PostScript

CORRESPONDENCE

Histopathology audit of chronic gastritis in India: need for objectivity and training

In India general surgical pathologists report on all tissue types, and subspecialty training is an exception. We undertook an internal audit of gastric biopsy reporting in a large teaching hospital to assess need for subspecialty training.

In a first phase, 228 consecutive gastric biopsies (two antral + one body bits in each) undertaken in our institution over six months were objectively evaluated by three experienced histopathologists, based on the updated Sydney classification.¹ The three histopathologists were "trained" over four to five sessions at a multihead microscope, each session lasting 20 to 30 minutes (less than two hours cumulative time). Thereafter each case took about eight minutes to score. All biopsies were evaluated independently and by consensus. Objectivity was validated by a κ analysis of interobserver and intraobserver variation. Intraobserver variation was analysed by a blinded repeat examination of 10% of the biopsies. Gastritis was present in 207 cases (90.8%), while *H pylori*, lymphoid aggregates, and intestinal metaplasia were seen in 190 (83.3%), 96 (42.1%), and 113 (49.0%, including 10% type III) cases, respectively. Dysplasia (seven cases; 3.1%) and lymphoepithelial lesions (four cases; 1.8%) were rare, supporting the view that *H pylori* infection leading to intestinal metaplasia is not a factor in gastric carcinogenesis in India.² We further qualified inflammation as neutrophilic activity (139 cases; 61.0%), eosinophilic activity (128 cases; 56.1%), and mast cell infiltration (130 cases; 57.0%). Lymphomononuclear infiltrate density correlated positively with atrophy, hyperplasia, and metaplasia. Both eosinophilic and neutrophilic activity independently correlated with *H pylori* density and hyperplasia. Mast

cell density was of no importance. Reproducibility was high for inflammation, metaplasia, dysplasia, and *H pylori* density, and intermediate for atrophy and hyperplasia, comparable with published reports.³

In a second phase, actual biopsy reports on these gastric biopsies were retrieved from our records and compared. Eight pathologists from our institution (including the three reviewing pathologists) had rendered the original reports. Omission of features ranged from 10.5% to 100% (fig 1). *H pylori* density had been mentioned in only 18.4% of the original reports, while the degree of lymphomononuclear and neutrophil infiltration was scored in only 26.8% and 25.9% of instances, respectively. Further, many errors (1.8% to 63.2%) were detected in the original reports, in the form of either incorrect observations or wrong grading of severity (table 1). Eosinophil activity was commonly missed, even though it correlates strongly with gastritis severity.⁴

In the final phase, clinical impact of the audit was analysed. Among features assessed, those with the greatest relevance to clinical management and prognosis were atrophy, metaplasia, dysplasia, lymphoepithelial lesions, *H pylori* density, and activity of gastritis. However, only a fraction of the original reports had correctly identified the presence and severity of these features. These

omissions and discrepancies could have directly affected treatment.

This study underscores the importance of routine and continuous in-house training and the use of objectivity in reporting, to prevent oversights and errors in surgical pathology. This could serve as a cost-effective alternative to long term subspecialty training.

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Figure 1 Features omitted in original reports.

Table 1 Errors in original reports in identifying individual features

Feature	Omitted	Wrong observation	Wrong grading	Correctly identified
Atrophy	173 (75.9%)	22 (9.7%)	9 (4.0%)	24 (10.5%)
Metaplasia	124 (54.4%)	36 (15.8%)	17 (7.5%)	51 (22.4%)
Dysplasia	170 (74.6%)	4 (1.8%)	NA	54 (23.7%)
LEL	228 (100%)	NA	NA	Nil (0%)
<i>H pylori</i>	24 (10.5%)	56 (24.6%)	88 (38.6%)	60 (26.3%)
Neutrophil activity	108 (47.4%)	14 (6.1%)	61 (26.8%)	45 (19.7%)
Eosinophil activity	221 (96.9%)	Nil (0%)	06 (2.6%)	01 (0.4%)

LEL, lymphoepithelial lesion; NA, not applicable.

References

- Dixon MF, Genta RM, Yardley JH, et al. Classification and grading of gastritis. The updated Sydney System. International Workshop on the Histopathology of Gastritis, Houston 1994. *Am J Surg Pathol* 1996;20:1161-81.
- Satarkar RP, Sawant P, Nanivadekar S, et al. Helicobacter pylori and intestinal metaplasia of gastric mucosa. *Indian J Gastroenterol* 1997;16:16-17.
- Chen XY, van der Hulst RW, Bruno MJ, et al. Interobserver variation in the histopathological scoring of Helicobacter pylori related gastritis. *J Clin Pathol* 1999;52:612-15.
- McGovern TW, Talley NJ, Kephart GM, et al. Eosinophil infiltration and degranulation in Helicobacter pylori-associated chronic gastritis. *Dig Dis Sci* 1991;36:435-40.