First, we agree that stone size and impaction are established predictors of conventional technique failure, as demonstrated by Garg *et al* [3]. In our study [2], DBS were defined as stones >1.5 cm and/or impacted, consistent with validated criteria. Beyond confirming their relevance, our analysis identified DBS as statistically significant predictors of CG-EHL prioritization across all models. These findings support the view that stone size and impaction are central determinants in decision-making for EHL.

Second, the structure of our classification tree reflects this hierarchy. The initial split prioritizes DBS over other anatomical factors, such as papillary size or stone wedging (Fig. 1,2 in our study [2]), thereby supporting simplified clinical decision-making. Ridge regression further confirmed the predominant influence of DBS compared with lower-level predictors. Collectively, these findings reinforce the central role of DBS in guiding CG-EHL selection (Fig. 3).

Third, regarding cost considerations, our study does not advocate indiscriminate first-line use of EHL, but rather supports its earlier positioning in the therapeutic algorithm for appropriately selected patients. Conventional techniques should remain the initial approach given their established efficacy and lower immediate costs. However, when predictors suggest a high likelihood of failure with standard methods, early EHL may reduce repeat endoscopic retrograde cholangiopancreatography procedures, prolonged hospitalization, and cumulative adverse events.

Finally, no statistically significant differences were observed in adverse events or length of hospital stay. We agree that multicenter validation and formal cost-effectiveness analyses would further substantiate this approach and look forward to future studies addressing these important considerations.

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[a]First Department of Internal Medicine, Democritus University of Thrace, University Hospital of Alexandroupolis, Greece (Dionysios Kogias, Nikolaos Kafalis, Georgios Kouklakis); [b]Otolaryngology – Head and Neck, Chatzikosta Hospital of Ioannina, Greece (Vaia Karapepera); [c]Laboratory of Anatomy, Department of Medicine, Democritus University of Thrace, Alexandroupolis, Greece (Vasileios Papadopoulos)

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Correspondence to: Dionysios Kogias, First Department of Internal Medicine, Democritus University of Thrace, Department of Medicine, Dragana 1, General University Hospital of Alexandroupolis, Alexandroupolis, 68100, Greece, e-mail: dionkogi@gmail.com

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Impact of *Helicobacter pylori* eradication on the progression of Correa's cascade

Jannis Kountouras[a], Stergios A. Polyzos[a,b], Ioannis S. Papanikolaou[a,c], Michael Doulberis[a,d,e], Christos Liatsos[a,f], Elisabeth Vardaka[a,g]

Aristotle University of Thessaloniki, Ippokration Hospital, Thessaloniki, Macedonia, Greece; School of Medicine, Aristotle University of Thessaloniki, Thessaloniki, Macedonia, Greece; Medical School, National and Kapodistrian University of Athens, Athens, Greece; University Hospital Zurich, University of Zurich, Zurich, Switzerland; Medical University Department, Kantonsspital Aarau, Aarau, Switzerland; 401 General Military Hospital of Athens, Athens, Greece; School of Health Sciences, International Hellenic University, Thessaloniki, Greece

Kouvaras *et al* [1] demonstrated an association between *Helicobacter pylori* infection (*Hp*-I) and the development of gastric intestinal metaplasia (IM)—complete, incomplete or mixed forms—a premalignant lesion in the Correa cascade of gastric carcinogenesis. Beyond *Hp*, oral *Porphyromonas gingivalis* (Pg) can translocate to the stomach further promoting progression along the Correa cascade [2,3].

Focusing on IM and gastric cancer (GC) associated with *Hp* and/or Pg, several significant findings are noteworthy:

Hp can also inhabit the oral cavity, which may serve as a main extragastric reservoir. Similarly, the oral cavity serves as a primary reservoir for Pg [2]. Pg and its virulence factors, including gingipains, appear to contribute to the aforementioned sequence of gastric carcinogenesis [4].

Among the types of IM, the incomplete type is more

strongly associated with GC progression than the complete type [5].

Hp-induced inflammation, demonstrated in both mice (Houghton's theory) and humans, triggers the migration of bone-marrow–derived stem cells to the gastric mucosa. There, these cells undergo metaplastic and dysplastic changes that can lead to GC, in line with Correa's cascade [6].

Areas exhibiting severe inflammation, IM, atrophy, and GC also show increased mast cell density, correlated with *Hp*-induced gastritis [7]. The CagA and VacA cytotoxins of *Hp* play central roles in promoting oncogenesis, consistently with Correa's model.

The absence of such features in cases of IM suggests a more favorable prognosis. For example, Hwang *et al* [8] reported that IM disappeared ≥5 years after *Hp* eradication. *Hp* eradication protects against GC in patients with IM or dysplasia (follow-up range: 2-26.5 years), and may reverse these conditions Furthermore, incomplete IM regresses within 10 years following *Hp* eradication [5].

Notably, radiofrequency ablation can eradicate incomplete IM [9], and endoscopic grading of IM, as a valuable surveillance tool, reduces the need for routine biopsy sampling [10].

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[a]Department of Medicine, Second Medical Clinic, Aristotle University of Thessaloniki, Ippokration Hospital, Thessaloniki, Macedonia, Greece (Jannis Kountouras, Stergios A. Polyzos, Ioannis S. Papanikolaou, Michael Doulberis, Christos Liatsos, Elisabeth Vardaka); [b]First Laboratory of Pharmacology, School of Medicine, Aristotle University of Thessaloniki, Thessaloniki, Macedonia, Greece (Stergios A. Polyzos); [c]Hepatogastroenterology Unit, Second Department of Internal Medicine-Propaedeutic, Medical School, National and Kapodistrian University of Athens, Athens, Greece (Ioannis S. Papanikolaou); [d]Department of Gastroenterology & Hepatology, University Hospital Zurich, University of Zurich, Zurich, Switzerland (Michael Doulberis); [e]Division of Gastroenterology and Hepatology, Medical University Department, Kantonsspital Aarau, Aarau, Switzerland (Michael Doulberis); [f]Department of Gastroenterology, 401 General Military Hospital of Athens, Athens, Greece (Christos Liatsos); [g]Department of Nutritional Sciences and Dietetics, School of Health Sciences, International Hellenic University, Thessaloniki, Greece (Elisabeth Vardaka)

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Correspondence to: Jannis Kountouras, MD, PhD, Professor of Medicine, Gastroenterologist, 8 Fanariou St, Byzantio, 551 33, Thessaloniki, Macedonia, Greece, e-mail: jannis@auth.gr, ancoratus2010@gmail.com

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Authors' reply

Stergios N. Kouvaras[a,b], Ioannis G. Koumarianos[a], Konstantinos Ekmektzoglou[b], George A. Kounis[c], Charikleia Spiliadi[d], Sotirios D. Georgopoulos[e], Theodoros Rokkas[b]

Private Endoscopy Clinic; Henry Dunant Hospital, Athens; Statistical Analysis; Athens Medical Group; GI Dept Athens Medical, Paleo Faliron Hospital

The comments raised in the letter by Prof. Kountouras *et al* [1] are fully consistent with the findings of our study [2]. Nevertheless, several issues discussed in that letter were beyond the scope of our investigation. In this context, 3 points merit attention:

1. In addition to the inflammation induced by *Helicobacter pylori* (*H. pylori*), other bacteria—such as *Porphyromonas gingivalis* (Pg)—have been reported to accelerate the progression of the Correa cascade. In our study, the presence