Histological assessment of the Sydney classification of endoscopic gastritis

S I Khakoo, A J Lobo, N A Shepherd, S P Wilkinson

Abstract

To determine the significance of the endoscopic classification of gastritis proposed by a working party at the World Congress of Gastroenterology in Sydney 1990, 167 patients undergoing upper alimentary endoscopy were prospectively assessed by comprehensive endoscopic and histological methods. Ninety eight patients had endoscopic mucosal changes of gastritis according to the Sydney classification. Twenty six (27%) of these had histologically normal biopsy specimens. This was not statistically significantly different to the 26 (38%) of 69 with normal endoscopies whose biopsy specimens were histologically normal ($\chi^2 = 1.857$, p>0.1). Forty three (62.5%) patients with normal endoscopies had histological gastritis. No histological counterpart was found for the macroscopic appearances of the gastric mucosa said to show inflammation proposed by the Sydney classification of gastritis. These findings confirm the inappropriate-ness of an endoscopic diagnosis of gastritis and it is suggested such a term should be reserved for the histological findings.

(Gut 1994; 35: 1172–1175)

Up to 50% of the asymptomatic population may have histological evidence of gastritis and 45% of patients with dyspepsia have endoscopic appearances interpreted as gastritis. It has been suggested that the frequency of these mucosal abnormalities are under reported. Several endoscopic criteria said to point to gastritis have been suggested, with varying correlation to histological abnormalities. These include mucosal erythema, superficial breaks in the mucosa ("erosions"), and visible gastric vasculature. Histological lymphocytic gastritis has been suggested to correlate well with a distinct form of endoscopic abnormality, namely varioliform gastritis. Conversely it is well recognised that endoscopy may commonly be normal in the presence of histological gastritis. The heterogeneity of published works on gastritis may be because of inconsistencies in the classification of the appearance of mucosal abnormalities, and to a paucity of biopsy material obtained at endoscopy.

Methods

Two endoscopists participated in the study. One (SIK), a trainee in gastroenterology who had performed about 100 procedures himself carried out the assessment under the supervision of a consultant (SPW) with 20 years experience. The other (AJL) was a senior registrar in gastroenterology, who had performed more than 1500 procedures unsupervised. Any patient endoscoped by the above was eligible for inclusion, and no further selection criteria were invoked. All patients were questioned about the presence of dyspepsia, smoking and drinking habits, non-steroidal anti-inflammatory drugs (NSAIDs) and ulcer healing drug use, previous ulcer surgery, and previous endoscopy. An Olympus fibreoptic endoscope system was used, and standard 7 FG sized biopsy forces were used to take biopsy specimens from the following sites as described by the Sydney system: two each from the anterior and posterior antrum, 2–5 cm from the pylorus; two each from the anterior and posterior body, 10 cm from the cardia; and two from any additional area of abnormality. A further antral biopsy specimen was taken for assessment for Helicobacter pylori by the urease test, Gram staining, and culture.

All endoscopies were reported according to the criteria of endoscopic gastritis described by the Sydney classification, which includes a subjective assessment of severity as mild, moderate or severe, and then classified into one of the following eight categories: erythematous/exudative gastritis, atrophic gastritis, raised erosive gastritis, flat erosive gastritis, haemorrhagic gastritis, rugal hyperplastic gastritis, enterogastric reflux gastritis, and congestive gastroenteropathy.

The eight biopsy specimens from each patient were prepared for histological assessment according to standard histopathological processes. Optical orientation was ensured at the embedding stage. Five μg paraffin sections were stained with haematoxylin and eosin. The histopathological features were reported according to the Sydney classification by one histopathologist (NAS) without knowledge of erythema, friability, punctate and confluent exudate, flat and raised erosions, rugal hyperplasia and atrophy, visibility of the vascular pattern, punctate and confluent intramural bleeding spots, and fine and coarse nodularity. It also re-categories portal hypertensive gastropathy as congestive gastroenteropathy.

