

Comparison of OLGA and OLGIM as predictors of gastric cancer in a Latin American population: the ECHOS Study

We read with great interest the article by Lee *et al*¹ evaluating the risk of progression from chronic atrophic gastritis (CAG) with intestinal metaplasia (IM) to high-grade dysplasia (HGD) or gastric adenocarcinoma (GA), according to the Operative Link on Gastric Intestinal Metaplasia (OLGIM) staging system. Patients with OLGIM III–IV had a substantially increased risk of HGD/GA, with a median time to progression of 22.7 months, while patients with OLGIM II had an intermediate risk. These findings complement the results by Rugge *et al*,² demonstrating a higher risk of neoplastic progression among patients with Operative Link on Gastritis Assessment (OLGA) III–IV, but not OLGA II.

Studies directly comparing the predictive value of both OLGA and OLGIM systems in the same cohort are scarce.^{3,4} We leveraged the ‘Endoscopic Cohort and Histological OLGA staging’ (trial: NCT05969444) to compare the predictive capability of OLGA versus OLGIM for HGD/GA. We included 685 Chilean adults who underwent an oesophagogastroduodenoscopy with gastric mapping biopsies, and who subsequently had at least one endoscopic surveillance examination with mapping biopsies completed at least 6 months apart (table 1). The primary outcome was histologically confirmed incident HGD or GA according to baseline OLGA and OLGIM stages (assessed by two experienced pathologists). The kappa values for interpathologist and intropathologist agreement were 0.63 and 0.71 for OLGA, and 0.75 and 0.63 for OLGIM. We calculated HRs and 95% CIs using Cox regression adjusted for age, sex and active *Helicobacter pylori* infection.

During a median of 3 years of follow-up (IQR 1.8–4.3), four HGD and seven GA were diagnosed (online supplemental figure 1). Patients with OLGA III–IV (1.8/100 person-years) and OLGIM III–IV (3.3/100 person-years) had substantially higher rates of HGD/GA compared with patients with respective OLGA or OLGIM 0–I. The adjusted HRs for HGD/GA were 14.8 (95% CI 1.8 to 125) and 34 (95% CI 4 to 286), respectively (figure 1A–D and online supplemental table 1). These results are consistent with those reported by Lee *et al* (OLGIM III–IV, HR 20.7; 95% CI 5.04 to 85.6).¹ In contrast, we

Table 1 Demographical, histological and follow-up variables according to OLGA groups

	OLGA 0–I n=366	OLGA II n=169	OLGA III–IV n=150	P value*
Sex, n (%)				
Female	240 (65.6)	117 (69.2)	88 (58.7)	0.13
Male	126 (34.4)	52 (30.8)	62 (41.3)	
Age in years, mean (SD)	55 (11.1)	55 (12.6)	60 (10.3)	<0.001
Tobacco, n (%)				
Active smoker	76 (20.8)	33 (19.5)	27 (18)	
Former smoker	36 (9.8)	23 (13.6)	33 (22)	0.009
Never smoker	254 (69.4)	113 (66.9)	90 (60)	
Active alcohol consumption, n (%)	171 (46.7)	78 (46.1)	66 (44)	0.85
First-degree family history of gastric cancer, n (%)	120 (32.8)	31 (18.3)	31 (20.7)	0.001
Baseline active infection by <i>Helicobacter pylori</i> †, n (%)	114 (31.2)	48 (28.4)	51 (34)	0.56
Persistent <i>H. pylori</i> infection, n (%)	29 (7.9)	11 (6.5)	4 (2.7)	0.09
Intestinal metaplasia, n (%)	91 (24.9)	127 (75.2)	140 (93.3)	<0.001
Anatomical extent				
Antrum-restricted	70 (19.1)	61 (36.1)	56 (37.3)	<0.001
Corpus-extended	21 (5.7)	66 (39.1)	84 (56)	
Histopathological subtype§				
Complete-type	20 (5.5)	46 (27.2)	32 (21.3)	
Incomplete-type	53 (14.5)	55 (32.5)	76 (50.7)	<0.001
Unavailable	18 (4.9)	26 (15.4)	32 (21.3)	
OLGIM				
0–I	366 (100)	104 (61.5)	31 (20.7)	
II	0 (0)	65 (38.5)	36 (24)	<0.001
III–IV	0 (0)	0 (0)	83 (55.3)	
Baseline low-grade dysplasia (LGD)¶, n (%)	0 (0)	3 (1.8)	9 (6)	<0.001
Autoimmune gastritis, n (%)	5 (1.4)	61 (36.1)	18 (12)	<0.001
Follow-up in months, median (IQR)	36 (22–52)	38 (22–52)	33 (17–51)	0.23
Number of surveillance endoscopies, median (range)	1 (1–1)	1 (1–2)	2 (1–2)	<0.001

