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ORIGINAL ARTICLE

Assessment of The Relationship Between Helicobacter Pylori Infection and Hyperemesis Gravidarum.

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ABSTRACT

Background: Hyperemesis gravidarum is one of the many complications of pregnancy; its etiology has not been fully understood. Inflammatory factors such as helicobacter pylori infection have been found a risk factor in several studies. The objective of this analysis was to assess the relationship between infection with Helicobacter pylori (H.P) and Hyperemesis Gravidarum (HEG).

Methods A case/control study, conducted at the department of Obstetrics and Gynecology, Zagazig university hospitals, during the period from May 2017 to February 2018. It included 190 pregnant women who were in the first trimester of pregnancy. 95 pregnant women with hyperemesis gravidarum, and the other 95 pregnant women of the same gestational age but without hyperemesis gravidarum. IgG for H.P was calculated and compared between the two groups.

Results: There were a statistically insignificant difference in age or BMI, parity, gestational age, gravidity and history of abortion between the study groups. There were significant difference between both groups regarding Serum H. pylori IgG (p.value: 0.047) and stool *H.pylori* antigen (P.value <0.001) where both Serum *H. pylori* IgG and stool *H. pylori* antigen were higher in the study group than the control group. There was highly statistically significant difference between study groups regarding hemoglobin level (p.value: 0.001), ketone bodies in urine (P.value: <0.001) and vomiting, nausea, retching domains and total PUQE score (P.value <0.001).

Conclusions: There was an association between H. pylori infection and hyperemesis gravidarum, and H. pylori was considered a risk factor for H.G, not the sole cause of H.G.

Key words: Pregnancy, Hyperemesis gravidarum, Helicobacter pylori.

INTRODUCTION

Nausea and pregnancy vomiting have become a very common condition from a long period. While not well known, it occurs in about 70% of pregnant women. Although "morning sickness" considered a usual complain, it is typically more serious when it's severe. While the etiology still unknown, the efficacy of treatment is uncertain. Over the years, the conventional method of providing symptomatic anti-emetic treatment without much awareness and trust has not changed **[1]**.

Hyperemesis gravidarum (HG) is the sever presentation of nausea and vomiting in pregnancy and is characterized by intractable nausea and vomiting leading to dehydration, electrolyte and metabolic disorders, and nutritional deficiency that may require hospitalization **[2]**.

Relatively little is understood about hyperemesis etiology. The effects of sex hormones, thyroid hormones, *H. Pylori* infections and paternal genes have been proposed, but no consensus has been achieved. Human chorionic gonadotropin (HCG) levels are positively correlated with the frequency and extent of hyperemesis symptoms as seen in multiple-or molar pregnancy **[3]**.

Estimates of extreme nausea and pregnancy vomiting differ widely and range from 0.3 percent in the Swedish registry to as high as 10.8 percent in

the Chinese registry of pregnant women, with most researchers recording nearly 0.5 percent incidence **[4]**.

Recent systematic reviews show that HG raises the risk of low birth weight and small for gestational age (SGA) by 42 per cent and 28 per cent, respectively **[5].**

Helicobacter pylori (*H.pylori*) are a Gram-negative, microaerobic human pathogen. *H Pylori* infection is closely associated with many gastroduodenal diseases, including chronic active gastritis, atrophic gastritis, peptic ulcer disease, mucosal associated lymphoid tissue (MALT) lymphoma **[6]**.

High prevalence of *H. pylori* was observed in the women having preeclampsia during pregnancy. The main gastrointestinal symptom, hyperemesis gravidarum, was also associated with this bacterium, although the other research showed contradictory findings. These findings raise questions about the need to know the status of *H. pylori* infection in pregnant women. A meta-analysis revealed that there was a clear correlation between *H. pylori* infection and HG and concluded that *H. pylori* could therefore be considered as one of the risk factors for HG. Screening for H pylori should be added to the HG investigations, especially in developing countries. Appropriate treatment regimens for the eradication of *H. pylori* can be considered to alleviate symptoms of HG in certain intractable conditions **[7]**.

The rate of *H. pylori* detection was nearly five times higher in HG cases than in asymptomatic cases. According to these results, it was proposed that the *H. pylori* diagnostic tests should be a part of the investigation of HG. The Helicobacter pylori Stool Antigen (HpSA) test considered as a non-invasive method that offers more dependable data to detect *H. pylori* infection during pregnancy than antibodies found in the serum **[8]**.

