ARTÍCULO ORIGINAL

Differences in the detection of gastric premalignant conditions and its correlation with gastric cancer after a negative esophagogastroduodenoscopy in a low-risk gastric cancer country

Diferencias en la detección de lesiones premalignas gástricas y su correlación con el cáncer gástrico tras una endoscopia digestiva alta negativa en un país de bajo riesgo para cáncer gástrico

Pedro Genaro Delgado-Guillena[1,a,b], Víctor Jair Morales-Alvarado[1,a], Beatriz De-Riba-Soler[1,a], Gemma Llibre-Nieto[1,a], Indira Armas-Ramírez[2,a], Terry Guillena-Castañeda[3,a], Ivana Levy-Ríos[3,a], Mireya Jimeno-Ramiro[4,a], Joaquim Rigau-Cañardo[1,a,b], Albert García-Rodríguez[1,a], Esteve Llargués Rocabruna[1,a,b], Henry Córdova[5,6,7,8,a,b], Gloria Fernández-Esparrach[5,6,7,8,a,b]

[1] Department of Gastroenterology. Hospital General de Granollers, Barcelona, Spain.
[2] Universidad Nacional de Trujillo, Trujillo, Perú.
[3] Universidad Nacional de Ucayali, Pucallpa, Perú.
[4] Department of Pathology. Hospital General de Granollers, Barcelona, Spain.
[5] Endoscopy Unit, Gastroenterology Department, ICMDM, Hospital Clínic, Universitat de Barcelona, Barcelona, Spain.
[6] Centro de Investigación Biomédica en Red de Enfermedades Hepáticas y Digestivas (CIBEREHD), Spain.
[7] Instituto de Investigaciones Biomédicas August Pi i Sunyer (IDIBAPS), Barcelona, Spain.
[8] Facultat de Medicina i Ciències de la Salut, Universitat de Barcelona.
[a] MD, [b] PhD

Received: 26/03/2025
Accepted: 03/06/2025
Online: 30/06/2025

Author contribution
The work reported in the paper has been performed by the authors, unless clearly specified in the text.

Conflict of interest
The authors declare that they have no conflict of interest.

Funding
No funding was received for conducting this study.

Cite as
Delgado-Guillena PG, Morales-Alvarado VJ, De-Riba-Soler B, Llibre-Nieto G, Armas-Ramírez I, Guillena-Castañeda T, *et al.* Differences in the detection of gastric premalignant conditions and its correlation with gastric cancer after a negative esophagogastroduodenoscopy in a low-risk gastric cancer country. Rev Gastroenterol Peru. 2025;45(2):109-19. doi: 10.47892/rgp.2025.452.1909.

Correspondence:
Pedro Genaro Delgado-Guillena
Gastroenterology Department.
Hospital General de Granollers,
Carrer de Francesc Ribas s/n, 08402,
Granollers, Spain.
E-mail: pgdg20@gmail.com

ABSTRACT

Introduction: Gastric cancer (GC), with nearly 90% being sporadic adenocarcinomas, is preceded by gastric premalignant conditions (GPC). Accurate detection of GPC during esophagogastroduodenoscopy (EGD) can enhance the identification of high-risk patients and improve early GC diagnosis. However, GPC detection rates during EGD vary among endoscopists, potentially leading to differences in GC rates after a negative EGD (GC post-EGD). **Objective:** This study aimed to assess the correlation between the GPC detection rate and the rate of GC post-EGD among endoscopists. **Materials and methods:** We conducted an observational study of EGDs at a community hospital between 2010 and 2019. GPC were defined as glandular atrophy, intestinal metaplasia, and dysplasia. EGDs were categorized into three groups: (i) benign, (ii) GPC, and (iii) malignant findings. GC post-EGD was defined as a diagnosis of gastric adenocarcinoma within three years of an EGD negative for malignancy. GPC detection rates and GC post-EGD were calculated for each endoscopist. **Results:** A total of 18,635 EGDs were performed by nine endoscopists. Gastric biopsies were obtained in 2,415 (13%) EGDs, identifying 533 GPCs (2.9%). The GC post-EGD rate was 1.26 per 1,000 EGDs. The detection rate of GPC varied between 1.8% and 5.8%, while GC post-EGD rates ranged from 0 to 3.36 per 1,000 EGDs. A negative correlation trend was observed between GC post-EGD and GPC detection rate (rs=-0.65, p=0.057), which was statistically significant for dysplasia (rs=-0.69, p=0.037). **Conclusion:** The detection rate of GPC—particularly dysplasia—showed a negative correlation with GC post-EGD in a community hospital within a low-risk setting during the period from 2010 to 2019.

Keywords: *Cancer, Gastric; Misdiagnosis; Quality Indicators, Health Care (source: MeSH NLM).*

RESUMEN

Introducción: El cáncer gástrico (CG), del cual cerca del 90% son adenocarcinomas esporádicos, está precedido por lesiones premalignas gástricas (LPMG). La detección oportuna de LPMG durante la esofagogastroduodenoscopia (EGD) puede mejorar la identificación de pacientes de alto riesgo y optimizar el diagnóstico precoz de CG. Sin embargo, las tasas de detección de LPMG durante la EGD varían entre endoscopistas, lo que podría conllevar diferencias en las tasas de CG tras una EGD negativa (CG post-EGD). **Objetivo:** Este estudio tuvo como objetivo primario evaluar la correlación entre la tasa de detección de CPG y la tasa de CG post-EGD entre endoscopistas. **Materiales y métodos:** Se realizó un estudio observacional de EGDs en un hospital comarcal entre 2010 y 2019. Las LPMG se definieron como atrofia glandular, metaplasia intestinal y displasia. Las EGDs se clasificaron en tres grupos: (i) hallazgos benignos, (ii) LPMG y (iii) hallazgos malignos. Se definió CG post-EGD como el diagnóstico de adenocarcinoma gástrico dentro de los tres años posteriores a una EGD sin datos de malignidad. Se calcularon las tasas de detección de LPMG y las tasas de CG post-EGD para cada endoscopista. **Resultados:** Un total de 18 635 EGDs fueron

realizadas por nueve endoscopistas. Se obtuvieron biopsias gástricas en 2415 (13%) EGDs, identificándose 533 LPMG (2,9%). La tasa de CG post-EGD fue de 1,26 por cada 1000 EGDs. La tasa de detección de LPMG varió entre 1,8% y 5,8%, mientras que las tasas de CG post-EGD oscilaron entre 0 y 3,36 por cada 1000 EGDs. Se observó una tendencia a la correlación negativa entre la CG post-EGD y la tasa de detección de LPMG (rs=-0,65, p=0,057), siendo estadísticamente significativa para la displasia (rs=-0,69, p=0,037). **Conclusión:** La tasa de detección de LPMG —especialmente de displasia— mostró una correlación negativa con la CG post-EGD en un hospital comarcal de un país de bajo riesgo durante el período comprendido entre 2010 y 2019.
Palabras clave: *Cáncer Gástrico; Errores Diagnósticos; Indicadores de Calidad de la Atención de Salud (fuente: DeCS Bireme).*

INTRODUCTION

Gastric cancer (GC), with nearly 90% being sporadic adenocarcinomas, is considered to be the result of a multistep and sequential process of gastric premalignant conditions (GPC) such as glandular atrophy, intestinal metaplasia (IM), and dysplasia, which ultimately result in adenocarcinoma [1]. The endoscopic recognition of GPC during an esophagogastroduodenoscopy (EGD) is quite variable and endoscopist-dependent [2,3]. Protocolized gastric biopsies are recommended for the histological identification of patients requiring surveillance, such as those with advanced glandular atrophy, intestinal metaplasia (OLGA/OLGIM stage III–IV), or dysplasia [4-6]. While histologic risk stratification has long been the standard, the recent MAPS III and ACG guidelines, both published in 2025, now also advocate for endoscopic risk stratification of GPC in Europe and other Western countries [7,8].

