MOSAIC APPEARANCE OF GASTRIC MUCOSA AS A PREDICTOR FOR HELICOBACTER PYLORI INFECTION

Ali Adnan Mohsin* & Sarkis K Strak®
*MBChB Registrar, ©MBChB, MRCP, FRCP, Professor, Department of Medicine, University of Basrah, Basrah, IRAQ.

Abstract
Experimental evidence supports a significant association between H. pylori infection and mosaic appearance of gastric mucosa. This study was carried out to find the significance of gastric mosaic mucosal pattern as a predictor of H. pylori related infection.
A total of one hundred consecutive patients were selected from those attending endoscopy unit at Al-Sader Teaching Hospital for various dyspeptic symptoms; fifty with mosaic gastric mucosa as patients and fifty with normal gastric mucosa as controls. They were classified according to their ages, gender, and smoking habits.
Two endoscopic biopsies from the antrum and corpus were taken from each patient and control and were tested for H. pylori by rapid urease test.
Mosaic gastric mucosal pattern was significantly associated with H. pylori infection, as compared with normal mucosa. Age was an important determinant for mosaic mucosal pattern and H. pylori infection, however; gender did not contribute to mosaic mucosal appearances and H. pylori infection, while smoking can contribute to mosaic mucosal appearance but not H. pylori infection.
In conclusion, mosaic mucosal pattern is a good indicator in predicting H. pylori related infection.

Introduction
Helicobacter pylori (H. pylori) are gastric organisms were first observed more than 100 years ago and their association with gastritis has been recognized since the 1970s. The true implication of these microbes was not fully realized, however, until 1982 when Marshall and Warren identified and subsequently cultured the gastric bacterium, Campylobacter pyloridis, later reclassified as H. pylori.
Colonization with this organism is the main risk factor for gastritis, peptic ulceration, gastric adenocarcinoma and gastric MALT (mucosa-associated lymphoid tissue) lymphoma. H. pylori is the most common chronic bacterial infection in humans. It has been demonstrated worldwide and in individuals of all ages. Conservative estimates suggest that 50% of the world's population are affected.
In addition to its morphologic characterization, the organism can be biochemically characterized as catalase, oxidase, and urease positive. Urease appears to be vital for its survival and colonization. Bacterial urease activity is clinically important because it forms the basis for several invasive and noninvasive tests to diagnose infection.
Although endoscopic features of H. pylori have been reported in the literatures, there is still some debate over whether H. pylori-related gastritis can be diagnosed via endoscopic features alone. Most studies concluded that it is possible to diagnose H. pylori-related gastritis on the basis of high resolution magnification endoscopy. If specific mucosal patterns
of H. pylori-related gastritis can be identified using standard endoscopy, they may be applicable to targeted biopsy of suspected H. pylori infection in daily practice. The previously observed endoscopic mucosal morphology of the gastric body was classified into 4 patterns; a cleft-like appearance mainly extending along the longitudinal axis of gastric body, a regular arrangement of red dots, the mosaic mucosal pattern without a focal area of hyperemia, and the mosaic pattern with a focal area of hyperemia (figure 1 A, B, C, and D). The first 2 types were not associated with H. pylori infection but the last two was significantly associated with H. pylori infection.

Gastric biopsies can be tested for urease activity. With this technique, one or two pieces of tissue are placed in a urease solution which contains urea and a pH reagent. Urease cleaves urea to liberate ammonia, producing an alkaline pH and a resultant color change. The test may become positive as early as one hour after collection, but a final reading at 24 hours is recommended. The sensitivity of biopsy urease tests is approximately 90 to 95 percent, and specificity is 95 to 100 percent. False positive tests are unusual. However, false negative results can occur in patients with recent gastrointestinal bleeding or with the use of proton pump inhibitors (PPIs), anti- H. pylori antibiotics, or bismuth containing compounds. Obtaining tissue samples from the antrum and the fundus may increase the sensitivity of the test.