In another study, no association was found between Helicobacter *pylori* and hyperemesis gravidarum. The poor social status of women in both groups may be one explanation for the high prevalence of *H.pylori* infection. **[9]**.

As *H. pylori* infection may occur before pregnancy, it is widely believed that hormonal and immunological changes occurring during pregnancy may initiate latent *H. pylori* with adverse effects not only on maternal health (nutritional deficiency, organ injury, death), but also on the fetus (insufficient development, malformation, death) **[10].**

Therefore, if *H. pylori* infection is established as a significant risk factor for pregnancy complications, the traditional *H. pylori* eradication, triple therapy, should preferably be completed several months before conception in order to achieve seronegativity. This strategy will prevent cross-reaction between anti-*H. Pylori* antibodies and host tissue antigens, while awaiting the development of novel effective vaccines [10]**.**

The objective of this analysis was to assess the relationship between infection with Helicobacter pylori (H.P) and Hyperemesis Gravidarum (HEG).

PATIENTS AND METHODS:

After obtaining approval of the ethics committee, the study was conducted as a Comparative observational (case/control) study on pregnant women who referred to department of Obstetrics and Gynecology, Zagazig university hospitals, during the period from May 2017 to February 2018. It included 190 pregnant women in the first trimester of pregnancy (6-14 w). 95 pregnant women with hyperemesis gravidarum, and 95 pregnant women of the same gestational age but without hyperemesis gravidarum.

Written informed consent was obtained from all participants and the study was accepted by the Research Ethics Committee of the Faculty of Medicine, Zagazig University. Research has been carried out on research concerning human subjects in compliance with the Code of Ethics of the World Scientific Association (Declaration Helsinki).

Inclusion criteria: PUQE score (Pregnancy-Unique Quantification of Emesis) was used in diagnosis of cases of HEG. Then both groups have gestational age of 6-16 weeks of pregnancy (depending on accurate LMP and ultrasound confirmation), singleton, desired gestation, and BMI between 18.5-24.9 kg / m^2. **Exclusion criteria:** Includes molar pregnancy, history of any systemic disease or any medication other than ordinary treatment (i.e. folic acid), history of any gastrointestinal (GI) disease or, smoking or drug misuse, hyperthyroidism, and any psychological problems.

Method: PUQE score (Pregnancy-Unique Quantification of Emesis): PUQE was designed to determine the extent of emesis (nausea and vomiting) during pregnancy in all patients included in the study. This questionnaire contains three questions regarding the time-span of nausea, vomiting and retching respectively, as well as one question assessing the global psychological and physical quality of life (QOL).

The three PUQE questions each have a rating from 1–5, thus the composite sum (PUQEscore) ranged from 3–15. A score between 3–6 points was defined as mild nausea and vomiting in pregnancy (NVP), 7–12 points as moderate NVP and scores 13-15 points was classified as severe NVP/HG as shown in figure (1) [11].

Full history taking from all participants in the study. Thorough clinical examination including the height and weight to determine the body mass index (BMI=weight kg/height m^2)and an ultrasound evaluation.

Laboratory investigations: Blood samples were drawn on admission for routine laboratory investigations. Complete blood picture (hemoglobin levels, TLC, platelet count) was assayed. The tests were sent for analysis in Zagazig university hospital laboratories and results were compared between the two groups. Also urine ketones were measured for all patients.

H pylori antibody (Ig G) in serum;

Two milliliters of venous blood have been taken from all participants. blood samples were drawn into the Gel & Clot, yellow colored, vacuum tubes(4–ml) ,without anti-coagulant, and serum was separated by centrifugation immediately then kept in dry clean eppendorf tubes and freezed at -18oc until all sampls are collected. Then samples have been sent to zagazig university clinical pathology department and analyzed under supervision of clinical pathology department teamwork. Serum Immunoassay for H. Pylori IgG seropositivity was done for all patients using Enzyme Linked ImmunoSorbent Assay (ELIZA) technique.

This is the Calbiotech H. Pylori IgG ELISA kit is designed for the detection of IgG antibody to H. pylori human serum and plasma (figure 2a).

Explanation of the test: Dilute patient serum is applied to wells filled with a filtered antigen. When IgG-specific antibody is present, it binds to the antigen. All unbound materials are washed away and the enzyme conjugate is added to bind the antibody-antigen complex. Excess enzyme conjugate is washed away and the substrate is added. The plate is incubated to allow the enzyme to hydrolyse the substrate. The strength of the color produced is proportional to the amount of Ig G specific antibody in the sample.

