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Study the protective effect of milk on the stomach

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Abstract

Stomach cancer and other deadly sequelae from chronic gastritis remain among the most prevalent catastrophic pandemic infections. Currently, perhaps more than half of the world's population may suffer from chronic gastritis. With time (years and decades), a lifelong, severe inflammation in gastritis causes stomach mucosa to deteriorate (atrophic gastritis). Stomach mucosa dysfunction follows the atrophic gastritis' gradual deterioration. The biggest independent risk factors for gastric cancer that have been identified so far are severe atrophic gastritis and a free acid stomach. In addition to the dangers of cancer and peptic ulcer, severe cases of atrophic gastritis and free acid stomach may be linked to deficiencies in the absorption of micro-nutrients (such as Ca, Fe, Zn, Mg,), food, and medications. These findings imply that study the protective effect of milk on the stomach.

Keywords: Bovine Milk, gastric cancer, upset stomach, Helicobacter pylori, peptic ulcer

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Introduction

One of the most prevalent chronic, risky, and sneaky diseases affecting people is chronic gastritis. Numerous 100s of millions of individuals global may have chronic gastritis in one way or another, according to estimates that more than half of the world's population has this disease to some degree or extent. Although the function of gastritis in the etiology of common Cancers peptic is known, the relevance of chronic gastritis as a dangerous disease is usually underestimated in clinical practice [1-4]. A millions of people global die pre-maturely each year as a consequence of cancer and ulcers, which are chronic gastritis's side effects. Either the nonatrophic or atrophic forms of Erosive Gastritis might be seen. These upset stomach phenotypes and morphologies indicate various stages of the same chronic disease [2,3,5-7]. Because gastritis's documented morphological manifestations are so similar over the world, chronic gastritis and its complications appear to be the same condition everywhere. Although Erosive Gastritis has been investigated and known about since the early 20^{th} century, Warren and Marshall's discovery of H.P in 1982 was what really brought the condition to the public's notice [8]. With the probable exception of autoimmune gastritis, it is now abundantly known that the vast majority of instances of gastritis are caused by bacteria. [3,9]. Therefore, it is now clear that treating chronic gastritis with the removal of H.P can resolve the condition, at least in situations where the gastritis has not progressed to the atrophic (atrophic gastritis) end stages [8–13]. Even though the fundamentals of chronic gastritis are well understood, there are still many unsolved concerns. For instance, we are unsure of the role that genetics or immunology play in the onset and progression of chronic H.P gastritis. The molecular mechanisms underlying chronic gastritis, as well as the effects of environmental factors including nutrition and other bacteria besides H.P , are largely unclear. We are unable to accurately anticipate who will develop atrophic end stages of chronic gastritis. [17,27,28].The antral region is "antral predominant" in gastritis, which is the term used to describe the early manifestations of the H.P pathogenesis [24,25,27]. The inflammation is "superficial" and takes up the upper layers of the mucosa, particularly in the corpus [2,20,27-29]. The cytotoxicity of the H.P strain is most likely a factor in the potency of this acute and "active" component of gastritis, which is linked to a neutrophilic inflammation. [22,30]. The chronic gastritis is more active and evidently more aggressive the more cytotoxic the strain. The most aggressive types of chronic gastritis are those that are most likely to proceed to advanced stages of atrophic gastritis, or are types of H.P gastritis that are most likely to do so. [5,6]. From one stage to the next, the annual risk of chronic gastritis development is thought to be between two and three percent [16,20,23,27]. Atrophic pan gastritis, also known as "corpus predominant gastritis," and multifocal atrophic gastritis are variations of H.P. gastritis that gradually move from a solely antral phenotype to variants that also affect the corpus and fundus. [7,15,19,21,23,27,31]. The progression of atrophic gastritis and this proximal (pyloro-cardial) dissemination are associated with significant morphological and physiological changes in the stomach mucosa. In corpus atrophy caused by the diseases results in failures in HCL and intrinsic factor secretion from oxyntic glands, while antral atrophy results in failures in gastrin-17 production and secretion from antral G cells [23,27,32-34]. The expansion of atrophic gastritis to the corpus and fundus causes a lifelong and irreversible achlorhydria or hypo because the loss of oxyntic glands and parietal cells in corpus atrophy. The initial H. pylori infection may then spontaneously decrease due to reduced intragastric acidity and may eventually heal in cases with extensive atrophy, as shown by serology testing in the follow-ups [29,36]. As chronic non-atrophic gastritis develops into atrophic phenotypes, a phenomenon in sc, numerous coincidental pathogenetic processes, including some carcinogenic ones, may be encouraged. The "Correa cascade" may ultimately play a role in the mechanisms connecting chronic gastritis with such severe aftereffects as cancer. [1,7,35,37–41].

