

B12 Deficiency Secondary to Thyrogastric Syndrome: A Case Report

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Introduction

Atrophic gastritis occurs when the gastric mucosa undergoes metaplasia forming intestinal type epithelium and fibrous tissue resulting in reduced gastric acid, intrinsic factor production, and malabsorptive syndromes. B12 deficiency, the most common nutritional deficiency seen in atrophic gastritis, presents with the clinical picture of early satiety, abdominal pain, iron deficiency and macrocytic anemia. We present a case of B12 deficiency secondary to thyrogastric syndrome in an otherwise healthy male.

Patient Presentation

A 49-year-old male with a history of Hashimoto's thyroiditis, non-adherent to his levothyroxine was admitted after presenting with a near syncope event. Patient reports he was in his usual state of health until 6 months ago when he started to develop early satiety, post prandial nausea, vomiting, 30-pound unintentional weight loss, fatigue and decreased exercise tolerance.

Clinical Course

On the day of presentation, he was walking up a flight of stairs when he suddenly felt lightheaded and had "tunnel like vision." He never fell or any episodes of loss of consciousness. Review of systems was negative for fevers, chills, night sweats, trouble chewing or swallowing, abdominal pain, melena, or hematochezia. Aside from progressive lightheadedness and early satiety, he was overall healthy. On arrival to the ER, he was afebrile with heart rate of 83 and blood pressure of 133/83. Physical exam was notable for catechetic appearing male in no acute distress. There was no evidence thyromegaly or goiter, nor petechiae or mucosal bleeding. Labs revealed a CBC of 3.9/6.4/17.9/81 with MCV of 119.5; macrocytic anemia. On peripheral blood smear, there were no schistocytes, blasts cells or other abnormal blood cells seen. Iron studies demonstrated serum iron of 211mcg/dL and ferritin of 990.4ng/mL. B12 level was undetectable. Thyroid studies included a TSH of 0.015 and free T4 of 0.8. Patient underwent endoscopy and colonoscopy which revealed atrophic gastritis. At this time, biopsies were obtained and found to be negative for malignancy or *helicobacter pylori*. The patient was diagnosed with B12 deficiency and started on IV b12 1000 mcg daily. Patient responded quickly to the IV b12 with complete resolution of his symptoms. He was later found to have significant serum levels of intrinsic factor autoantibodies, which are presumed to be the cause of this acute presentation of atrophic gastritis complicated by B12 deficiency.

Endoscopic Findings



Figure 1: Comparing images of atrophic gastritis to normal gastric mucosa.

1a demonstrates the diffuse atrophy of the gastric mucosa and overall loss of rugae in the setting of atrophic gastritis.

1b is a comparative image of healthy, typical gastric mucosa.

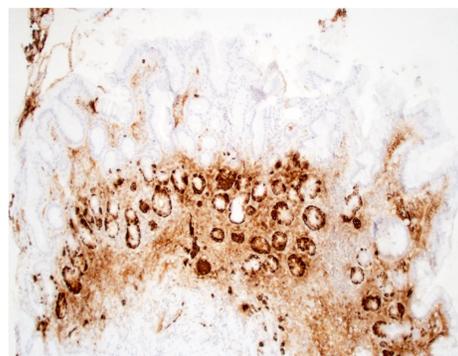


Figure 2: Histology changes seen in the setting of atrophic gastritis.

This figure demonstrates the linear hyperplasia of the enterochromaffin like cells distinguishing autoimmune gastritis from *H pylori* gastritis. Additionally, can be signs of intestinal type dysplasia-adenocarcinoma sequence not visualized on this slide.

Future Studies

Further investigation that would improve the management of patients with thyrogastric syndrome could include,

- Screening patients with autoimmune thyroiditis for autoimmune gastritis; determine the rate of occurrence of these concomitant diseases
- Identify if a genetic connection between thyroiditis and autoimmune gastritis
- Determine the impact of long-term outcomes of annual endoscopic gastric screening on patients with thyrogastric syndrome

Discussions

We presented a case of B12 deficiency secondary to thyrogastric syndrome. ~33% of patients with autoimmune thyroid disease will develop an autoimmune atrophic gastritis due to cross reaction of the gastric parietal cell antigens and thyroid antigens. Chronic atrophic gastritis results in elevated gastrin levels leading to antral and pyloric G cells hyperplasia, and further increased gastrin levels. The clinical significance of this syndrome includes both nutritional deficiencies and malignancy. The incidence of gastric adenocarcinoma is 14.2 cases per 1000 person years compared to 0.073 in patients without atrophic gastritis. 17.5% of patients with polyendocrine syndrome with predominant atrophic gastritis will develop gastrin induced enterochromaffin cell hyperplasia and have an annual incidence of 0.68% of gastric neuroendocrine tumors. Clinicians should be aware of the relationship between autoimmune thyroid disease and atrophic gastritis due to the increased risk of both nutritional deficiencies and malignancy.

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