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Research Article

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Assessing CA Stomach and H. Pylori Relationship: Outcomes and Implications

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Abstract: Background: The outlook for patients with stomach cancer is generally poor, with gastric cancer being the second most common cause of cancer death. Although advances in diagnosis and treatment have led to a drastic decrease in the incidence of gastric cancer in developed countries in the past few decades. Aim: Our current study aims to evaluate the clinical and pathological outcomes associated with the effect of H. pylori on gastritis (gastric cancer) patients. Methods: To evaluate the association between gastritis (gastric cancer) and H. pylori, demographic and diagnostic parameters of 103 patients were recorded during the follow-up period, which ranged from October 2023 to October 2024, In several hospitals in Iraq. Our study detected the degree and severity of H. pylori by polymerase chain reaction. The quality of life of patients was assessed by a general health assessment (SF - 36) questionnaire, and a curve was made to show the survival rate of patients during the follow-up period. Results: Our outcomes found patients with ages above 45 years were the most group participated, with 42.72%, males with 62 cases and females with 41 cases, obesity with 42.72%, smokers with 36.89%, and alcohol consumption with 12.62%. According of gastric cancer, the upper tumor got 40.78%, tumor size had 6.12 ± 1.42, lymph node metastasis of N1 was 32.04%, N3 was 11.65%, and N2 was 18.45%. Clinical of tumor signs were CEA ≥6 µg/L include 86.41%. Conclusion: Based on our outcomes, gastric cancer remains a large global health problem because H. pylori implicates it as a major risk factor for it.

Keywords: Gastric Cancer; H. Pylori; Lymph-node metastasis; Tumor signs; and Cum survival.

INTRODUCTION

With a remarkable contribution in cancer-related morbidity and death, gastric cancer, also referred to as stomach cancer, is one of the most prevalent and fatal malignancies globally. There is still much to learn about the genesis and risk factors of gastric cancer since, despite advancements in detection along with treatment, the prognosis is still poor, particularly when the disease is advanced (Garcia, J.A.M., and Ward, E.M 2007; Xu, D.Z., and Geng, Q.R 2009; Marrelli, D., and De Stefano, A 2005; Kattan, M.W., and Karpeh, M.S 2003; NCCN 2009).

One of the most prevalent chronic bacterial infections is H. pylori, a Gram-negative bacterium which colonizes the stomach mucosa in about half of the world's population (Marshall, B.J., and Warren, J.R 1984; IARC 1994; Group HaCC 2001). Many individuals do not experience symptoms at all, but others get peptic ulcers, chronic gastritis, and sometimes stomach cancer (Tajima, K 2002; Parsonnet, J., and Friedman, G.D 1997; Honda, S., and Fujioka, T 1998; Correa, P., and Fontham, E.T 2000;Uemura, N., and Okamoto, S 2001).

The results from H. pylori infections were also predominantly influenced by the cumulative effect of certain bacterial virulence factors, innate genetic predisposition of the host, and outside environment components such as diet and smoking (Ohnishi, N., and Yuasa, H 2008).

Elimination of H. pylori has shown evidence in lowering the risk of gastric cancer in high-risk groups, emphasizing early detection and intervention. Other consequences associated with H. pylori eradication increase questions concerning its implications regarding gastric microbiome diversity, antibiotic resistance, or the subsequent emergence of other gastrointestinal disorders (Yang, Y., and Deng, C.S 2003).

This exploration delves into the complex relationship between H. pylori and gastric cancer while tackling the pathophysiological mechanisms, clinical results, and wider implications for public health and clinical practice (Calcagno, D.Q., and Leal, M.F 2008).

PATIENTS AND METHODS

I. Study Design

A population comprising 104 patients (60% male and 40 percent female) that were scheduled for surgery at In several hospitals in Iraq between October 2023 and October 2024 made up the cross-sectional study. The patients' median age ranged from 25 to 60 years old. Many variables were included in the demographic data, including age, sex, body mass index, marital status,

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socioeconomic status, body mass index, smoking or alcohol use, and education level. All patients had either a subtotal or complete gastrectomy. Every surgical resection and lymphadenectomy were carried out following to a stated procedure. The pathologists recorded the breadth of the fresh specimen and the length of the tumor.

II. Exclusion data

Additionally, patients who had antibiotics, bismuth-containing drugs, H2-receptor blockers, proton pump inhibitors, or H. pylori eradication therapy within the previous five weeks were not included in this study.

III. Follow-up of Patients

Each patient had a follow-up within the first few months following surgery, which included a clinical examination ten months later, routine blood tests, an evaluation of the tumor markers' concentration, and either a CT scan or an abdominal ultrasound. Since the procedure, endoscopies have been carried out every six months. This required an endoscopy every 12 months and a follow-up every 4 months. All patients had comprehensive metastases staging to determine whether the disease was present elsewhere with the return of relapse, which is defined as a local recurrence as well as metastasis in a distant site. From the date of tumor excision to the date of death to the patient's final known date of life, the time for survival was calculated.