H.pylori stool antigen (HpSA): New fecal samples should be obtained in a stool sample storage jar. A minimum of 1-2 ml of liquid stool sample or 1-2 g of solid sample should be obtained. Then all samples have been sent to the clinical pathology lab and analysed by ELIZA technique according to the manufacture procedure as before (figure 2b).

Outcomes measures: Assessing the association between maternal infections with Helicobcter pylori, and the occurrence of Hyperemisis Gravidarum with bad maternal or fetal perinatal outcomes.

Statistical Analysis: All data were statistically analyzed and tabulated using the SPSS (Statistics System for Social Science Version 20) of IBM computer. The statistical significance point was set at $P<0.05$. Highly important differences were present when $p\leq0.001$.

RESULTS

The mean age was 27.27 ± 5.82 years for the Hyperemesis group and 28.43 ± 6.07 years for the control group with a statistically non-significant difference between the study groups (P=0.181). The mean of the body mass index was 23.15 ± 1.4 for the Hyperemesis group and 23.16 ± 1.41 for the control group a statistically non-significant difference between both groups (P=0.188) (Table 1).

The mean of the hemoglobin level was 10.69 ± 1.54 mg/dl for the Hyperemesis group and 11.39 ± 1.23 mg/dl for the control group with highly statistically significant difference between both groups (P= 0.001). Urine ketones were 2 (1-3)mmol/ L for the Hyperemesis group and Zero for the control group showing highly statistically significant difference between both groups (P <0.001).There are statistically non-significant differences between both groups regarding parity (P= 0.335), gestational age (P=0.109), gravidity (P= 0.05) and history of abortion (P= 0.444) (table 2).

Regarding PUQE score in relation to H.pylori positive (82) cases and H.pylori negative (108) cases, there is highly statistically significant difference between both groups (P <0.001), especially in the severe class where 51.2% of the H.pylori positive cases are presenting severe score while 21.5% of the H.pylori negative cases are presenting severe score (figure 3) revealing the relation the H.pylori infection and hyperemesis gravidarum .

The serum IgG in the hyperemesis group was negative in 24 (25.3%) cases, borderline in 11 (11.6%) cases and positive in 60 (63.1%) cases, while in the control group the serum IgG was negative in 28 (29.4%) cases, borderline in 22 (23.2%) cases and positive in 45 (47.4%) cases with statistically significant difference between the study groups (P=0.047). Stool H.pylori antigen in the hyperemesis group was negative in 33 (34.7%) cases and positive in 62 (65.3%) cases but in the control group the stool H.pylori antigen was negative in 75 (78.9%) cases and positive in 20 (21.1%) cases with highly statistically significant difference between both the study groups revealing a relation between the infection with H.pylori and hyperemesis gravidarum (table 3). Odds ratio for occurrence of hyperemesis showed that Presence of stool H.pylori antigen significantly increases risk of hyperemesis by about 7 times (table4).

Table (1) Demographic criteria of study groups:

	Hyperemesis group	Control group	T	p
Age: Mean ± SD range	 27.27 **± 5.82** **19 – 38**	 28.43 **± 6.07** **19 -39**	 -1.343	 0.181
BMI: Mean ± SD range	 23.15 **± 1.4** **18.6 – 24.6**	 23.16 **± 1.41** **18.6 – 24.9**	 -0.014	 0.188

Table (2) Obstetric data of study groups (Gestational age, parity, Gravidity and abortion):

	Hyperemesis group	Control group	Test	p
Gestational age: Mean ± SD range	 10.28 ± 2.08 7 – 14	 9.8 ± 2.03 6-14	 1.609	 0.109
Parity: Median Mean ± SD range	 1 1.16 ± 1.16 0 – 4	 1 1.25 ± 1.04 0 -5	 -0.965	 0.335
Gravidity: Median Mean ± SD range	 2 2.42 ± 1.57 0 – 7	 3 2.86 ± 1.69 0 -7	 -1.957	 0.05
Abortion: Median Mean ± SD range	 0 0.55 ± 0.85 0 – 4	 0 0.52 ± 0.86 0 - 3	 -0.765	 0.444

*p<0.05 is statistically significant

Table (3) Serum H.pylori IgG and stool H.pylori antigen in study groups:

	Hyperemesis group	Control group	X^2	P
	N (%)	**N (%)**		
Serum IgG: Negative Borderline Positive	 24 (25.3) 11 (11.6) 60 (63.1)	 28 (29.4) 22 (23.2) 45 (47.4)	6.117	0.047*
Stool H.pylori antigen: Negative Positive	 33 (34.7) 62 (65.3)	 75 (78.9) 20 (21.1)	37.846	<0.001**

*p<0.05 is statistically significant
**p≤0.001 is highly significant

Table (4) Odds ratio for occurrence of hyperemesis:

	OR	CI	P
Stool antigen	7.045	3.68 – 13.487	<0.001**

**p≤0.001 is highly significant

PUQE form:

Pregnancy-Unique Quantification of Emesis and nausea

Circle the answer that suit the best your situation for the last 24 hours.

