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A REVIEW: CLINICAL MANIFESTATIONS OF CHRONIC ATROPHIC GASTRITIS

[1]Dr Raina Amin,[2]Dr Muhammad Awais, [3]Dr Ruqaya Bashir.
[1,3]MBBS, King Edward Medical University, Lahore.
[2]MBBS, University College of Medicine and Dentistry, University of Lahore, Lahore.

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Abstract
The prevalence of chronic atrophic gastritis is obscure as it is found that most of the patients of atrophic gastritis are usually anticipated with progressive phases of disease while at early phases diagnosis is uncommon. The demolition of parietal cells, may be autoimmune directed or outcome of H. Pylori infection, establish a decreased in the secretion of acid. The threat of neuroendocrine tumor development increases with enhanced levels of gastrin in serum originated by Hypo/achlorhydria. The fate of pernicious and megaloblastic anemia occurrence connected with persistent development hypochromic and microcytic anemia. Furthermore, a high level of homocysteine may be by depletion of cobalamin, with an elevated risk to develop CVD and might have a connection with neurological indications, basically atrophy, and demyelinated spinal cord recognition build the abnormal sensory-motor. Gastrointestinal manifestations are related to non-acid reflux.

Corresponding author:
Dr. Raina Amin,
MBBS, King Edward Medical University, Lahore.

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INTRODUCTION:

Chronic atrophic gastritis (CAG) is characterized an inflammatory process leads to loss of mucosal glands and it is might be because of an autoimmune medication reaction of parietal cells and their component or may be due to the *Helicobacter pylori* infection. There are no exact criteria to elucidate and clinical distinguish the autoimmune gastritis disease from *H. pylori* and multifactorial atrophic gastritis.[1] However, few traditional properties used to differentiate including intrinsic factor and parietal cell antibodies positivity, the existence of entero-chromaffin like cells, lack of infection associated with *H. pylori*, all resembling with historical measures of autoimmune gastritis reported in patients of body restricted atrophic gastritis and with antral and body atrophic gastritis therefore the distinct attributes linked with autoimmune gastritis can't describe well.[2] The *Helicobacter pylori* infection cause a gradual diminution of the indigenous structure of gastric portion in scattered patches of whole stomach such condition is known as multifocal atrophic gastritis. In a study of 40 patients, the pyloric region of stomach occupied by atrophic and metaplastic mucosa due to the depletion of specific parietal cell mass.[6] The etiology is far being to clear the condition, it is deemed to be associated with autoimmune phenomena including some unknown environmental factors and also with primary autoimmune situation. It is an eminent condition of autoimmune metaplastic atrophic gastritis that speedily grow with unknown cause from mid to chronic inflammation of gastric corpus to more severe complication associated with vitamins B_{12} deficit anemia knowns as pernicious anemia[3]. The clinical regimen of pernicious anemia has been explored for several decades now from the past three decades, more attention has been given to *H. pylori* infection that obscures further research of autoimmune gastritis to some extent. Thus, lack of intimacy of this entity by the gastroenterologists and pathologists.[4] The depreciation is recorded for the prevalence of *H. pylori* infection, especially in Western countries untended conditions are rising more problem therefore we should be to ratify and treat them. Moreover, many researchers concluded that maintaining the more hygienic environment results in lesser experience to infectious and non-infectious antigen during childhood enhance the incidence of autoimmune and allergic conditions including coeliac disease, eosinophilic, oesophagitis, asthma and IBD.[5]

Clinical manifestation of chronic atrophic gastritis

The serological studies with markers of gastric function like pepsinogen I, ration of pepsinogen I and pepsinogen II by inclusion or omission of gastrin-17 and antibodies against *H. pylori* and invasive studies considered histological assay of biopsy samples of the patient's upper esophagogastroduodenoscopy, both methods were considered important for the diagnosis.[7] The patients of stage three and four have the more severe stage of atrophy distinguished by extensive atrophy of antrum and oxyntic mucosa which can develop a greater risk of gastric neoplasms.[8] Even though the lesser numbers of such tumor, it is a leading reason for deaths linked with cancers, 32.4% of cases with 5 years of survival rate in Italy. The recognition of a subgroup of patients is indispensable who is at greater risk due to its high mortality rate and its silent progressive nature and endoscopy is the best way to monitor these patients. The early diagnosis of patients with CAG allows them to identify neoplasms at the initial stage and reduced the death rate caused by gastric cancer.[9] In clinical observation of 30 patients with CAG, demolishing parietal cells bring the reduction in acid secretion which helps to identify the patients with iron deficits lead to developed microcytic anemia which, can be assured through clinical extra-digestive manifestation. Abolition of intrinsic factor produced by parietal cells leads to vitamin B_{12} deficiency aids to determine the megaloblastic anemia might be associated with peripheral neuropathy and with low platelet counts[10]. High level of homocysteine as a result of cobalamin deficiency associated with the risk factors of cardiovascular disease. The elevated gastrin serum level finds by hypo/achlorhydria, this hormone trigger to develop the enterochromaffin-like cells cause to develop hyperplasia, which subsequently deemed precursor of neuroendocrine tumors of gastric mucosa.[11]