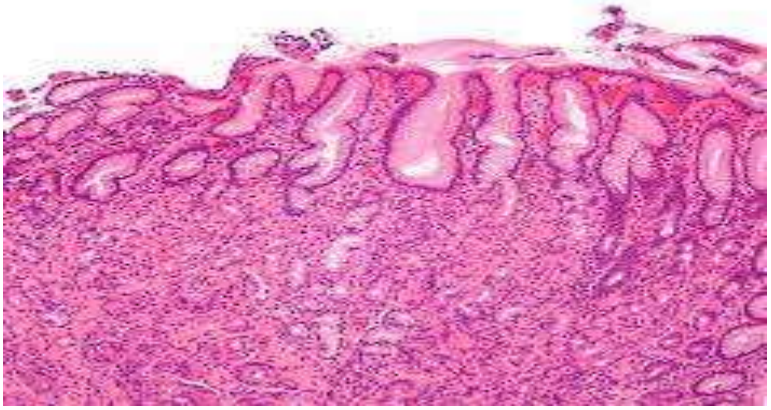

Figure No.1 Active Chronic Gastritis

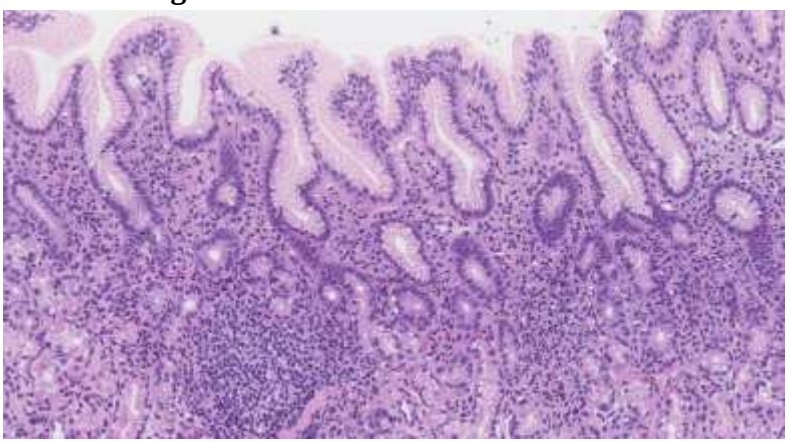

Figure No.2 Chronic inactive gastritis

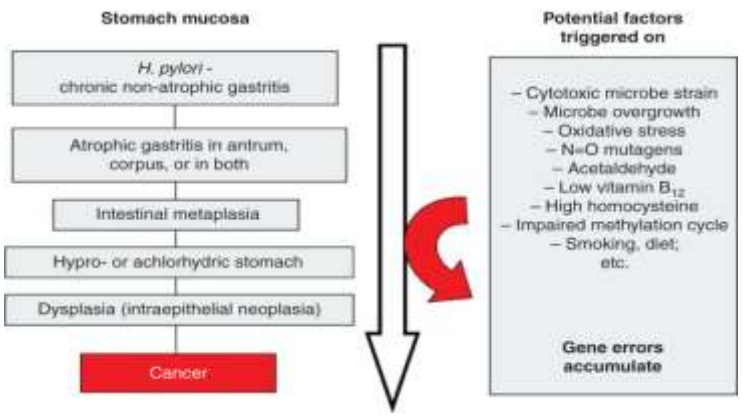

Figure. 3 Schematic depicting the development of gastritis caused by H.P from a non-atrophic state to stomach cancer (sc. "Correa cascade"). Several potentially pathogenic elements and processes that are connected to the development of cancer play a part and are progressively activated as the cascade develops.

THE BIRTH COHORT PHENOMENON AND EPIDEMIOLOGY

Some essential conclusions about the epidemiology of chronic gastritis and its consequences may be supported by the findings from population studies on the condition. Despite a considerable drop in prevalence and a concomitant decline in the prevalence of H.P, chronic gastritis is nevertheless a reasonably prevalent condition, particularly in wealthy nations [42,43]. In contrast to today, where the prevalence may