A population-based endoscopic screening strategy is not cost-effective in low to intermediate risk regions for GC, as seen in most European countries [9]. However, EGD, commonly performed for evaluating various digestive symptoms, provides an opportunity to detect GPC that can potentially lead to GC [4]. Unfortunately, some at-risk individuals remain undetected, and around 10% of GC cases are missed during EGD [10-12]. The underdiagnosis of high-risk patients may potentially lead to a lack of surveillance and the diagnosis of GC at advanced stages, thereby underscoring the importance of implementing risk stratification strategies in routine clinical practice [13-15].

The MAPS I and II guidelines (published in 2012 and 2019, respectively) highlighted the weak correlation between endoscopic findings and histology in the diagnosis of GPC. As a result, they recommended protocolized gastric biopsies during the initial diagnostic EGD [4,5]. Between the MAPS I and II guidelines, the ESGE quality guideline (2016) also recommended the use of the MAPS protocol for gastric biopsies [6]. Despite this recommendation, gastric biopsy rates vary significantly across European tertiary hospitals [16], particularly when the gastric mucosa appears "normal" or shows no visible abnormalities [17-19]. This variability in biopsy rates, which are essential for the histological risk stratification of GPC, may result not only from inconsistent adherence to guidelines but also from differences in endoscopic expertise in recognizing GPC [2,3].

Several factors can improve the endoscopic recognition of GPC, including the use of high-definition (HD) endoscopes, chromoendoscopy, adequate examination time, gastric mucosal cleansing, and patient sedation, among others [20-24]. However, in routine practice, the decision to perform gastric biopsies is primarily guided by the endoscopist's suspicion of abnormalities. In settings where biopsy sampling is based on the endoscopist's judgment, a higher GPC detection rate may reflect greater expertise in recognizing these conditions, leading to more thorough examinations and a reduced risk of GC post-EGD. Conversely, a lower GPC detection rate may indicate limited diagnostic expertise, increasing the likelihood of GC post-EGD. Therefore, this study aimed to assess the correlation between the GPC detection rate from 2010 to 2019 and GC post-EGD rates, as well as to identify factors associated with GPC detection in routine clinical practice within a community-based setting in a low-risk GC country.

MATERIALS AND METHODS

All consecutive EGDs performed for any indication between January 2010 to December 2019 at the Endoscopy Unit of the Hospital General of Granollers (a community hospital) were retrieved from an electronic database (Endobase©, Olympus, Europe) used for editing endoscopic reports. Patients < 18 years and therapeutic EGDs were excluded. This study was approved by the Ethics Committee of the Hospital de Granollers (Code 20203002).

EGDs were performed with standard (GIF-Q160 and GIF-Q165, Olympus, Germany) and high-definition

endoscopes (GIF-HQ180 and GIF-HQ185, Olympus, Germany), along with videoprocessors Exera II and Exera III (Olympus, Germany). No systematic exploration with NBI or chromoendoscopy was performed. All patients received sedation with propofol administered by an anesthesiologist. The examinations were carried out by nine gastroenterologists, and the gender, age, and experience of the endoscopists were documented. EGD data were extracted from Endobase©, while histological information of gastric biopsies was obtained from the hospital pathology database. EGDs were subsequently classified according to the most advanced histology and grouped into one of the following three categories: (i) benignity (normal, hyperplastic polyp, fundic gland polyp, foveolar hyperplasia, acute gastritis, ulcer debris, chronic gastritis); (ii) GPC (glandular atrophy, IM, and any grade of dysplasia); and (iii) malignancy (adenocarcinoma, gastrointestinal stromal tumor, lymphoma, neuroendocrine tumor/endocrine cell hyperplasia, and metastasis).

The detection rates of GPC, benignity, malignancy, and the rate of gastric biopsies were calculated. For the rate of GC after a negative EGD (post-EGD), cases of adenocarcinomas were additionally identified from the hospital pathology database up to December 2022. EGDs performed in cases of GC post-EGD were reviewed in detail, and we excluded EGDs that were therapeutic, incomplete (due to reasons such as poor mucosal visualization caused by food, blood, active bleeding, patient intolerance, or anesthetic complications), had a suspicious malignant lesion (whether confirmed by biopsies or not), and patients previously diagnosed with dysplasia.

Definitions

Detection rate of GPC: Defined as the percentage of EGDs in which at least one GPC was identified. This variable was calculated by dividing the number of EGDs with GPC by the total number of EGDs performed by each endoscopist [23]. The detection rate of dysplasia was also calculated by dividing the number of EGDs in which any grade of dysplasia was diagnosed by the total number of EGDs.

Rate of gastric biopsy: Defined as the percentage of EGDs in which gastric biopsies were obtained [16]. This variable was calculated by dividing the number of EGDs with gastric biopsies by the total number of EGDs performed by each endoscopist. This concept differs from the Endoscopic Biopsy Rate (EBR), which includes any biopsy taken during EGD, regardless of the anatomical site [23].

GC post-EGD: Defined as a diagnosis of gastric adenocarcinoma made within three years following an EGD that showed no endoscopic or histological suspicion of malignancy [25,26].

Rate of GC post-EGD: Defined as the number of EGDs initially negative for malignancy or dysplasia in which a subsequent diagnosis of gastric adenocarcinoma was made, divided by the total number of EGDs performed by each endoscopist during the study period (2010–2019). The rate is expressed per 1,000 procedures [27]. It is important to note that this differs from the *missed GC* rate.

Missed GC rate: A retrospective concept defined as the percentage of patients diagnosed with GC during a specified time period who had a previously negative EGD within the three years prior to diagnosis [25]. This definition also focuses on cases of gastric adenocarcinoma and assesses the proportion of patients who had a prior endoscopy that failed to detect GC, reflecting the potential for delayed or missed diagnosis.

Statistical analysis

Continuous variables are presented as the median along with the range. Categorical and continuous variables were compared using the Chi-squared (X^2) test and the Wilcoxon test, respectively. Various rates were computed for the entire series and on a per-endoscopist basis. The correlation between different rates and the rate of GC post-EGD was assessed using Spearman's correlation coefficient. Furthermore, a univariate logistic regression analysis was conducted to identify factors that increased the detection rate of GPC. Subsequently, all significant factors were analysed through a stepwise multivariate logistic regression model. The adjusted odds ratio (OR) was calculated to Indicate the associated risk. SPSS version 25 (IBM Corp, Armonk, NY, USA) was used for all statistical analyses, and statistical significance was considered at $p \leq 0.05$.