1. On average in a day, for how long do you feel nauseated or sick to your stomach?

> 6 hours 5 points	4-6 hours 4 points	2-3 hours 3 points	≤1 hour 2 points	Not at all 1 point

2. On average in a day, how many times do you vomit or throw up?

≥7 times 5 points	5-6 times 4 points	3-4 times 3 points	1-2 times 2 points	Not at all 1 point

3. On average in a day, how many times have you had retching or dry heaves without bringing anything up?

≥7 times 5 points	5-6 times 4 points	3-4 times 3 points	1-2 times 2 points	Not at all 1 point

Total score (sum of replies to 1, 2, and 3): mild NVP ≤6; moderate NVP, 7-12; severe NVP ≥13.

Quality of life question:
On a scale of 0 to 10, how would you rate your well-being:_____
0 (worst possible) 10 (As good as you felt before pregnancy)

PUQE form modified from: Koren G, Boskovic R, Hard M, Maltepe C, Navioz Y, Einarson A. Motherisk-PUQE (pregnancy-unique quantification of emesis and nausea) scoring system for nausea and vomiting of pregnancy. American journal of obstetrics and gynecology. 2002;186:S228-31, with permission.

Figure (1). PUQE-questionnaire [11].

Figure (2) showing a photograph of ELISA kits for (a) serum H pylori antibody (IgG), (b) H pylori stool antigen.

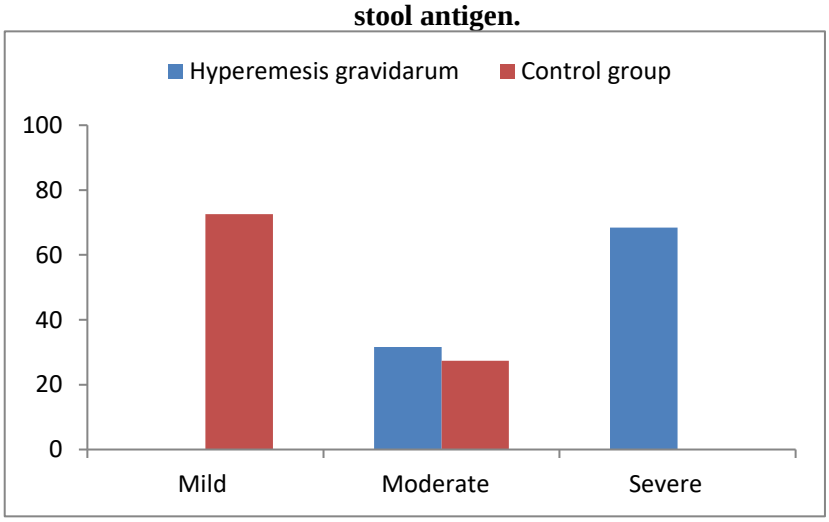

Figure (3) Combined bar chart showing severity of PUQE score in study groups

DISCUSSION

Hyperemesis gravidarum describes nausea and vomiting that are severe enough to cause fluid and electrolyte imbalance and frequently require hospitalization. This affects up to 1% of pregnant women and is associated with frequent vomiting (more than three times a day) resulting in extreme dehydration, ketonuria, electrolyte disorders such as hypokalemia, and weight loss of more than 5% [12].

The exact etiology of HEG is not clearly established, but can be regarded as a multi-factorial problem under investigation. Relationships between HP and HEG have recently been recorded [13]**.**

In the current study the age range of the hyperemesis group was 19-38 y (and mean age of 27.27 ± 5.82), while in the control group the age range was 19-39 y (and mean age of 28.43 ± 6.07) with statistically non-significant difference between both groups. This is in agreement of the results of **Elmahdy M et al.,** [14]**; Abd Alwahed et al.,** [15]**,** while **Guven M.A et al.,** [16] concluded that young maternal age was associated with an increased rate of HG and H pylori.