be less than 50% on average, it is possible that nearly everyone, even in affluent nations, had H.P gastritis some 100 years ago. Most often, H. P gastritis develops in childhood, and at that point, it is a painless, symptomless simple chronic mononuclear ("superficial") inflammation. [25,29]. The severe symptoms of chronic gastritis don't usually start to manifest until later in life and tend to affect people who have extensive end-stage lesions [1,39,43]. Following the identification of H.P, it became clear that knowledge of the epidemiology of H. pylori infection is essential to knowledge of the epidemiology of chronic gastritis and its aftereffects. The epidemiological features of chronic gastritis and its sequelae (peptic ulcer and stomach cancer), which were once pandemic pediatric bacterial infections, adhere to the same "pandemic" principles as those of H.P infection by itself. The two most significant underlying causes of H.P infection transmission globally are high socioeconomic status and environmental hygiene, which also serve as underlying causes of chronic gastritis and its complications [43–45]. The likelihood of contracting the H.P infection in childhood is likely influenced by a number of factors, including household density, cooking practices, environmental and family-bound cleanliness, socioeconomic situation in childhood, and others. Improvements in these conditions reduce the risk of contracting the infection or spreading it further through the mouth, most likely. [46].

GASTRITIS AUTOIMMUNE CHRONIC

Even though H. pylori is the primary cause of gastritis (in more than 90% of cases), a chronic mononuclear inflammation (gastritis) without an active H. pylori infection that also co-occurs with severe atrophic corpus gastritis, an achlorhydric stomach, and auto-antibodies against parietal cells (the proton pump) and/or intrinsic factor is a recognized medical condition.[3,32,47-50]. Uncertainty exists regarding the existence of this "autoimmune" gastritis and whether or not some vulnerable individuals with a typical H. pylori infection also have autoimmune reactions. [3,50–52]. When atrophic gastritis reaches a severely atrophic and achlorhydric stage, the initial H. pylori infection may spontaneously disappear, creating the mistaken impression that the disease is solely autoimmune in origin. In any case, a gastritis associated to an inflammatory phenomenon may advance more quickly and last longer than a pure H. pylori gastritis [3,23].

INFLUENCES ON STOMACH PHYSIOLOGY

A persistent (lifelong) chronic and active inflammation cannot be benign and will lead to the destruction of the stomach mucosa through a number of mechanisms that may interact with the gastric epithelium's renewal, development, integrity, differentiation, and function [1,24,39,53,54]. Because intrinsic factor secretion is impaired in an acid-free and atrophic stomach, vital vitamins like vitamin B12 absorption is severely compromised. [55]. Methionine, homocysteine, or folate metabolism can be affected by malabsorption, which can lead to a long-lasting and permanent vitamin B12 deficiency. This mechanism may be what causes epigenetic DNA damages to manifest by impairing the methylation cycle's functionality in epithelial cells [55,56]. Acid-free stomachs also have reduced dietary metabolism and absorption of micronutrients such Fe, Ca, Mg, and Zn, as well as some medications (e.g., dipyridamole, some iron formulations, anti-fungal medications like fluconazole thyroxine or Iitraconazole, and atazanovir) [57]. A microbial flora from the mouth eventually colonizes the acid-free stomach. [58]. Acid-free stomachs and microorganisms have the ability to create class carcinogens such acetaldehyde and nitrosamines. [39,59]

PEPTIC ULCER AND CANCER

The most dangerous conditions associated with chronic H. P gastritis include gastric cancer and peptic ulcer disorders (apart from ulcers caused by NSAIDs) [60-65]. In addition to preventing cancer, H.P eradication is effective in preventing ulcer recurrences [66,67]. Despite the correlations between H.P

gastritis and peptic ulcers and cancer, there are also notable differences regarding these connections [62,68]. Peptic ulcers are common in patients with non-atrophic H.P gastritis, but they are rare or nonexistent in people whose stomachs have become hypochlorhydric as a result of the gastritis progressing to atrophic forms. On the other hand, gastric ulcers frequently co-occur with the phenotypes of gastritis in which the antrum and incisura exhibit considerable atrophy and IM, and in which the corpus mucosa may even exhibit mild atrophy but never severe atrophy (and the stomach is never achlorhydric) [62]. Patients with hypochlorhydric or achlorhydric stomachs are more likely to develop stomach cancer, and subjects with severe stages of atrophy and IM in both the antrum and corpus are more likely to develop intestinal type stomach cancer [39, 69]. At the molecular level, gastric carcinogenesis is highly heterogeneous and variable [53,54]. While extensive IM in the underlying mucosa, a hypochlorhydric stomach, a visibly atrophic gastritis, and overt tumors or precancer lesions of the intestinal cancer type are frequently associated. [37,38,70,71].