Ethical considerations

This is an observational study. The Hospital de Granollers's Research Ethics Committee (Code 20203002) has confirmed that informed consent was not required.

RESULTS

A total of 19,375 EGDs were performed during the 10-year study period (2010–2019). After excluding procedures that did not meet the inclusion criteria, 18,635 EGDs performed in 14,776 patients were included in the analysis. Among these, 12,226 patients underwent one EGD, 1,811 underwent two, and 739 patients had three or more EGDs. Only 3,825 procedures (20.5%) were performed using high-definition endoscopes. Gastric biopsies were obtained in 2,415 EGDs (13%). Histopathological findings

were benign in 1,582 cases (8.5%), showed GPC in 533 cases (2.9%), and revealed malignancy in 300 cases (1.6%) (Table 1).

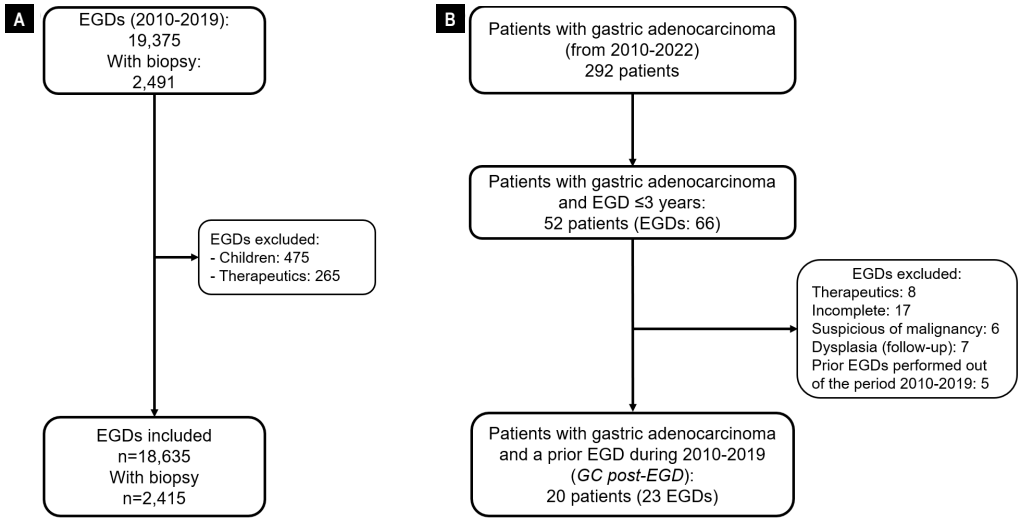
From the pathology database (2010–2022), a total of 292 patients with gastric adenocarcinoma were identified, of whom 20 had at least one previous EGD negative for

Table 1. EGD characteristics and histological results.

EGD characteristics	n=18,635	%
Patient's sex		
Female	9,778	52.5
Male	8,857	47.5
Patient's age		
≤ 40 years old	2,861	15.4
> 40 - ≤ 60 years old	6,354	34.1
> 60 years old	9,408	50.5
Endoscopist's sex		
Female	5,185	27.8
Male	13,450	72.2
Endoscopist's age*		
≤ 51 years old	9,369	50.3
> 51 years old	9,266	49.7
Endoscopist's experience*		
≤ 20 years	9,411	50.5
> 20 years	9,224	49.5
Period of time		
From 2010 to 2012	4,991	26.8
From 2013 to 2015	5,357	28.7
From 2016 to 2019	8,287	44.5
Type of EGD		
First-time EGD	14,776	79.3
Follow-up EGD	3,859	20.7
Endoscope quality		
Standard definition	14,810	79.5
High definition	3,825	20.5
Gastric biopsy during EGD		
Without gastric biopsy	16,220	87.0
With gastric biopsy	2,415	13.0
Gastric protocolized biopsies		
Non-protocolized biopsies	2,172	11.7
Protocolized biopsies	243	1.3
Histological results from gastric biopsies	N=2,415	13.0
Benign findings	1582	8.5
Normal	195	1.0
Non-atrophic chronic gastritis	774	4.2
Polyps (fundic or hyperplastic)	394	2.1
Miscellanea (ulcer debris, foveolar hyperplasia, acute gastritis)	219	1.2
Gastric premalignant conditions	533	2.9
Glandular atrophy	94	0.5
Intestinal metaplasia	373	2.0
Dysplasia	66	0.4
Malignancy	300	1.6
Adenocarcinoma	249	1.3
Other (GIST, lymphoma, neuroendocrine tumor, metastasis)	51	0.3

EGD, esophagogastroduodenoscopy; GIST, gastrointestinal stromal tumor; *Quantitative variable was split-up into two groups base on the median.

 https://doi.org/10.47892/rgp.2025.452.1909



EGD: esophago-gastro-duodenoscopy; GC: gastric cancer.

Figure 1. Flowchart of the study. (**A**) EGDs included during the period from 2010 to 2019. (**B**) GC post-EGD included (considering all cases of gastric adenocarcinomas from 2010 to 2022) which happened in patients with a negative EGD performed during the period from 2010 to 2019.

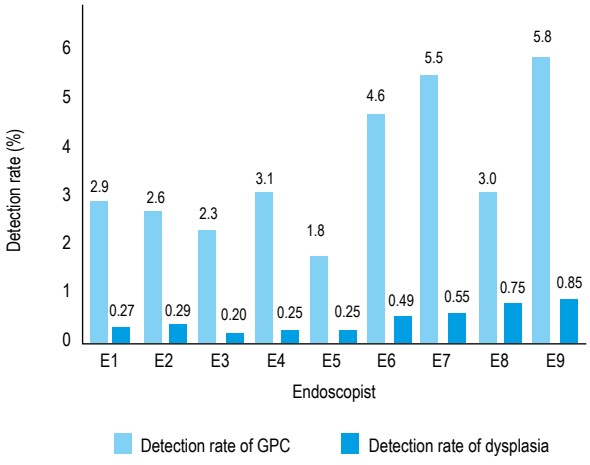
malignancy. Thus, the missed GC rate during this period was 6.8% (20/292) (Figure 1). Among these 20 patients, a total of 23 EGDs without evidence of malignancy or dysplasia were performed between 2010 and 2019, resulting in a GC post-EGD rate of 1.26 per 1,000 EGDs (23/18,269) (Table 2).

Among the nine endoscopists, the GPC detection rate ranged from 1.8% to 5.8%, while the dysplasia detection rate varied between 0.20% and 0.85% (Table 2 and Figure 2). Conversely, the rate of GC post-EGD across EGDs performed by individual endoscopists was 1.26 per

Table 2. Number of EGDs with gastric biopsies performed per endoscopist, histological findings, GC post-EGD, and correlation between the rate of GC post-EGD and the detection rates of gastric biopsy, benignity, GPC/dysplasia and malignancy.

