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MINIREVIEWS

History of chronic gastritis: How our perceptions have changed

Dmitry Bordin, Maria Livzan

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Abstract

Currently, the diagnostic strategy for chronic gastritis (CG) is aimed not just at fixing the presence of gastric mucosal inflammation, but also at gastric cancer (GC) risk stratification in a particular patient. Modern classification approach with the definition of the stage of gastritis determines the need, activities and frequency of dynamic monitoring of a patient. However, this attitude to the patient suffering from CG was far from always. The present publication is a literature review describing the key milestones in the history of CG research, from the description of the first observations of inflammation of the gastric mucosa, assessment of gastritis as a predominantly functional disease, to the advent of endoscopy of the upper digestive tract and diagnostic gastric biopsy, assessment of the role of *Helicobacter pylori* infection in progression of inflammatory changes to atrophy, intestinal metaplasia, dysplasia and GC.

Key Words: Chronic gastritis; Intestinal metaplasia; Dysplasia; Gastric cancer; *Helicobacter pylori*

Core Tip: For more than a century, physicians have noted the relationship of chronic gastritis (CG) with the development of gastric cancer, which prompted great interest in the study and systematization of CG in order to better understand the prognosis and develop approaches for cancer prevention. The accumulated knowledge about the etiology, pathogenesis and morphology of gastritis has made it possible to coordinate the general ideas about gastritis in the classifications used by practicing physicians today.

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INTRODUCTION

The history of studying chronic gastritis (CG) is the history of Nobel Prize-winning scientific discoveries and their implementation in real clinical practice, the history of the emergence and development of endoscopic examination of the stomach and related histopathological findings in gastric mucosa (GM) biopsy material, the formation and realization of predictive approach in medicine in terms of gastric cancer (GC) prognosis based on the assessment of precancerous changes in the GM. Only less than a century ago the idea of CG was formed on the basis of existing diagnostic capabilities of that time: "The subject of gastritis is one very difficult to approach because the scientific physician feels that nothing is known of its etiology, its morbid anatomy is obscure, symptoms are often completely absent, the prognosis is doubtful and, in fact, the one thing about which we can speak with certitude is the treatment there is none"[1]. It was due to the emergence of new methods of studying the function of the stomach and the structure of its mucosa that new knowledge about various etiologic factors of gastritis, the peculiarities of its course, clinical manifestations and prognosis, approaches to classification were developed, international interdisciplinary expert communities were formed with the adoption of agreements and consensus to determine the most effective tactics of patient management.

MAIN PART

The first mention of CG is found in the works of German physician Stahl[2] in "Collegium practicum" in 1732, who noted that some febrile diseases are associated with superficial gastric irritation and ulcerative tendencies[3]. However, the history of the term CG has its origins in the work of Broussais[4], a physician in the French Republican Army. In his book "History of chronic phlegmoses or inflammations" (1808), Broussais described common inflammations in the stomach found in almost every deceased soldier, calling them "gastritides" and singled out gastritis (gastritides) as a separate nosologic form[4]. However, his assumptions that these inflammatory changes were formed during life were later refuted by the Scottish professor of pathology Carswell[5] (1838), indicating that the gastric changes described by Broussais were the result of postmortem changes.

The first microscopic description of inflammation of the GM belongs to Charles H. Jones and Edward H. Sieveking (1854)[6] and Wilson Fox[7] (1858), who distinguished diffuse and segmental form of the lesion. Later, the British physician Brinton[8] (1859) in his book "Diseases of Stomach" subdivided the lesion of GM into acute, subacute and chronic, presented their histological differences and compared them with clinical manifestations.

In 1868, the German therapist Kussmaul proposed the use of a gastric tube, which gave rise to the period of studies of gastric functions, including the study of its motility and secretory activity of glands[9]. For the first time there were statements in favor of functional disorders, more and more often gastritis was identified with dyspepsia[10]. Despite this, Fenwick[11] (1880) suggested that CG can rightly be considered an organic pathology, and the presence of pernicious anemia in patients of this group is probably explained by gastric gland atrophy.

