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# Association of H. Pylori Infection with Changes in Molecular Markers of Inflammation in Patients with Incident Hypertension Attending Federal University Teaching Hospital Owerri, imo state

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**Abstract**

Helicobacter pylori (H. pylori) infection and hypertension are associated with chronic inflammation. This study investigates the association of molecular markers of inflammation in H. pylori-infected patients with incident hypertension attending selected hospitals in Imo State, Nigeria. A total of 100 adult participants (aged 20-60 years) were recruited and categorized into four groups (n 25 each): Group 1 (Control): Normotensive, H. pylori-negative. Group 2: Hypertensive, H. pylori-positive. Group 3: Hypertensive, H. pylori-negative. Group 4: Normotensive, H. pylori-positive. Parameters assessed included: C-reactive protein (CRP), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-a), Immunoglobulin M(IgM), Immunoglobulin G(IgG). Data were analyzed using one-way ANOVA ($p<0.05$). Group 2 (hypertensive, H. pylori+) consistently showed the most elevated values across inflammatory and lipid markers but a decreased patelet values. Key findings (mean ± SD) include:. CRP (mg/L): G1: 2.10 ± 0.51; G2: 6.78 ± 0.87; G3: 5.96 ± 0.79; G4: 4.84 ± 0.69. IL-6 (pg/mL): G1: 3.44±0.62; G2: 8.76 ± 0.91; G3: 7.10 ±0.80; G4: 6.34±0.74. TNF-a (pg/mL): G1: 4.26 ± 0.58; G2: 9.12 ± 1.04; G3: 8.02 ± 0.88; G4: 6.98 ±0.81. IgM(mg/dl): G1:93.08±17.59; G2:134.44±13.48;G3:97.80±16.76;G4:118.20±16.83.

IgG(mg/dl):G1:1174.84±153.28;G2:1705.40±143.21;G3:1275.36±161.83;G4:1649.68±110.16.Statistical analysis revealed significant differences across all groups in all parameter ($p<0.05$), with Group 2 showing the most pronounced elevations in inflammatory. The combination of H. pylori infection and hypertension is associated with significant upregulation of molecular markers of inflammation. Group 2 displayed a synergistic exacerbation of systemic

inflammation, suggesting an increased risk of cardiovascular events.

**Key words:** pylori infection; inflammation; hypertension owerri

## Introduction:

The World Health Organisation has classified Helicobacter pylori as a group I carcinogen, and the estimated 50% global burden of Helicobacter pylori infection makes the bacterium a pathogen of concern. The World Health Organisation has classified Helicobacter pylori as a group I carcinogen, and the estimated 50% global burden of Helicobacter pylori infection makes the bacterium a pathogen of concern. [1] A gram-negative, spiral-shaped bacterium called Helicobacter pylori (H. Pylori) colonises the human stomach and, if left unchecked, can often last a lifetime. Affecting over 44% of the world's population, it is one of the most prevalent illnesses worldwide. Because of variations in socioeconomic level, hygienic habits, and availability to healthcare, prevalence rates range greatly between locations, with greater rates in underdeveloped nations (70–90%) compared to industrialised nations (20–40%) [2].

Numerous studies have documented the prevalence of H. pylori infection in Nigeria throughout time, taking into account variations in the country's population, age groups, occupation, gender, marital status, educational attainment, and illness state. It has been demonstrated that the largest frequency of H. pylori infection occurs in the northern region of the nation [3].

Despite the fact that more than half of the world's population has an H. pylori infection, the majority of people do not exhibit any symptoms. Numerous gastric and non-gastric illnesses can be brought on by persistent colonisation with increasingly aggressive bacteria. Inflammation of the stomach lining, or gastritis, is the first of the infection-related gastric illnesses. Chronic gastritis will develop from the protracted inflammation caused by a persistent infection. Although this will initially be non-atrophic gastritis, the duodenum (the closest portion of the intestine) or the stomach itself may develop atrophic gastritis and ulcers as a result of the damage to the stomach lining [4]. The chance of getting stomach cancer is high at this point. However, there is a significantly lower risk of cancer when a duodenal ulcer develops. Helicobacter pylori is a class 1 carcinogen, and it can cause stomach cancer and mucosa-associated lymphoid tissue (MALT) lymphoma. An estimated 89% of all stomach cancers are caused by H. pylori infection, which is also connected to the development of 5.5% of all cancer cases globally. H. pylori is the only known bacterium that causes cancer. [5]