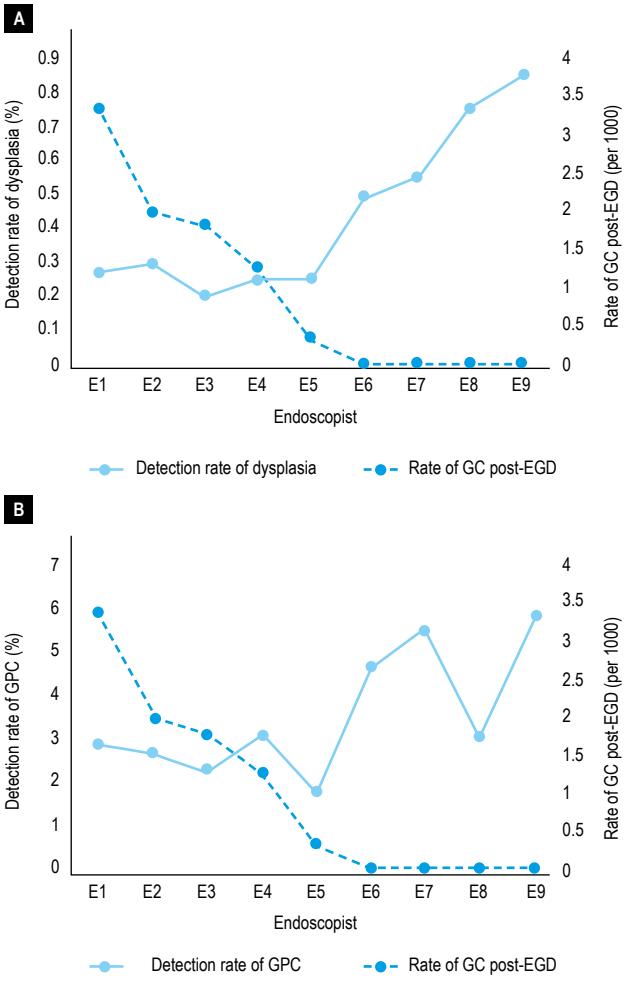
Endoscopist	Nº of all EGDs	Annual mean number of EGDs	EGD with gastric biopsy (%)	Findings in gastric biopsies				GC post-EGD (rate per 1000 EGDs*)
				Benignity (%)	GPC (%)		Malignancy (%)	
					All types	Dysplasia		
E1	1,822	364	304 (16.7)	223 (12.2)	52 (2.9)	5 (0.27)	29 (1.6)	6 (3.36)
E2	4,087	409	441 (10.8)	270 (6.6)	108 (2.6)	12 (0.29)	63 (1.5)	8 (1.99)
E3	3,995	666	446 (11.2)	275 (6.9)	90 (2.3)	8 (0.20)	81 (2.0)	7 (1.79)
E4	3,217	460	351 (10.9)	242 (7.5)	57 (1.8)	8 (0.25)	52 (1.6)	1 (0.32)
E5	814	81	138 (17.0)	104 (12.8)	25 (3.1)	2 (0.25)	9 (1.1)	1 (1.25)
E6	1,875	469	204 (10.9)	119 (6.3)	57 (3.0)	14 (0.75)	28 (1.5)	0
E7	1,635	409	284 (17.4)	187 (11.4)	76 (4.6)	8 (0.49)	21 (1.3)	0
E8	366	366	63 (17.2)	39 (10.7)	20 (5.5)	2 (0.55)	4 (1.1)	0
E9	824	412	184 (22.3)	123 (14.9)	48 (5.8)	7 (0.85)	13 (1.6)	0
Total	18,635	-	2,415 (13.0)	1,582 (8.5)	533 (2.9)	66 (0.35)	300 (1.6)	23 (1.26)
Correlation (p-value)	NA	NA	-0.49 (0.18)	-0.08 (0.84)	-0.65 (0.05)	-0.69 (0.03)	0.59 (0.09)	Reference

EGD, esophagogastroduodenoscopy; GC, gastric cancer; GPC, gastric premalignant condition (glandular atrophy, intestinal metaplasia and dysplasia); HD, high-definition endoscopes; NA, not applicable. *Cases with malignancy and dysplasia at the initial EGD were excluded for this rate.



GPC: gastric premalignant conditions. E: endoscopist.

Figure 2. The detection rate of GPC and dysplasia per endoscopist.



GPC: gastric premalignant conditions; GC: gastric cancer; EGD: esophagogastroduodenoscopy; rs: Spearman's correlation.

Figure 3. The trend of the rate of gastric cancer post-EGD and the detection rate of dysplasia (**A**) and GPC (**B**).

1,000 procedures, with variations ranging from 0 to 3.36 per 1,000 EGDs. A non-significant negative correlation was observed between the GPC detection rate and GC post-EGD (rs=–0.65, p=0.057); however, this correlation became statistically significant when considering the dysplasia detection rate (rs=–0.69, p=0.037) (Table 2 and Figure 3).

In the stepwise multivariate analysis, GPC detection was associated with three categories of factors: patient-related, endoscopist-related, and the time period in which the EGD was performed. Among patient-related factors, male sex was associated with an increased risk [OR 1.28; 95% CI: 1.05-1.56], as was older age, with elevated risk observed in patients aged >40 to ≤60 years [OR 2.25; 95% CI: 1.38-3.67] and in those over 60 years [OR 2.79; 95% CI: 1.74-4.49].

Regarding endoscopist-related factors, adherence to a standardized gastric biopsy protocol significantly increased the likelihood of detecting GPC [OR 2.02; 95% CI: 1.50-2.71]. The time period was also relevant, with EGDs performed between 2016 and 2019 being associated with a higher detection rate [OR 1.42; 95% CI: 1.10-1.84].

No significant associations were found with other endoscopist characteristics, such as age, sex, years of experience, annual EGD volume, or biopsy rate, nor with non-endoscopist factors such as EGD indication or endoscope resolution (Table 3).

DISCUSSION

This study aimed to explore the correlation between variations in GPC detection rates and GC post-EGD rates among endoscopists in a community hospital in a low-risk GC country, covering the period from 2010 to 2019. A notable negative correlation trend was observed between GPC detection rates and GC post-EGD, reaching statistical significance in the case of advanced GPC, such as dysplasia. No correlation was found between the gastric biopsy rate and GC post-EGD. These findings highlight the importance of accurate identification of GPC—particularly through targeted biopsies—and underscore the need for structured training programs to improve diagnostic performance, especially in non-academic or non-specialized centers.

Similar to the role of adenomas in the pathogenesis of colorectal cancer, GPCs are key precursors in the development of GC, especially adenocarcinomas. In colonoscopy, the adenoma detection rate (ADR)—which is intrinsically linked to polypectomy—is a well-established quality indicator due to its association with reduced incidence and mortality of post-colonoscopy colorectal cancer [28]. However, unlike in colonoscopy, consistent

Table 3. Factors related to the detection of GPC.