This study was devised to correlate the endoscopic features described by the Sydney system with the histological findings.
TABLE I  Cases with two endoscopic diagnoses

<table>
<thead>
<tr>
<th>Antrum diagnosis</th>
<th>Body diagnosis</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Raised erosive</td>
<td>Erythematous/exudative</td>
<td>4</td>
</tr>
<tr>
<td>Erythematous/exudative</td>
<td>Congestive gastritis</td>
<td>4</td>
</tr>
<tr>
<td>Atrophic</td>
<td>Erythematous/exudative</td>
<td>3</td>
</tr>
<tr>
<td>Erythematous/exudative</td>
<td>Atrophic</td>
<td>2</td>
</tr>
<tr>
<td>Atrophic</td>
<td>Haemorrhagic</td>
<td>1</td>
</tr>
<tr>
<td>Raised erosive</td>
<td>Atrophic</td>
<td>1</td>
</tr>
<tr>
<td>Raised erosive</td>
<td>Haemorrhagic</td>
<td>1</td>
</tr>
</tbody>
</table>

TABLE II  Distribution of endoscopic findings

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Antr frequency</th>
<th>Body frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>75</td>
<td>111</td>
</tr>
<tr>
<td>Erythematous/exudative</td>
<td>61</td>
<td>39</td>
</tr>
<tr>
<td>Atrophic</td>
<td>13</td>
<td>7</td>
</tr>
<tr>
<td>Raised erosive</td>
<td>12</td>
<td>1</td>
</tr>
<tr>
<td>Enterogastric reflux</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Congestive gastritis</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Haemorrhagic</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Flat erosive</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

Results

ENDOSCOPY FINDINGS
In 98 of 167 (58-6%) patients the following endoscopic mucosal changes considered by the Sydney classification to point to gastritis were found: erythematous/exudative gastritis 73 (44%) patients, atrophic gastritis 16 (10%), raised erosive gastritis 12 (7%), enterogastric reflux gastritis five (3%), congestive gastro-enteropathy five (3%), haemorrhagic gastritis two (1-2%), and flat erosive gastritis one (0-6%). The commonest single mucosal abnormality was punctate erythema, which was found in 73 (44%) patients. Sixteen patients had evidence of two different forms of 'endoscopic gastritis' affecting the antrum and body (Table I). Sixty nine (41%) had no gastric mucosal abnormality.

The distribution of endoscopic mucosal change was predominantly antral in 41 (42%) cases or pangastric 50 (51%), being confined to the body in only seven (7%) of cases (Table II).

The severity of the endoscopic mucosal changes was categorised as mild in 66 (67%), moderate in 29 (30%), and severe in only three cases (3%).

There was no significant difference between the populations with and without 'endoscopic gastritis' with respect to age, sex, smoking, the presence of dyspepsia, previous surgery or NSAID use.

HISTOPATHOLOGICAL FINDINGS
One hundred and fifteen (69%) cases had histological gastritis, classified as follows: Helicobacter pylori associated chronic active gastritis 71 (43%), chronic active gastritis with no H pylori detected nine (5%), reactive gastritis 31 (19%), mild to moderate atrophic gastritis 11 (7%), severe, probably autoimmune associated, atrophic gastritis three (2%), and chronic gastritis two (1-2%).

The distribution of histological gastritis was antral in 39 (34%) cases and pangastric in 73 (63%). Histological abnormalities were deemed to be mild in 34 (30%) cases, moderate in 54 (47%), and severe in 27 (23%).

The H pylori associated chronic active gastritis was antral in 13 (18%), pangastric in 57 (81%), and found in the body only in one (1%) patient, who had had an antrectomy. Thirty (42%) of these patients had an endoscopically normal mucosa.

CORRELATION OF ENDOSCOPIC AND HISTOLOGICAL FINDINGS
Of the 98 cases of 'endoscopic gastritis' 26 (27%) had normal histology, and 43 (62%) of patients with normal endoscopies had abnormal histology. There was thus no significant correlation overall between the presence of endoscopic and histological gastritis (\(\chi^2=1-857\) (Yates's correction), \(p>0-1\)).

Of the three most commonly found types of 'endoscopic gastritis', the association with histological gastritis was: erythematous/
exudative gastritis 50 of 73 patients (68%), atrophic gastritis 12 of 16 patients (75%), and raised erosive gastritis 12 of 12 patients (100%). (Figure). The last category was significantly associated with histological gastritis ($\chi^2=4.39$ (Yates’s correction), $p<0.05$), but not any specific type. Other endoscopic categories contained too few cases to draw meaningful conclusions (Table III).

