Original Research Article

The role of *Helicobacter pylori* in preeclampsia and in gastric diseases in pregnant women

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ABSTRACT

**Background:** *Helicobacter pylori* is a gastric organism was first observed more than 100 years ago. It may cause chronic gastritis, peptic ulcers and gastric adenocarcinoma and lymphoma. It can produce some extragastric disorders including preeclampsia. This study aimed to focus on the importance of *H. pylori* and its relationship with preeclampsia and gastric illnesses in pregnant patients.

**Methods:** This study included 100 pregnant women, half of them were healthy and the other half with preeclampsia. Patients with chronic medical illnesses were excluded. Data was collected and laboratory investigations were done including that for *H. pylori*. Oesophagogastroduodenoscopy then was done 6 weeks post-delivery or termination of pregnancy for symptomatic *H. pylori* positive women.

**Results:** 50% of the total number were healthy, 17% had mild preeclampsia, 33% suffering from severe preeclampsia. Seropositivity for *H. pylori* is significantly higher in preeclampsia and significantly related to severity of preeclampsia and complications of pregnancy. Positive *H. pylori* test in stool was seen in (45%) of total number of patients. Epigastric pain persist in 41 (91%) of patients with positive *H. pylori* test in stool. Gastritis, duodenal ulcer, and gastric ulcer were found in 85.3%, 12.1% and 2.4% of symptomatic patients respectively.

**Conclusions:** Significant correlation between *Helicobacter pylori* seropositivity and preeclampsia may indicate the benefit of using this parameter in the prediction and management of preeclampsia and its severity. *H. pylori* infection plays an important role in gastric pathologies in pregnant women that can be dealt with or may be prevented more efficiently in future.

**Keywords:** Preeclampsia, *H. pylori*, Gastric

INTRODUCTION

*Helicobacter pylori* is a gastric organisms were first observed more than 100 years ago and their association with gastritis has been recognized since the 1970s.1 This organism is now known to cause many other gastric pathologies.2 *H. pylori* is a spiral shaped, microaerophilic, gram negative bacterium measuring approximately 3.5 microns in length and 0.5 microns in width.3 This bacteria is characterized by the production of catalase, oxidase, and urease. Urease activity plays an important role for its survival and colonization and it is of clinical benefit because it forms the basis for several invasive and noninvasive tests to diagnose infection. The organism's urease, motility, and ability to adhere to gastric epithelium are factors that allow it to survive and proliferate in the gastric mucosa.4

*H. pylori* infection, through a chronic stimulation of the immune system and the occurrence of molecular mimicry...
mechanisms, is responsible for the majority of the gastroduodenal diseases and also for some extragastric disorders. It has been demonstrated that this pathogen enhances platelet activation and thrombus formation thus inducing endothelial inflammatory reaction and damage that may lead to neurological, dermatological, hematologic, ocular, cardiovascular, metabolic, allergic, and hepatobiliary diseases. Therefore, *H. pylori* could directly cause or intensify the generalized inflammation and endothelial dysfunction which is typical in pre eclampsia. *H. pylori* has been found worldwide and in population of all ages. Fecal/oral transmission of bacteria is possible. Contaminated water supplies in developing countries may be accused to be an important source of infection.

*H. pylori* infection can cause a wide spectrum of gastroduodenal diseases ranging from mild antrum and body gastritis which usually associated with mild clinical conditions to prevalent antrum gastritis with the possibility of duodenal ulceration to prevalent body gastritis in response to the infection that is associated with multifocal atrophic gastritis and the possible development of gastric adenocarcinoma. However, Mendall et al in was the first to notice the association between *H. pylori* infection and extra-gastric diseases. The aim of the study was to focus on the relationship between *H. pylori* seropositivity and preeclampsia, the incidence of different gastric pathologies in symptomatic pregnant patients with positive *H. pylori* test in stool.