* χ^2 (categorical variables); one-way ANOVA or Wilcoxon (numerical variables).

†Any amount.

‡*H. pylori* infection based on Giemsa staining in any of the gastric samples.

§The highest histopathological grade observed was considered for diagnosis.

¶No cases of indefinite for dysplasia were observed and all LGDs were confirmed by two expert pathologists. ANOVA, analysis of variance; OLGA, Operative Link on Gastritis Assessment; OLGIM, Operative Link on Gastric Intestinal Metaplasia.

did not observe a significantly increased risk of HGD/GA in patients with OLGA or OLGIM II. Outcomes of patients with low-grade dysplasia and autoimmune gastritis are described in the online supplemental material. The progression of CAG with or without IM varied according to OLGA and OLGIM stage, with a large proportion of patients remaining stable or even histologically regressing during follow-up (figure 1E,F). Notably, OLGIM demonstrated greater stability over time compared with OLGA (62% OLGIM remain stable during follow-up vs 47% OLGA; $p<0.001$), possibly related to lower interpathologist variability. These findings suggest that OLGIM may perform better than OLGA for real-world risk stratification.

From the vantage point of evaluating the benefit of endoscopic surveillance, nearly all (91%, 10 of 11) HGD/GAs

were detected at early stages (online supplemental table 2). This observation contrasts with the reality in most Western countries, where GAs are mostly diagnosed at advanced stages.^{5–7} International guidelines generally recommend surveillance at least every 3 years for advanced stages of CAG/IM,^{8–10} but adherence to guidelines is suboptimal, and timely access to endoscopy and concerns regarding resource overutilisation pose challenges. Our findings emphasise the need to concentrate resources on patients with OLGA/OLGIM III–IV. Furthermore, during the first years of follow-up, only one early-stage GA was diagnosed (15 months) and the median times to HGD/GA in patients with OLGA III–IV and OLGIM III–IV were 33 months. Taken together, surveillance every 3 years in patients with OLGA/OLGIM III–IV might

