International Surgery Journal
Hasan JS et al. Int Surg J. 2020 Jul;7(7):2097-2102
http://www.ijsurgery.com
pISSN 2349-3305 | eISSN 2349-2902

DOI: http://dx.doi.org/10.18203/2349-2902.isj20202810

Original Research Article

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

Jehan Sabah Hasan[1]*, Mohammed Ahmed Alshami[2]

[1]Department of Gynecology, Karbalaa Health Directorate. Karbala, Iraq
[2]Department of General Surgery, Al-Hussain Medical City, Karbala, Iraq

Received: 25 February 2020
Revised: 09 May 2020
Accepted: 11 May 2020

***Correspondence:**
Dr. Jehan Sabah Hasan,
E-mail: geehansabah76@gmail.com

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 includeed 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.[5] It has been demonstrated that this pathogen enhances platelet activation and thrombus formation6 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.[7] *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.[8]

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.[9] However, Mendall et al in was the first to notice the association between *H. pylori* infection and extra-gastric diseases.[10]

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 2 to 4+ on dip stick test appeared after twenty weeks of gestational age on two occasions in previously normotensive non proteinuric women.

D.M., chronic renal disease and chronic hypertension were excluded in the selected sample. For all cases following data were collected: maternal age, maternal weight, parity, infant birth weight, type of delivery, B.P, urinary protein, drug intake in pregnancy such as antihypertensive, corticosteroids, aspirin) risk factors for P. E. (previous history for P. E., auto immune disease, multiple pregnancy, primegravida and grand multipara), family history and maternal and fetal complication. The B.P. was taken on the right arm in the sitting position with the patient at rest using a mercury sphygmomanometer with an appropriate cuff placed at heart level.

Investigations and procedures included urine sample collection for estimation of albumin in urine (mid-stream) 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 immunosorbant 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 (x^2) 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 proteinurea (p<0.001).

Table 1: Patients characteristics of each group.

Characteristics	Normal	Mild PE	Sever PE	P value
Number (patients)	**n=50**	**n=17**	**n=33**	
Age	27.34±4.62	26.69±5.33	26.443±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 N (%)	Mild PE n=17 N (%)	Sever PE n=33 N (%)	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

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

Characteristics	Normal	Mild PE	Severe PE	P value
Number (patients)	**n=50**	**n=17**	**n=33**	
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%)	

Table 4: Lab results of each group.

Characteristic	Normal	Mild PE	Sever PE	P value
Blood urea (mg/dl)	25.427±3.76	27.581±4.34	36.794±4.859	<0.001
S.creatinine (mg/dl)	0.571±0.194	0.821±0.259	1.089±0.368	<0.001
S.uric acid (mg/dl)	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 ($p>0.05$).

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 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.

Charact-eristic	Normal N (%)	Mild PE N (%)	Sever PE N (%)	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
Placental 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).[11] 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 Shiadeh et al.[21] Several studies have found indirect evidence of the involvement of the infection in the pathogenesis of pre-eclampsia.[22,23] Aqudelo et al has found that the risk of preeclampsia was increased in pregnant women with urinary tract infection and periodontal disease.[19]