**METHODS**

This was a prospective case control study. It was done in Karbalaa Maternity Teaching Hospital and Alhussein Medical City between March and December 2017. It included a total number of 100 pregnant women, half of them were healthy and the other half with preeclampsia who were admitted at time of delivery or termination of pregnancy. Preeclampsia was diagnosed when hypertension (systolic B.P. ≥140 and diastolic B.P. ≥90 mmHg) and proteinuria (≥300 mg per 24 hours) or hypothyroidism which is performed by dip stick test, venous blood sample was taken for biochemical and hematological investigation which are Hb%, platelet count, liver function test, renal function test, random blood sugar and serum anti *H. pylori* (IgG) antibody titer were assessed by commercial enzyme-linked immunosorbsant assay (ELISA) by mini VIDAS technique to assess seropositivity and the titer value for anti *H. pylori* (IgG) antibodies. The titer is negative when it is <0.75, equivocal when it is ≥0.75 and <1.00, positive when it is ≥1.00, stool sample was collected for *H pylori* antigen test, Oesophago-gastro-duodenoscopy was done to symptomatic patient (epigastric pain) with positive *H pylori* stool test after 6 weeks follow-up post-delivery or termination of pregnancy.

Statistical analysis was done by using SPSS in which analysis of variance (ANOVA) for measurement data and chi (x²) for categorical data was used.

All the investigations and the endoscopic procedures were performed within the standard indications and after taking the permission from the patient. No new experimental medications or procedures have been tried in this study.

**RESULTS**

The study consist of 100 pregnant women, 50 (50%) of them were healthy, 17 (17%) had mild preeclampsia and 33 (33%) suffering from severe preeclampsia.

The result in Table 1 showed no significant difference between the groups regarding age of mother (p=0.361) also no significant difference in parity (p=0.746). There is high significant difference between the groups regarding systolic and diastolic blood pressure and proteinuria (p<0.001).

**Table 1: Patients characteristics of each group.**

| Characteristics | Normal (n=50) | Mild PE (n=17) | Severe PE (n=33) | P value |
|-----------------|--------------|---------------|-----------------|---------|
| **Number (patients)** |              |               |                 |         |
| Age             | 27.3±4.62    | 26.69±5.33    | 26.44±6.32      | 0.361   |
| Parity          | 1.52±1.3     | 1.3±1.6149    | 1.4±1.7         | 0.746   |
| Systolic BP     | 115.67±11.36 | 41±8.76       | 172.91±12.82    | <0.001  |
| Diastolic BP    | 72.69±9.19   | 104.71±7.58   | 116.26±5.61     | <0.001  |
| Proteinuria     | 0            | 1.32±0.476    | 2.28±0.454      | <0.001  |
Table 2: Comparison between different groups according to different risk factors.

| Risks                  | Normal (N=50) | Mild PE (N=17) | Sever PE (N=33) | P value   |
|------------------------|---------------|----------------|-----------------|-----------|
| **Maternal medication**|               |                |                 |           |
| Antihypertensive        | 0 (0)         | 9 (52.9)       | 18 (54.54)      | <0.001    |
| Aspirin                | 0 (0)         | 2 (11.7)       | 4 (12.12)       | 0.041     |
| Corticosteroid         | 6 (12)        | 0 (0)          | 11 (33.3)       | 0.004     |
| **Maternal risk factors** |             |                |                 |           |
| Primipara              | 16 (32)       | 10 (58.8)      | 20 (60.6)       | 0.019     |
| Grandmultipara         | 4 (8)         | 2 (11.76)      | 11 (33.3)       | 0.008     |
| Previous PE            | 3 (6)         | 5 (29.4)       | 8 (24.24)       | 0.021     |
| Twin                   | 2 (4)         | 0 (0)          | 7 (21.21)       | 0.009     |
| **Family risk factors**|               |                |                 |           |
| Hypertension           | 20 (40)       | 9 (52.9)       | 21 (63.63)      | 0.104     |
| D.M                    | 9 (18)        | 6 (35.3)       | 7 (21.21)       | 0.328     |
| C.V.D                  | 10 (20)       | 7 (41.17)      | 13 (39.39)      | 0.091     |

In Table 2, regarding maternal medication (antihypertensive, aspirin, and corticosteroid) it is more commonly used by those suffering from severe PE (p<0.05). Regarding maternal risk factors like primipara, grandmultipara, previous PE and twin pregnancy also more significant with severity of PE (p<0.05). Family risk factors like hypertension, diabetes mellitus and cardiovascular disease show no significant difference between the groups (p>0.05).

In Table 3 regarding gestational age there is high significant difference between the groups (p=0.001) where the mean is higher in healthy group and lower in those with sever preeclampsia. Birth weight also lower in sever PE which is significantly differ from other groups (p<0.001). The mode of delivery significantly differ between the groups (p<0.001) with higher cesarean section rate in sever PE (78.8%).