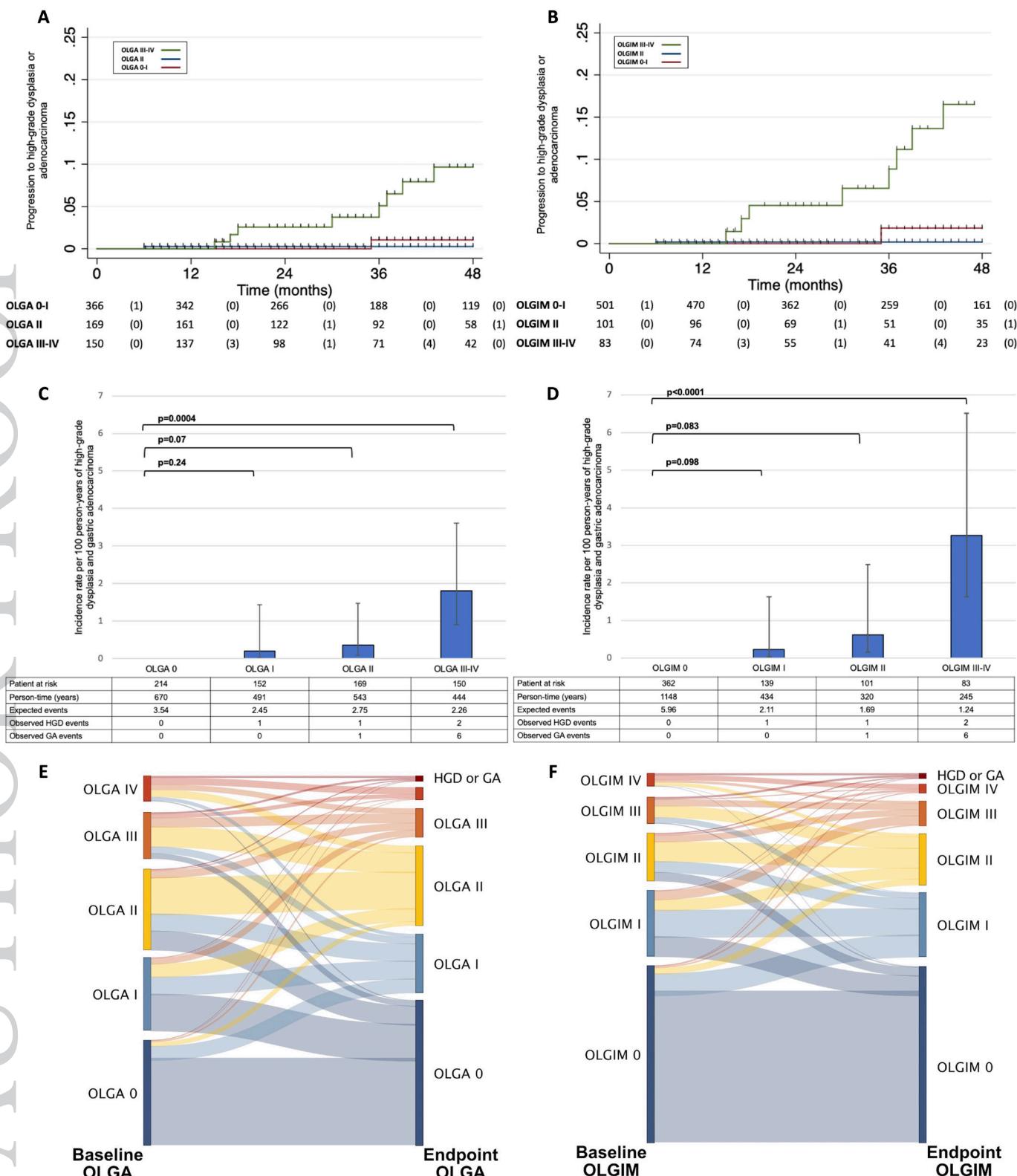


Figure 1 Comparison of performance of OLGA (Operative Link on Gastritis Assessment) and OLGIM (Operative Link on Gastric Intestinal Metaplasia). (A,B) Cumulative risk curves of the primary composite outcome high-grade dysplasia (HGD) or gastric adenocarcinoma (GA) according to the baseline OLGA (left) and OLGIM (right) with at-risk tables and events in parenthesis below. (C,D) Incidence rates per 100 person-years of HGD and GA according to OLGA (left) and OLGIM (right). Error bars represent the 95% CIs and differences are established by log-rank test for equality survival function. (E,F) Dynamics of OLGA (left) and OLGIM (right) during the follow-up.

strike the optimal balance between resource allocation and early detection of GA, even in high-risk Latin American

populations. Nevertheless, randomised trials evaluating surveillance strategies are pressingly needed to determine the

optimal endoscopic intervals for patients with high-risk gastric premalignant conditions.

Gonzalo Latorre,¹ Felipe Silva,^{1,2}
 Isabella Montero,³ Miguel Bustamante,³
 Eitan Dukes,³ Javier Uribe,³
 Oscar Corsi Sotelo,³ Diego Reyes,³
 Eduardo Fuentes-López,⁴ Margarita Pizarro,³
 Patricio Medel,⁵ Javiera Torres,⁶
 Juan Carlos Roa,^{7,8} Sebastián Pizarro,⁷
 Pablo Achurra,⁹ Andrés Donoso,⁹
 Ignacio Wichmann,^{10,11} Alejandro H Corvalán,^{10,11}
 Javier Chahuan,¹² Roberto Candia,¹³
 Carlos Agüero,³ Robinson Gonzalez,¹⁴
 Jose Ignacio Vargas,¹⁰ Alberto Espino,¹⁵
 M Constanza Camargo,¹⁶ Shailja Shah,¹⁰,
 Arnaldo Riquelme,^{10,4,6}

