Editorial

IDIOPATHIC CHRONIC GASTRITIS

The association of chronic inflammation of the gastric mucosa with other diseases as iron deficiency anaemia, or pernicious anaemia is well known. A similar lesion with an independent involvement of the fundus and the body of the stomach has been called chronic idiopathic gastritis. The cause has been speculated as being traumatic in origin or related to tobacco and alcohol. Recently, thought has been given to it being an autoimmune disease.

With the introduction of the fibreoptic gastroscope the diagnosis of the condition has become easy and accurate. Every corner of the mucosa can be viewed along-with biopsies which can be taken from different points giving a final diagnosis. Roentgenography is helpful only in very advanced cases (Kirkpatrick et al., 1973). Serological examination is valuable as parietal cell antibodies indicate chronic gastritis. Other organ specific or non-organ specific antibodies present, give a clue to an associated autoimmune disease.

The etiology of chronic gastritis is still debatable. Minor trauma resulting from drinking hot tea (Edwards and Edwards, 1956) or taking hot food (Ivy 1955) has been attributed to be (Joske et al., 1955; Roberts, 1972) the causative factor. Chronic alcoholics have been observed by some workers to be suffering from chronic gastritis. Smoking and chronic infections as tuberculosis and syphilis have been considered to be a cause of this condition (Edwards and Coghill, 1966; Jefferies, 1973). Malnutrition especially of proteins and vitamins giving rise to glossitis and stomatis, in a similar manner may lead to chronic inflammation of the gastric mucosa (Wood and Taft, 1958).

The barrier to absorption of hydrogen ions from the lumen into the mucosa may be broken by drugs as aspirin, hypochlorhydria associated with fundal gastric ulcers, and pernicious anaemia. This situation leads to the manifestation of chronic gastritis (Davenport 1964, 1965).

An increased gastrin secretion, which may be secondary to hypochlorhydria, reflux of bile into the antrum or reduced secretion of cholecystokinin and pancreozymin gives rise to chronic gastritis (Gillespie 1974; Grossman 1970).

Emotional stress and psychological factors causing gastric erosions are contributory factors to chronic gastritis (Wood and Taft, 1958).

The autoimmune nature of chronic gastritis is still under consideration. Evidence of gastric auto-antibody formation has been found by some workers. Round cell infiltration and glandular damage has been reported suggesting the auto-immune phenomenon.

The treatment of chronic gastritis may be prophylactic as not taking drugs on an empty stomach especially those which alter the mucosal barrier to hydrogen ions. Cigarettes, tea and coffee causing bile reflux into the stomach should be avoided. Curative therapy is correction of the cause of ion deficiency anaemia, pernicious anaemia or any local cause affecting the pyloric motility.

Induced chronic gastritis produced by immunization against gastric mucosal antigen has been tried in duodenal ulcer patients. The aim is to produce hypochlorhydria by biochemical damage to the parietal cells.

With the auto-immune factor in view, a supportive treatment along-with immuno-suppressive drugs may bring a considerable advancement in the management of this clinical condition (Jorge and Sanchez, 1973).

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