

## The Classification of Chronic Gastritis

Chronic gastritis (CG), described more than a century ago,<sup>1</sup> is now a known precursor of gastric carcinoma. Besides the non-erosive, non-specific forms, the term includes infective, eosinophilic, hypertrophic and granulomatous gastritis. While the specific chronic gastritides have distinct features and treatment, the non-erosive, non-specific types pose problems in management. An accurate classification of CG would help to understand its aetiopathogenesis, to differentiate it from other similar conditions, to enable comparison of data from different centres and to devise effective treatment. At least eight different classifications<sup>1-8</sup> of CG have been proposed based on its clinical features, pathogenesis and endoscopic and histological changes. Most of these classifications<sup>4-8</sup> are inadequate as they ignore the immunological changes which are important in its aetiology.

Twenty years ago, we<sup>1</sup> and others<sup>2</sup> proposed a classification of CG, emphasizing the immunological changes associated with the condition. We divided CG into three types:

Type I: in which parietal cell (PCA) and intrinsic factor antibodies (IFA) are absent (e.g. postoperative or corrosive gastritis).

Type II: in which PCAs are present but IFAs are absent. This form involves the antrum with extension to the body and fundus of the stomach.

Type III: in which PCAs and IFAs are present. This includes pernicious anaemia (PA), in which the fundus and body are involved but the antrum is spared.

Using this classification, PA can be differentiated from severe CG with achlorhydria and vitamin B<sub>12</sub> malabsorption.<sup>9</sup>

The widely used classification of Strickland and Mackay<sup>2</sup> divides CG into two types: Type A (autoimmune) and Type B (environmental; later attributed to infection with *Helicobacter pylori*). Type A CG affects the fundus and body with sparing of the antrum. Histamine-fast achlorhydria, hypergastrinaemia and severe vitamin B<sub>12</sub> malabsorption are present. PCAs are present in the sera of 95% of the patients and IFAs in 75%. Type A gastritis is one-fourth as common as Type B.

Type B CG affects the antrum initially with extension into the body and gastric haemorrhage, ulcer and carcinoma are prominent sequelae. Strickland and Mackay,<sup>2</sup> in order to emphasize the differences between the two types, reported the absence of PCAs in Type B CG. Subsequently, they realized their error and reported the incidence of PCAs to be 60% in Type B gastritis.<sup>10</sup>

Glass and Pitchumoni<sup>3</sup> proposed a Type AB negative (PCAs absent) and Type AB positive (PCAs present), in addition to the classical Type A and Type B.

Correa<sup>4</sup> divided CG into the autoimmune, environmental and hypersecretory types; the last also known as antral gastritis is seen in patients with duodenal ulcer. Correa<sup>5</sup> then re-classified CG on a morphological and aetiological basis. The morphological classification included the atrophic and non-atrophic forms, further subdivided into superficial, diffuse antral, diffuse corporal and multifocal atrophic. Multifocal atrophic gastritis is associated with intestinal metaplasia and gastric carcinoma.

Whitehead<sup>6</sup> classified CG according to (i) the site of mucosa involved: pyloric, body, cardiac, transitional, indeterminate; (ii) the degree of gastritis: superficial or atrophic; and (iii) the presence of metaplasia: pseudopyloric or intestinal.

Siurala *et al.*<sup>7</sup> classified CG based on the rate of its progress; gastritis in the body of the stomach progressed rapidly in PA but slowly in other forms. CG was graded as:

Score 0: Normal mucosa with no round cell infiltration or loss of glands

Score 1: Superficial gastritis; round cell infiltration and no loss of glands—slight, moderate, severe

Score 2: Atrophic gastritis with slight loss of glands

Score 3: Atrophic gastritis with moderate loss of glands

Score 4: Atrophic gastritis with severe loss of glands

A simple and comprehensive classification based on histological and endoscopic observations was reported by the working party on gastritis in Sydney in 1990.<sup>8</sup> The histological findings are suffixed by the morphological characteristics and prefixed by the aetiology.

Rubin<sup>11</sup> suggested that a close cooperation between clinicians and pathologists would make the classification of CG meaningful. However, clinical and endoscopic diagnoses show a poor correlation with histology and hence any classification including these will be confusing.