Anaemia from iron or vitamin B12 deficiency, diabetes mellitus, cardiovascular disease, and several neurological conditions are examples of extragastric problems that have been connected to H. pylori. [6]

In Sub-Saharan Africa, hypertension is a major contributor to non-communicable morbidity. According to recent research, hypertension is becoming more common in rural Sub-Saharan Africa. Hypertension is a worldwide public health issue that increases the risk of heart disease, stroke, renal failure, and other illnesses. It is seen as a complex causative disorder that is impacted by the interplay of numerous factors, including genetics, bad eating habits, hazardous alcohol consumption, physical inactivity, and tobacco use. [8]

Because of its high incidence, role in the burden of disease, and substantial economic impact, hypertension—also known as high blood pressure—is a serious global public health concern. Known as the "Silent Killer," it is characterised by a persistent blood pressure reading of 140/90 mmHg or above and is frequently asymptomatic. [9]

One of the biggest risk factors for many serious illnesses, such as heart disease, stroke, and renal failure, is still high blood pressure. Systemic inflammation has been linked to H. pylori infection and may have a role in the development of hypertension. By triggering immunological responses and releasing inflammatory cytokines like interleukin-6 (IL-6), tissue necrosis factor-alpha (TNF-α), and C-reactive proteins (CRP), the bacterium creates a persistent inflammatory state. Endothelial dysfunction, a crucial factor in hypertension, can result from this inflammatory mediation's disruption of vascular haemostasis [10].

In order to comprehend disease causes, facilitate early detection, and create tailored therapeutics, molecular makers of inflammation are essential. These biomarkers, which include cytokines, chemokines, and other signalling molecules, provide important insights into disease processes by reflecting the dynamic interaction between the immune system and physiological homeostasis [11].

The identification of chronic inflammatory problems, such as infections, autoimmune disorders, and cardiovascular diseases, depends on inflammatory markers including interleukin-6 (IL-6), C-reactive protein (CRP), tumour necrosis factor-alpha (TNF-α), interleukin-8, interleukin-10, and interferon-gamma, among others. Since elevated levels of these indicators frequently occur before clinical symptoms appear, early detection and intervention are made possible. [12]

Chronic gastritis brought on by Helicobacter pylori infection can develop into serious gastroduodenal diseases such as peptic ulcers, gastric cancer, and gastric mucosa-associated lymphoid tissue lymphoma. In addition to its well-established function in gastrointestinal problems, Helicobacter pylori infection has been linked to a number of non-gastric systemic disorders, such as inflammatory syndromes, metabolic disorders, and cardiovascular diseases.

Although some research has looked into the possible connection between H. pylori and hypertension, nothing is known about how it affects haemostasis and inflammatory markers. It is well recognised that inflammatory indicators such C-reactive protein (CRP), interleukin-6 (IL-6), and tumour necrosis factor-alpha (TNF-a) are important in the pathophysiology of hypertension as well as chronic infections [13]. However, not enough research has been done on how much an H. pylori infection affects these

markers in people who have incident hypertension.
Even though earlier studies may have examined the connection between H. pylori infection and hypertension, more research is necessary given the recent rise in H. pylori-associated stomach ulcers and the rising incidence of early-onset hypertension in young adults, particularly in the study area. Evaluating whether H. pylori infection contributes to inflammatory and homeostatic abnormalities that may predispose people to hypertension is crucial given the changing epidemiological patterns. Knowing this link may help identify new risk factors and possible treatment targets for hypertension, especially in populations where the prevalence of both illnesses is high [14, 15].
By assessing the relationship between H. pylori infection and certain inflammatory markers in individuals with incident hypertension, this study seeks to close the current knowledge gap. For those impacted by both illnesses, the results may have implications for risk assessment, early diagnosis, and the creation of integrated management plans.
Establishment [1] A gram-negative, spiral-shaped bacterium called Helicobacter pylori (H. Pylori) colonises the human stomach and, if left unchecked, can often last a lifetime. Affecting over 44% of the world's population, it is one of the most prevalent illnesses worldwide. Because of variations in socioeconomic level, hygienic habits, and availability to healthcare, prevalence rates range greatly between locations, with greater rates in underdeveloped nations (70–90%) compared to industrialised nations (20–40%) [2].
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## Materials And Methods:

### Study Area

The study was conducted between February, 2025 to April, 2025 at the Federal University Teaching Hospital, Owerri, Imo State, Nigeria.

