Definition

Definition of gastritis has its basis in histological features of the gastric mucosa. It is not erythema observed during gastroscopy, and there are no specific clinical presentations or symptoms defining it. The current classification of gastritis centers on time course (acute versus chronic), histological features, anatomic distribution, and underlying pathological mechanisms.

Epidermiology

Socioeconomic and environmental hygiene are the essential factors in the transmission of H. pylori infection worldwide. These factors include family-bound hygiene, household density, and cooking habits. The pediatric origin of H. pylori infection is currently considered the primary determinant of H. pylori-associated gastritis in a community

Pathophysiology

H.pylori-associated gastritis transmission is via the fecal-oral route. H. pylori possess several virulence factors which facilitate cell adhesion (e.g., BabA/B, sabA, OipA), cell damage and disruption of tight junctions (e.g., Ure A/B), and evasion from the immune response (e.g. LPS). In particular, the cytotoxin-associated gene a (CagA) is considered a potent inducer of inflammation and correlate with gastric cancer development.[12]
Another factor influencing H. pylori pathogenic effects is host factors. The host susceptible factors such as polymorphism in genes coding for tall receptors or specific cytokines. The infection with H. pylori triggers IL-8, which attracts neutrophils which release oxyradicals leading to cell damages. Lymphocyte infiltration is also present in H. pylori infection.
Chronic gastritis mostly results from H. pylori infection and appears either as non-atrophic or atrophic form. These two forms are phenotypes of gastritis at different stages of the same life-long disease.[13]
The progression from acute to chronic gastritis begins in childhood as a simple chronic superficial mononuclear inflammation of gastric mucosa which progress in years or decades to atrophic gastritis characterized by loss of normal mucosal glands in the antrum, corpus, fundus or all.
Factors that determine progression to atrophic gastritis and sequelae such as a peptic ulcer or gastric cancer are not clearly understood and unpredictable.

Pathophysiology (cont)

However, Epstein-Barr virus (EBV) and human cytomegalovirus (HCMV) have been identified in gastric tumors and DNA from H. pylori, EBV, and PCR determined the presence of HCMV in biopsies from patients with gastric cancer complicating chronic gastritis.[14] Some researchers have confirmed the involvement of EBV and H. pylori in the development of gastric cancer in patients with chronic gastritis. They found no role for human papillomavirus (HPV) in gastric tumorigenesis.[15]. NSAIDs cause gastritis through inhibition of prostaglandin synthesis. Prostaglandins are responsible for the maintenance of protective mechanisms of gastric mucosa from injuries caused by hydrochloric acid.
The pathogenesis of autoimmune gastritis focuses on two theories. According to the first theory, an immune response against superimposed H. pylori antigen gets triggered, antigen cross-reacting with antigens within the proton-pump protein or the intrinsic factor, leading to a cascade of cellular changes and causing damages to the parietal cells and stopping hydrochloric acid secretion and thus these cells gradually become atrophic and not functioning. The second theory assumes that the autoimmune disorder develops irrespective of H. pylori infection, and it directs itself against the proteins of the proton-pump. As per both theories, the autoimmune gastritis is the result of a complex interaction between genetic susceptibility and environmental factors resulting in immunological dysregulation involving sensitized T lymphocytes and autoantibodies directed against parietal cells and the intrinsic factor.

Histopathology

Histologically, gastritis definitively demonstrates by the presence of at least grade 2 neutrophils or mononuclear cells in at least one gastric biopsy site or grade 1 neutrophils or mononuclear cells in at least two sites.[17] Sampling comes from five gastric biopsy specimens from the following locations: antrum greater and lesser curvature, incisura, and corpus greater and lesser curvature. Specimens must be put into separate vials and grouped for each site of the lesion. The aim is to maximize the opportunity to identify H. pylori and hence not to miss the diagnosis..

History

There are no typical clinical manifestations of gastritis. Sudden onset of epigastric pain, nausea, and vomiting have been described to accompany acute gastritis. Many people are asymptomatic or develop minimal dyspeptic symptoms. If not treated the picture may evolve to chronic gastritis. History of smoking, consumption of alcohol, intake of NSAIDs or steroids, allergies, radiotherapy or gall bladder disorders should all be considerations. A history of treatment for inflammatory bowel disease, vasculitic disorders, or eosinophilic gastrointestinal disorders might require exploration if no cause of gastritis is apparent.. The most common initial findings for chronic and autoimmune gastritis are (1) hematological disorders such as anemia (iron-deficiency) detected on routine check-up, (2) positive histological examination of gastric biopsies, (3) clinical suspect based on the presence of other autoimmune disorders, neurological symptoms (related to vitamin B12 deficiency) or positive family history.[19] Iron-deficiency anemia (based on blood film showing microscopic hypochromic changes as well as iron studies) commonly presents in the early stages of autoimmune gastritis. Achlorhydria causing impairment of iron absorption in the duodenum and early jejunum is the main cause.[20] Iron-deficiency anemia could also occur in other types of chronic gastritis..

Investigations

The diagnosis of gastritis has its basis in histopathological examination of gastric biopsy tissues. While medical history and laboratory tests are helpful, endoscopy and biopsy is the gold standard in making the diagnosis, identifying its distribution, severity, and cause.

Treatment

Treatment regimens differ from antibiotics (in H. pylori gastritis) to vitamin supplementation (in autoimmune metaplastic atrophic gastritis) to immunomodulatory therapy (in autoimmune enteropathy) to dietary modifications (in eosinophilic gastritis).

Complications

eptic ulcer
Chronic atrophic gastritis (loss of appropriate glands resulting mainly from long-standing H. pylori infection)
Gastric metaplasia/dysplasia
Gastric cancer (adenocarcinoma)
Iron-deficiency anemia (chronic gastritis and early stages of gastric autoimmunity)
Vitamin B12 deficiency (autoimmune gastritis)
Gastric bleeding
Achlorhydria (autoimmune gastritis, chronic gastritis)
Gastric perforation
Mucosa-associated lymphoid tissue (MALT) lymphoma
Neuroendocrine tumors (NET) (previously referred to as gastric carcinoid; complicates autoimmune gastritis)