In 1904, Faber and Bloch[12], continuing the works of Fenwick, described in detail atrophic changes in the GI tract in a patient with pernicious anemia, indicating their possible relationship. Later Whipple *et al*[13], who at that time studied the role of the liver in hematopoiesis, found that raw liver leads to an increase in the level of erythrocytes in the blood in dogs with posthemorrhagic anemia. In 1926, Minot and Murphy[14], having learned about the discovery of Whipple *et al* [13], applied raw liver to treat pernicious anemia in humans. For their discovery in 1934, Whipple, Minot and Murphy were awarded the Nobel Prize in Physiology and Medicine[15]. In 1948, biochemists Smith[16] and Rickes[17] isolated vitamin B12 from the liver, which gave a new impetus to the study of the causes of this vitamin deficiency in GM atrophy.

At the beginning of the 20th century independently of each other two scientists Faber and Konjetzny made a serious attempt to prove the presence of a morphologic substrate in CG[18]. Faber by introducing 10% formalin solution into the abdominal cavity protected GM from postmortem autolysis and putrefaction. Konjetzny[19] studied resected stomachs in patients with peptic ulcer disease (PUD) and GC by developing a special technique that prevented the possibility of postmortem autolysis of tissue. In his works Konjetzny[19] wrote: "...Ulcer and GC can develop against the background of silent inflammation of the GI tract. We can not distinguish between gastritis, on the background of which appears GC. If we can prevent the development of gastritis or treat it, we can prevent the formation of ulcers and the development of GC. Prevention of gastritis is prevention of ulcers and stomach cancer...". In 1944, Warren and Meissner[20] published

data on a groundbreaking change that can be found in patients with gastritis – intestinal metaplasia (IM).

In vivo diagnosis of gastritis became possible after the invention of the semi-rigid gastroscope by Schindler in 1932. In his monograph "Gastritis", he classified gastritis into acute and chronic, subdividing the latter into superficial, atrophic and hypertrophic (1947)[21,22]. Since the time of Schindler, the term "superficial gastritis" was included in the medical dictionary and is still used today to denote non-atrophic gastritis.

In 1949, Wood *et al*[23] reported the invention of a simple biopsy tube, and soon (1957) Hirschowitz[24], a flexible fiber optic fibrogastroscope, which made it possible to perform targeted forceps biopsy under visual control from any part of the stomach. The emergence of this method can rightly be considered a revolutionary event in gastroenterology. In a short time it became universally recognized and widespread, expanding the range of possibilities in the diagnosis of various gastric diseases. Since that time, a new chapter in the history of CG research begins.

In 1956, Cheli and Dodero[25] proposed to classify gastritis into "superficial", "interstitial" and "atrophic" ones. In 1959, Wood and Taft[26] outlined possible etiologic factors of CG: alcohol, diet, stress, radiation and other. A little later, in the 60s of the last century with the help of immunologic studies it was possible to detect in some CG patients the presence of autoantibodies to parietal cells of gastric glands and to Castle's intrinsic factor, which allowed to explain the pathogenesis of autoimmune CG and its connection with vitamin B12 deficiency[27].

In 1972, Whitehead *et al*[28] divided CG topographically into antral, fundal, cardiac, and pyloric, and proposed the division of CG into "active" and "inactive" based on the presence of inflammatory infiltration of the GM and introduced the evaluation of intestinal and pseudopyloric metaplasia into the routine practice of pathologists. A year later, Strickland and MacKay[29] proposed to supplement the classification of CG with etiologic data. They used the terms "type A gastritis" (autoimmune) to denote gastritis of the stomach body, and "type B gastritis" (non-autoimmune) to denote antral gastritis, presumably caused by duodeno-gastric reflux. In 1975, Jerzy Glass and Pitchumoni[30] added "type AB gastritis" to the classification to denote CG extended from the body of the stomach to the prepyloric region.

Another truly revolutionary event was the publication in "*The Lancet*" of an article by Correa *et al*[31,32] where he presented a sequence of pathologic changes in GM (P. Correa's cascade) from the formation of non-atrophic CG and slow, over 20-25 years, development and progression of atrophy (at a rate of 0%-3%-3% per year) to the appearance of specialized intestinal-type epithelium, and then to dysplasia/intraepithelial neoplasia and GC.

