

Current recommendations for analyzing and reporting gastric biopsies

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Dear editor,

After carefully reading the article “Frequency of morphological changes in gastric biopsies associated with *Helicobacter pylori* infection.” It is medically known that gastric cancer is a frequent condition in our setting; it is the fifth most common cancer worldwide, and the third cause of cancer deaths. In its global distribution, it is more frequent in East Asia, Eastern Europe, and Central and South America. Unfortunately, it is not diagnosed early, which leads to an unfavorable prognosis for the patients (1).

Thanks to the ongoing study of gastric cancer, its significant association with inflammation and *Helicobacter pylori* infection has been established; several changes occur in the gastric mucosa prior to developing cancer, including superficial chronic gastritis, atrophic gastritis, gastritis with lymphoid follicles or follicular gastritis, and intestinal metaplasia. In the conclusions of their paper, the authors remark that 42% of the pathological anatomy reports do not align with the internationally described parameters for histological interpretation of the morphological changes (based on the premise of variability between the pathologists examining them), and recommend harmonizing concepts when reporting histopathological findings. A review of the biopsy reporting system used in the study confirmed that the Sydney protocol was used, which is known today to have some problems in its interobserver reproducibility, and this probably also affected the results (2).

Currently, different international consensuses recommend using the OLGA/OLGIM protocol for reporting gastric biopsies, due to its easy application and reproducibility. In this system, atrophy (the loss of total glandular volume or of its normal function due to pyloric/intestinal metaplasia) is the lesion which indicates actual progression of the gastritis, which is scored from 0 to IV, with 0 being no atrophy, I minimal atrophy, and IV the most severe atrophy. Its purpose is to stratify the risk of developing gastric cancer and thus determine interventions, especially for patients with OLGA III or IV scores, who are monitored with endoscopy for early detection of GC (3).

This reporting system is already being used in Colombia, and its applicability has been proven. In 2016, Martinez et al. analyzed a sample of more than 5,000 patients collected from 2010 to 2013 and showed that the OLGA protocol identified 61.8% more cases of atrophy than the protocols which used fewer biopsies (42% versus 26%). They also confirmed the association between dysplasia and advanced OLGA stages (III/IV), which concurs with what has been reported in studies in other countries with a high incidence of gastric cancer, like Japan (4).

Thus, the community of gastroenterologists, endoscopists and pathologists is invited to set up working groups to stay updated on the most recent gastric cancer risk stratification protocols and thus provide a better diagnosis, treatment and follow up for patients, to guarantee their comprehensive care.

de estratificación de riesgo de cáncer gástrico para así ofrecer un mejor diagnóstico, tratamiento y seguimiento de los pacientes; con lo que se garantiza una atención integral de los mismos.

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References

1. Sarem M, Corti R. ¿Por qué es importante detectar la gastritis atrófica y la metaplasia intestinal gástrica? ¿Cuál es la forma adecuada de hacerlo?. *Rev Gastroenterol Peru.* 2020;40(3):260-6
2. Melo-Peña MA, Mendoza-Rodríguez A. Frecuencia de cambios morfológicos en biopsias gástricas asociadas a infección por *Helicobacter Pylori*. *Acta Med Colomb.* 2021; 46. DOI: <https://doi.org/10.36104/amc.2021.1987>.
3. Crafa P, Russo M, Miraglia C, Barchi A, Moccia F, Nouvenne A, et al. From Sidney to OLGA: an overview of atrophic gastritis. *Acta Biomed.* 2018; 89(8-S): 93-99. DOI:10.23750/abm.v89i8-S.7946
4. Martínez D, Otero W, Ricaurte O. Impacto del sistema OLGA en la detección de gastritis crónica atrófica en Colombia: un estudio de casos y controles. *Rev Col Gastroenterol.* 2016; 31(4): 360-367.



ANSWER

Response to the editor

First, I would like to thank the reader for his careful reading of the article “Frequency of morphological changes in gastric biopsies associated with *Helicobacter pylori* infection.”

Second, I would like to say that I agree completely with his suggestion of promoting the use of the OLGA or OLGIM methodology in Colombia for gastric biopsy sampling and histological interpretation to seek and adequately stratify atrophic gastritis and intestinal metaplasia, in order to conduct follow up and possibly detect gastric cancer cases in their early stages (1).

