Duodenitis: A Clinical, Endoscopic, Acid Secretory and Histological Study in Patients with Non-Ulcer Dyspepsia

S P ATMAKURI, M MATHUR, R K TANDON

Departments of Gastroenterology and Pathology, All India Institute of Medical Sciences, New Delhi 110 029

Abstract

Forty-two patients with non-ulcer dyspepsia were prospectively studied endoscopically and histologically for the presence of duodenitis. Endoscopic abnormalities compatible with duodenitis were seen in 21 of them (mild in 16, marked in 5). Histologically, the duodenal biopsies from these patients were graded as 1+ (normal) in 26, 2+ (definitely abnormal) in 12 and 3+ (markedly abnormal) in four. Although correlation between the specific endoscopic and histological features of duodenitis was poor, the overall matching between degrees of severity by endoscopy and histology was excellent (p< 0.001). Acid secretory status in the various groups of patients was however not discriminatory. 'Ulcer like' symptoms were rare in patients with duodenitis and therefore, when present, should raise suspicion of peptic ulcer disease.

Key words: Duodenitis, peptic ulcer, dyspepsia, endoscopy.

Introduction

Non-specific duodenitis is a condition of unknown cause characterised by inflammation involving the mucosa of the first part of the duodenum without evidence of an active duodenal ulcer. Its association with dyspeptic symptoms, as well as its place in the evolving or healing phases of duodenal ulceration, is however not clear.13 There is a poor correlation between the endoscopic and histopathological features of duodenitis and hence the criteria for its diagnosis remain controversial.69 Furthermore, the pathogenesis of duodenitis is uncertain; unlike in duodenal ulcer, gastric acid hypersecretion has not been demonstrated in patients with duodenitis.69

We, therefore, undertook the present study to correlate endoscopic and histological features of duodenitis in patients with dyspepsia, and to define the profile of the gastric acid secretory status of these patients.

Material and Methods

Patients presenting to the Gastroenterology Clinic with symptoms referable to the upper gastrointestinal tract excluding the oesophagus were studied. Patients with organic diseases, like duodenal ulcer, gastric ulcer, gastric carcinoma and reflux oesophagitis by endoscopic or radiological investigations done earlier or at the present, were excluded. Those with evidence of giardiasis or hookworm infestation were also excluded.

The patients were divided into two groups depending on their symptoms.1 The symptoms in Group I were 'ulcer-type', viz., post-prandial epigastric pain (>1 h), periodicity of pain, relief of pain with food or antacids, nocturnal pain, history of gastrointestinal bleeding, perforation or obstruction, epigastric tenderness and a family history of peptic ulcer. Group II patients had the following symptoms: non-localised upper abdominal pain related to or unrelated to meals, non-periodic pain, abdominal fullness, nausea, belching, bloating, inability to finish meals because of discomfort, vomiting and diffuse upper abdominal tenderness. The presence of two or more of Group I symptoms would categorise the patient as 'ulcer-type'. Otherwise the patients would be categorised as non-ulcer type.

Upper gastrointestinal endoscopy was performed with an Olympus GIF-Q endoscope by an independent investigator who was unaware of the patients' symptoms and who was not involved in the histological interpretation of the biopsies obtained. The endoscopic features of duodenitis looked for were nodularity, erosions, contact bleeding, haemorrhages, atrophy, mammillations and hypertrophic folds. Three to four biopsies were taken from various parts of the duodenal bulb, including the normal areas when present.

The histological features were evaluated on the basis of classifications reported earlier.11 12 In particular the biopsies were analysed for: (a) the intensity of round cell infiltrate of the lamina propria and its extension to the Brunner's glands; (b) retention of villous morphology or its complete atrophy; (c) invasion of the epithelium by neutrophils; and (d) erosive and regenerative epithelial changes.

The biopsies were, however, classified chiefly on the basis of the chronic inflammatory infiltrate; presence of a mild uniform infiltrate was graded as 1+ (Fig 1), a moderate increase causing a wider than normal separation of the glandular elements as 2+, and a marked increase, densely involving the lamina propria and submucosa with destruction of villous morphology, as 3+ (Fig 2). Focal aggregates were ignored.

Basal and peak acid outputs were measured by the augmented histamine test.13

Results

Of the 42 patients with non-ulcer dyspepsia (NUD) studied, 29 were male and 13 female, aged 18-54 years.
Of 42 study subjects, 38 had non-ulcer dyspeptic symptoms (Group II) and only 4 patients had ulcer-like symptoms (Group I).

Endoscopy

Twenty-one patients had entirely normal endoscopy. The findings in the remaining 21 patients were as follows. Mild abnormalities (16 patients) included mild nodularity (9), atrophic mucosa (9), hypertrophic folds (1) and nodularity and petechiae (1). Marked abnormalities (5 patients) included severe nodularity, erosions, hyperaemia, friability (2), congestion and diffuse mamilations (1), diffuse salt and pepper erosions (1), and marked hyperaemia with exudation (1).

Histology

Biopsies of 26 cases were categorised as 1+, 12 cases as 2+, and 4 cases as 3+. In the last group, extension of inflammatory infiltrate to the Brunner’s glands was also seen, villous epithelial changes in terms of regeneration, degeneration, erosions or gross morphologic changes could not be assessed because of marked artifacts present in the biopsies. Congestion of blood vessels was seen in almost all biopsies.