The discovery of the bacterium *Helicobacter pylori* (*H. pylori*) was a revolutionary event in gastroenterology and led to a rethinking of existing approaches to the diagnosis and treatment of gastric diseases. The presence of *H. pylori* in the human GM was described more than a century ago, but it was not until the end of the 20th century that the role of the bacterium as a leading etiological factor in CG, PUD, GC, and MALT lymphoma was recognized[33-35]. For a long time, there was a belief that due to low pH values in the stomach, microbial growth and reproduction were impossible, so many researchers thought that the bacteria they detected were accidental representatives of the oral microbiota that had nothing to do with the stomach itself[36].

It is believed that the first to discover colonies of spiral-shaped bacteria in the bottom of dog gastric ulcers were German bacteriologist Bottcher and his colleague Letulle in 1875, and they first suggested that bacteria are the cause of gastric ulcers[37]. However, the bacteria discovered by them were not cultured on the known nutrient media of that time, due to which their hypothesis was criticized and was not further developed, even though later in 1881 the pathologist Klebs[38] reported a bacillus-like organism found in the lumen of the gastric glands and in the GM of dogs with the formation of a characteristic "inflammatory infiltration". And although Klebs noted only the presence of concomitant inflammatory infiltration without making specific conclusions, this report is considered by many researchers as the first description of gastritis caused by *H. pylori*.

In 1889, Jaworski, professor of medicine at the Jagiellonian University in Krakow (Poland), examined stomach flushes obtained from humans and discovered a characteristic spiral-shaped bacteria, which he called *Vibrio Rugula*[39]. Jaworski suggested that *Vibrio Rugula* may play a possible pathogenic role in the development of gastric diseases. The work of Jaworski were published in Polish, and perhaps that is why they were not widely disseminated and recognized.

In 1893, an Italian researcher, famous anatomist Bizzozero together with his student Camillo Golgi described spiral-shaped bacteria in the parietal cells and glands of the GM of dogs, later identified as *H. canis, H. Felis* and *H. heilmannii*. Bizzozero[40] noted that these microorganisms could infect the mucosa of both the pyloric and fundal parts of the stomach. Three years later, in 1896, in an article entitled "Spirillum of the mammalian stomach and its behavior with respect to the parietal cells". Salomon[41] reported the presence of spirochetes in the GM of dogs, cats, and rats and described a series of experiments where he managed to transfer the spirochete bacterium detected in the stomach of dogs to white mice.

In 1906, Krienitz[42] identified spirochetes in the stomach of a patient with carcinoma. 9 years later, similar bacteria were found in patients with gastric and duodenal ulcers. Around the same time, the presence of urease activity in the human stomach was documented, but it was thought to occur directly in mucosal cells and was not related to the presence and activity of bacteria[43].

In 1923, the scientist Edkins[44] from London, who had previously gained fame for the discovery of the hormone gastrin, using Giemsa staining identified spiral-shaped bacteria in the bottom of gastric ulcers, as well as in the antral part of the stomach and put forward a theory about the relationship between the development of PUD and the bacterium he discovered, which he named *Spirochete regaudi*.

In 1938 in the United States Doenges[45] found spiral-shaped bacteria in the GM of the rhesus macaque he studied and in 43% of resected human stomach samples.

In 1974, Ito and Schofield[46] from Harvard Medical School (United States), who made the first anatomical description of the GM under an electron microscope, published a photograph of the gastric parietal cell, which showed a bacterium later identified as *H. pylori*.

The coccoid and vegetative bacterial forms later identified also as H. pylori were discovered in Russia in 1974 by I.A. Morozov and L.I. Aruin during electron microscopic observation of gastric mucosa sections from patients after proximal vagotomy in connection with peptic ulcer[47].

