Investigations Proposed to Accurately Classify Chronic Gastritis

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Abstract

Patients of chronic gastritis should be investigated with gastric mucosal biopsy, parietal cell antibody, intrinsic factor antibody, *Helicobacter pylori* antibody, urea breath test or faecal antigen test for *Helicobacter pylori*, to accurately classify them. The results of these tests will indicate *Helicobacter pylori* infection (present or past), the role of hereditary factor (intrinsic factor antibody present or absent) and the success or failure of *Helicobacter pylori* eradication treatment.

**INTRODUCTION**

An accurate classification of chronic gastritis (CG) into different subgroups is desirable to understand its aetiopathogenesis, to compare data from different centers, to study the life-history, to suggest surveillance programme (if any), to plan treatment (in future), to prevent its progress or complications (gastric carcinoma, lymphoma), with a view to improve or cure this disease.1-3 Two major advances in our understanding of CG are (a) detection of immunological parameters-parietal cell antibody (PCA) (1962),4 intrinsic factor antibody (IFA) (1963)5 and (b) discovery of *Helicobacter pylori* (*H. pylori*) in endoscopic gastric mucosal biopsy (1983).6 The commonest cause of CG (more than 80%) is *H. pylori* infection; it affects about half the world population.3,7 Uncommon causes of CG - granulomatous, eosinophilic, lymphocytic, reflux (postoperative), corrosive, radiation-induced, Crohn’s disease, sarcoidosis, tobacco-induced8 - are not discussed.

**INVESTIGATIONS**

Five investigations are required to accurately classify subgroups of patients with CG and the significance of each of them are shown in Table 1.

Gastric mucosal biopsy on upper gastrointestinal endoscopy is preferred and at least five biopsies are obtained – two each from antrum and body mucosa and one from incisura angularis. The diagnosis of CG is established as superficial gastritis, atrophic gastritis or gastric atrophy (pernicious anaemia : PA). The aetiology of *H. pylori* infection is indicated by presence of lymphoid follicles and/or polymorphonuclear leucocyte infiltration.9 *H. pylori* can be detected on rapid urease test (RUT) or identified near surface epithelium with routine haematoxylin-eosin stain and with greater ease on special stains (modified Giemsa, acridine orange, Warthin-Starry). To diagnose *H. pylori* infection in gastric mucosa, RUT, faecal antigen test (FAT),10 urea breath test (UBT)11 have a sensitivity of approximately 85%, 90%, 95% respectively.12 Culture of *H. pylori* in gastric mucosal biopsy or stool, helps to assess bacterial sensitivity to different antibiotics.

PCA detected on complement fixation test is present in serum or gastric juice.4 The antibody is formed against the antigen-alpha and beta subunits of proton pump.13 Its presence indicates CG even in healthy control subjects; its absence does not exclude CG (e.g. post-operative or corrosive).1 PCA in serum is present in 5-15% of control population, 20% with iron deficiency anaemia, 25% with diabetes mellitus, 30% with thyrotoxicosis, 35% with idiopathic Addison’s disease;14,15 the presence of PCA indicates the prevalence of CG in these diseases.

IFA detected on radioimmunoassay is of 2 types.5

**Table 1 : Investigations in a patient of chronic gastritis (CG)**

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Significance</th>
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<tbody>
<tr>
<td>Gastric mucosal biopsy</td>
<td>for diagnosis of CG and indicates aetiology</td>
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<tr>
<td>Parietal cell antibody (serum)</td>
<td>presence indicates chronic gastritis</td>
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<tr>
<td>Intrinsic factor antibody*</td>
<td>presence indicates pernicious anaemia</td>
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<tr>
<td><em>H. pylori</em> antibody (IgG)</td>
<td>indicates past <em>H. pylori</em> infection</td>
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<tr>
<td>Urea breath test (or)</td>
<td>confirms eradication or presence of *H. pylori</td>
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<tr>
<td><em>H. pylori</em> faecal antigen test*</td>
<td>confirms eradication or presence of *H. pylori</td>
</tr>
</tbody>
</table>

+ = serum or gastric juice, * after eradication treatment of *H. pylori* in peptic ulcer
Type I: Blocking antibody blocks the union of IF to free vitamin B₁₂; Type II: Binding antibody interferes with the binding of IF-vitamin B₁₂ complex to ileal receptors. In serum, Type II IFA is found only in patients with Type I antibody and hence the latter is only looked for. In Western countries, the incidence of IFA in serum of patients with PA, thyrotoxicosis, diabetes mellitus, iron deficiency anaemia is 75%, 7%, 5%, 2% respectively. The rarity of IFA in Indian patients, including those with severe atrophic gastritis and histamine fast achlorhydria (HFA) with severe vitamin B₁₂ malabsorption, was emphasized. Formation of IFA is independent of sex, age, duration of disease, degree of atrophic gastritis or the titre of PCA and is determined by hereditary factors. In relatives of patients with PA, the prevalence of IFA is higher than in control population. The diagnosis of PA should be restricted to only these patients with IFA in serum or gastric juice.

Helicobacter pylori antibodies (HpA) (IgG) indicate past H. pylori infection. HpA crossreact with gastric autoantigens such as alpha and beta subunits of proton pump and determines the progress and localization (body or antrum) of gastric mucosal damage and explains the pathogenic link between H. pylori infection and CG. In patients with corporal atrophic gastritis, the incidence of positive serum HpA and negative tissue staining of H. pylori is present in more than 50% of patients, indicating H. pylori disappears as intestinal metaplasia develops in them.

UBT can be performed with ¹⁴C or ¹³C urea meal test;¹¹ ¹⁴CO₂ liberated by H. pylori is absorbed and exhaled in breath and is measured with liquid scintillation. The test should not be performed in pregnant women and children. ¹³CO₂ is measured with mass spectrometry. UBT also provides some useful quantitative estimation of H. pylori infection in the whole stomach. One month after completion of H. pylori eradication treatment, the negative UBT confirms its eradication.

FAT for H. pylori is another alternative method to confirm the absence or presence of H. pylori in the gastric mucosa;¹⁰ in children, this test is preferred. To confirm eradication of H. pylori in patients of duodenal ulcer (associated with antral gastritis) or gastric ulcer (associated with corpus gastritis), UBT or FAT is absolutely necessary, as sensitivity of histology or RUT on gastric mucosal biopsy is poor, when few organisms are present in the gastric mucosa.

To conclude, the result of these five tests (Table 1) will indicate present or past H. pylori infection, the contribution of hereditary factors (IFA present or absent), and the success or failure of H. pylori eradication treatment. These investigations should be available in institutions desiring to diagnose and classify patients of CG accurately. Future progress of CG depends on the availability of such information from various countries.
Crosby- Kugler capsule), augmented histamine test to detect hypochlorhydria or HFA, $^{58}$Co vitamin B$_{12}$ excretion (Schilling test) to detect mild (>5% excretion) or severe (<5% excretion) vitamin B$_{12}$ malabsorption, to separate patients of atrophic gastritis from those with PA respectively (Fig. 1). At present, patients of CG should be investigated with endoscopic gastric mucosal biopsy (histology + RUT) and immunological parameters (PCA, IFA, HpA) (Table 1).

REFERENCES


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**Announcement**

**APICON 2008 Kochi**

**Venue :** Le Meridien Hotel and International Convention Centre, Maradu, N H Bypass, Kochi.

**Dates:** 10th to 13th January 2008

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