Table 3: Outcome of pregnancy and H. pylori titer in normal and PE mothers.

| Characteristics | Normal (N=50) | Mild PE (N=17) | Sever PE (N=33) | P value   |
|-----------------|--------------|---------------|-----------------|-----------|
| Number (patients) |              |               |                 |           |
| Gestational age (weeks) | 38.2±1.4 | 37.1±1.3 | 34.4±2.23 | 0.001 |
| Birth weight (Kg) | 3.25±0.34 | 2.49±0.4 | 2.1±0.51 | <0.001 |
| C/S (patients) | 13 (26%) | 5 (29.4%) | 26 (78.8%) | <0.001 |
| Vaginal delivery (patients) | 37 (74%) | 12 (70.6%) | 7 (21.2%) | <0.001 |

Table 4: Lab results of each group.

| Characteristic       | Normal (mg/dl) | Mild PE (mg/dl) | Sever PE (mg/dl) | P value   |
|----------------------|---------------|-----------------|-----------------|-----------|
| Blood urea           | 25.427±3.76   | 27.581±4.34     | 36.794±4.859    | <0.001    |
| S.creatinine         | 0.571±0.194   | 0.821±0.259     | 1.089±0.368     | <0.001    |
| S.uric acid          | 3.416±0.683   | 4.513±0.742     | 4.913±7.891     | 0.024     |
| Hb %                 | 11.361±1.074  | 11.298±1.083    | 10.298±1.184    | 0.032     |
| SGPT U/l             | 16.631±5.372  | 19.842±6.794    | 32.583±11.913   | <0.001    |
| SGOT U/L             | 14.521±9.732  | 24.742±8.418    | 42.684±12.873   | <0.001    |
| S.A.P mg/dl          | 182.621±22.924 | 187.794±20.947 | 210.853±29.985 | <0.001    |
| Platelets (*10^9)    | 239±18        | 191±16          | 126±15          | <0.001    |
| Total serum bilirubin| 0.531±0.429   | 0.549±0.298     | 0.615±0.351     | 0.399     |

In Table 2, regarding maternal medication (antihypertensive, aspirin, and corticosteroid) it is more commonly used by those suffering from severe PE (p<0.05). Regarding maternal risk factors like primipara, grandmultipara, previous PE and twin pregnancy also more significant with severity of PE (p<0.05). Family risk factors like hypertension, diabetes mellitus and cardiovascular disease show no significant difference between the groups (p>0.05).

Table 4 showed that in sever PE there is significantly higher blood urea, serum creatinine, serum uric acid, SGPT, SGOT, and S.A.P than other groups(p<0.05). Sever PE shows lower hemoglobin and platelet count than other groups (p<0.001), while there is no significant difference regarding total serum bilirubin (p=0.399).

Table 5: Distribution of groups (number of patients) according to H.pylori titer.

| Characteristic | Normal (N=50) | Mild PE (N=17) | Sever PE (N=33) | P value   |
|---------------|--------------|---------------|-----------------|-----------|
| Negative      | 31 (65.9)    | 5 (10.6)      | 11 (23.5)       | 0.007     |
| Equivocal     | 6 (85.7)     | 1 (14.3)      | 0 (0)           |           |
| Positive      | 13 (28.3)    | 11 (23.9)     | 22 (47.8)       |           |
### Table 6: H. pylori titer and severity of preeclampsia.

| Characteristic     | Normal  | Mild PE | Sever PE | P value |
|--------------------|---------|---------|----------|---------|
| H. pylori titer    | 0.44±0.05 | 0.92±0.12 | 2.19±0.17 | <0.001 |

### Table 7: Relation between specific pregnancy complications with H. pylori titer

| Complication            | H. pylori titer |      |      | P value |
|-------------------------|-----------------|------|------|---------|
|                         | Positive (n=46) | Equivocal (n=7) | Negative (n=47) |      |
| Eclamptic fit           | 3               | 0    | 1    | 0.706   |
| Immenent eclampsia      | 11              | 1    | 0    | 0.033   |
| FGR                     | 16              | 4    | 4    | 0.001   |
| NEED N.C.U              | 16              | 1    | 10   | 0.114   |
| HELLP syndrome          | 4               | 0    | 1    | 0.620   |
| Placentral abruption    | 10              | 0    | 0    | 0.016   |
| IUD                     | 11              | 0    | 1    | 0.04    |