In 1979, during a routine histologic examination, Warren, a pathologist at the Royal Perth Hospital (Australia), noticed a blue line on the surface of the GM of a patient with active CG. After analyzing a large volume of biopsy material, he assumed that these were bacteria that somehow played a role in gastric disease. However, in light of the prevailing dogma at the time about hydrochloric acid-induced ulcers and the skepticism of his colleagues, Warren was reluctant to discuss this controversial observation in the wider gastroenterologic community[48]. Coincidentally, at this time, a young research fellow gastroenterologist, Marshall was looking for a project to complete his internship at the Royal Perth Hospital, he was attracted to the hypothesis of Warren and they embarked on a collaborative research journey to explore ways of culturing *Unidentified bacilli*[49]. Attempts to grow the bacterium were unsuccessful until April 1982. This event occurred by chance, when cultures were left in the thermostat over the long Easter weekend and bacterial colonies appeared on the 5th day after biopsy. Among the first 135 patients studied by Warren and Marshall, nearly all were diagnosed with gastritis, and more than 65% were found to be infected with the bacterium. The microorganism was found in all 13 patients with duodenal ulcers and in 18 of 22 patients with gastric ulcers. In 1982, Marshall submitted a summary detailing his observations to the Australian Gastroenterological Association, where he was categorically rejected. However, on October 22, 1982, he managed to report his work at the International Workshop on Campylobacter infections, and later in 1983 these data were published as 2 Letters in "*The Lancet*"[50]. The microorganism was initially named *Campylobacter pyloridis* because of its similarity to other *Campylobacter* in morphology and structure.

In order to establish the causal relationship between infection and gastritis, Marshall ventured into a self-infection experiment. In 1984, after a preliminary endoscopy, the results of which showed that *Campylobacter pyloridis* was absent in the scientist's stomach, Marshall intentionally infected himself with *Campylobacter pyloridis* by drinking the contents of a petri dish in which he had cultured the microorganism, and on the 7th day he woke up with severe nausea and vomiting, and on the 10th day he underwent a repeat endoscopy and biopsy, which showed massive inflammation, and a culture revealed *Campylobacter pyloridis*. On the 14th day of the experiment, the physician started therapy with bismuth salts and metronidazole for 14 d, after which the complaints disappeared. At the control endoscopy one month after the completion of treatment, the histology returned to normal, and the bacteria disappeared[51].

Further research allowed scientists to accumulate a sufficient database proving the undeniable role of the bacterium in the development of gastritis and PUD. Subsequently, it was shown that *Campylobacter pyloridis* does not belong to the genus *Campylobacter*, and in 1989 the bacterium received its current name *H. pylori*.

In October 1987, in Copenhagen, Denmark, to promote interdisciplinary research into the pathogenesis of *H. pylori*-associated diseases The European *Helicobacter pylori* Study Group (EHSG) was founded with the active participation of Peter Malfertheiner and Francis Megraud. And this has been followed by an exponential increase in the number of studies on the role of bacteria in the development of gastric diseases. Since then, the EHSG has organized a series of consensus conferences with leading experts to develop approaches to the diagnosis and treatment of infection based on evidence-based medicine standards[52].

For the "discovery of *H. pylori* and elucidation of its role in the development of gastritis and peptic ulcer disease" Australian scientists Barry Marshall and Robin Warren in 2005, the Nobel Assembly of the Karolinska Institute in Stockholm awarded the prize in the field of medicine and physiology[49].

In parallel with the study of the role of *H. pylori* as a leading etiologic factor of gastritis, other possible causes of the disease were also evaluated. In 1988, Wyatt and Dixon[53] proposed to use the term "gastritis due to duodenogastric reflux". Dixon proposed to use the term "type C gastritis" or "chemical gastritis". However, several years later it was shown that most cases of chemical gastritis in the intact (unoperated) stomach were caused not by bile reflux but by the intake of nonsteroidal anti-inflammatory drugs, after which "type C gastritis" or "chemical gastritis" was used for a long time to describe inflammatory changes of the GM caused by both etiologic factors[54].

At the International Congress of Gastroenterology held in Sydney (1990), the "Sydney system" of gastritis classification was adopted[55], where it was proposed to classify gastritis according to three characteristics, presented by analogy with morpheme parsing of a word, where the "prefix" means the etiology of gastritis, the "root" - topography (stomach body, antral section), the "suffix" - morphological characteristics (degree of inflammation activity, severity of inflammation, severity of atrophy and metaplasia, presence and degree of colonization by *H. pylori*)[56]. The system also established a four-level scale to determine the severity of histopathological changes. It should be noted that the new classification was criticized because some of the frequently used descriptive names such as "multifocal atrophic gastritis" or "diffuse antral gastritis" were not included in the system[57,58].