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.[9] 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 upper 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.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Graham DY. History of *Helicobacter pylori*, duodenal ulcer, gastric ulcer and gastric cancer. World J Gastroenterol. 2014;20(18):5191-204.
2. Kivi M, Johansson AL, Reilly M, Tindberg Y. Helicobacter pylori status in family members as risk factors for infection in children. Epidemiol Infect. 2005;133:645.
3. Ehab HN, Ghada MM. *Helicobacter pylori* and *Hyperemesis Gravidarum*. Nature Sci. 2010;8(8):44-9.
4. Amieva MR, El-Omar EM. Host-bacterial interactions in *Helicobacter pylori* infection. Gastroenterol. 2008;134:306.
5. Gasbarrini G, Racco S, Franceschi F, Miele L, Cammarota G, Grieco A, et al. *Helicobacter pylori* infection: from gastric to systemic disease. Recenti Prog Med. 2010;101(1):27-33.
6. Xia HH, Lam SK, Chan AO, Lin MC, Kung HF, Ogura K, et al. Macrophage migration inhibitory factor stimulated by *Helicobacter pylori* increases proliferation of gastric epithelial cells. World J Gastroenterol. 2005;11:1946-50.
7. Cardaropoli S, Rolfo A, Piazzese A, Ponzetto A, Todros T. *Helicobacter pylori's* virulence and infection persistence define pre-eclampsia complicated by fetal growth retardation. World J Gastroenterol. 2011;17(47):5156-65.
8. Bellack NR, Koehoorn MW, MacNab YC, Morshed MG. A conceptual model of water's role as a reservoir in *Helicobacter pylori* transmission: a review of the evidence. Epidemiol Infect. 2006;134:439.
9. Gravina AG, Zagari RM, De Musis C, Romano L, Loguercio C, Romano M. *Helicobacter pylori* and extragastric diseases: a review. World J Gastroenterol. 2018;24(29):3204-21.
10. Mendall MA, Goggin PM, Molineaux N, Levy J, Toosy T, Strachan D, Camm AJ, et al. Relation of *Helicobacter pylori* infection and coronary heart disease. Br Heart J. 1994;71:437-9.
11. Mayburry M, Waugh J. Proteinuria in pregnancy. Fetal Andmaternal Med Review. 2004;16:71-95.
12. Duckitt K, Harrington D. Risk factors for pre-eclampsia at antenatal booking: systematic review of controlled studies. BMJ. 2005;330:565.
13. Kenny LC, Myers J. Epidemiology of preeclampsia. In Obstetrics by ten teachers. 19th edition. Hodder Arnold. 2011: 122.
14. Mustafa R, Ahmed S, Gupta A. Rocco C, Venuto A. Comprehensive review of hypertension in pregnancy. J Preg. 2012;5:105918.
15. Sibai BM, Mercer B, Sarinoglu C. Severe preeclampsia in the second trimester: recurrence risk and long-term prognosis. Am J Obstet Gynecol. 1991;165:1408.
16. Ponzetto A, Cardarpoli S. Preeclampsiais associated with *H pylori* seropositivity in Italy. J Hypertens. 2006;24:2445-9.
17. Hauth JC, Ewell MG, Levine RJ. Pregnancy outcomes in healthy nulliparas who developed hypertension. Calcium for preeclampsia prevention study group. Obstet Gynecol. 2000;95:24.
18. Norwitz ER, Hsu CD, John T. Acute complications of preeclampsia. Clin Obst Gynecol. 2002;45(2):308-29.
19. Aqudelo C, Villar J. Maternal infection and risk factor of preeclampsia systemic review. Am J Obst Gyn. 2008;198(1):7-22.
20. Tytgat GN. Endoscopic transmission of *Helicobacter pylori*. Aliment Pharmacol Ther. 1995;9(2):105.
21. Shiadeh MN, Riahi SM, Adam I, Saber V, Behboodi Z. Armon B. *Helicobacter pylori* infection and risk of preeclampsia: a systematic review and meta-analysis. J Maternal-Fetal Neonatal Med. 2019;32(2):324-31.
22. Trogstad LIS, Eskild A, Bruu AL, Jeansson S, Jenum PA. Is preeclampsia an infectious disease? Acta Obstet Gynecol Scand. 2001;80:1036-8.
23. Todros T, Verdiglione P, Ogge G, Paladini D, Vergani P, Cardaropoli S. Low incidence of hypertensive disorders of pregnancy in women treated with spiramycin for toxoplasma infection. Br J Clin Pharmacol. 2006;336:340-61.
24. Oung B, Chea K, Oung C, Saurin JC, Ko CW. Endoscopic yield of chronic dyspepsia in outpatients: a single-center experience in Cambodia. JGH Open. 2020;4(1):61-8.

Cite this article as: Hasan JS, Alshami MA. The role of *Helicobacter pylori* in preeclampsia and in gastric diseases in pregnant women. Int Surg J 2020;7:2097-102.