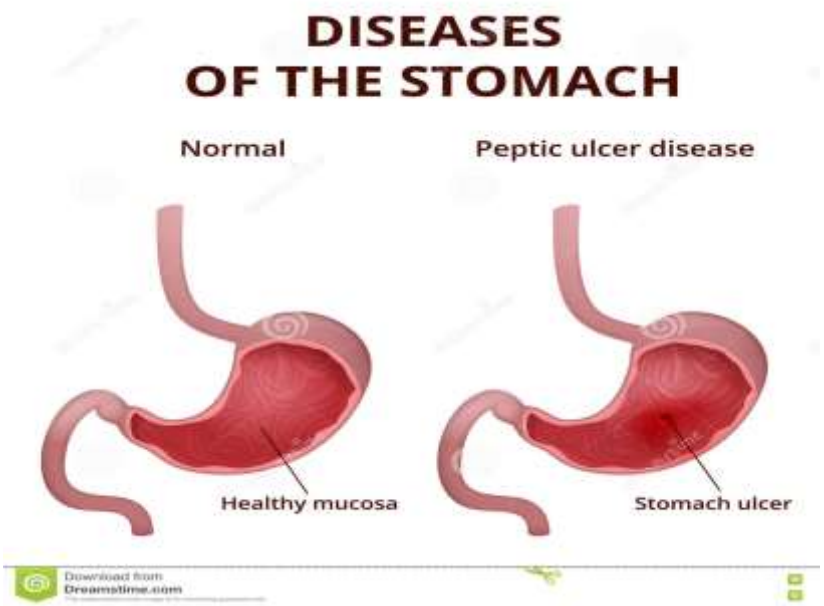

Figure.4 healthy and unhealthy stomach

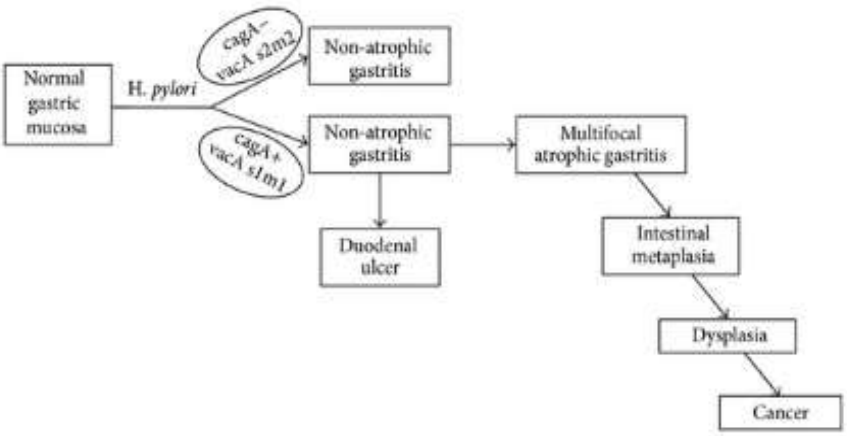

Figure. 5 Multistep cascade of gastric cancer. This sequence begins with the infection of H. pylori to sequential steps of the precancerous cascades. Taken from Correa and Piazuelo.

BOVINE MILK

Milk has been used as a great food source since it contains a number of healthy components (Chung, 2010) [85]. Drinking milk is typically seen as a crucial component of a nutritious diet. Milk includes a variety of

bioactive peptides, including peptides that are beneficial for digestive health (Mohanty et al., 2016) [86]. To the best of our knowledge, however, there has never been a comprehensive evaluation of a study the protective effect of milk on the stomach. Our hypothesis was that by regulating antioxidant enzymes and anti-inflammatory activities, bovine milk had protective benefits against stomach mucosal injury. We study the protective effect of milk on the stomach.[87]

CANCER RISK

The two most significant independent risk factors for stomach cancer that have been identified so far are atrophic gastritis and an acid-free stomach. According to statistics, atrophic gastritis carries a larger chance of developing cancer than a simple H.P infection [69]. People without H.P gastritis, or in subjects with a healthy stomach mucosa, have a relatively low risk of developing cancer. Patients with a pure H.P infection, or individuals with a non-atrophic H.P gastritis, are at an almost twofold increased risk [60,61,69].

The Sydney system and operational interface for classifying gastritis

The realization that H. pylori infection, the ensuing atrophic gastritis, and an acid-free stomach are important pre-cancerous disorders makes it possible to arrange patients in clinical practice into meaningful groups according to their cancer risk. This classification involves endoscopy inspection and endoscopy biopsy, but it can also be carried out using non-invasive blood testing using stomach-specific biomarkers [72–77].