### Table 8: Distribution of groups according to H. pylori test in stool.

| Distribution of groups      | Normal (%) | Mild PE (%) | Sever PE (%) | Total |
|-----------------------------|------------|-------------|--------------|-------|
| Negative H. pylori test in stool | 38 (69)    | 6 (10.9)    | 11 (20)      | 55    |
| Positive H. pylori test in stool | 12 (26)    | 11 (24)     | 22 (48.8)    | 45    |

### Table 9: Incidence of persistent epigastric pain in patients with positive H. pylori test in stool.

| Asymptomatic | Mild pain | Moderate pain | Severe pain |
|--------------|-----------|---------------|-------------|
|              | 4 (8%)    | 15 (33.3%)    | 20 (44.4%)  |
|              | 6 (13.3%) |

### Table 10: Endoscopic findings for symptomatic patients with positive H. pylori test in stool.

|               | Gastritis (%) | Duodenal ulcer (%) | Gastric ulcer (%) | Atrophic gastritis |
|---------------|--------------|--------------------|-------------------|-------------------|
| Mild pain     | 30 (73)      | 1 (2.4)            | 0                 | 0                 |
| Moderate pain | 4 (9.7)      | 3 (7.3)            | 1 (2.4)           | 0                 |
| Severe pain   | 1 (2.4)      | 1 (2.4)            | 0                 | 0                 |
| Total         | 35 (85.3)    | 5 (12.1)           | 1 (2.4)           | 0                 |

Table 5 and 6 showed that sever PE associated significantly with higher seropositivity of H. pylori (IgG) titer (47.8%) than other groups, (28.3%) in normal and (23.9%) in mild preeclampsia (p=0.007). The titer of H. pylori significantly higher with severity of PE (p<0.001). Table 7 showed that there is significant difference between those with positive H. pylori titer and those with negative and equivocal result regarding imminent eclampsia, intrauterine death, placental abruption and fetal growth retardation (p<0.05). There is no significant difference between negative, equivocal and positive titer regarding eclampsia with eclamptic fit, HELLP syndrome and need for neonatal care unit (p>0.05).

Table 8 showed that positive H. pylori test in stool was seen in 45 (45%) of total number of patients. 22 (48.8%) out of the 45 with positive stool test are severe preeclampsia against 11 (24%) with mild preeclampsia and 12 (26%) with no preeclampsia. Table 9 showed that symptoms as epigastric pain persist in 41 (92%) patients out of 45 patients with positive for H. pylori test in stool after six weeks follow up (post-delivery or after termination of pregnancy), while 4 (8%) were asymptomatic.

When endoscopy done, most of the cases with H. pylori positive stool test that complaining of epigastric pain are found to have gastritis (73%) followed by duodenal ulcer (12.1%) and gastric ulcer (2.4%). Atrophic gastritis or other serious pathologies were not encountered (Table 10).

### DISCUSSION

Table 1 showed that blood pressure and proteinuria are significantly higher in cases of preeclampsia than in normal pregnant women. These results are supported by Maybury et al and reflects that preeclampsia is a multiorgan disease (systemic endothelial dysfunction affecting renal glomeruli leading to increase protein loss to the urine). It is noticed in the same table that there is no significant association between preeclampsia in one hand and age of the mothers and parity in the other hand.
These findings were different from other studies because we excluded the cases of preeclampsia who had already affected by other diseases like diabetes, essential hypertension or any other medical illness that occur more in older age group which increase the incidence risk of preeclampsia.12

Table 2 showed that preeclampsia was found to be significantly affecting primipara more than subsequent pregnancies. It is thought that the normal fetal-maternal transfusion that occurs during pregnancy and particularly during delivery exposes the mother to products of the fetal (and hence placental) genome, protecting her in subsequent pregnancies.13 Preeclampsia is found significantly more in cases of multiple pregnancies. This can be explained by the larger placental tissue size that occur in multiple pregnancy cases which reflects the fact that the placenta plays a central role in preeclampsia pathogenesis.14

Preeclampsia is found significantly more in grandmultipara mothers (tended to be older and heavier than those with fewer children and had significantly higher systolic blood pressure readings than those with less than five children) and in mothers with history of preeclampsia in previous pregnancies. These results agreed by Sibai et al.15 Family history of diabetes, hypertension and cardiovascular disease in our study showed no significant association with preeclampsia. This is comparable with what is found by Antonio et al.16

