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A Rare Case of Atrophic Gastritis with Normal Vitamin B12 Levels

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Abstract: Long standing infection of *Helicobacter pylori* leads to atrophic gastritis most of the times. Atrophic gastritis most commonly causes Vitamin B12 deficiency. We report a case of atrophic gastritis in a young male in whom no vitamin B12 deficiency was seen.
Keywords: *Helicobacter pylori*, atrophic gastritis, Vitamin B12 deficiency.

INTRODUCTION

Atrophic gastritis is a histopathologic entity characterized by chronic inflammation of the gastric mucosa with loss of gastric glandular cells and replacement by intestinal-type epithelium and fibrous tissue. Both autoimmune and *Helicobacter pylori*-related atrophic gastritis are common diseases everywhere in the world and increase in prevalence with age. Atrophic gastritis is a well-known cause of vitamin B12 deficiency [1]. But hereby we report a case of atrophic gastritis in a young male in whom no vitamin B12 deficiency was seen.

CASE REPORT

A 28 year old unmarried, Hindu male from low socio economic status having mixed diet presented to the out patient department of Medicine in May 2013 with symptoms of severe abdominal pain and vomiting. He had previous such episodes intermittently for which he took treatment on his own. He occasionally took proton pump inhibitors. In the past Endoscopy was done and the biopsied mucosa was also tested for the presence of *Helicobacter pylori* by Rapid Urease Test and it was positive then.

The routine physical examination was normal with per abdomen examination not suggestive of any organomegaly. The symptoms this time persisted with self treatment so the patient presented to our hospital. He was afebrile and all the routine laboratory parameters were well within range. The hematological examinations were all normal. Ultrasonography of abdomen was also normal. He was then referred to gastroenterology unit of Surgery department. On endoscopy pale gastric mucosa was seen and it was suggestive of Atrophic gastritis, this finding correlated with histological findings of the same. The biopsied mucosa was also tested for the presence of Helicobacter pylori by Rapid Urease Test and it was negative. The patient's serum vitamin B12 level was measured by Chemiluminiscent immunoassay and it was found to be 365 pg/ml (normal 160-800 pg/ml). The endoscopic and histopathological finding did not correlate with Vitamin B12 levels. The patient has never been on Vitamin B12 tablets or injectables.

DISCUSSION

H. pylori infection is the main cause of chronic active gastritis. Long-lasting infection may lead to atrophic gastritis, which may in turn impair vitamin B12 absorption owing to diminished acid secretion, lower ascorbic acid levels in gastric juice and reduced secretion of intrinsic factor. A significant association between the presence of atrophic gastritis and low vitamin B12 levels has been suggested [2]. There is little doubt that in most stomachs, multifocal atrophic gastritis develops secondary to long-standing superficial chronic active gastritis by *H. pylori* infection [3-6].

As atrophic gastritis develops from *H. pylori* gastritis, the inflammation becomes more extensive and spreads to involve the full thickness of the mucosa [7]. Initially, glands are compressed and separated by the inflammatory infiltrate, but ultimately, they are destroyed. This destruction occurs in a patchy fashion. When atrophy occurs, the lamina propria will become fibrotic, and the epithelium will undergo intestinal metaplasia.5 This process of atrophic gastritis and intestinal metaplasia occurs in approximately half of the *H. pylori*-colonized population, first in those subjects and at those sites where inflammation is most severe [8].

Atrophic gastritis was more common in the B12-deficient patients. Eradication of *H pylori* infection

alone has been reported to correct B12 status and improve anemia in B12-deficient individuals. Thus, the possible role of *H pylori* infection in cases of severe food cobalamin malabsorption suggests specific options to prevent and treat B12 deficiency when associated with *H pylori* infection [9].

The epidemiology of *H. pylori* infection in developing countries, such as India is characterized by a rapid rate of acquisition of the infection such that approximately 80% of the population is infected by the age of 20 yrs [10] because the disease is most often acquired in childhood [11]. In developing countries the prevalence of infection peaks in the 20 to 30 year old age group. The prevalence of *H. pylori* is inversely related to socioeconomic status [12], the major variable being the status during childhood, the period of highest risk.

CONCLUSION

We want to bring in notice to the physicians that all the diagnostic possibilities should be pondered upon. Atrophic gastritis is generally seen in middle aged to elderly people. Atrophic gastritis and vitamin B12 defeciency go hand in hand. In this case report atrophic gastritis was reported on histopathology and endoscopy. *H. pylori* were detected in the first biopsy done in the past. *H. pylori* infection may lead to atrophic gastritis in long standing cases. Patients having Atrophic gastritis generally have vitamin B12 deficiency. In our case report we did not find vitamin B12 deficiency and possibly not all cases of *H. pylori* induced atrophic gastritis have low vitamin B12 deficiency levels.

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