RESULTS

Table 1: Enroll clinical features of patients {n = 103}.

Variables	Participants, {n = 103}	Percentage, %
Age		
25 – 35	23	22.33%
36 – 45	37	35.92%
> 45	44	42.72%
Gender		
Men	62	60.19%
Women	41	39.81%
Body mass index, {kg/m2}		
Underweight	12	11.65%
Normal weight	18	17.48%
Overweight	29	28.16%
Obesity	44	42.72%
Smoking		
Present	38	36.89%
Absent	65	63.11%
Alcohol consumption		
Present	13	12.62%
Absent	90	87.38%
Comorbidity		
No	38	36.89%
Hypertension	34	33.01%
Asthma	11	10.68%
Diabetes	14	13.59%
Others	6	5.83%
Marital status		
Single	18	17.48%
Married	75	72.82%
Divorced/widow	10	9.71%
Education status		
Primary	18	17.48%
Secondary	29	28.16%
University	56	54.37%
Economic level, $		

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< 400	35	33.98%
400 – 800	40	38.83%
> 800	28	27.18%

Table 2. Distribution of tumor data overall patients.

Items	Variables	No. of patients {n = 103}	%
Tumor location			
	Upper	42	40.78%
	Middle	32	31.07%
	Lower	29	28.16%
Tumor size		6.12 ± 1.42	
Depth of tumor invasion			
	T1	7	6.80%
	T2	11	10.68%
	T3	75	72.82%
	T4	10	9.71%
Surgery			
	Curative	84	81.55%
	Palliative	19	18.45%

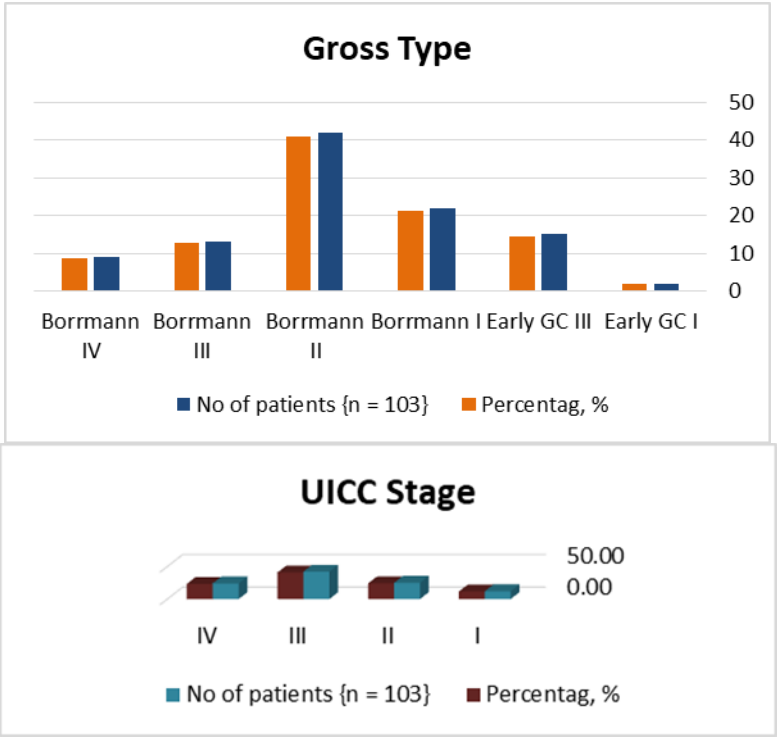

Figure 1: Classification of tumor degree on all patients in terms of Gross type and UICC stage.

Table 3: Distribution of clinical characteristics metastasis into patients.

Variables	No. of patients {n = 103}	%
Lymph-node metastasis		
NO	39	37.86%
N1	33	32.04%
N2	19	18.45%
N3	12	11.65%
Distant metastases		
MO	87	84.47%
M1	16	15.53%

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Table 4: Identification of clinical findings tumor signs of patients.

Items	Variables	No. of patients {n = 103}	%
CEA			
	<6 µg/L	14	13.59%
	≥6 µg/L	89	86.41%
CA199			
	<28 µg/L	65	63.11%
	≥28 µg/L	38	36.89%
P53			
	Positive	34	33.01%
	Negative	69	66.99%

Table 5: Determining the relationship between tumor and degree of H. Pylori.