The body mass index (BMI) of the hyperemesis group in the current study is 23.15 ± 1.4 and BMI of the control group is 23.16 ± 1.41 with statistically non-significant difference between both groups. This is in accordance with **Abd Alwahed et al., [15]** while **Elmahdy M, et al., [14]** found that body weight among patients with hyperemesis was significantly lower compared to controls [17]. And concluded that low pre-pregnancy weight: height ratio can develop hyperemesis.

According to hemoglobin concentration in our study, there is a high statistically significant difference between the two groups. **Kabir S, et al., [18]** found that the patient with Hyperemesis gravidarum usually has illness presentation, vomiting, marked body weight loss and anemia. In the other hand, **Fatma Beyazit et al., [19]** found a non-significant difference between hyperemesis and control classes. Further in the **Agmon N et al., [20]** study, hematocrit and hemoglobin values were significantly higher in hyperemesis patients, where vomiting episodes was longer, this may be due to haemoconcentration resulted from severe vomiting and dehydration.

There was highly statistically significant difference between study groups regarding urine ketones. This was in accordance with **Tan PC et al., [21]** who reported a rise in urinary ketone levels in patients with HG. Also **Arslan S. et al., [22]** concluded that fast, bedside capillary blood ketone measurements can accurately help diagnosis of hyperemesis gravidarum in pregnancy-induced nausea and vomiting. There is a clear correlation between capillary blood ketone and urinary ketone values, especially at low levels.

In the current study there is statistically non-significant difference between both groups regarding the gestational age this is consistent with the study of **Fatma Beyazit et al., [19].**

Regarding the gravidity in this study, there is statistically non-significant difference between both groups. This is consistent with the study of **Elmahdy M et al., [14] and Fatma Beyazit et al., [19].**

The parity in the hyperemesis group in our study was1.16 **± 1.16**, and1.25 **± 1.04** in the control group with statistically non-significant difference between both groups**.** It was consistent with the findings of **Elmahdy M et al., [14]** while **Giri et al.,[23]** concluded that hyperemesis gravidarum decreased with increased parity indicating that it was predominantly a disease of nulliparous women. The disease rarely occur in women with parity more than three

Regarding abortion, in the current study, there was no statistically significant difference between the two classes and this was in accordance with **Elmahdy M et al., [14].**

No single test can be easily specified, quantified or measured for the treatment of hyperemesis, but the English Pregnancy Specific Questionnaire PUQE (Pregnancy-Unique Quantification of Emesis) has been developed to determine the extent of emesis (nausea and vomiting) in pregnancy **[24].** Detailed history and accurate examination are needed to exclude differential diagnosis. Moreover, lab investigations are useful tools to assess the complications **[25]**.

In the current analysis, considering PUQE, there was a high statistically significant difference between the two groups in the vomiting score, nausea score and retching score. The overall PUQE score was (11.44 ± 3.73) for the positive *H.pylori* group and (7.72 ± 4.22) for the negative *H.pylori* group. This was in line with **Birkeland et al., [11],** where PUQE scores were significantly higher in patients with extreme HG compared to the control group.

Nashaat et al., [26] concluded that chronic infection with Helicobacter pylori may have a role in hyperemesis gravidarum. However, **Sandven I et al., [27]** were unable to demonstrate a connection between seropositivity for Helicobacter pylori and time of onset or period of hyperemesis.

In our study, regarding the serum *H.Pylori* IgG; for the hyperemesis gravidarum group 24 (25.3%) cases were negative and 60 (63.1%) cases were positive, while for the control group 28 (29.4%)

cases were negative and 45 (47.4%) cases were positive with statistically significant difference between both groups (P. value=0.047). this was in agreement with the study of **El-Garhy et al., [28]** where the serum IG were positive in 80% of hyperemesis patients compared with 30% in the controls with statistically significant difference between the study groups (P<0.001)

For stool *H.pylori* antigen in our study, 33 (34.7%) cases were negative and 62 (65.3%) cases were positive in the hyperemesis group, while in the control group 75 (78.9%) cases were negative and 20 (21.1%) cases were positive with statistically high significant difference between both groups (P.value <0.001). then by considering the Odds ratio for occurrence of hyperemesis, the presence of stool *H.pylori* antigen significantly increases risk of hyperemesis by about 7.045.