Later, Houston (1994) revised the Sydney classification, restored the division of CG into types A, B, and C, and added drug-induced CG to the chemical gastritis (type C) section. Among other things, this updated system introduced a visual analog scale to assess the severity of histopathologic elements, and a protocol for taking biopsy specimens during endoscopic examination was proposed[55].

In 2005, international experts gastroenterologists and pathologists developed a system to determine the stage of CG - Operative Link for Gastritis Assessment (OLGA)-system[59]. It was based on the biopsy sampling protocol defined in Houston (1994): Two biopsy specimens from the antral part of the stomach at a distance of 2-3 cm from the gatekeeper along the lesser and greater curvature, one biopsy specimen from the gastric angle and two biopsy specimens from the stomach body at a distance of 8 cm from the cardia rosette along the lesser and greater curvature[60].

The purpose of the established OLGA system is to translate histopathological data into a standardized report with information about the state of the stomach (topography and degree of atrophic changes) and to divide patients according to the risk of GC. In 2008, based on the results of OLGA system application, a number of papers appeared in which it was convincingly demonstrated that patients with severe atrophy (OLGA III and IV) have a higher risk of developing GC.

Table 1 Etiological classification of gastritis presented in the ICD XI[63]

Etiological group of gastritis	Subgroup	Note
I. Autoimmune CG		Etiology unknown, autoimmune pathogenesis
II. Infectious CG	Induced by *H. pylori*	
	Bacterial non-helicobacter	Caused by enterococci
		Caused by mycobacteria
		Caused by Treponema pallidum
	Viral	Caused by enterovirus
		Caused by cytomegalovirus
	Fungal	For gastric mucormycosis
		For gastric candidiasis
		For gastric histoplasmosis
	Parasitic	Caused by Cryptosporidium
		Caused by strongyloidiasis
		Caused by anisokiasis
III. Caused by external causes	Drug-induced gastritis	
	Alcoholic	
	Radiation	
	Chemical	
	Caused by biliary reflux	
	Caused by other specified external causes	
IV. Caused by special reasons	Lymphocytic	
	Ménétrier's disease	
	Allergic	
	Eosinophilic	
V. Gastritis due to other classified diseases	For sarcoidosis	
	For vasculitis	
	For Crohn's disease	

CG: Chronic gastritis.

This allowed us to conclude that patients with atrophy stages III-IV belong to a high-risk group for the development of GC and require different approaches to curation, as well as dynamic monitoring[61,62].

Along with the OLGA system, the Operative Link on Gastritic Intestinal Metaplasia assessment (OLGIM) system for evaluation of CG stage has also entered clinical practice. In the modified OLGIM system atrophy assessment is based on the consideration of IM only as an indicator of CG stage, because IM is the most reproducible criterion of atrophy[63,64]. The question of comparing these classifications from the point of view of sensitivity is still debatable, since the OLGIM, which is more reproducible in practical application, may underestimate the true severity of absolute (nonmetaplastic) atrophy[65]. Modifications of these classifications are based on similar principles of integral assessment[66].

In 2015, an international meeting of experts in the field of gastroenterology was held in Kyoto (Japan)[67]. During the conference, the data on etiological factors of CG were systematized, which were later fully transferred to ICD XI[68]. The etiologic classification of gastritis reflected in ICD XI is presented in Table 1.

The most relevant topics related to inflammatory gastric diseases were addressed in the work of the initiative group - the Real-world Gastritis Initiative, or RE.GA.IN. - from redefining the disease to clinical diagnosis and assessing prognosis[69]. After lively debates on the most controversial aspects, the RE. GA.IN. consensus summarized existing published and additional scientific evidence to produce patient-centered, evidence-based statements that will help health professionals in their actual clinical practice.

CONCLUSION

Thus, the problem of CG has been discussed by the medical community for about 300 years (Supplementary Table 1). Historically, this diagnosis did not immediately gain the right to exist. For a long time it was attributed to the number of functional diseases, thus slowing down the detailed study of CG. The data accumulated to date allow not only to detect changes in the GM, but also to predict the course of CG, and the progress achieved in the field of molecular biology and genetic engineering opens new opportunities for early diagnosis and prevention of GC.

FOOTNOTES

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