Factors	Nº EGD (%)	EGD with GPC		Univariant analysis		Multivariant analysis	
	n=18,635	Yes (%)	No (%)	OR (95% CI)	*p*-value	Adjusted OR (95% CI)	p-value
Patient's age							
≤40	2,861 (15.4)	21 (0.7)	2,840 (99.3)	Ref	-	Ref	-
>40 - ≤60	6,354 (34.1)	170 (2.7)	6,184 (97.3)	3.71 (2.35 - 5.86)	<0.001	2.25 (1.38 – 3.67)	0.001
>60	9,408 (50.5)	333 (3.5)	9,075 (96.5)	4.96 (3.18 - 7.72)	<0.001	2.79 (1.74 – 4.49)	<0.001
Patient' sex							
Female	9,778 (52.5)	251 (2.6)	9,527 (97.4)	Ref	-	Ref	-
Male	8,857 (47.5)	282 (3.2)	8575 (96.8)	1.24 (1.05-1.48)	0.012	1.28 (1.05 – 1.56)	0.013
Endoscopist's age (years)**							
≤51	9,369 (50.3)	306 (3.3)	9,063 (96.7)	Ref	-	-	-
>51	9,266 (49.7)	227 (2.4)	9,039 (97.6)	0.74 (0.63 – 0.89)	<0.001	-	-
Endoscopist's sex							
Female	5,185 (27.8)	158 (3.0)	5,027 (97.0)	Ref	-	-	-
Male	13,450 (72.2)	375 (2.8)	13,075(97.2)	0.91 (0.76 – 1.10)	0.34	-	-
Endoscopist's experience (years)**							
≤ 20	9,411 (50.5)	315 (3.3)	9,096 (96.7)	Ref	-	-	-
>20	9,224 (49.5)	218 (2.4)	9,006 (97.6)	0.69 (0.59 – 0.83)	<0.001	-	-
Endoscopist EGD's annual mean**							
≤ 412	9,548 (51.2)	329 (3.4)	9,219 (96.6)	Ref	-	-	-
> 412	9,087 (48.8)	204 (2.2)	8,883 (97.8)	0.64 (0.54 – 0.77)	<0.001	-	-
Period of time							
2010 – 2012	4,991 (26.8)	101 (2.0)	4,890 (98.0)	Ref	-	Ref	-
2013 – 2015	5,357 (28.7)	150 (2.8)	5,207 (97.2)	1.39 (1.08 - 1.80)	0.011	1.22 (0.91 – 1.62)	0.178
2016 – 2019	8,287 (44.5)	282 (3.4)	8,005 (96.6)	1.71 (1.35 – 2.15)	<0.001	1.42 (1.10 – 1.84)	0.007
Type of EGD							
1st EGD	14,776 (79.3)	346 (2.3)	14,430 (97.7)	Ref	-	-	-
Follow-up	3,859 (20.7)	187 (4.8)	3,672 (95.2)	2.12 (1.77 – 2.55)	<0.001	-	-
Endoscope definition							
SD	14,810 (79.5)	395 (2.7)	14,415 (97.3)	Ref	-	-	-
HD	3,825 (20.5)	138 (3.6)	3,687 (96.4)	1.37 (1.12 – 1.66)	0.002	-	-
Endoscopist gastric biopsy rate*							
≤ 11.2	9,179 (49.3)	222 (2.4)	8,957 (97.6)	Ref	-	-	-
>11.2	9,456 (50.7)	311 (3.3)	9,145 (96.7)	1.37 (1.15 – 1.63)	<0.001	-	-
Gastric protocolized biopsy**							
No	2,172 (89.9)	453 (20.9)	1,719 (79.1)	Ref	-	Ref	-
Yes	243 (10.1)	80 (32.9)	163 (67.1)	1.86 (1.39 – 2.48)	<0.001	2.02 (1.50 – 2.71)	<0.001

EGD, esophagogastroduodenoscopy; GPC, gastric premalignant condition; SD, standard-definition; HD, high-definition. CI, Confidence interval. *Quantitative variable was split-up into two groups base on the median. **Gastric protocolized biopsies from the antrum and the body among patients in whom biopsies were taken (n=2,415).

evidence linking GPC detection rates to the occurrence of GC post-EGD is lacking. This gap has hindered the establishment of clear benchmarks for GPC detection in routine EGD practice [6,29,30].

In our single-centre study, the overall GPC detection rate (2.9%) was consistent with findings from other European settings, such as the 3% reported in real-world practice at a French tertiary hospital [31]. Notably, in Poland, Januszewicz et al. observed a substantial difference in both the EBR and the GPC detection rate between a general hospital (2%) and a cancer-specialised institute (12.7%) [23]. These variations may be attributed to differences in patient populations (e.g., inclusion of high-risk patients in the cancer institute) as well as varying levels of awareness and expertise among endoscopists.

Studies reporting GC cases with a prior negative EGD typically refer to the concept of missed GC, commonly expressed as the missed GC rate. In our study, this rate was 6.8%, consistent with estimates from the three meta-analyses published to date [10-12]. This metric is widely used to raise awareness about EGD quality and allows for comparisons across endoscopy units or institutions. However, it is not appropriate for evaluating performance at the level of individual practitioners, where the total number of EGDs performed must be taken into account. Although this approach has been relatively underexplored

in the literature, some recent studies have incorporated procedure volume per endoscopist into their analyses [23,27]. Following this rationale, our study adopted the concept of GC post-EGD, calculated per 1,000 EGDs performed by each endoscopist, to better reflect individual-level outcomes [27].

Considering this perspective, the overall rate of GC post-EGD in our series (1.26 cases per 1,000 procedures) is also consistent with rates reported in other low-risk GC countries, such as Australia (35 cases out of 28,064 EGDs; 1.25 per 1,000), Denmark (26 out of 27,368; 0.9 per 1,000), and, more recently, Poland (36 cases out of 29,634; 1.21 per 1,000) [10,23,32,33]. In contrast, higher rates have been reported in high-risk countries such as Japan, with 3.99 cases per 1,000 EGDs (199 cases of GC out of 29,775 EGDs performed in symptomatic patients) [34]. This difference is primarily attributable to the higher prevalence of GC in these populations, as well as to variations in histopathological criteria—particularly in Japan. These findings suggest that the likelihood of GC post-EGD is lower in countries with a lower overall prevalence of GC.

Before the MAPS III (2025) and RE.GA.IN (2024) consensuses, the diagnosis and risk stratification of GPC in Europe and other Western countries relied exclusively on the histological evaluation of gastric biopsies [7,35]. The diagnosis of GPC is intrinsically linked to gastric biopsy—much like ADR is to polypectomy in colonoscopy. In this context, the EBR has been associated with improved GPC detection and a reduced incidence of GC post-EGD [23]. While the EBR, defined as any biopsy performed during EGD, has been proposed as a potential quality indicator, our study did not find a correlation between the specific gastric biopsy rate and GC post-EGD. Two factors may explain this lack of correlation: first, the low-risk setting for GC in our community hospital—malignancy was diagnosed in only 1.6% of EGDs—which reduces the likelihood of missing a cancer during the procedure; and second, the relatively low gastric biopsy rate observed in our cohort (13%) compared to previously published data [16,23]. The importance of this study lies in the identification of a connection—a negative correlation—between the GPC detection rate (with a significant association observed in cases of dysplasia) and GC post-EGD, despite a relatively low gastric biopsy rate.