### Ethics Advocacy and pre survey contacts

An ethical approval was obtained from the Federal University teaching Hospital Owerri Imo State for the collection of samples. Those who qualified to participate in the study were given an informed consent letter and those who have their consent by signing the letter were recruited as participants and a day was fixed for sample collection.

### Study Population/ sample size

### Study population

This case study comprises a total of 100 adult men and women between the age of 20-60yrs attending the out-patient department (OPD), Federal University teaching Hospital Owerri Imo State. Participants are divided equally or proportionally (25 participants per group) into four (4 ) groups
Participants assigned to one of four groups based on their hypertension status and H. pylori infection status:
Group 1: Normotensive, H.pylori negative subjects, serves as the control group.
Group 2: Hypertensive, H.pylori positive subjects.
Group 3: Hypertensive, H.pylori negative subjects.
Group 4: Normotensive, H.pylori positive subjects.

### Sample Size Determination

Sample technique used was targeted random sampling.The sample size was calculated using the formula given below at 95% confidence interval (CI) level = 1.96, an expected prevalence of 0.5% and 9.8% marginal error.

$$N = \frac{Z^2P(1-P)}{d^2}$$

Where N= Sample size
Z= degree of confidence=1.96
P= expected prevalence= 0.098
d= maximum tolerated error= 0.05

$$N = \frac{(1.96)^2 \times 0.54(1-0.54))}{(0.098)^2}$$

$$\frac{3.84 \times 0.25}{0.0009604} = \frac{3.84(0.54)}{0.0009604} = \frac{0.9604}{0.0009604} = 99.4$$

However, for this study, 100 participants were recruited.

### Selection Criteria

### Inclusion Criteria

i. Patients newly diagnosed of hypertension (incident hypertension) based on clinical guideline (systolic BP ≥140mmHg or diastolic BP≥90mmHg) (WHO, 2021).
ii. Subjects confirmed with H.pylori infection via validated diagnostic test (Stool test).
iii. Adults aged 20-60years
iv. Subjects who have not previously received antihypertensive medications.

v. Subjects who are willing to provide written informed consent to participate in the study

**Exclusion Criteria:**

i. Patients who have undergone treatment for H.pylori infection in the past.
ii. Patients with chronic inflammatory or autoimmune diseases and subject not within the ages of 20-60yrs.
iii. Patients with regular use of NSAIDS, corticosteroids or immunosuppressants within the past 3 months.
iv. Pregnant and lactating subjects
v. Subjects with active infection or Vaccinations within the past 4months that could influence inflammatory markers.
vi. Patients with history of gastric resection or bariatric surgery
vii. Subjects with recent use of proton pump inhibitors (PPIs) or antibiotics
viii. Subjects who are unwilling to comply to study protocols or follow-up visits.

**Study Design**

This is a comparative, cross-sectional case-control study designed to evaluate the association between Helicobacter pylori infection with changes in markers of inflammation and hemostasis in patients with incident hypertension. This case study comprises of a total of 100 adult men and women between the age of 20-60yrs attending the out-patient department(OPD),Federal University teaching Hospital Owerri Imo State. Participants are divided equally or proportionally (25 participants per group) into four (4) groups

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Group 2: Hypertensive, H.pylori positive subjects.
Group 3: Hypertensive, H.pylori negative subjects.
Group 4: Normotensive, H.pylori positive subjects.

**Sample Collection**

Participants were informed too fast for 8-12 hours before sample collection, other procedures for the study were communicated to participants using a questionnaire method and their consents were obtained

Volume of blood: 10ml

**Collection site:** median cubital vein (antecubital fossa).

**Procedure:**

The puncture site was cleaned with 70% alcohol in an absorbent cooton wool and allowed to dry. Vacutainer needle was used to draw blood into appropriate tubes: EDTA tube (for platelet count), Citrate tube (for fibrinogen and vWF analysis), Plain tube (for CRP, IL-6, TNF-a,H.Pylori, IgM, IgG and Lipid profile). The various tubes were labelled appropriately, the samples in plain tube centrifuged within 2hrs of collection at 3000rpm for 10-15minutes accordingly and serum stored at -20oC or -80oC until analysis.

**Laboratory Investigation**

All reagents were commercially purchase and the manufacturer's standard operating procedures were strictly adhered to.