Elmahdy M et al., [14] concluded that Helicobacter pylori was found to be positive in stool samples in 75 percent of HEG cases and positive in 37.50 percent of normal pregnant women. The findings were statistically significant (p = 0.001). The prevalence of Hp IgG AB and HpS AB was 77.5 per cent in HEG patients and 55.0 per cent in control patients (P = 0.05). Therefore, *H.pylori* infection can be one of the risk factors for HEG. The prevalence of *H.pylori* AB in both serum and stool is higher in HEG cases than in normal pregnant women.

Salimi et al., [29] concluded that Positive serum *H. pylori* IgG antibody was observed in 88.9 percent of the experimental patients compared to 40.7 percent of the controls (P<0.001). While more HG patients were seropositive to *H. pylori* infection than controls, they were unable to show an association between *H. pylori* seropositivity and onset of HG symptoms and duration of disease. While *H. pylori* infection may be an important factor in exacerbating HG, it may not be the only cause of the disease.

The meta-analysis of **Lin Li et al., [7]** agreed with our study and indicated that there was a clear correlation between *H. pylori* infection and HG and concluded that *H. pylori* should therefore be considered as one of the risk factors for HG. Screening for *H. pylori* should be added to the HG investigations, especially in developing countries. Appropriate treatment regimens for the eradication of *H. pylori* can be considered to alleviate symptoms of HG in certain intractable situations.

Also, **Mashaallah K et al., [13]** concluded that HP infection is higher in HEG cases and can be seen as a risk factor. The pathophysiology of HG remains controversial. However, pregnancy can be associated with increased susceptibility to *H. pylori* infection, and it has been hypothesized that a change in the gastrointestinal tract pH during early pregnancy as a result of increased body fluid deposition, shifts in steroid hormones, and immunological tolerance may lead to the activation of latent *H. pylori* infection, which may exaggerate the symptoms of nausea and vomiting **[16].**

Bezircioğlu, et al., [30] concluded that the prevalence of *Helicobacter pylori* was also shown to be significantly higher in pregnant women with Hyperemesis gravidarum relative to control groups.

At the other hand, **Karadeniz RS, et al., [9]** was unable to consider a relation between Hp and HG. The poor social status of women in both groups may be an explanation for the high prevalence of Hp infection.

In contrast to our research, **Vikanes et al., [31]** did not find that *H. pylori* exposure was significantly correlated with extreme HG among immigrant women in Norway. It was either when *H. pylori* exposure was examined by IgG seropositivity, CagA and VacA seropositivity, or when *H. pylori* antigens were found in stools. These findings may show that the association between *H. pylori* and HG is lower than previously thought, especially in populations with a high prevalence of *H pylori* infection.

In a study by **Shirin et al., [32]** the relationship between *H. pylori* infection and the occurrence of gastric upset during pregnancy was investigated and a substantial correlation was found between this infection and hyperemesis. However, not all gastric symptoms of pregnancy were associated with *H. pylori* infection. The findings of this analysis were identical to our study.

The causal association between the development of *H. pylori* infection and the persistence of symptoms of dyspepsia and hyperemesis after the first three months of pregnancy has been established **Poveda et al., [33]**. Pregnant women with dyspepsia have been reported to have a weak to moderate response to *H. pylori* infection, whereas women with hyperemesis have shown an elevated response to infection; supporting the association between the frequency of symptoms and the degree of infection **Shaban et al., [34]**, in which there is a clear connection between *H. pylori* and hyperemesis *gravidarum*.

Ahmed et al., [35] recommended that Screening for Helicobacter pylori should be added to the HG investigations, particularly if it is prolonged or refractory to conventional management. Modified, high-dose, non-teratogenic dual therapy for eradication of Helicobacter pylori may be considered to relieve HG in intractable cases with minimal side effects.

Abd Alwahed et al., [15] have concluded that the eradication of Helicobacter pylori greatly improves the outcomes for the treatment of hyperemesis gravidarum. The role of Helicobacter pylori in the pathogenesis of hyperemesis gravidarum is therefore highly suggestive.

CONCLUSIONS

There was an association between *H. pylori* infection and hyperemesis gravidarum, and *H. pylori* was considered a risk factor for H.G, not the sole cause of H.G.

RECOMMENDATION

H. pylori infection screening is recommended for women of child-bearing age. Many found to be positive for *H. pylori* infection should be treated before becoming pregnant, as the current treatment regimens for *H. pylori* are not safe during pregnancy. If *H. pylori* infection is established as a contributing factor to H.G, the potential challenge will be to determine whether the eradication of *H. pylori* will improve the occurrence of H.G or decrease the frequency of its clinical presentation.

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