¹Department of Gastroenterology, School of Medicine, Pontificia Universidad Católica de Chile, Santiago, Chile

²Pontificia Universidad Católica de Chile Facultad de Medicina, Santiago, Chile

³Departamento de Gastroenterología, Facultad de Medicina, Pontificia Universidad Católica de Chile, Santiago, Chile

⁴Department of Health Sciences, Pontificia Universidad Católica de Chile, Santiago, Chile

⁵Pharmacology and Toxicology Program, Faculty of Medicine, Pontificia Universidad Católica de Chile, Santiago, Chile

⁶Department of Pathology, School of Medicine, Pontificia Universidad Católica de Chile, Santiago, Chile

⁷Departamento de Anatomía Patológica, Facultad de Medicina, Pontificia Universidad Católica de Chile, Santiago, Chile

⁸Centro para la Prevención y el Control del Cáncer (CECAN), Santiago, Chile

⁹Departamento de Cirugía Digestiva, Facultad de Medicina, Pontificia Universidad Católica de Chile, Santiago, Chile

¹⁰Departamento de Hematología y Oncología, Facultad de Medicina, Pontificia Universidad Católica de Chile, Advanced Center for Chronic Diseases (ACCDIS), Santiago, Chile

¹¹Division of Oncology, Department of Medicine, Stanford University School of Medicine, Stanford, California, USA

¹²Department of Gastroenterology, Pontificia Universidad Católica de Chile, Santiago, Chile

¹³Departamento de Gastroenterología, Pontificia Universidad Católica de Chile, Santiago, Chile

¹⁴Pontificia Universidad Católica de Chile, Santiago, Chile

¹⁵Department of Gastroenterology, Universidad Católica de Chile, Santiago, Chile

¹⁶Division of Cancer Epidemiology and Genetics, National Cancer Institute, Bethesda, Maryland, USA

¹⁷Department of Gastroenterology, Veterans Affairs, VA San Diego Healthcare System, San Diego, California, USA

¹⁸Department of Gastroenterology, University of California San Diego, La Jolla, California, USA

Correspondence to Dr Arnaldo Riquelme, Department of Gastroenterology, Pontificia Universidad Católica de Chile, Santiago, Chile; a.riquelme.perez@gmail.com

Twitter Gonzalo Latorre @GonzaloLatorreS and Jose Ignacio Vargas @jivargasd

Contributors GL, FS, JU, MP, PA, AHC, RC, AE, MCC, SS and AR conceived and designed the study. GL, JU, OCS, DR, PA, AD, RC, CA, RG, JIV, AE and AR recruited patients and performed oesophagogastroduodenoscopy. GL, FS, IM, MB, ED, JU, OCS, DR and SP collected data. JT, JCR and SP performed histopathological analysis. GL, EF-L, IW, AHC, RC, AE, MCC, SS and AR analysed the data. GL, FS, MP, PM, SP, PA, IW, AHC, RC, CA, AE, MCC, SS and AR wrote the manuscript. GL, FS, IM, MB, ED, JU, OCS, DR, EF-L, MP, PM, JT, JCR, SP, PA, AD, IW, AHC, JC, RC, CA, RG, JIV, AE, MCC, SS and AR reviewed the manuscript.

Funding FONIS SA19/0188 (AR), FONDECYT no 11201338 (PA), European Union's Horizon 2020 research and innovation program grant agreement no 825832 (AR), FONDECYT 1230504 (AR, GL, AHC, JCR, SS), ANID FONDAP 152220002 (AR, JCR), FONDECYT 1231773 (AHC, AR), CONICYT-FONDAP 15130011 (AHC, IW), American Gastroenterological Association Research Scholar Award (2019) (SS), Veterans Affairs Career Development Award (ICX002027A01) (SS) and San Diego Digestive Diseases Research Center (NIH P30 DK120515) (SS).

Competing interests None declared.

Patient consent for publication Obtained.