Despite the quite low rate of gastric biopsies, we observed a significant increase in GPC detection during the more recent period (2016–2019), particularly when biopsies were performed following a standardized protocol. The importance of protocolized biopsies on the detection of GPC was demonstrated in a recent Chilean study [36]. Although this improvement may reflect increased adherence to existing guidelines (MAPS I – 2012 and the ESGE Upper GI Quality Guideline – 2016) [4,6], the key factor may not be the frequency of biopsies per se, but rather the appropriateness and quality of sampling—specifically, targeted biopsies guided by endoscopic recognition rather than random sampling. Recent evidence from China supports this interpretation, showing that higher GPC detection rates were more closely related to endoscopist qualification than to EBR alone [37].

Due to advances in endoscopic imaging and chromoendoscopic techniques, an endoscopic model for gastric carcinogenesis has been proposed for use during gastroscopy examination [38]. The ACG guideline also recommends that gastric exploration be guided by the identification of GPC [8]. During the study period (2010–2019), endoscopic classification systems such as Kimura-Takemoto for glandular atrophy and EGGIM for intestinal metaplasia were not routinely used. Today, endoscopic risk stratification should be properly complemented by standardized gastric biopsy protocols [7,8,35]. Increasing adherence to MAPS guideline recommendations would enhance GPC detection and improve the chances of diagnosing GC at early stages. However, poor adherence to biopsy sampling recommendations during EGD has been documented across Europe, even in academic centers. A survey conducted by Bornschein *et al.* [16], involving ten academic centers with significant expertise in gastric pathology, revealed that gastric biopsies were performed in only 43.8% of cases, with biopsy rates ranging from 13% to 76.6%. Notably, centers that implemented the MAPS guidelines showed significantly higher biopsy rates. These centers also demonstrated superior sampling practices, with biopsies more frequently obtained from both the antrum and the body, compared to centers not adhering to MAPS recommendations. Although the primary objective of that study was not GPC detection, another study demonstrated a clear improvement in GPC detection when protocolized biopsies were performed [36].

In studies involving symptomatic patients—mainly those with dyspepsia—up to 80% of EGDs are labelled as normal [39]. Our findings are consistent with these results, as gastric biopsies were not taken in more than 80% of cases. This suggests that endoscopists often refrain from taking biopsies in the absence of significant or visible abnormalities. In this context, limited awareness or ability to recognize GPCs may influence adherence to biopsy protocols. Without the endoscopic recognition of GPC or mucosal abnormalities, biopsies are frequently omitted, resulting in poor compliance with guideline recommendations. This explanation is supported by studies on missed GC, which have shown that approximately one-third of prior EGDs were reported as "normal" [17-19], and biopsies were omitted in nearly two-thirds of these cases [17,40]. Although high-definition (HD) endoscopy and virtual chromoendoscopy have been shown to improve the diagnostic accuracy of GPC [2,3], their use remains limited in routine clinical practice. In our study, these technologies were not used systematically, and the availability of HD endoscopes was not associated with improved GPC detection rates.

Our findings are consistent with previous reports showing that male sex and increasing age are established risk factors for the presence of GPC, mirroring the epidemiological patterns observed in GC [13,31]. Contrary to

other studies in which endoscopic expertise was associated with higher detection rates of high-risk lesions [10], our findings showed no significant differences based on the endoscopist's age or experience. However, the influence of the MAPS I and ESGE Upper GI quality guidelines may have encouraged younger endoscopists to adhere more closely to best practices—such as standardized biopsy protocols—leading to increased GPC detection rates during the most recent period, from 2016 to 2019 [4,6]. These findings highlight the importance of continuous education and training in upper GI endoscopy [41,42]. The establishment of a quality indicator analogous to the ADR in colonoscopy should be considered, taking into account the population risk of GC, the national prevalence of GPC, and the healthcare setting, with a particular focus on first-time diagnostic EGDs. Ensuring high-quality endoscopy requires that endoscopists develop competence in the recognition of GPC, dysplasia, and early GC [43], which would, in turn, enhance the diagnostic yield of gastric biopsies.

The main strength of our study lies in the fact that the Hospital General of Granollers is integrated into the Spanish National Health System and serves as the reference center for GC treatment in a well-defined geographic area. This integration minimizes the risk of data loss in GC post-EGD cases. Furthermore, all EGDs involving gastric biopsies or resulting in a GC post-EGD diagnosis were individually reviewed.

However, this study has several limitations. Its retrospective design limits the ability to assess the impact of critical quality factors such as procedure time or gastric mucosal cleansing on GPC detection. Additional limitations include: (i) the single-centre design, which restricts the representativeness and generalizability of our findings to broader populations; (ii) limitations in the electronic registry, which prevented the collection of specific indications for EGD—factors that may have influenced biopsy decisions. Additionally, some therapeutic endoscopies may have been miscoded and included, potentially leading to an underestimation of the endoscopists' detection rates; (iii) variation in endoscopist workload (measured as the endoscopist's annual mean number of EGDs) may have affected the results, as those performing fewer procedures or working only in the most recent years were less likely to be linked to GC post-EGD cases. To mitigate this bias, we reviewed all gastric adenocarcinomas recorded in the pathology database from 2010 to 2022 and only considered GC post-EGD cases in patients who had undergone EGD between 2010 and 2019. And finally, due to the lack of consensus on how to report GC post-EGD rates at the individual level, we opted to express this outcome as the number of cases per 1,000 EGDs [27].

Our results reflect routine clinical practice before and after the publication of MAPS I (2012) and the ESGE Upper GI Quality Guideline (2016). These findings may serve as a benchmark for evaluating the impact of MAPS II (2019), MAPS III (2025), and the national clinical guidelines published in 2021 on the quality of EGDs performed thereafter [44,45].

In conclusion, the detection rate of GPC, especially advanced cases like dysplasia, showed a negative correlation with the rate of GC post-EGD in a community hospital within a low-risk GC country from 2010 to 2019. We proposed that the detection rate of GPC could serve as a valuable quality indicator for monitoring EGD performance. Therefore, strategies to enhance GPC detection and minimize variability among endoscopists are warranted.

Use of artificial intelligence

Artificial intelligence was used to correct grammar and enhance the academic fluency of the English text.

Data availability

The datasets generated or analysed during the current study are available from the corresponding author on reasonable request.