This study was aimed to find the significance of gastric mosaic mucosal pattern as a predictor for H. pylori infection.

Patients and Methods
A comparative study was conducted from January 2011 to September 2012. A total of 100 consecutive patients were selected from those attending Endoscopy Unit for various dyspeptic symptoms. Twenty-eight males and 22 females with mosaic mucosal pattern as patients (group A), and 20 males and 30 females with normal looking gastric mucosa as controls (group B) were enrolled in the study. The study was carried out in the Endoscopy Unit at Al-Sader Teaching Hospital, Basrah; Southern Iraq. Ages of (group A) patients were ranging from 21–66 years with a mean age of 38.42 year, while those of (group B) ranged from 20–80 years with a mean age of 33.26 years. Patients were classified according to their ages as groups of 20 – 39 years. 40 – 59 years. 60 years and above.

Patients of both groups receiving NSAIDs, proton pump inhibitors, antibiotics for H. pylori eradication, or with bleeding tendency, pervious gastric surgery, gastric or duodenal ulcer, gastric malignancy were excluded from the study. Gender and smoking habits were reported and correlated with endoscopic findings. Current smoking was considered present if the patient had smoked any number of cigarettes with in the previous one month. Endoscopic examination was performed by one endoscopist using upper GI video scope (Olympus CLV 260). The whole stomach and duodenum were examined first, then gastric body (corpus) was chosen and only the mucosal morphology of mosaic pattern with or without a focal area of hyperemia was considered for the study.

Two endoscopic gastric biopsies from antrum and gastric body (corpus) were taken from each patient with normal and mosaic gastric mucosa and tested for H. pylori by rapid urease test. Urease test was read one and 24 hours after biopsy specimen insertion into the reagent. Patients with normal and mosaic gastric mucosa were considered as H. pylori positive when the color of the reagent changed from faint yellow to bright pink in the test tube. Statistical analysis was done by using the SPSS V 20 multilingual
Results

Number of patients with mosaic gastric mucosa and normal mucosa at the age group of 20–39 years were 24 (13 males and 11 females), and 38 (12 males and 26 females) respectively. At the age group of 40–59 years, were 18 (11 males and 7 females), and 10 (7 males and 3 females) patients respectively, while at the age group of 60 years and above were 8 (4 males and 4 females) patients with mosaic gastric mucosa, and 2 (1 male and 1 female) patients with normal gastric mucosa as shown in table (IA, IB).

Thirty six (72%) patients with mosaic gastric mucosa had H. pylori infection as shown by positive urease test, and 14 (28%) of them were H. pylori negative. 19 (38%) patients with normal gastric mucosa had H. pylori, and 31 (62%) of them were negative. These differences were significant P=0.001 as shown in table (II).

In the age group (20–39 years), out of 24 patients with mosaic gastric mucosa 15 (62.5%) patients were infected with H. pylori while out of 38 patients with normal looking gastric mucosa 14 (36.8%) were infected, the differences were significant P=0.049. In the age group (40–59 years), out of 18 patients with mosaic gastric mucosa 17 (94.4%) patients were infected while out of 10 patients with normal gastric mucosal appearance, 4 (40%) were infected. The differences were highly significant P=0.001. In the age group of 60 years and above, out of 8 patients with mosaic mucosal appearance, 4 (50%) patient was infected, and out of 2 patients with normal gastric mucosal appearance, 1 (50%) patients were infected. The differences were not significant P=1.0 (see table (III)).

Out of 48 studied males, 26 (54.17%) patients were infected with H. pylori while out of 52 females 29 (55.7%) were infected. The difference were insignificant P=0.872. Out of 48 males 28 (58.33%) had mosaic appearance of their gastric mucosa, and from the 52 studied females 22 (42.3%) had mosaic gastric mucosa. The difference here where insignificant P = 0.109 as shown in table (IVA, and IVB).

