between H. pylori and DU is strong, it is not specific as it is also causally linked with GU.[13] The reasons may be that negative test for H pylori were false negatives because patients were probably on PPI or antibiotics bought from over the counter but were not aware. Although these were part of the exclusion criteria. Studies shows that PPI and the unrelated use of antibiotics may suppress, although not eradicate, the microorganisms, rendering a false negative Helicobacter diagnosis.[14-15] In addition, studies conducted in United State of America shows that most patients with peptic ulcers are infected with H. pylori, but may not be responsible for the ulcer. The H. pylori infection may not have caused the peptic ulcer in the previous study as was originally thought to have done so. This may be another reason why there were no association between H. pylori infection and peptic ulcer disease in this study.

Risk factors significantly associated with GDs in this study includes male sex, age ≥ 50 years, cigarette smoking and NSAIDs used. These findings are similar to previous studies published in the country and other countries.[16-17]

Gastric cancer (GCA) did not achieve statistical significance with H. pylori in this study. This is similar to previous studies conducted in Accra, Ghana, which did not find an association between H. pylori and gastric cancer.[16,18] On the contrary, epidemiological studies demonstrate a strong correlation between H. pylori infection and non-cardia gastric cancer.[19] Additionally, adenocarcinoma has been shown to be more strongly associated with H. pylori in comparison with diffuse-type GCA.[20-21] The reasons for non-association of H. pylori and gastric cancer in this study and the previous studies may be due to the fact that the number of GCA cases were relatively small, possibly under-estimating the H. pylori prevalence. Moreover gastric cancer demonstrates marked geographic variability, both regionally and within countries, which may relate to differences between the H. pylori strain virulence and its interaction with host response and genotypes, diet, and environmental factors.[22-24] It is likely that the environmental or genetic factors, or both, which cause the change from gastritis to intestinal metaplasia and subsequently dysplasia and cancer are not present in our participants or Ghana

Two hundred and twenty eight (228, 53.2%) of the participants had a history of NSAID-use, mainly as an analgesic for musculoskeletal pain. This is higher than 32.6% reported in a

study conducted at Korle-Bu Teaching Hospital in Accra, Ghana.[16] Study in North-American reported that 27% of elderly people were prescribed NSAIDs over a six-month period.[25] The frequency of its use was reportedly higher in another study, where 40% of elderly people were taken NSAIDs.[25] NSAIDs damage the gastroduodenal mucosa through both systemic and local mechanisms, but the systemic inhibition of constitutively expressed cyclooxygenase 1 (COX-1)-derived prostaglandins are regarded as the main mechanism. Reduced mucosal prostaglandin values are associated with low mucus and bicarbonate secretion, inhibition of cell proliferation, and decreased mucosal blood flow, which are essential to maintenance of mucosal integrity. NSAIDs initiate mucosal damage in the cell through disruption of mucus phospholipids or the cell membrane and by uncoupling of mitochondrial oxidative phosphorylation. The loss of mucosal integrity is followed by tissue reaction amplified by luminal content such as acid, pepsin, food, bile, and H pylori.[26-28] Therefore, COX derived prostaglandin inhibition, vascular damage, and topical effects are the main players in the pathogenesis of ulcers caused by NSAIDs. In this study, there were significant association between peptic ulcer disease and NSAIDs used. This is similar to report of previous study in this country.

We also found a statistically significant association between NSAID-use and GU but not DU in H. pylori-positive patient. This is contrary to previous study by Acheampong *et al*,[16] they reported association between DU but not GU in patients infected with H, pylori and using NSAIDs at the same time. The interaction between NSAID-use, H. pylori and peptic ulcer disease is controversial: The role of H. pylori in NSAID-naïve patients seems to be different from those on long-term therapy. Randomised controlled trials have shown that eradication of H pylori is beneficial in patients who start taking NSAIDs but not in those who are on long-term NSAID treatment.[29] H. pylori therefore exert a greater influence on ulcer risk on initiation of NSAID therapy than during long-term use.

There was a significant association between cigarette smoking and gastric ulcer in this study. This is contrary to study by Acheampong *et al*.[16] which smoking did not show any demonstrable association with gastric ulcer. Cigarette smoking is considered to be one of the major contributors to ulcer disease. According to study conducted in US, current and former smokers is almost doubled that of never smoked. According to clinical observations, cigarette smokers are more likely to develop ulcers which are more difficult to heal.[30] The risk of peptic ulcers also increases in smokers who have a large daily intake of tobacco compared with never smokers.[31] However there were no association between alcohol users and peptic ulcer disease.

In conclusion, NSAID, male sex, age ≥ 50 years, NSAID and H. pylori infection in combination and cigarette smoking were the main risk factors associated with GDs and GU in this study. The risk factors associated with DU were NSAID, male sex and age ≥ 50 years. H. pylori alone were not associated with gastro-duodenal diseases in this study. This strengthens the need for awareness on the adverse GI effects of NSAID.

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