No association was found between the severity of 'endoscopic gastritis' and the presence of histological abnormality ($\chi^2=2.30$, $p>0.1$).

Considering individual endoscopic findings in more detail, the commonest endoscopic abnormality was punctate erythema. This was found in 50 of 167 antrums and 34 of 116 bodies. Of these 94 endoscopically distinct areas, 58 had associated histological gastritis. This gives a 61.7% specificity of this abnormality for histological gastritis. Using the same criteria the presence of prominent lineae gastricae gave a specificity of 57-6% (15 of 26 areas), visible vessels a 66-6% specificity (20 of 30 areas), raised erosions a 100% specificity (13 of 13 areas), confluent erythema 85% (17 of 23 areas), and oedema 46-9% (15 of 32 areas).

Of the 52 patients with normal histology, 26 (50%) had an endoscopic mucosal abnormality that would be classified as 'gastritis' according to the Sydney classification including 23 with 'erythematous/exudative gastritis' (Table III). The patients with and without histological gastritis did not differ significantly with respect to the endoscopic diagnosis or distribution of endoscopic abnormality ($\chi^2=3.42$, $p>0.1$).

**Discussion**

No overall association was found between the endoscopic abnormalities described by the Sydney classification and histological gastritis. Both endoscopists participating in this study were in good agreement with their findings. This lack of association results from two main factors. Endoscopy is an insensitive method for diagnosing gastritis, and in our study despite the use of a comprehensive classification of gastritis we could not increase this sensitivity from the previously reported figures. The endoscopic abnormalities described in the Sydney classification are also not specific for the diagnosis of gastritis, and such abnormalities were often found in the absence of histological changes. Sampling error in cases where histological abnormality might have been patchy, was minimised by taking biopsy specimens from specific areas of endoscopic abnormality in addition to the multiple biopsy specimens from the antrum and body taken as part of the endoscopic protocol.

‘Erythematous/exudative gastritis’, the commonest endoscopic finding had the poorest (60-6%) association with histological gastritis. Erythema has been suggested previously to have a poor association with histological abnormality and it was unlikely to have been over diagnosed by us as a fibroptic endoscope, compared with a video system, was used to reduce machine bias, and the number of normal endoscopies associated
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with abnormal histology in our study was similar, or slightly greater, than has previously been reported.3 5 21 Furthermore, erythema may not be reported consistently by independent observers.6 It may represent a transient vasodilatation of the gastric vasculature.

Apart from patients with end stage renal failure undergoing maintenance haemodialysis, endoscopic mucosal breaks (‘erosions’) have been reported to have a stronger association with histological gastritis than erythema.7 13 Our findings agree with this, as endoscopic ‘raised erosive gastritis’ had a 100% association with histological abnormality. It did not, however, have an association with any specific histological type of gastritis (Table III).

Nodularity of the mucosa has been proposed to show the presence of H pylori in children23 and more recently in adults.24 We found a 76-9% association of nodules for H pylori, not the 100% association reported previously in adults. These mucosal findings, and those categories of ‘endoscopic gastritis’ in which we had only small numbers constitute areas for further investigation.

No type of histological gastritis was specifically associated with a single category of endoscopic abnormality as described by the Sydney classification, although five of seven cases of visible vessels in the body were associated with histological atrophy.

Our study suggests that the endoscopic division of the Sydney classification has not helped to clarify the reporting of gastritis. Cases where nodularity, oedema or prominent lineae gastricae are found in isolation, are difficult to classify according to the Sydney system. The 15 mucosal findings it lists may be well employed as descriptive terms, but other than raised erosions, would not necessarily point to gastritis. Grouping endoscopic abnormalities into different categories of gastritis is misleading as they have no histopathological correlates. Misleading reports may lead to inappropriate treatment, especially with the increasing number of open access endoscopy units where reports must be interpreted by non-gastroenterologists. Endoscopy reports are more meaningful if the endoscopic mucosal findings are accurately described and biopsy specimens taken. We therefore suggest that the term ‘gastritis’ is reserved for histological abnormality.

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