Guidelines for interpreting the microscopical appearances in biopsy specimens in endoscopic practice include the Sydney System and its updates. [5,6]. The correct and successful classification of patients into the clinically significant risk groups depends on a proper biopsy process and accurate microscopical analysis of the samples [78,79]. Such classifications or lineations cannot be made just through endoscopy or with H.P testing. The Operative Link on Gastritis/IM Assessment (OLGA/OLGIM staging) of atrophic gastritis is a useful system for classifying patients into low-, medium-, and high-risk categories for stomach cancer [79]. The patients are divided into 5 sub-groups (0–IV) according to this classification, each with a significantly different chance of developing stomach cancer. Which patient would require and benefit the most from endoscopic surveillances and follow-ups can be determined objectively using the classification and sorting. [79,80].

Plasma biomarkers for atrophic gastritis diagnosis and screening

Purification of stomach pepsinogens in the 1970s, knowledge of the course of H. pylori infection, novel advancements in microwell plate vertical measurement techniques, and development of ELISA tests with particular monoclonal antibodies have all contributed to the applications of using gastric biomarkers in noninvasive diagnosis and screening of atrophic gastritis from a simple blood sample. [81–83]. These blood tests may now be successfully completed in any laboratory without the requirement for specific setup. This and other medical journals have previously published a number of thorough and in-depth reviews, consensus reports, and guidelines on the practical application of the biomarker tests in the diagnosis of atrophic gastritis (see references [72,80,84]). For pepsinogen 1 and 2, there have been numerous commercial radioimmunoassays and ELISA tests available for quite some time. However, due to the fact that they can only be used to diagnose atrophic corpus gastritis, these tests have limits [74]. In the stomach antrum, where the atrophic and metaplastic abnormalities emerge, the blood levels of pepsinogens 1 and 2 do not indicate any potential atrophic gastritis; nonetheless, first, in a more thorough test (Gastro Panel), in addition to the pepsinogen 1 and 2 assays and the H. pylori serology test, the plasma levels of G-17 are also measured. In addition to providing information on potential corpus atrophy, this test panel also allows for assessments of atrophy in the gastric antrum and provides hints of intragastric acidity [72, 73, 77, 78, 80].

Result and Discussion

the protection of milk on the gastric mucosa and its effect on inhibiting gastric acid.

When gastric Juice Hydrochloric acid is increased in the stomach and the gastric mucosa has effected but actually causes Gastric cancer, Gastric ulcer, heart burn, stomach burn. So therefore the Bovine milk is Gastroprotective in the gastric mucosa.

Milk is frequently suggested as a treatment for peptic ulcer patients. Milk's frequent use by peptic ulcer sufferers should be questioned because it includes both protein and calcium, both of which stimulate the release of stomach acid.[88]

According to these findings, milk may have strong antiulcer properties due to the phospholipids that make up its phospholipid composition. [89]

Therefore, it's probable that HMGF III caused a pattern of changes in gastric acid and/or duodenal mucus synthesis that was different from what cysteamine elicited in mice to produce its anti-ulcer effects. The current work investigates the effects of HMGF III on gastric acid secretion and mucus production in the mouse stomach and duodenum to understand the mechanisms of the anti-ulcer properties. [90]

the protective effect of nutrients on the stomach.

The different type of nutrients that protect the stomach cancer such as Ca, Fe, Zn, Mg,), food, and medications.

The amino acids glutamine and arginine, the essential micronutrients vitamin A and zinc, and the two classes of food additives known as prebiotics and probiotics were all examined in recent in vitro, animal, and clinical experiments. [91]

Natural antioxidants and prebiotics are abundant in plant polyphenols. Following consumption, the majority of polyphenols are absorbed in the intestine where they interact with the gut microbiota and modified metabolites like short-chain fatty acids (SCFAs) formed by bacterial fermentation. This work serves as a foundation for more research into the Gastro intestinal system's defense mechanisms against polyphenol damage. [92]

The purpose of this study was to look into any potential protective benefits of walnut oligopeptides (WOPs) on rat stomach ulcers brought on by indomethacin. The participation of arachidonic acid metabolism and elevation of tryptophan, phenylalanine, tyrosine, and alpha-Linolenic acid metabolism production in vivo may be related to the Gastroprotective mechanism. [93]

CONCLUSION

Approximately 90–100% of the youngest native citizens in western country birth cohorts currently have and will have a normal, healthy stomach for the rest of their lives, leading to populations where the overwhelming majority of people have a healthy stomach and are, therefore, free from risks of peptic ulcer or stomach cancer. This study suggests that dietary variables are a part of the complex etiology of peptic ulcer. High consumption of fermented milk products was linked to a lower incidence of ulcers, whereas high consumption of milk was linked to an increased risk.

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