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Research Article

Pattern of C-Reactive Protein (CRP) and some Haematological Characteristics in Patients with Helicobacter pylori Infection at the Specialist Hospital Owerri

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Abstract

Helicobacter pylori (H. pylori) infection continues to pose a significant public health challenge worldwide, especially in poorer nations. Chronic infection has been associated with gastrointestinal issues and systemic inflammatory responses that may affect haematological and biochemical markers, including C-reactive protein (CRP) levels. This study examined the trend of several haematological indicators and serum CRP levels in H. pylori-infected patients at Specialist Hospital, Owerri. A cross-sectional analytical investigation was performed involving 100 persons aged 18 to 60 years, comprising 50 seropositive H. pylori patients and 50 seemingly healthy controls. Blood samples were taken for a full blood count using an automated haematology analyser and for CRP quantification with an enzyme-linked immunosorbent test (ELISA). The data were examined with SPSS version 25.0, and a p-value of less than 0.05 was considered significant. The mean haemoglobin concentration, packed cell volume (PCV), and red blood cell (RBC) counts were significantly decreased in H. pylori-infected patients relative to controls ($p < 0.05$), indicating moderate anaemia. On the other hand, the number of white blood cells (WBC) and the proportion of neutrophils were higher, which suggests systemic inflammation. The average serum CRP level in infected individuals was markedly elevated compared to controls ($p < 0.01$). H. pylori infection is linked to modified haematological parameters indicative of anaemia and an enhanced inflammatory response, as seen by raised CRP levels. Regular examination of haematological indices and CRP may serve as valuable supplementary tools in evaluating disease severity and tracking therapy response in H. pylori infection.

Keywords: C-reactive protein, haematological parameters, and Helicobacter pylori, Owerri.

INTRODUCTION

Helicobacter pylori (H. pylori) is a Gram-negative, spiral-shaped, microaerophilic bacteria that only lives in the mucous layer of the human stomach. It has several unipolar flagella that let it move through the thick gastric mucus, which lets it start an infection in the stomach's severe acidic environment. H. pylori has been acknowledged as one of the most widespread bacterial illnesses globally, impacting almost fifty percent of the world's population since its identification. The frequency is significantly elevated in underdeveloped nations, where inadequate sanitation, overcrowding, and restricted healthcare access facilitate transmission, especially through oral–oral or fecal–oral pathways [1]. H. pylori is related with a range of gastrointestinal problems, including chronic gastritis and peptic ulcer disease, as well as more severe conditions such as gastric adenocarcinoma and mucosa-associated lymphoid tissue (MALT) lymphoma. The bacterium's pathogenicity is facilitated by a consortium of virulence factors, including cytotoxin-associated gene A (CagA), vacuolating cytotoxin A (VacA), urease, and different outer membrane proteins, which allow it to circumvent the host immune response, provoke inflammation, and inflict epithelial damage. Chronic colonisation leads to enduring mucosal inflammation, epithelial cell injury, and the eventual disturbance of normal stomach function [2]. In addition to its recognised involvement in gastrointestinal disorders, recent findings suggest that H. pylori infection has considerable systemic impacts, especially on haematological and immunological functions. Chronic low-grade inflammation is caused