In Table 3, there was significant relation between severity of pre-eclampsia and low gestational age due to the increase risk of preterm labour and increase tendency to iatrogenic termination of pregnancy to prevent and decrease maternal and fetal complications. (In this study 78.8% of severe pre-eclampsia delivered by C.S in comparison with 41.6% of mild and 26% of normal women). In the same time severe pre-eclampsia had significantly low birth weight and this is due to placental insufficiency and fetal growth restriction as a complication of preeclampsia and iatrogenic prematurity. This agreed by Hauth et al who found that the risk of preterm labour, caesarean section and prematurity increase with severity of pre-eclampsia.17

In our study, Table 4 demonstrated that severe pre-eclampsia show high blood urea, serum creatinine and serum uric acid which means renal dysfunction due to ischemic changes as shown by Mustafa et al.14 Liver enzymes (SGPT, SGOT, SALP) showed marked elevation in severe pre-eclampsia than in other groups and as Errol et al said, this is due to liver injury which is caused by endothelial dysfunction and hepatic sinusoidal obstruction leading increase intrahepatic pressure.18 It is noticed that in severe pre-eclampsia both Hb% and platelet count decrease (especially in HELLP syndrome) if compared with their levels in the other two groups. This may be explained by microangiopathic haemolytic anaemia. The same results were found also in studies done by Errol et al.18,19

In Our study, Table 5 has shown that there is significant difference in H. pylori seropositivity among normal (28.3%), mild and severe pre-eclampsia where positive titre was found in 71.7% of the total cases of preeclampsia. These results are comparable with that of Ponzetto et al where seropositivity for H. pylori was found in 51.1% of mothers with pre-eclampsia while only 31.9% of normal pregnant women were found to be seropositive for H. pylori.16 These results reflected the significant correlation between pre-eclampsia and H. pylori infection which is predominantly acquired during childhood and it is lifelong when untreated. This finding supported the speculation that H. pylori positive women may have underlying vascular damage; such subclinical dysfunction might augment the inflammatory changes in pregnancy, thus contributing to the symptoms of pre-eclampsia.20

The level of H. pylori titre in this study (Table 6) was found to be significantly higher in mothers with preeclampsia if compared with normal mothers group and the level increase as the severity of preeclampsia increase. This may drew the attention to the importance of this microorganism in the pathogenesis of this disorder. This is supported by Shiaedeh et al.21 Several studies have found indirect evidence of the involvement of the infection in the pathogenesis of pre-eclampsia.22,23 Aquedo et al has found that the risk of preeclampsia was increased in pregnant women with urinary tract infection and periodontal disease.19

This this study (Table 7) showed significant association between positive titer for H. pylori and imminent eclampsia, IUD (intrauterine death), IUGR (intrauterine growth retardation) and placental abruption. Other complications like eclamptic fit, HELLP syndrome and the need for NCU (neonatal care unit) have no significant association with H. pylori seropositivity. This is very important to choose the proper time for delivery or termination of pregnancy complicated by hypertension and need further evaluation.

This study (Table 8 and 9) showed that 45 patients (45% of total number of patients) had positive H. pylori test in stool, of which 4% were asymptomatic and 41 patients (96%) show different severity epigastric pain. Oung et al found that only 46% of patients with dyspepsia/pain symptoms had H. pylori infection.24 This difference may be due to the type of the study population.

Table 10 showed that most of the cases with positive H pylori stool test that complaining of epigastric pain are found to have gastritis (73%) followed by duodenal ulcer (12.1%). This is supported by the results by Gravina et al who noticed that 80-85% of the infected patients had mild gastritis and 10-15% had more severe antral gastritis with the possibility to develop duodenal ulceration due to
increased acid secretion. Lower percentage may develop body gastritis with decreased acid secretion that may complicate to atrophic gastritis and adenocarcinoma.

CONCLUSION

The results of our study concluded that there is significant correlation between H. pylori seropositivity and preeclampsia. There is also a linear association between the level of H. pylori antibody titer and the severity of preeclampsia and its complication. These parameters may be used as a predictor of the disease and its severity and its future therapeutic options and pregnancy outcome.

This study showed the importance of H. pylori infection that can lead to persistent gastrointestinal symptoms with increasing frequency in pregnancy and puerperium that can end with serious gastric (medical and surgical) complication and drew the attention to the need for further studies on larger samples to assess the benefit of prophylactic eradication of H. pylori before getting pregnant.

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