REFERENCES

1. Correa P, Piazuelo MB. The gastric precancerous cascade. J Dig Dis. 2012;13(1):2-9. doi: 10.1111/j.1751-2980.2011.00550.x.
2. Delgado-Guillena PG, Morales-Alvarado VJ, Elosua-González A, Murcia Pomares O, Pérez-Aisa A, Córdova H, *et al*. Gastroenterologists' attitudes on the detection and management of gastric premalignant conditions: results of a nationwide survey in Spain. Eur J Cancer Prev. 2021;30(6):431-436. doi: 10.1097/CEJ.0000000000000648.
3. Yip HC, Uedo N, Chan SM, Teoh AYB, Wong SKH, Chiu PW, *et al*. An international survey on recognition and characterization of atrophic gastritis and intestinal metaplasia. Endosc Int Open. 2020;8(10):E1365-E1370. doi: 10.1055/a-1230-3586.
4. Dinis-Ribeiro M, Areia M, de Vries AC, Marcos-Pinto R, Monteiro-Soares M, O'Connor A, *et al*. Management of precancerous conditions and lesions in the stomach (MAPS): guideline from the European Society of Gastrointestinal Endoscopy (ESGE), European Helicobacter Study Group (EHSG), European Society of Pathology (ESP), and the Sociedade Portuguesa de Endoscopia Digestiva (SPED). Endoscopy. 2012;44(1):74-94. doi: 10.1055/s-0031-1291491.
5. Pimentel-Nunes P, Libânio D, Marcos-Pinto R, Areia M, Leja M, Esposito G, *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(4):365-388. doi: 10.1055/a-0859-1883.
6. Bisschops R, Areia M, Coron E, Dobru D, Kaskas B, Kuvaev R, *et al*. Performance measures for upper gastrointestinal endoscopy: A European Society of Gastrointestinal Endoscopy quality improvement initiative. United European Gastroenterol J. 2016;4(5):629-656. doi: 10.1177/2050640616664843.
7. Dinis-Ribeiro M, Libânio D, Uchima H, Spaander MCW, Bornschein J, Matysiak-Budnik T, *et al*. Management of epithelial precancerous conditions and early neoplasia of the stomach (MAPS III): European Society of Gastrointestinal Endoscopy (ESGE), European Helicobacter and Microbiota Study Group (EHMSG) and European Society of Pathology (ESP) Guideline update 2025. Endoscopy. 2025;57(5):504-554. doi: 10.1055/a-2529-5025.

8. Morgan DR, Corral JE, Li D, Montgomery EA, Riquelme A, Kim JJ, *et al*. ACG Clinical Guideline: Diagnosis and Management of Gastric Premalignant Conditions. Am J Gastroenterol. 2025;120(4):709-737. doi: 10.14309/ajg.0000000000003350.
9. Areia M, Dinis-Ribeiro M, Rocha Gonçalves F. Cost-utility analysis of endoscopic surveillance of patients with gastric premalignant conditions. Helicobacter. 2014;19(6):425-36. doi: 10.1111/hel.12150.
10. Pimenta-Melo AR, Monteiro-Soares M, Libânio D, Dinis-Ribeiro M. Missing rate for gastric cancer during upper gastrointestinal endoscopy: a systematic review and meta-analysis. Eur J Gastroenterol Hepatol. 2016;28(9):1041-9. doi: 10.1097/MEG.0000000000000657.
11. Menon S, Trudgill N. How commonly is upper gastrointestinal cancer missed at endoscopy? A meta-analysis. Endosc Int Open. 2014;2(2):E46-50. doi: 10.1055/s-0034-1365524.
12. Alexandre L, Tsilegeridis-Legeris T, Lam S. Clinical and Endoscopic Characteristics Associated With Post-Endoscopy Upper Gastrointestinal Cancers: A Systematic Review and Meta-analysis. Gastroenterology. 2022;162(4):1123-1135. doi: 10.1053/j.gastro.2021.12.270.
13. Delgado-Guillena P, Morales-Alvarado V, Ramírez Salazar C, Jimeno Ramiro M, Llibre Nieto G, Galvez-Olortegui J, *et al*. Frequency and clinical characteristics of early gastric cancer in comparison to advanced gastric cancer in a health area of Spain. Gastroenterol Hepatol. 2020;43(9):506-514. English, Spanish. doi: 10.1016/j.gastrohep.2020.01.015.
14. Chapelle N, Bouvier A-M, Manfredi S, Drouillard A, Lepage C, Faivre J, et al. Early Gastric Cancer: Trends in Incidence, Management, and Survival in a Well-Defined French Population. Ann Surg Oncol. 2016;23(11):3677-3683. doi: 10.1245/s10434-016-5279-z.
15. Dassen AE, Dikken JL, Bosscha K, Wouters MW, Cats A, van de Velde CJ, *et al*. Gastric cancer: decreasing incidence but stable survival in the Netherlands. Acta Oncol. 2014;53(1):138-42. doi: 10.3109/0284186X.2013.789139.
16. Bornschein J, Tran-Nguyen T, Fernandez-Esparrach G, Ash S, Balaguer F, Bird-Lieberman E, *et al*. Biopsy Sampling in Upper Gastrointestinal Endoscopy: A Survey from 10 Tertiary Referral Centres Across Europe. Dig Dis. 2021;39(3):179-189. doi: 10.1159/000511867.
17. Delgado Guillena PG, Morales Alvarado VJ, Jimeno Ramiro M, Rigau Cañardo J, Ramírez Salazar C, García Rodríguez A, *et al*. Gastric cancer missed at esophagogastroduodenoscopy in a well-defined Spanish population. Dig Liver Dis. 2019;51(8):1123-1129. doi: 10.1016/j.dld.2019.03.005.
18. Hernanz N, Rodríguez de Santiago E, Marcos Prieto HM, Jorge Turrión MÁ, Barreiro Alonso E, Rodríguez Escaja C, *et al*. Characteristics and consequences of missed gastric cancer: A multicentric cohort study. Dig Liver Dis. 2019;51(6):894-900. doi: 10.1016/j.dld.2019.02.006.
19. Voutilainen ME, Juhola MT. Evaluation of the diagnostic accuracy of gastroscopy to detect gastric tumours: clinicopathological features and prognosis of patients with gastric cancer missed on endoscopy. Eur J Gastroenterol Hepatol. 2005;17(12):1345-9. doi: 10.1097/00042737-200512000-00013.
20. Teh JL, Tan JR, Lau LJF, Saxena N, Salim A, Tay A, *et al*. Longer examination time improves detection of gastric cancer during diagnostic upper gastrointestinal endoscopy. Clin Gastroenterol Hepatol. 2015;13(3):480-487.e2. doi: 10.1016/j.cgh.2014.07.059.
21. Park JM, Huo SM, Lee HH, Lee BI, Song HJ, Choi MG. Longer Observation Time Increases Proportion of Neoplasms Detected by Esophagogastroduodenoscopy. Gastroenterology. 2017;153(2):460-469.e1. doi: 10.1053/j.gastro.2017.05.009.
22. Buxbaum JL, Hormozdi D, Dinis-Ribeiro M, Lane C, Dias-Silva D, Sahakian A, *et al*. Narrow-band imaging versus white light versus mapping biopsy for gastric intestinal metaplasia: a prospective blinded trial. Gastrointest Endosc. 2017;86(5):857-865. doi: 10.1016/j.gie.2017.03.1528.
23. Januszewicz W, Wieszczy P, Bialek A, Karpinska K, Szlak J, Szymonik J, *et al*. Endoscopist biopsy rate as a quality indicator for outpatient gastroscopy: a multicenter cohort study with validation. Gastrointest Endosc. 2019;89(6):1141–9. doi: 10.1016/j.gie.2019.01.008.
24. Zhou J, Li Z, Ji R, Wang P, Zhang A, Wu K, *et al*. Influence of Sedation on the Detection Rate of Early Cancer and Precancerous Lesions During Diagnostic Upper Gastrointestinal Endoscopies: A Multicenter Retrospective Study. Am J Gastroenterol. 2021;116(6):1230-1237. doi: 10.14309/ajg.0000000000001201.
25. Fujita S. Biology of early gastric carcinoma. Pathol Res Pract. 1978;163(4):297-309. doi: 10.1016/S0344-0338(78)80028-4.
26. Hosokawa O, Tsuda S, Kidani E, Watanabe K, Tanigawa Y, Shirasaki S, *et al*. Diagnosis of gastric cancer up to three years after negative upper gastrointestinal endoscopy. Endoscopy. 1998;30(8):669-74. doi: 10.1055/s-2007-1001386.
27. Kamran U, King D, Abbasi A, Coupland B, Umar N, Chapman WC, *et al*. A root cause analysis system to establish the most plausible explanation for post-endoscopy upper gastrointestinal cancer. Endoscopy. 2023;55(2):109-118. doi: 10.1055/a-1917-0192.
28. Kaminski MF, Wieszczy P, Rupinski M, Wojciechowska U, Didkowska J, Kraszewska E, *et al*. Increased Rate of Adenoma Detection Associates With Reduced Risk of Colorectal Cancer and Death. Gastroenterology. 2017;153(1):98-105. doi: 10.1053/j.gastro.2017.04.006.
29. Park CH, Kim B, Chung H, Lee H, Park JC, Shin SK, *et al*. Endoscopic quality indicators for esophagogastroduodenoscopy in gastric cancer screening. Dig Dis Sci. 2015;60(1):38-46. doi: 10.1007/s10620-014-3288-y.
30. Beg S, Ragunath K, Wyman A, Banks M, Trudgill N, Mark Pritchard D, *et al*. Quality standards in upper gastrointestinal endoscopy: a position statement of the British Society of Gastroenterology (BSG) and Association of Upper Gastrointestinal Surgeons of Great Britain and Ireland (AUGIS). Gut. 2017;66(11):1886-1899. doi: 10.1136/gutjnl-2017-314109.
31. Chapelle N, Péron M, Mosnier J-F, Quénéhervé L, Coron E, Bourget A, *et al*. Prevalence, Characteristics and Endoscopic Management of Gastric Premalignant Lesions in France. Dig Dis. 2020;38(4):286-292. doi: 10.1159/000503748.
32. Raftopoulos SC, Segarajasingam DS, Burke V, Ee HC, Yusoff IF. A cohort study of missed and new cancers after esophagogastroduodenoscopy. Am J Gastroenterol. 2010;105(6):1292-7. doi: 10.1038/ajg.2009.736.
33. Lassen A, Hallas J, de Muckadell OB. The risk of missed gastroesophageal cancer diagnoses in users and nonusers of antisecretory medication. Gastroenterology. 2005;129(4):1179-86. doi: 10.1053/j.gastro.2005.07.028.
34. Hosokawa O, Tsuda S, Kidani E, Watanabe K, Tanigawa Y, Shirasaki S, *et al*. Diagnosis of gastric cancer up to three years after negative upper gastrointestinal endoscopy. Endoscopy. 1998;30(8):669-74. doi: 10.1055/s-2007-1001386.
35. Rugge M, Genta RM, Malfertheiner P, Dinis-Ribeiro M, El-Serag H, Graham DY, *et al*. RE.GA.IN.: the Real-world Gastritis Initiative-updating the updates. Gut. 2024;73(3):407-441. doi: 10.1136/gutjnl-2023-331164.
36. Latorre G, Vargas JI, Shah SC, Ivanovic-Zuvic D, Achurra P, Fritzsche M, *et al*. Implementation of the updated Sydney system biopsy protocol improves the diagnostic yield of gastric preneoplastic conditions: Results from a real-world