by a long-term infection with H. pylori. This keeps immune cells active and releases proinflammatory cytokines such interleukin-1β (IL-1β), interleukin-6 (IL-6), tumour necrosis factor-alpha (TNF-α), and interferon-gamma (IFN-γ). These cytokines not only maintain localised gastric inflammation but also infiltrate systemic circulation, potentially modifying haematopoiesis, iron metabolism, and erythropoietin activity [3]. Consequently, H. pylori infection has been linked to numerous haematological disorders, such as iron deficiency anaemia, anaemia of chronic illness, vitamin B_{12} deficiency anaemia, and immune thrombocytopenic purpura (ITP). There are many factors that make the mechanisms work. For example, the bacterium's long-term inflammatory state boosts hepcidin production through IL-6 signalling. This makes it harder for the body to absorb iron from the intestines and keeps iron inside macrophages and hepatocytes. Additionally, stomach mucosal atrophy resulting from chronic infection hinders acid secretion and intrinsic factor synthesis, resulting in the malabsorption of iron and vitamin B_{12} [4]. Oxidative stress that occurs during an infection also harms the membranes of red blood cells and precursor cells in the bone marrow, which makes anaemia worse. The immune system's reaction to H. pylori might also cause the body to make autoantibodies against platelets, which can lead to thrombocytopenia. Changes in leukocyte differentials have been noted, marked by heightened neutrophil activity and relative lymphocytosis, indicative of persistent inflammatory and immunological responses [5]. Helicobacter pylori infection is not just a localised stomach problem; it is a systemic disease that affects the blood and immune systems in many ways. Chronic infection induces a persistent state of inflammation, oxidative stress, and dysregulated cytokine production, thus disrupting normal haematopoietic functions and iron homeostasis. Comprehending these mechanisms establishes a basis for recognising the extra-gastrointestinal manifestations of H. pylori and highlights the significance of prompt diagnosis and eradication therapy—not solely to avert gastrointestinal complications but also to alleviate related systemic disorders [6]. C-reactive protein (CRP) is a well-defined acute-phase reactant produced mostly by hepatocytes in response to proinflammatory cytokines, particularly interleukin-6 (IL-6), along with interleukin-1β (IL-1β) and tumour necrosis factor-alpha (TNF-α). The body makes CRP as part of its natural immune system. This system is meant to quickly respond to infections, inflammation, or tissue damage. CRP is usually only found in extremely low amounts in the blood. However, after an inflammatory stimulation, its levels can go up by a hundred times or more in just 6 to 8 hours. Because it works quickly, has a short half-life, and is sensitive to inflammatory mediators, CRP is one of the best biochemical markers of systemic inflammation [7]. CRP is very important for the body's defence and immune control. It attaches to phosphocholine residues on the surfaces of microbes, dead cells, and nuclear debris. This makes opsonisation easier and speeds up phagocytosis by macrophages and neutrophils by activating the classical complement pathway. CRP can also change how immune cells work, affect the generation of cytokines, and help get rid of invaders and damaged tissues. These characteristics render it a crucial component of the acute-phase response during infections, including those induced by Helicobacter pylori [8]. Numerous investigations have shown that individuals infected with H. pylori display markedly increased serum CRP levels, even in the absence of pronounced gastrointestinal symptoms. This observation highlights the bacterium's ability to elicit a systemic inflammatory response beyond the gastric mucosa. The long-term presence of H. pylori in the stomach keeps both local and systemic immune systems active. Cytokines including IL-6, IL-1β, and TNF-α are made in the gastric mucosa and then enter the bloodstream, where they cause the liver to make CRP. Increased CRP levels indicate both the severity of gastric mucosal inflammation and the level of systemic immune activation induced by the infection [9]. Moreover, elevated CRP levels in patients infected with H. pylori have been associated with the severity of gastritis, the occurrence of peptic ulcers, and the potential risk of gastric cancer. In addition to gastrointestinal pathology, increased CRP may indicate extra-gastric symptoms of infection, including haematological problems, metabolic changes, and cardiovascular risk. Consequently, evaluating CRP levels in H. pylori-infected individuals yields significant information regarding the systemic inflammatory load linked to the illness [10]. Although there is a wealth of global research on H. pylori and its inflammatory effects, there is a dearth of data from southeastern Nigeria, especially concerning the infection's influence on haematological profiles and serum CRP levels. Socioeconomic, environmental, and genetic factors specific to the location may affect both the incidence of infection and the host's immune-inflammatory response. Comprehending these variances is essential for context-specific diagnosis, management, and public health initiatives [11]. Thus, this study seeks to examine the correlation between several haematological indices and serum CRP levels in H. pylori-infected patients at Specialist Hospital, Owerri, Imo State, Nigeria. The research aims to clarify potential changes in haematological indices, including haemoglobin concentration, packed cell volume, total and differential white blood cell counts, and platelet count, in comparison between infected and non-infected persons, alongside CRP concentrations. The results are anticipated to yield significant baseline data for doctors and researchers in southeastern Nigeria, enhancing the comprehension of the systemic impacts of H. pylori infection.

Materials and Methods

Study Area

This study was conducted at the Imo Specialist Hospital, Owerri, Imo State, Nigeria. It is a tertiary health facility

Study Population

100 adults aged 18–60 years were enrolled and categorized into two groups: Group 1 (Control): 50 Non *H. pylori*-infected patients as well as Group 2: 50 *H. pylori*-infected patients serve as test group

Ethical Approval

The study was approved by the Ethical and Research Committees of the Specialist Hospital Owerri used in the study. Informed consent was also obtained from all participating patients.

Collection of Blood Samples

Blood samples were collected aseptically by venopuncture, using a 5ml sterile disposable syringe and needle from all the subjects. Two milliliters were dispensed into EDTA tubes for full blood count. The remaining 3 mL were transferred into plain tubes, allowed to clot, and centrifuged at 3000 rpm for 10 minutes to obtain serum for CRP estimation.

Laboratory Procedures

The following parameters Hemoglobin concentration (Hb), Packed Cell Volume (PCV), Red Blood Cell count (RBC), Total White Blood Cell count (WBC), Differential count (Neutrophils, Lymphocytes) and Platelet count were analyzed using an automated hematology. CRP was determined by ELISA method

Statistical Analysis

All data generated in this study was subjected to statistical analysis using SPSS version 23. Mean and standard deviation, student t-test and correlation were determined. The level of significant will be taken at $p<0.05$.

Results

Table 1: The Mean and Standard Deviation of the Levels of Hematological Parameters (Hb, PCV, RBC, WBC, Neutrophils, Lymphocytes, and Platelets) and CRP in H pylori, attending Imo Specialist Hospital, Owerri.

