Int.j.pathol.2023;21(2): 85 – 87

Case Report

Comorbidity of Celiac Disease and Autoimmune Hemolytic Anemia in a Patient

Neelam Ahmad[1], Asma Wasim[2] and Saira Malik[3]
[1, 3] Department of Pathology, Khyber Teaching Hospital, Peshawar,
[2] Colorado Technical University, United States.

ABSTRACT

Background: Celiac Disease (CD) is an autoimmune condition caused by consumption of food containing gluten. The condition gets worse while occurring simultaneously with another autoimmune disease known as Auto Immune Hemolytic Anemia (AIHA). In the study, a case is reported who had two conditions simultaneously.

Objectives: The objective is to report a rare case that has both CD and AIHA.

Material and Methods

Upon reception, a patient undergone testing and screenings to have first hand data. After series of screenings, when some of the serological tests showed connection with celiac diseases, Coombs test and biopsy was performed to rule out the possibility of autoimmune hemolytic anemia.

Results: The results were obtained from the serological and screenings and they confirmed the existence of co-morbidity of CD with AIHA. Retic count showed raise by 15%. Bone marrow showed all cell lines intact but increased erythroid hyperplasia. Iron showed an increase in bone marrow with siderocytes and sideroblasts.

Conclusion: It was concluded from the study that CD and AIHA can co-exist and cause more complications

Introduction

Celiac Disease is an autoimmune disorder that is caused by the gliadin and related prolamins found in the wheat, barley and rye grains.[1] Celiac disease is multisystemic, immune-mediated disease in those with certain genetic predispositions. Clinically speaking, CD is a complex chronic condition that affects many tissues and organs, including the skin, endocrine and exocrine glands, nervous system, joints, and muscles. These manifestations can range from mild to severe and resemble those of irritable bowel syndrome or severe malabsorption. As a result, CD continues to be a difficult illness to identify, delaying the implementation of the proper therapy and raising related morbidity.[2] Autoimmune hemolytic anaemia (AIHA) is an immune disorder that develops over time and is characterized by the production of antibodies that bind to the surface of circulating erythrocytes, causing hemolysis and a reduction in the lifespan of red blood cells (RBCs), which are then removed by the reticuloendothelial system. In the general community, there are 1-3 cases of AIHA per 100,000 persons each year. [3]

CORRESPONDENCE AUTHOR
Neelam Ahmad
Department of Pathology,
Khyber Teaching Hospital, Peshawar
Email: neelamahmad30@gmail.com

Case Report

A 30-year-old Pushtun guy reported to the emergency room with a 15-day history of widespread weakness and shortness of breath. He had no known co morbidities. According to the patient, his widespread weakness was growing and intensifying to such a degree that it impeded his ability to do everyday tasks. In addition to this, he was also suffering from shortness of breath. Nevertheless, he denied orthopnea, paroxysmal nocturnal dyspnea (PND), fever, rash, abnormal bowel habits, cough, joint discomfort, and any history of severe blood loss.

The patient's prior medical history revealed that he began experiencing intermittent loose stools at age 9 and that they stopped by the time he was 16. Additionally, he was taken to a local hospital two years ago with widespread weakness and jaundice. Although there is no confirmed paperwork, it is believed that two blood bags were also transfused into him. He was not obedient throughout his stay and left against medical advice, thus the workup and diagnosis were not finished. His vital signs were 110/60 mmHg (reference is 120/80 mmHg), 90 beats per minute (reference range is 70-100 beats per minute), 98 degrees Fahrenheit (reference range is 97-99 degrees Fahrenheit), and 22 breaths per minute (reference range is 12-20 breaths per minute).

An elevated jugular venous pulse, anemia, jaundice, and clubbing were all seen during his routine physical

examination. His central nervous system, cardiovascular system, and respiratory system were all in good condition. The remainder of the examination was normal, but the abdominal examination revealed hepatomegaly (liver palpable to one finger below the costal border) and splenomegaly (spleen palpable to three fingers below the costal margin).