**Interleukin 6 (IL-6) estimation using ELISA**

The human interleukin-6 ELISA standards and samples are incubated in microplate wells pre-coated with monoclonal anti-human IL-6 antibody. After another washing, streptavidin-HRP conjugate is added. After 30 minutes of incubation and the last washing step, the remaining conjugate is allowed to react with the substrate solution 3,3'5,5' tetramethyl-benzidine (TMB). The reaction is stopped by addition of acidic solution and absorbance of the resulting yellow product is measured. The absorbance is proportional to the concentration of IL-6. A standard curve is constructed by plotting absorbance values against concentration of standards, and concentration of unknown samples are determined using this standard curve

**C- Reactive Proteins using ELISA**

**Principle**

The essential reagents required for an immunoenzymometric assay include high affinity and specificity antibodies (enzyme and immobilized), with different and distinct epitope recognition, in excess, and native antigens. In this procedure, the immobilization takes place during the assay at the surface of the microplate well through the interaction of streptavidin coated on the well and exogenous added biotinylated monoclonal anti – CRP antibody. Upon mixing monoclonal biotinylated antibody, the enzyme-labelled antibody and a serum containing the native antigen, reaction occurs between the native antigen and antibodies, without competition or steric hinderance, to form a soluble sandwich complex. After equilibrium is attained, the antibody bound fraction is separated from the unbound antigen by decantation or aspiration. The enzyme activity in the antibody bound fraction is directly proportional to the native antigen concentration. By utilizing several different serum

references of known antigen values, a dose response curve can be generated from which the antigen concentration of an unknown can be ascertained (Kimberly et al., 2003).

**Determination of Serum Immunoglobulin IgG**

This test was carried out using Enzyme-linked Immunosorbent Assay Technique according to (Sood, 2006) as modified by Affymetrixe Bioscience, North America.
Principle
An anti-human total IgG coating antibody is adsorbed onto micro wells.
Human total IgG present in the sample or standard binds to antibodies adsorbed to the micro wells and a HRP- conjugated anti-human total IgG antibody is added and binds to human total IgG captured by the first antibody. Following incubation unbound HRP- conjugated anti-human total IgG antibody is removed during a wash step, and substrate solution reactive with HRP is added to the wells.
A coloured product is formed in proportion to the amount of human total IgG present in the sample or standard. The reaction is terminated by addition of acid and absorbance is measured at 450 nm. A standard curve is prepared from 7 human total IgG standard dilutions and human total IgG sample concentration determined.

**Determination of Tumor Necrosis factor alpha (TNF-α) using ELISA**

Principle
The Human TNF Alpha (TNF-α) ELISA employs the quantitative sandwich enzyme immunoassay technique. A monoclonal antibody specific for TNF-α has been pre-coated onto a microplate. Standards and samples are pipetted into the wells and any TNF-α present is bound by the immobilized antibody. Following incubation unbound samples are removed during a wash step, and then a detection antibody specific for TNF-α is added to the wells and binds to the combination of capture antibody- TNF-α in sample. Following a wash to remove any unbound combination, and enzyme conjugate is added to the wells. Following incubation and wash steps a substrate is added. A colored product is formed in proportion to the amount of TNF-α present in the sample. The reaction is terminated by addition of acid and absorbance is measured at 450nm. A standard curve is prepared from seven TNF-α standard dilutions and TNF-α sample concentration determined.

## Statistical Analysis:

Statistical analysis of results obtained was analyzed using the SPSS version 21. The means and standard deviations was calculated for all parameters under investigation. Statistical differences between the experimental and control groups were determined using one-way ANOVA analysis of variance followed by student t-test. Values were considered significant at $p<0.05$

## Results:

| | VARIABLE (UNITS) | | | | |
|---|---|---|---|---|---|
| **Groups** | **CRP (mg/dl)** | **IL-6 (pg/m)** | **TNF-α (pg/m)** | **IgM (mg/dl)** | **IgG (mg/dl)** |
| Group 1 | 1.96±0.87 | 4.16±1.03 | 4.28±1.36 | 93.08±17.59 | 1174.84±153.28 |
| Group 2 | 5.54±1.52 | 8.10±1.80 | 9.98±1.26 | 134.44±13.48 | 1705.40±143.21 |
| Group 3 | 4.81±0.97 | 7.07±1.09 | 7.15±1.00 | 97.80±16.76 | 1275.36±161.83 |
| Group 4 | 4.10±0.88 | 6.31±1.49 | 8.04±0.98 | 118.20±16.83 | 1649.68±110.16 |
| F-value | 58.29 | 34.66 | 45.45 | 35.01 | 91.20 |
| P-value | 0.00009* | 0.0007* | 0.00001* | 0.00003* | 0.0845 |
| G1 Vs G2 | 0.00001* | 0.00001* | 0.00001* | 0.00001* | 0.00001* |
| G1 Vs G3 | 0.00001* | 0.00001* | 0.00001* | 0.17267 | 0.0019* |
| G1 Vs G4 | 0.00001* | 0.00046* | 0.00001* | 0.00001* | 0.00001* |
| G2 Vs G3 | 0.17474 | 0.10497 | 0.00030* | 0.00001* | 0.00001* |
| G2 Vs G4 | 0.00952* | 0.01451* | 0.00119* | 0.00001* | 0.03160* |
| G3 Vs G4 | 0.07371 | 0.16788 | 0.05965 | 0.00001* | 0.00001* |