Vitamin B_{12} deficits

- The vitamin B_{12} plays an important role as a cofactor in the metabolic processes and its deficiency produced many cytological effects, one of the crucial conversion of homocysteine in methionine with the help of an enzyme homocysteine methyltransferase the deficiency of this vitamin interrupt normal conversion, which ultimately imposes a negative effect on nitrogen compounds synthesis and DNA synthesis. Cobalamin deficiency associated with hematopoiesis with megaloblastic anemia.[12]
- A series of studies reported that 50% of patients are seeking the medical advice when the disease severity reached an advanced stage with gradually rising symptoms of gastroenterological, neurological, and hematological. As repercussions of chronic atrophic gastritis digestive manifestation lead to post-prandial discomfort, diarrhea, and anorexia while anemia linked with vertigo,

dyspnea on exertion, and tachycardia. The light lemon color complexion (paleness) in case of anemia shows the correlation with mild jaundice.[13]

- Another important aspect cobalamin deficit is neuropathy, the lesion of central nervous system has evaluated in four patients out of 50 patient's diagnosis with pernicious anemia, without the history of hematological changes.[14] However, corresponding patients presenting the neurological changes, including spinal cord with damaged myelin atrophy evenly modification in axon structure. Thus, such patients experienced the spastic paraparesis, sensory ataxia, visual disturbances, unsteady gait, and altered nervous reflexes,[15] and cognitive impairment involving apathy, memory loss, depression and complexity of behavior was noticed in such patients.[16]

Iron deficiency

- The frequent development of megaloblastic anemia in patients with CAG was often observed through the clinical manifestation of patients with microcytic, hypochromic anemia.[17] There are four mechanisms associated with the pathophysiology of iron deficiency.

i. Bleeding from gastric micro-erosion
ii. Meet the dietary iron need in competition with *H. pylori*
iii. Hypochlorhydria
iv. Regulation of inflammatory hepcidin

A cohort study of patients with atrophic gastritis elucidate the genetic association such as both megaloblastic anemia and microcytic anemia are also developed due to genetic factors such as transcobalamin II is a genetic variant, associated with a reduction in the level of vitamin B_{12} was closely related to pernicious anemia.[18]

- A review study of CAG has been evaluated that round about 20-30% reported cases of those iron deficits patients who are refractory to iron supplementation. The impaired iron absorption observed because of parietal cell atrophy and negative effects of hypochlorhydria.
- Furthermore, 55% of patients have been diagnosed with an active *H. pylori* infection and, refractory to supplementation therapy for iron deficiency anemia. There is a substantial relation between the abolition of *H. pylori* infection and anemia resolution, which supports the fact that elimination of infection is associated with the treatment of anemia. In many guidelines, the eradication of *H. pylori* infection is advised, especially in patients who are refractory to iron supplementation with unknown cause of iron deficit anemia.[19]

Hyperhomocysteinemia

The metabolic enzyme involved in the regulation of homocysteine level of plasma depends on cofactors such as vitamin B_{12} and folate. However, elevated plasma concentration of homocysteine may be a cause of cardiovascular disease and it also has close associations to develop dementia, diabetes mellitus and somehow renal disorders.[12] Thus, deficiency of vitamin B_{12} and folate is the obvious cause of hyperhomocysteinemia, sometimes involved to develop CAG. Although, *H. pylori* and atrophy of gastric mucosa is interconnected with plasma concertation of vitamin B_{12} while few epidemiological studies evaluated the correlation of coronary heart disorder with *H. pylori* infection.[20]

Gastrointestinal symptoms

It has been reported that gastrointestinal symptoms exhibited the silent features in patients with CAG, approximately 24% heartburn and 12% regurgitation was observed in patients of CAG while other recurrent symptoms include postprandial fullness and early satiety in 7.1% and 10.1% was reported.[21] A recent study collected data elucidated that 56.7% of CAG patients experienced one more gastrointestinal symptoms e.g. dyspepsia, subtype postprandial distress syndrome considered most recurrent symptoms.[22] The acid reflux was seldom noticed in a case study of 41 patients with autoimmune CAG and elevation of non-acid reflux was commonly found in more than 50% of patients.[23] the prevalence rate for postprandial symptoms and epigastric pain syndrome was noticeably higher in male patients for corpus atrophy and atrophy of antrum in females. Hence, gender- dependent manner exhibit a prominent role to allocate symptoms of the atrophy gastritis.[24]

DISCUSSION:

- To manage the CAG at the initial stage it is essential to take the adequate supplements to overcome the vitamin B_{12}, folate, and iron deficits. Appropriate supplementation will help to prevent the development of anemia and cobalamin depletion**.**
- The timely diagnosis and treatment is essential especially in case of CAG in which corpus mucosa may develop iron and cobalamin deficiency may be associated with hematological changes as micro or macrocytosis or anisocytosis.[25]

CONCLUSION:

The information obtained from the different literature concluded that patients with CAG are associated with 90% impaired parietal cells, 70% H. pylori infection outcomes may be brought the

nutritional deficits that may cause more complexities related to hematological and neurological abnormalities. Furthermore, in 20 case studies, most patients have a high level of homocysteine may be by depletion of cobalamin, with an elevated risk, to develop CVD and might have connection with neurological indications, basically atrophy, and demyelinated spinal cord recognition builds abnormal sensory-motor. Although in 10 case studies patients with CAG developed gastrointestinal symptoms most of them are related to non-acid reflux.[26]

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