Based on the history and examination, we ordered pertinent laboratory work up along with other tests. His base line laboratory values were hemoglobin 9.2 g/dL (reference range, 11.5 – 17.5 g/dL); mean corpuscular volume (MCV) of 114.4 fl (reference range, 76 – 96); total lung capacity (TLC) 4.2×10^{9}/L; platelets 92000/mm ^{3} (reference range, 150 – 400000/mm ^{3}); sodium 140 meq/L (reference range, 135–145 meq/L); potassium 3.6 meq/L (reference range, 3.5–5 meq/L); chloride 108 meq/L (reference range, 97–107 meq/L); calcium 8.5 meq/L (8.5–10.2 meq/L); magnesium 2.2 (reference range, 1.5–2.5 meq/L); total bilirubin 1.53 (reference range, 0.1–1.2 meq/L); serum glutamic pyruvic transaminase (SGPT) 59 units/L (reference range, 7–56 units/L); alkaline phosphatase (ALP) 87 IU/L (reference range, 40–129 IU/L); serum glutamic-oxaloacetic transaminase (SGOT) 40 units/L (reference range, 5–40 units/L); total protein 7.7 g/dL (reference range, 6–8.3 g/dL); albumin 3.5 g/dL(reference range, 3.5–5 g/dL) and prothrombin time to international normalized ratio (PT/INR) 11 seconds (reference range, 11–13.5 seconds). The value of direct Bilirubin was 0.405 mg/dL (normal values range 0-0.25) an indirect bilirubin was 1.82 mg/dL (normal values range from 0-0.84).

We also did an echocardiogram and a chest x-ray because he had complained of having trouble breathing, and both results were within normal ranges. We initially believed that the patient's symptoms and signs could be caused by an autoimmune disorder (like SLE or rheumatoid arthritis (RA)), an immune-mediated disorder (like celiac disease), a thyroid disorder (for example, immune thrombocytopenia purpura), Evan syndrome (caused by thrombocytopenia and hemolytic anaemia), or even chronic liver disease. Therefore, we performed further tests in order to rule out the differentials and arrive at a potential diagnosis.

The patient's iron profile included ferritin levels of 115.5 ng/mL (normal range: 12-150 ng/mL), serum iron levels of 82 mcg/dL (normal range: 50-170 mcg/dL), total iron binding capacity (TIBC) levels of 258 mcg/dL (normal range: 250-370 mcg/dL), transferrin saturation levels of 37% (normal range: 25-35%), and B12 was 434.6 pg/mL with normal values ranging from (193 pg/mL -982 pg/mL). We examined his thyroid profile [thyroxine (fT4) 14.8 pmol/L with normal ranges from (10-28) pmol/L, tri-iodothyronine (fT3) 0.917 nmol/L with normal ranges from (0.6-0.2) nmol/L and thyroid stimulating hormone (TSH) 1.73 μIU/mL, (reference range, 0.3-4.2 μIU/mL] to rule out thyroid problems.

To rule out the existence of celiac disease in the patient, upper gastro intestinal endoscopy was performed. Esophagus was checked for any ulcers, growth, strictures or varices and no abnormality was seen. Stomach was found to be normal with nodular antrum and pylorus appeared normal. Scalloping of duodenal mucosa was found but no other abnormalities were seen in screening. Further, histopathology of duodenum and stomach biopsies was performed. Coombs test and bone marrow biopsy were performed to check if there is existence of autoimmune hemolytic anemia. Nevertheless, neither the screening nor any of the blood tests revealed the presence of autoimmune hemolytic anemia. It was simpler to rule out that just celiac disease was present, but with further tests, such as the Coombs test and bone marrow biopsy, the presence of AIHA, which may be dangerously harmful for the patient, was proven in the patient.

Discussion

Gluten, a number of environmental triggers, and immunological variables can all cause celiac disease. While gliadin is the portion of gluten that is alcohol-soluble, gluten is the sole protein found in wheat.[4]The immunological response to gliadin causes an inflammatory reaction in the small intestine, which includes shrinkage of the villi and infiltration of inflammatory cells into the lamina propria and epithelium. Furthermore, the two HLA genes in adults that produce the HLA-DQ2 and HLA-DQ8 proteins are strongly associated with the development of celiac disease.[5]Women are more susceptible to the sickness for unidentified causes. Additionally, women are more likely than males to have autoimmune illnesses in general and conditions like osteoporosis and iron deficiency anemia, which trigger tests for celiac disease, are particularly prevalent in women.[6]