Discussion

One study has addressed the endoscopic mucosal abnormalities as features of Helicobacter-related gastropathy and infection7, however; some of the studies concluded that H. pylori infection cannot be diagnosed based on endoscopic findings alone8-11. Recently, Yagi et al14 first described the characteristic magnification endoscopic findings of H. pylori-negative stomach. Further, Anagnostopoulos et al15 demonstrated the usefulness of magnifying endoscopy in the identification of H. pylori-associated gastritis in a western population. However, practicing magnification endoscopy takes more examination time and needs more training and experience. It is therefore not feasible to practice magnification endoscopy in daily endoscopy examinations7.

Mosaic gastric mucosa was a predictor for H. pylori infection as proved in this study which was supported by Sheng-Lei Yan and his colleagues who proved that mosaic gastric mucosa was statistically significant in predicting H. pyloripositive status as compared with normal and other mucosal types7. There was a significant association between the age groups of (20–39 years, and (40–59) years and mosaic mucosal
appearance, and hence, H. pylori infection. This result was supported by a study done in northern Iraq.\textsuperscript{16} Both males and females had neither significant association with mosaic gastric mucosa or H. pylori infection, therefore sex seems not to contribute to mosaic gastric mucosa. This was agreed with other studies.\textsuperscript{17-18} Khouri K et al assumed that smoking carries significant association with both mosaic gastric mucosa and H. pylori infection.\textsuperscript{19} In this study, however, both smokers and non-smokers had significant association with mosaic gastric mucosa but not H. pylori infection, therefore smoking seems to contribute to mosaic gastric mucosal appearance.

**Conclusion**

This study suggests that mosaic mucosal pattern of gastric mucosa seems to be a good indicator in predicting H. pylori infection. Age and smoking can contribute to mosaic gastric mucosal appearance but not gender.

**Recommendation**

It is recommended that endoscopists need to be aware of mucosal changes of H. pylori infection, and therefore guide them to targeted biopsy of suspected H. pylori infection.

| Table IA: The distribution of patients according to age group |
|-------------------------------------------------------------|
| **Age group** | **Patients** |  
| | Mosaic mucosa | Normal mucosa | Total 62 |
| 20 – 39 years | No. | 24 | 38 |
| | % | 48.0% | 76.0% | 62.0% |
| 40 – 59 years | No. | 18 | 10 |
| | % | 36.0% | 20.0% | 28.0% |
| 60+ years | No. | 8 | 2 |
| | % | 16.0% | 4.0% | 10.0% |
| Total | No. | 50 | 50 |
| | % | 100.0% | 100.0% | 100.0% |

| Table IB: The distribution of patients according to gender |
|-----------------------------------------------------------|
| **Gender** | **Patients** |  
| | Mosaic mucosa | Normal mucosa | Total 48 |
| Male | No. | 28 | 20 |
| | % | 56.0% | 40.0% | 48.0% |
| Female | No. | 22 | 30 |
| | % | 44.0% | 60.0% | 52.0% |
| Total | No. | 50 | 50 |
| | % | 100.0% | 100.0% | 100.0% |
### Table II: H. pylori infection in patients with normal and mosaic gastric mucosa

| Gastric mucosa | H. pylori infection | Total |
|----------------|---------------------|-------|
| Mosaic         | Positive | 36 | 14 | 50 | 72% | 28% | 100% |
| Normal         | No. | 36 | 14 | 50 | 72% | 28% | 100% |
| Total          | No. | 55 | 45 | 100 | 55% | 45% | 100% |