study. Gastroenterol Hepatol. 2024;47(8):793-803. doi: 10.1016/j.gastrohep.2023.08.005.

37. Shen Y, Gao X-J, Zhang X-X, Zhao J-M, Hu F-F, Han J-L, *et al*. Endoscopists and endoscopic assistants' qualifications, but not their biopsy rates, improve gastric precancerous lesions detection rate. World J Gastrointest Endosc. 2025;17(4):104097. doi: 10.4253/wjge.v17.i4.104097.
38. Delgado-Guillena P, Jimeno M, López-Nuñez A, Córdova H, Fernández-Esparrach G. The endoscopic model for gastric carcinogenesis and Helicobacter pylori infection: a potential visual mind-map during gastroscopy examination. Gastroenterol Hepatol. 2024 Dec;47(10):502214. doi: 10.1016/j.gastrohep.2024.502214.
39. Nasseri-Moghaddam S, Mousavian A-H, Kasaeian A, Kanno T, Yuan Y, Ford AC, *et al*. What is the Prevalence of Clinically Significant Endoscopic Findings in Subjects With Dyspepsia? Updated Systematic Review and Meta-analysis. Clin Gastroenterol Hepatol. 2023;21(7):1739-1749.e2. doi: 10.1016/j.cgh.2022.05.041.
40. Gavric A, Hanzel J, Zagar T, Zadnik V, Plut S, Stabuc B. Survival outcomes and rate of missed upper gastrointestinal cancers at routine endoscopy: a single centre retrospective cohort study. Eur J Gastroenterol Hepatol. 2020;32(10):1312-1321. doi: 10.1097/MEG.0000000000001863.
41. Córdova H, Sánchez-Montes C, Delgado-Guillena PG, Morales VJ, Sendino O, González-Suárez B, *et al*. Quality indicators for esophagogastroduodenoscopy: A comparative study of outcomes after an improvement programme in a tertiary hospital. Gastroenterol Hepatol. 2017;40(9):587-594. English, Spanish. doi: 10.1016/j.gastrohep.2017.05.007.
42. Alcaraz Serrat JA, Córdova H, Moreira L, Pocurrull A, Ureña R, Delgado-Guillena PG, et al. Evaluation of long-term adherence to oesophagogastroduodenoscopy quality indicators. Gastroenterol Hepatol. 2020 Dec;43(10):589-597. doi: 10.1016/j.gastrohep.2020.01.017.
43. Dekker E, Houwen BBSL, Puig I, Bustamante-Balén M, Coron E, Dobru DE, *et al*. Curriculum for optical diagnosis training in Europe: European Society of Gastrointestinal Endoscopy (ESGE) Position Statement. Endoscopy. 2020;52(10):899-923. doi: 10.1055/a-1231-5123.
44. Cubiella J, Pérez Aisa Á, Cuatrecasas M, Díez Redondo P, Fernández Esparrach G, Marín-Gabriel JC, *et al*. Gastric cancer screening in low incidence populations: Position statement of AEG, SEED and SEAP. Gastroenterol Hepatol. 2021;44(1):67-86. English, Spanish. doi: 10.1016/j.gastrohep.2020.08.004.
45. Fernández-Esparrach G, Marín-Gabriel JC, Díez Redondo P, Núñez H, Rodríguez de Santiago E, Rosón P, *et al*. Quality in diagnostic upper gastrointestinal endoscopy for the detection and surveillance of gastric cancer precursor lesions: Position paper of AEG, SEED and SEAP. Gastroenterol Hepatol. 2021;44(6):448-464. English, Spanish. doi: 10.1016/j.gastrohep.2021.01.002.