Variables	H.Pylori high-Risk {52}		H.Pylori Lower-Risk {51}	
	N	**%**	**N**	**%**
Tumor location				
Upper	25	48.08%	10	19.61%
Middle	15	28.85%	25	49.02%
Lower	12	23.08%	16	31.37%
Lymph-node metastasis				
NO	24	46.15%	12	23.53%
N1	17	32.69%	22	43.14%
N2	6	11.54%	10	19.61%
N3	4	7.69%	7	13.73%
Distant metastases				
MO	45	86.54%	39	76.47%
M1	7	13.46%	12	23.53%

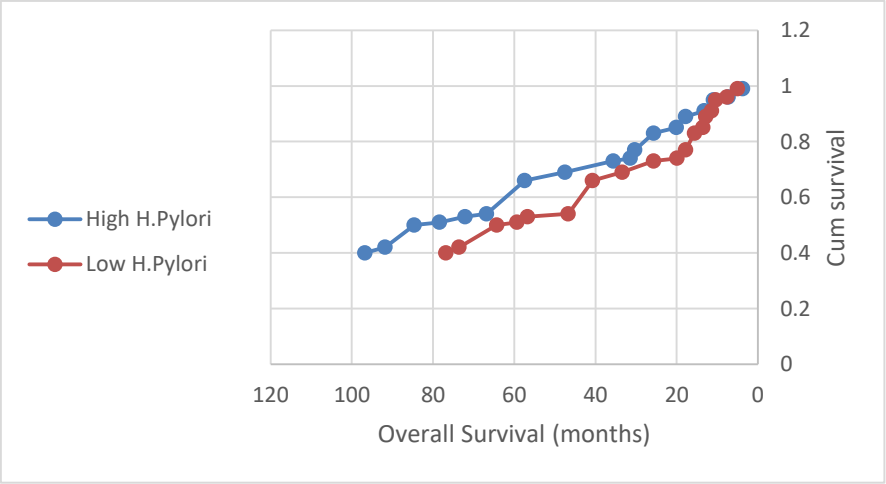

Figure 2: A conducting graph showing the survival rate during the follow-up period for patients.

DISCUSSION

Although it is one among the most prevalent malignancies worldwide, stomach cancer is still one of the deadliest; nonetheless, the prevalence varies by region (Murakami, K., and Fujioka, T 2006). - One of the primary causes of the risk for the development of stomach cancer is the gram-negative bacteria H. pylori. Nearly 50% of persons worldwide have an infection with H. pylori, with a larger prevalence among those who live in underdeveloped nations (Wu, M.S., and Hung, H.W 1998). The fourth most deadly and fifth most prevalent kind of cancer worldwide is gastric cancer. About 90% of non-cardia stomach malignancies are thought to be produced by an H. pylori infection. However, dietary practices, such as consuming a lot of salt and a few fruit and vegetables, smoking, heredity, and other environmental variables, are also important risk factors (Rocco, A., and Caruso, R 2006).

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Prolonged inflammation results from chronic gastritis, which is caused by H. pylori. By interfering at host cell signaling pathways, CagA promotes cell division and prevents apoptosis (Meimarakis, G., and Winter, H 2006). Chronic H. pylori infection can cause intestinal metaplasia and atrophic gastritis, both of which are precancerous conditions. Tumor suppressor genes may be silenced by this type of infection, which might encourage the development of epigenetic alterations such as DNA methylation (Marrelli, D., and Pedrazzani, C. 2009). The risk of stomach cancer is decreased by successful elimination, especially in individuals with precancerous lesions. In carriers who do not exhibit any symptoms, testing of H. pylori is necessary (Lehours, F.M., and P., Megraud 2007).

In contrast to CEAs, which are unconnected to H. Pylori copy number, individuals in an environment that is highly infected N states typically have tumor sites higher in the stomach as well as an earlier N stage, even though some characteristics regarding the tumor site along with N staging were associated to the copy quantity of H. Pylori (Sobin, L.H., and W.C. 2002; Marrelli, D., and Pinto, E 2001). There were no significant differences in overall survival or relapse-free survival between patients with positive H. Pylori status and those with negative H. Pylori status (Megraud, F. "H 2004). Although there was no statistically significant difference, patients with higher H. Pylori copy counts had a better prognosis than those with lower copy counts among H. Pylori-positive patients (Liu, H., and Rahman, A 2008).

The clinic pathological characteristics of the H. pylori-positive and negative H. pylori patient cohorts were contrasted (Lawson, A.J., and Elviss, N.C 2005). According to statistical analysis, these H. Pylori-positive individuals had more upper stomach locations among those who had H. Pylori, the upper stomach also had more copies of H.Pylori than the middle or lower stomach (Ernst, P 1999). Generally speaking, H. Pylori thrives best in the stomach, especially at the gastric pylorus. At least five centimeters distant from the tumor, non-neoplastic tissue was used for the diagnosis and measurement of H. pylori infections. Reducing the incidence of stomach cancer worldwide requires early H. pylori identification and elimination, lifestyle changes, and focused screenings (Elnemr, A., and Yonemura, Y 2003).

CONCLUSION

Helicobacter pylori (H. pylori) are known as a cause of gastric cancer as a Group 1 carcinogen, according to the International Agency for Research on Cancer. The pathogen's worldwide infection exists in large parts of the population and is strongly associated with non-cardia gastric cancer. The pathogenicity involves complex mechanisms of a molecular nature along with virulence factors causing chronic inflammation and alterations to cell structure permissive to cancer development. Such knowledge is vital for devising prevention and treatment modalities.

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