P – value = 0.001

### Table III: H. pylori infection as correlated to age groups in patients with normal and mosaic gastric mucosa

| Age Group | H. pylori infection | Total |
|-----------|---------------------|-------|
|           | Positive | Negative | Total | P value |
| 20 – 39 years | Mosaic | No. | 15 | 9 | 24 | 0.049 |
|             | % | 62.5% | 37.5% | 100% |
|             | Normal | No. | 14 | 24 | 38 | 0.049 |
|             | % | 36.8% | 63.2% | 100% |
|             | Total | No. | 29 | 33 | 62 | 0.049 |
|             | % | 46.8% | 53.2% | 100% |
| 40 – 59 years | Mosaic | No. | 17 | 1 | 18 | 0.001 |
|             | % | 94.4% | 5.6% | 100% |
|             | Normal | No. | 4 | 6 | 10 | 0.001 |
|             | % | 40% | 60% | 100% |
|             | Total | No. | 21 | 7 | 28 | 0.001 |
|             | % | 75% | 25% | 100% |
| 60+ years | Mosaic | No. | 4 | 4 | 8 | 1.00 |
|             | % | 50% | 50% | 100% |
|             | Normal | No. | 1 | 1 | 2 | 1.00 |
|             | % | 50% | 50% | 100% |
|             | Total | No. | 5 | 5 | 10 | 1.00 |
|             | % | 50% | 50% | 100% |
### Table IVA: The correlation between H. pylori infection and gender

| Gender | H. pylori infection | Total | P – value |
|--------|---------------------|-------|-----------|
|        | Positive | Negative |       |          |           |
| Male   | No. 26    | 22      | 48    | 0.872    |
|        | % 54.17% | 45.83%  | 100%  |           |
| Female | No. 29    | 23      | 52    |           |
|        | % 55.77% | 44.23%  | 100%  |           |
| Total  | No. 55    | 45      | 100   |           |
|        | % 55%     | 45%     | 100.0%|           |

### Table IVB: The correlation between mosaic gastric mucosa and gender

| Gender | Mosaic gastric mucosa | Total | P – value |
|--------|-----------------------|-------|-----------|
|        | Positive | Negative |       |          |           |
| Male   | No. 28    | 20      | 48    | 0.109    |
|        | % 58.33% | 41.67%  | 100%  |           |
| Female | No. 22    | 30      | 52    |           |
|        | % 42.3%   | 57.7%   | 100%  |           |
| Total  | No. 50    | 50      | 100   |           |
|        | % 50%     | 50%     | 100%  |           |

### Table VA: H. pylori infection as correlated to smoking

| Smoking habit | H. pylori infection | Total | P – value |
|---------------|---------------------|-------|-----------|
|               | Positive | Negative |       |          |           |
| Smoker        | No. 19    | 13      | 32    | 0.546    |
|               | % 59.38% | 40.62%  | 100%  |           |
| Non smoker    | No. 36    | 32      | 68    |           |
|               | % 52.94% | 47.06%  | 100%  |           |
| Total         | No. 55    | 45      | 100   |           |
|               | % 55%     | 45%     | 100%  |           |

### Table VB: Mosaic appearance of the gastric mucosa as correlated to smoking

| Smoking habit | Mosaic gastric mucosa | Total | P – value |
|---------------|-----------------------|-------|-----------|
|               | Positive | Negative |       |          |           |
| Smoker        | No. 21    | 11      | 32    | 0.023    |
|               | % 65.63% | 34.37%  | 100%  |           |
| Non smoker    | No. 29    | 39      | 68    |           |
|               | % 42.65% | 57.35%  | 100%  |           |
| Total         | No. 50    | 50      | 100   |           |
|               | % 50%     | 50%     | 100%  |           |
The observed mucosal morphology of the gastric body in 
H. pylori associated infection  
as seen by upper GI endoscopy (Figure 1).

Figure 1: Endoscopic mucosal morphology in H. pylori infection:

a. A cleft-like appearance mainly extending along the longitudinal axis of gastric body. 
b. A regular arrangement of red dots.  
c. The mosaic mucosal pattern without a focal area of hyperemia.  
d. The mosaic pattern with a focal area of hyperemia.