Parameter	*H. pylori*	Control	*p*-value
Hb (g/dL)	9.1 ± 1.2	12.9 ± 1.1	0.03
PCV (%)	31.7 ± 3.3	43.1 ± 4.1	0.03
RBC ($\times 10^{12}$/L)	3.2 ± 0.5	5.0 ± 0.7	0.02
WBC ($\times 10^{9}$/L)	8.7 ± 3.1	6.6 ± 1.2	0.02
Neutrophils (%)	63.8 ± 6.7	55.3 ± 5.8	0.02
Lymphocytes (%)	22.8 ± 4.2	37.1 ± 7.3	0.01
Platelets ($\times 10^{9}$/L)	230 ± 44	255 ± 48	0.05
CRP (mg/L)	10.9 ± 3.4	5.6 ± 1.9	0.01

Discussion

This study showed that patients with Helicobacter pylori infection had big changes in their blood cell counts and higher levels of C-reactive protein (CRP) in their blood than people who weren't affected. These results highlight the systemic effects of H. pylori infection, transcending localised gastric disease. The noted decreases in haemoglobin concentration, packed cell volume (PCV), and red blood cell (RBC) count in infected individuals indicate the onset of anaemia, aligning with previous findings that associate H. pylori with iron deficiency anaemia and anaemia of chronic illness [12]. The processes driving these haematological alterations are diverse. Chronic infection with H. pylori causes long-term inflammation of the gastric mucosa, which makes it hard for the stomach and duodenum to absorb iron normally. The organism's virulence factors, including cytotoxin-associated gene A (CagA) and vacuolating cytotoxin A (VacA), damage epithelial cells and produce the release of proinflammatory cytokines such as interleukin-1β (IL-1β), interleukin-6 (IL-6), and tumour necrosis factor-alpha (TNF-α). These cytokines, especially IL-6, cause the liver to make hepcidin, a regulatory peptide that stops iron from being absorbed in the intestines and keeps it in macrophages and hepatocytes. This iron sequestration results in functional iron deficit despite sufficient iron reserves, which is indicative of anaemia of chronic inflammation [13]. H. pylori infection can also cause chronic irritation of the stomach and erosions of the mucosa, which can lead to hidden bleeding in the gastrointestinal tract. This can induce iron loss and lower the production of red blood cells. The bacterium has been demonstrated to compete for available iron, exacerbating anaemia [14]. These processes collectively elucidate the reduction in erythrocytic indices noted in infected patients in this investigation. The increased total white blood cell (WBC) count and neutrophil predominance seen in H. pylori-positive patients indicate a stronger systemic inflammatory response. The lipopolysaccharide (LPS) part of the bacteria activates macrophages and other immune cells through Toll-like receptor 4 (TLR4), which makes proinflammatory cytokines more likely to be made. These cytokines facilitate localised inflammation in the stomach mucosa and additionally encourage leukocyte mobilisation and bone marrow activation, leading to peripheral leukocytosis [15]. The significant increase in CRP levels seen in this study further corroborates the existence of systemic inflammation in H. pylori-infected patients. CRP, an acute-phase reactant synthesised by the liver in response to IL-6, functions as a sensitive biomarker for both acute and chronic inflammatory conditions. The current findings align with those of [16], which indicated markedly increased serum CRP levels in individuals with H. pylori-associated gastritis. The elevated CRP levels indicate the body's inflammatory response to the illness and the systemic spread of inflammatory mediators from the stomach mucosa [17]. Interestingly, CRP levels

had a positive relationship with the total number of WBCs and neutrophils. This shows that they all come from the same inflammatory source and supports the idea that cytokine-mediated immune activation is important in how H. pylori causes disease. On the other hand, the negative association between CRP and haemoglobin concentration shows that inflammation and erythropoiesis are related in the opposite way. This condition is facilitated by cytokine-induced suppression of erythropoietin synthesis and the inhibition of iron utilisation, both of which contribute to anaemia in persistently infected patients [18]. In terms of platelet counts, there was no significant difference between the H. pylori-infected and non-infected groups, which is consistent with the findings of [19]. This indicates that H. pylori-associated thrombocytopenia may not be a consistent characteristic of infection, but rather may vary based on particular bacterial strains, host immunological responses, or genetic predisposition [20].

The results of this investigation underscore the systemic haematological and inflammatory changes linked to H. pylori infection. The bacterium's ability to cause chronic inflammation, change how iron is processed, and raise acute-phase proteins shows that an H. pylori infection is not only a problem in the stomach, but one that affects the whole body. These changes have significant diagnostic and therapeutic ramifications, indicating that individuals exhibiting unexplained anaemia or heightened inflammatory markers ought to be assessed for potential H. pylori infection [21, 22].

Conclusion

This study finds that Helicobacter pylori infection in patients at Specialist Hospital, Owerri, correlates with mild anaemia, neutrophilia, and higher serum CRP levels, all of which signify a systemic inflammatory response. The infection's effect on erythropoietic and immunological parameters highlights the necessity for regular assessment of haematological and inflammatory markers in H. pylori-positive people. Keeping an eye on these factors may help find haematological problems early, point doctors in the right direction for treatment, and improve patient outcomes overall.

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