Association between specific endoscopic findings and histology

1. Nodularity: Of nine patients with this finding, the mucosal changes were classified as 1+ in four and 2+ in five. A significant extension of the inflammatory cells into the epithelial layer was seen in four cases. No correlation was thus possible.

2. Atrophic mucosa (defined as a thin mucosa through which the vascular pattern could be easily made out): Of five patients with this finding, the grading by histology was 1+ in three, 2+ in one and 3+ in one. Although deformed or desquamated, the villous structure could be identified in some parts of the biopsy.

The overall association is shown in the Table. A matching of grades was found in 26 of 42 patients (62%). There was an over-estimation of one grade by endoscopy in 11 patients (25-2%) and by histology in five patients (11.9%). The overall matching was found to be highly significant ($p<0.001$), using the chi square test.

<table>
<thead>
<tr>
<th>Endoscopy</th>
<th>Histology</th>
<th>++</th>
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<th>Total</th>
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<td>4</td>
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<td>21</td>
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<tr>
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<td>9</td>
<td>6</td>
<td>1</td>
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<td>16</td>
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<tr>
<td>Markedly abnormal</td>
<td>2</td>
<td>3</td>
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<td>5</td>
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<tr>
<td><strong>Total</strong></td>
<td>26</td>
<td>12</td>
<td>4</td>
<td>5</td>
<td>42</td>
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$X^2=21.3; p<0.001$

Acid secretory studies

AHT was performed on 30 study patients. The mean peak acid output (PAO) in them was 18-9 mEq/h. On grouping the PAO data according to endoscopic abnormalities, however, it was observed that the mean PAO in those with normal endoscopy (n=14) was 18.23 mEq/h, in those with nodularity (n=7) 19-32 mEq/h, in those with atrophy (n=4) 20-02 mEq/h, and in those with erosions (n=3) 21-3 mEq/h. The differences were not significant.

The mean PAO values in the 3 groups of histological severity were as follows: 1+ histology (n=18) 18-11 mEq/h; 2+ histology (n=8) 20-49 mEq/h; 3+ histology (n=2) 14-30 mEq/h. The differences were not significant.

Discussion

Much controversy exists as regards the association between duodenitis and duodenal ulcer. Some authors believe that duodenitis identified by endoscopy is the forerunner of a duodenal ulcer. Peptic ulceration has been suggested to be a symptomatic syndrome—a hypothetical Muyinbah’s disease—and symptoms suggestive of duodenal ulcer disease identify patients with both duodenitis and duodenal ulcer.67,15 These observations are, however, not corroborated by others who...
have found a poor correlation between duodenal ulcer and duodenitis.\(^1\)\(^,\)\(^2\)

One-half of our NUD patients had one or the other abnormal finding at endoscopy. This is a high percentage compared to Western investigators' figures for abnormal endoscopies in dyspeptic patients.\(^2\)\(^,\)\(^3\) However, Naik et al\(^7\) in a study conducted in the same geographical area as ours found endoscopic abnormalities in 20 of 25 dyspeptic patients.

Histological assessment was difficult because of a number of artifacts induced by the punch biopsy technique. Classification based on the density of chronic inflammatory cells was subjective and arbitrary. Quantitative methods\(^8\) would probably be the only way of obviating this problem. It would also be necessary to interpret biopsies against the findings in healthy volunteer controls, to establish criteria for 'abnormal.' A certain degree of 'chronic inflammation' is 'normal' in comparison with animals grown in germ-free environment, perhaps as a result of various dietary and other luminal irritants.\(^9\)

The absence of correlation between the specific endoscopic and histological features in our patients was surprising since most other authors have found a fair correlation between the two, especially when visually abnormal areas of the duodenum were biopsied.\(^2\)\(^,\)\(^3\)\(^,\)\(^6\)\(^,\)\(^7\) Even in biopsies taken from the apparently atrophic mucosa in our patients, the villous structure could be identified in some part of the biopsy. This would imply that such endoscopic appearance may not be pathologically significant. The overall correlation between the grades of abnormality was, however, excellent (p<0.0001). Thus in the severe cases of duodenitis, which are probably the significant ones, one could possibly rely on endoscopy alone for the assessment of duodenitis.

One major assumption in the study was that there existed a group of patients who, in spite of having symptoms suggestive of an ulcer (ulcer-like symptoms), did not have ulcer.\(^2\) However, only four such patients could be identified in our group of 42 patients. Possibly most patients with ulcer-like symptoms had ulcers and were thus excluded from the present study.

Gastric acid secretory studies failed to discriminate between the groups, although the size of some subgroups was too small to derive such a conclusion confidently.

In conclusion, we found endoscopic abnormalities in 21 of 42 patients with non-ulcer dyspepsia. Though the overall correlation between the grades of severity on endoscopy and histology was excellent, there was a poor correlation in terms of specific features like nodularity and atrophy. Screening patients for endoscopy on the basis of ulcer-like symptoms is probably most rewarding for diagnosing peptic ulcer disease.

References