There is much more to rely on histology than the sole endoscopy tells us

We read with great interest the article by Banks and coworkers, who presented the British Society of Gastroenterology guidelines on the diagnosis and management of patients at risk of gastric carcinoma (GC).[1] This work exhaustively provides statements and recommendations on the prevalence, risk factors, diagnosis and management of gastric premalignant lesions (ie, gastric atrophy (GA) and gastric intestinal metaplasia (GIM)) as well as early gastric cancer. Relevant suggestions for improving our clinical practice were made; however, some data have been underestimated and given the lack of useful predictors of cancer progression, we believe they are important to emphasise.

In these guidelines GA and GIM are grouped as chronic atrophic gastritis (CAG), which was staged based on its extension in the antrum-incisura or even in the gastric body. According to this endoscopic assessment, two groups of patients are identified, respectively, at low and high risks for evolution in GC. The authors stated that the endoscopic staging of the severity CAG can be sufficient to stratify the GC risk, since histology assessment lacks reproducibility (ie, high level of complexity and low consistency between pathologists) and insufficient validation data.[1]

The Operative Link on Gastritis Assessment (OLGA) system is a score able to quantify the degree of atrophy and to stage the CAG, based on biopsies taken using the updated Sydney protocol. It is assessed analysing specimens from both mucosecreting and oxyntic compartments separately, which are scored from 0 (no atrophy) to 3 (atrophy involving >60% of the samples). Scores from antrum and corpus are then cross-matched for an overall stage of CAG (I–IV).[2] Data to validate the usefulness of OLGA use in clinical practice have been recently published.[3 4] In particular, Rugge *et al* examined 7436 patients who underwent endoscopy with histological examination. At the first evaluation patients were stratified in terms of CAG severity, by OLGA staging system: stage 0 (80.8%), stage I (12.6%), stage II (4.3%), stage III (2.0%) and stage IV (0.3%).[4] At the end of the follow-up (mean/median=6.3/6.6 years), the rate of incident neoplasia was significantly stage related (incidence rate 10^3/person-years): stage 0=0.03; stage I=0.34; stage II=1.48; stage III=19.1; stage IV=41.2. The strong data emerging from this study suggested that OLGA assessment was able to predict risk of developing gastric cancer, based on the severity of CAG.

Concerning interobserver agreement variations between pathologists in CAG evaluation, recent studies were not unanimous and demonstrated that in experienced hands, accuracy is moderate to excellent both for OLGA and operative link on gastritic intestinal metaplasia assessment (OLGIM), even if it is better for the second one.[5 6] Also, in the previously mentioned validation study there was a substantial agreement despite different pathologists involved and the great number of specimens analysed.[4]

In conclusion, we think that in this laudable attempt of synthesis, the CAG staging with OLGA/OLGIM is a critical point that cannot be underestimated. We believe that considering all the specimens containing atrophy, obtained from the same compartment (antrum-incisura/corpus), would be as relevant as the 'simplified' partition based only on extension at endoscopy. Indeed, we want to underline that we do not see 'endoscopy' and 'histology' as two distinct and alternative methods to stratify the GC risk, but two complementary tests to be always used in conjunction in order to optimise the management of these

patients, reducing the risk of following up patients who do not need revaluations, and vice versa. Thus, OLGA staging should be considered in the management of these patients, in order to improve their prognosis.

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