Ethics approval This study involves human participants and was approved by the Ethics Committee of Hospital Clínico Universidad Católica de Chile (ID: 16-34118-0806007; 22-0601004). Participants gave informed consent to participate in the study before taking part.

Provenance and peer review Not commissioned; externally peer reviewed.

Supplemental material This content has been supplied by the author(s). It has not been vetted by BMJ Publishing Group Limited (BMJ) and may not have been peer-reviewed. Any opinions or recommendations discussed are solely those of the author(s) and are not endorsed by BMJ. BMJ disclaims all liability and responsibility arising from any reliance placed on the content. Where the content includes any translated material, BMJ does not warrant the accuracy and reliability of the translations (including but not limited to local regulations, clinical guidelines, terminology, drug names and drug dosages), and is not responsible for any error and/or omissions arising from translation and adaptation or otherwise.

© Author(s) (or their employer(s)) 2023. No commercial re-use. See rights and permissions. Published by BMJ.

► Additional supplemental material is published online only. To view, please visit the journal online (<http://dx.doi.org/10.1136/gutjnl-2023-331059>).

SS and AR contributed equally.

SS and AR are joint senior authors.

To cite Latorre G, Silva F, Montero I, et al. Gut Epub ahead of print: [please include Day Month Year]. doi:10.1136/gutjnl-2023-331059

Received 2 September 2023

Accepted 15 December 2023

Gut 2023;0:1–3. doi:10.1136/gutjnl-2023-331059

ORCID iDs

Felipe Silva <http://orcid.org/0009-0003-8074-5781>
 Oscar Corsi Sotelo <http://orcid.org/0000-0002-3631-9326>

Juan Carlos Roa <http://orcid.org/0000-0001-8313-8774>

Jose Ignacio Vargas <http://orcid.org/0000-0002-1547-2292>

Shailja Shah <http://orcid.org/0000-0002-2049-9959>
 Arnaldo Riquelme <http://orcid.org/0000-0002-8259-8960>

REFERENCES

- Lee JWJ, Zhu F, Srivastava S, et al. Severity of gastric intestinal Metaplasia predicts the risk of gastric cancer: a prospective Multicentre cohort study (GCEP). *Gut* 2022;71:854–63.
- Rugge M, Genta RM, Fassan M, et al. OLGA Gastritis staging for the prediction of gastric cancer risk: A long-term follow-up study of 7436 patients. *Am J Gastroenterol* 2018;113:1621–8.
- Piazuelo MB, Bravo LE, Mera RM, et al. The Colombian Chemoprevention trial: 20-year follow-up of a cohort of patients with gastric precancerous lesions. *Gastroenterology* 2021;160:1106–17.
- Gawron AJ, Shah SC, Altayar O, et al. AGA technical review on gastric intestinal Metaplasia—natural history and clinical outcomes. *Gastroenterology* 2020;158:705–731.
- Whiting JL, Sigurdsson A, Rowlands DC, et al. The long term results of endoscopic surveillance of Premalignant gastric lesions. *Gut* 2002;50:378–81.
- Sano T, Katai H, Sasako M, et al. The management of early gastric cancer. *Surg Oncol* 2000;9:17–22.
- Bolkschweiler E, Berlth F, Baltin C, et al. Treatment of early gastric cancer in the Western world. *World J Gastroenterol* 2014;20:5672–8.
- Gupta S, Li D, El Serag HB, et al. AGA clinical practice guidelines on management of gastric intestinal Metaplasia. *Gastroenterology* 2020;158:693–702.
- Pimentel-Nunes P, Libânia D, Marcos-Pinto R, et al. Management of epithelial precancerous conditions and lesions in the stomach (MAPS II): European society of gastrointestinal Endoscopy (ESGE), European Helicobacter and Microbiota study group (EHMSG), European society of Pathology (ESP), and Sociedade Portuguesa de Endoscopia Digestiva (SPED) guideline update 2019. *Endoscopy* 2019;51:365–88.
- Banks M, Graham D, Jansen M, et al. British society of Gastroenterology guidelines on the diagnosis and management of patients at risk of gastric adenocarcinoma. *Gut* 2019;68:1545–75.