References
1. Graham DY. Helicobacter Pylori infection in the pathogenesis of duodenal ulcer and gastric cancer: a model. Gastroenterology. 1997; 113: 1983-1991. Citation.  
2. Marshall BJ, Warren JR. Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. Lancet. 1984; 1:1311.  
3. John C.A, Martin J.B. Helicobacter pylori infection. Harrison's Principles of Internal Medicine (18th Ed.). USA. 2012. p 1261 – 1265. 
4. Cave DR. Transmission and epidemiology of Helicobacter Pylori. Am J Med. 1996; 100 (Suppl 5A):125.  
5. Pounder RE, Ng D. The prevalence of Helicobacter Pylori infection in different countries. Aliment Pharmacol Ther. 1995; 9 (Suppl 2):33.  
6. Abood R.A, Strak S.K, Al Haroon S.S. Evaluation of endoscopy based methods (histopathology, cytology and urease test) for the detection of 
H. pylori. Basrah Journal of Surgery. 2009; vol.15 No.1: 25 – 31. 
7. Sheng-Lei Yan, Shwu-Tzy Wu, Chien-Hua Chen, Yeh-Huang Hung, Tsung-Hsuan Yang, Yun-Siew Pang, et al. Mucosal patterns of Helicobacter pylori related gastritis without atrophy in the gastric corpus using standard endoscopy. World J Gastroenterol 2010 January 28; 16(4): 496-500. 
8. Bah A, Saraga E, Armstrong D, Vouillamoz D, Dorta G, Duroux P, et al. Endoscopic features of Helicobacter pylori-related gastritis. Endoscopy. 1995;27:593–596. 
9. Calabrese C, Di Febo G, Brandi G, Morselli-Labate AM, Areni A, Scialpi C, et al. Correlation between endoscopic features of gastric antrum, histology and Helicobacter pylori infection in adults. Ital J Gastroenterol Hepatol. 1999;31:359–365. 
10. Loffeld RJ. Diagnostic value of endoscopic signs of gastritis: with special emphasis to nodular antritis. Neth J Med. 1999;54:96–100. 
11. Redeem S, Petersson F, Jönsson KA, Borch K. Relationship of gastroscopic features to histological findings in gastritis and Helicobacter pylori infection in a general population sample. Endoscopy. 2003;35:946–950. 
12. Howden CW, Hunt RH. Guidelines for the management of Helicobacter Pylori infection. Am J Gastroenterol. 1998; 93: 2330. 
13. Weston AP, Campbell DR, Hassanein RES, Cherian R, Dixon A, McGregor DH. Prospective multivariate evaluation of CLO test performance. Am J Gastroenterol. 1997; 92: 1210. 
14. Yagi K, Nakamura A, Sekine A. Characteristic endoscopic and magnified endoscopic findings in the normal stomach without Helicobacter pylori infection. J Gastroenterol Hepatol. 2002;17:39-45. 
15. Anagnostopoulos GK, Yao K, Kaye P, Fogden E, Fortun P, Shorde A, et al. High-resolution magnification endoscopy can reliably identify normal gastric mucosa, Helicobacter pylori-associated gastritis, and gastric atrophy. Endoscopy. 2007;39:202–207. 
16. Nawfel R H. The Pathogenesis Of Helicobacter Pylori Associated Diseases In Kurdistan Region, Iraq. 2009. PhD. Thesis. University of Nottingham. 
17. Slaat, M. A., Krouzon-Moran, D., McQuillan, G. M. & Kaslow, R. A. A population-based serologic survey of Helicobacter pylori infection in children and adolescents in the United States. J Infect Dis, 1996; 174, 1120-3. 
18. Al-Moaelg, M. A., Evans, D. G., Abdul-Ghani, M. E., Adam, E., Evans, D. J., JR., Malaty, H. M. et al. Prevalence of Helicobacter (formerly Campylobacter) pylori infection in Saudia Arabia, and comparison of those with and without upper gastrointestinal symptoms. Am J Gastroenterol, 1990; 85, 944-8. 
19. Khouri K, Sayegh R, Yaghi C, Honen K, Gedeon E, Bou Jaoude J, et al. Role of endoscopic gastric biopsies in the management of gastritis. J Med Liban. 2002 Jul-Aug;50(4):149.