The major breakthrough in our understanding of CG followed the discovery of *Helicobacter pylori* (HP) in 1983.<sup>12</sup> HP-related damage to the gastric mucosa is an immunological phenomenon as HP antibodies cross-react with parietal cell antigen.<sup>13</sup> Immune-mediated damage is important for the localization as well as the progression of CG. As HP antibodies cross-react with the parietal cell antigen, the changes are restricted to the fundus and body of the stomach which contain these cells. The antrum is spared. In patients with PA, the prevalence of HP in the gastric mucosa is lower than in a control population but the prevalence of HP antibodies in the sera is high, indicating past exposure and clearance of HP from the gastric mucosa, perhaps over decades.<sup>14</sup> A genetic predisposition determines the development of IFA in patients with CG.<sup>15</sup> This explains the rarity of IFA and PA in Indian patients, despite the wide prevalence of CG.<sup>16</sup>

Rapid developments in immunology have emphasized the role of immune mechanisms in the pathogenesis of diseases such as acute and chronic hepatitis, glomerulonephritis, arthritis and thyroiditis and their classification is based on immunological responses. Any attempt at classifying CG without including immunological criteria will invariably be inaccurate. It is rather strange that what was obvious to some<sup>1-3,9</sup> of us decades ago is still not generally acceptable to the majority of experts classifying CG.<sup>4-8</sup>

## REFERENCES

- Desai HG, Antia FP. A second look at chronic gastritis: A new classification. *Indian J Med Sci* 1973;27:217-24.
- Strickland RG, Mackay IR. A reappraisal of the nature and significance of chronic atrophic gastritis. *Am J Dig Dis* 1973;18:426-40.
- Jerzy-Glass GB, Pitchumoni CS. Structural and ultrastructural alterations, exfoliative cytology and enzyme cytochemistry and histochemistry, proliferation kinetics, immunological derangements and other causes, and clinical associations and sequelae. *Hum Pathol* 1975;6:219-50.
- Correa P. The epidemiology and pathogenesis of chronic gastritis: Three etiologic entities. *Frontiers Gastrointest Res* 1980;6:98-108.
- Correa P. Chronic gastritis: A clinico-pathological classification. *Am J Gastroenterol* 1988;83:504-9.
- Whitehead R. *Mucosal biopsy of the gastrointestinal tract*. Philadelphia:Saunders, 1985.
- Siurala M, Sipponen P, Kekki M. Chronic gastritis: Dynamic and clinical aspects. *Scand J Gastroenterol (Suppl)* 1985;109:69-76.
- Misiewicz JJ, Tytgat GNJ, Goodwin CS, Price A, Sipponen P, Strickland R. *The Sydney system. A new classification of gastritis, working party report*. Sydney: Ninth World Congress of Gastroenterology, 1990:1-10.
- Desai HG. Definition of pernicious anaemia. *Indian J Med Sci* 1968;22:477-9.
- Strickland RG. Gastritis. In: Van der Reis L (ed). *Immune disorders*. Basel:Karger, 1975:12-18.
- Rubin CE. Histological classification of chronic gastritis: An iconoclastic view. *Gastroenterology* 1992;102:360-1.
- Warren JR, Marshall B. Unidentified curved bacilli on gastric epithelium in active chronic gastritis. *Lancet* 1983;1:1273-5.
- Negrini R, Lisato L, Zanella I, Cavazzini L, Gullini S, Villanacci V, et al. *Helicobacter pylori* infection induces antibodies cross-reacting with human gastric mucosa. *Gastroenterology* 1991;101:437-45.
- Fong TL, Dooley CP, Dehesa M, Cohen H, Carmel R, Fitzgibbons PL, et al. *Helicobacter pylori* infection in pernicious anaemia. A prospective controlled study. *Gastroenterology* 1991;100:328-32.
- Desai HG, Dighe PK, Borkar AV. Parietal cell and intrinsic-factor antibodies in Indian subjects. *Scand J Gastroenterol* 1968;3:321-6.
- Desai HG, Antia FP. Vitamin B<sub>12</sub> malabsorption due to intrinsic factor deficiency in Indian subjects. *Blood* 1972;40:747-53.

D. N. AMARAPURKAR

H. G. DESAI

B. Y. L. Nair Charitable Hospital  
Bombay  
Maharashtra