However, we must remember that this was a retrospective study; the chart review was carried out from 2008 to 2017, a period of time in which both the performance of biopsies as well as their interpretation employed the system which was in force at that time, which was the Sydney protocol (2, 3).

As this was a retrospective study, we must accept that both the sampling as well as the histological diagnosis may vary widely, since several pathologists reported the biopsies, and several endoscopists took the samples. Thus, this was noted at the end of the paper as a limitation of the study, since neither the number nor exact location of the biopsies was able to be determined.

This is also a barrier which has been improving significantly in our setting, since the pathology reports increasingly differentiate their findings between the body and the antrum, but especially also the incisura biopsy, and classify the gastric biopsies according to the OLGA or OLGIM system.

Since Colombia is a country with a high incidence of gastric cancer, the detection of atrophy in its different degrees and intestinal metaplasia, but especially that which affects both the antrum as well as the body, are of high interest in follow up, as the article recognizes (1).

I also think it is important to note that the study focused on observing the variation in morphological changes for both helicobacter positive and negative results, since in daily clinical practice we take the negative to be an unimportant clinical situation, since it is not treated with antibiotics. However, follow up is not carried out, either, and we must highlight that some of these cases also have atrophy and metaplasia, and therefore there may be false negatives, true negatives, or we may be faced with a different condition in which there is no interaction with *Helicobacter pylori*, but there may be a risk of developing gastric cancer through other mechanisms which are under study and for which there is no clear follow up method (4, 5).

It has been thought that a negative result may represent a different condition than *H. pylori-related gastritis*, but which may have similar risks if atrophy and metaplasia are present. In this regard, in a 2015 study of 895,323 patients, Sonneber found that 1.5% of the patients were *Helicobacter* negative, but that 13% of these had intestinal metaplasia, suggesting that this is a different condition to which future studies must be directed (6).

In terms of the stomach microbiome, it has recently been found that 96% of the normal stomach microbiome is made up of firmicutes (42%), bacteroidetes (24%), proteobacteria (17%), actinobacteria (7%) and fusobacteria (6%), which interact around the presence or absence of *Helicobacter pylori*. Studies are being carried out which aim to show that a positive or negative *H. pylori* status may develop different microbiotic conformations which may contribute to the superficial changes of atrophic or non-atrophic gastritis through mechanisms which have yet to be explained (4, 5).

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References

1. Rugge M, de Boni M, Pennelli G, de Bona M, Giacomelli L, Fassan M, Bassi D, Plebani M, Graham DY. Gastritis OLGA-staging and gastric cancer risk: a twelve-year clinico-pathological follow-up study. *Aliment Pharmacol Ther*. 2010 May;31(10):1104-11. doi: 10.1111/j.1365-2036.2010.04277.x. Epub 2010 Feb 23. PMID: 20180784.
2. Dixon MF, Genta RM, Yardley JH, Correa P. Classification and grading of gastritis. The updated Sydney System. International Workshop on the Histopathology of Gastritis, Houston 1994. *Am J Surg Pathol*. 1996 Oct;20(10):1161-81. doi: 10.1097/00000478-199610000-00001. PMID: 8827022.
3. Price AB. The Sydney System: histological division. *J Gastroenterol Hepatol*. 1991 May-Jun;6(3):209-22. doi: 10.1111/j.1440-1746.1991.tb01468.x. PMID: 1912431.
4. Gunathilake M, Lee J, Choi IJ, Kim YI, Kim J. Association between bacteria other than *Helicobacter pylori* and the risk of gastric cancer. *Helicobacter*. 2021 Oct;26(5):e12836. doi: 10.1111/hel.12836. Epub 2021 Jul 15. PMID: 34268831.
5. Ndegwa N, Ploner A, Andersson AF, Zagai U, Andreasson A, Vieth M, Talley NJ, Agreus L, Ye W. Gastric Microbiota in a Low-*Helicobacter pylori* Prevalence General Population and Their Associations With Gastric Lesions. *Clin Transl Gastroenterol*. 2020 Jul;11(7):e00191. doi: 10.14309/ctg.0000000000000191. PMID: 32764211; PMCID: PMC7431247.
6. Genta RM, Sonnenberg A. *Helicobacter*-negative gastritis: a distinct entity unrelated to *Helicobacter pylori* infection. *Aliment Pharmacol Ther*. 2015 Jan;41(2):218-26. doi: 10.1111/apt.13007. Epub 2014 Nov 6. PMID: 25376264