Diarrhea is the typical manifestation, and stomach discomfort may also be present. However, investigations over the last ten years have shown that less than 50% of patients had diarrhea as their main

symptom. Additionally, with the introduction of serologic screening, the idea of silent celiac disease has emerged due to a lack of information on the illness's characteristic symptoms. Iron deficiency anaemia, accidental endoscopic discoveries for other symptoms such as gastroesophageal reflux, and osteoporosis are examples of silent presentations.[7]

Constipation, neurologic symptoms, increased liver enzymes, hypoproteinemia, and hypocalcaemia are a few more, less frequent results. A third of celiac disease patients were identified by serological screening during the previous five years without experiencing any gastrointestinal symptoms, according to another study, proving that the condition can also be asymptomatic.[8]

Additionally, anemia from iron, folate, or vitamin B12 insufficiency, coagulopathy from vitamin K deficiency, and, very infrequently, leukopenia and thrombocytopenia are hematologic symptoms of celiac disease. Serum folate, vitamin B12, and ferritin levels can be normal in silent compared to typical celiac disease. A second indication of the prevalence of silent celiac disease was the fact that our patient did not have a folate, vitamin B12, iron, or vitamin K shortage. Additionally, it was believed that the anemia in our instance was caused by the autoimmune destruction of red blood cells.[9]

Only widespread weakness, shortness of breath, and slight jaundice were observed in our patient at presentation, who was a male and so at increased risk of celiac disease. A duodenal biopsy is the gold standard test for diagnosing celiac disease.[10]

Conclusions

The study concluded that if an individual have celiac disease with hemolytic anemia then the individual will develop more complications such as constipation, gastroesophageal reflux, osteoporosis and diarrhea. Although blood tests with endoscopy can be performed to diagnose the celiac disease with hemolytic anemia but duodenal biopsy is the gold standard test for diagnosing celiac disease.

HISTORY	
Date received:	22-03-2023
Date sent for review:	24-03-2023
Date received reviewers comments:	04-04-2023
Date received revised manuscript:	20-06-2023
Date accepted:	20-06-2023

References

1. Lindfors K, Ciacci C, Kurppa K, Lundin KE, Makharia GK, Mearin ML, et al. Coeliac disease. Nature Reviews Disease Primers. 2019; 5(1).
2. Guarino, Matteo, et al. "Life-Threatening Onset of Coeliac Disease: A Case Report and Literature Review." BMJ Open Gastroenterology, vol. 7, no. 1, 2020, https://doi.org/10.1136/bmjgast-2020-000406.
3. Hayat AS, Humaira M, Pathan GN, Bawany MA. Autoimmune hemolytic anemia (AIHA). The Professional Medical Journal. 2016; 23(11):1377–81.
4. Sweetser S. Evaluating the patient with diarrhea: a case-based approach. InMayo Clinic Proceedings 2012 Jun 1 (Vol. 87, No. 6, pp. 596-602).
5. Gujral N, Freeman HJ, Thomson AB. Celiac disease: prevalence, diagnosis, pathogenesis and treatment. World journal of gastroenterology: WJG. 2012 Nov 11;18(42):6036.
6. Angum, Fariha, et al. "The Prevalence of Autoimmune Disorders in Women: A Narrative Review." Cureus, 2020, https://doi.org/10.7759/cureus.8094.
7. Ludvigsson JF, Leffler DA, Bai JC, Biagi F, Fasano A, Green PH, et al. The Oslo definitions for coeliac disease and related terms. Gut. 2012; 62(1):43–52.
8. Sadeghi, Amir et al. An unusual cause of constipation in a patient without any underlying disorders. Gastroenterology and hepatology from bed to bench (2015); 8 (2): 167-70.
9. Baydoun A, Maakaron JE, Halawi H, Abou Rahal J, Taher AT. Hematological manifestations of celiac disease. Scandinavian Journal of Gastroenterology. 2012;47(12):1401–11.
10. Freeman, H.J. (2018). Role of biopsy in diagnosis and treatment of adult celiac disease. GastroenterolHepatol Bed Bench. 11(3):191-196