**Table 1:** *Mean±SD for molecular markers (CRP, IL-6, TNF-α, IgM and IgG) in hypertensive patients with H.pylori infection.*

Key: Group 1 (normotensive, *H.pylori* negative participants) serves as control
Group 2 (Hypertensive, *H.pylori* positive participants).
Group 3 (hypertensive, *H.pylori* negative participants).
Group 4(normotensive, *H.pylori* positive participants).
* = statistically significant ($P<0.05$)

Table 1: shows the mean, standard deviation, F-value, post-hoc pairwise comparison (student t-test) and p-value for molecular markers (C-reactive protein, Interleukin-6, Tissue Necrosis Factor-α, Imunoglobin M, Imuniglobin G). Group 2 – 4 mean and standard deviation for C-reactive Protein (5.54±1.52, 4.81±0.97 and 4.10±0.88 respectively), Interleukin-6 (8.10±1.80, 7.07±1.09 and 6.31±1.49 respectively) and Tisuue necrosis factor-alpha (9.98±1.26, 7.15±1.00 and 8.04±0.98 respectively) increased as compared to the control (1.96±0.87,4.16±1.03 and 4.28±1.36 respectively).
Also groups 2-4 mean and standard deviation Imunoglobin M (134.44±13.48, 97.80±16.76 and 118.20±16.83 respectively) shows

a statistically significant difference as compared to the control group 1 (93.08±17.59).
There is no significant difference observed for Imunoglobin G, the mean and standard deviation for groups 2 – 4 (1705.40±143.21, 1275.36±161.83 and 1649.68±110.16 respectively) as compared with the control group 1(117.84±153.28).
Post hoc anaylsis using student t-test shows significant difference between group 1 and all other group for CRP, IL-6, TNF-α, IgM, IgG (except G1vsG3 for IgG) ($p<0.05$).
Post hoc anaylsis shows significant difference between group 2 for all parameters except G2vsG3 for CRP and IL-6 respectively. No significant difference observed between G3vsG4 for CRP, IL-6 and TNF-α but there is a significant difference between G3vsG4 for IgG and IgM

## Discussion:

Interleukin 6 (IL-6), tissue necrosis factor Alpha (TNF-α), C-reactive protein (CRP), and immunoglobulins (1gG, 1gM) are among the molecular indicators evaluated.
Group 2 (hypertensive + H.pylori positive) had considerably higher levels of the pre-inflammatory cytokines (TNF-α, IL-6), whereas Group 4 (normotensive + H.pylori positive) had mild increases, while Group 1 (controls) showed no significant increase. The pathophysiology of hypertension has been linked to nitric oxide production factors, endothelial function, and chronic low-grade inflammation, as well as increased cytokines such TNF-α and IL-6 [15, 16]. This is consistent with research on clinical hypertension and animals that demonstrates the role of cytokines in vascular and metabolic processes. [17]
In comparison to controls, C-Reaction Protein (CRP) was greatest in Group 2, elevated in Group 3, and moderately elevated in Group 4. Elevated CRP and IL-6 are independent of BMI predictors of the onset of hypertension, according to multiple meta-analyses. Higher serum CRP is associated with H. pylori infection. This result is consistent with [18,19].

## Conclusion :

This study shows a strong correlation between Helicobacter pylori infection, hypertension, and negative alterations in inflammatory markers (TNF-α, IL-6, and CRP). It shows that hypertension patients with an H. pylori infection have an inflated rise in pro-inflammatory markers. The bacterial infection may result in a persistent inflammatory state.
Hypertension (Group 2) and H. pylori infection work together to create a synergistic inflammatory effect that raises the risk of cardiovascular problems.

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