TURKISH JOURNAL OF INTERNAL MEDICINE

ISSN:2687-4245

Case Report

Acute Tubular Necrosis Associated with Autoimmune Hemolytic Anemia due to Acute Gastroenteritis

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Abstract

Autoimmune hemolytic anemia (AIHA) is a rare disease and defined as primary (idiopathic) or secondary depending on the presence or absence of accompanying disease. Here, we reported a case of AIHA due to acute gastroenteritis and acute tubular insufficiency.

Turk J Int Med 2021;3(Supplement 1):S120-S122
DOI: 10.46310/tjim.877028

Keywords: *Acute Tubular Necrosis, Autoimmune Hemolytic Anemia, Acute Gastroenteritis*

Introduction

Autoimmune hemolytic anemia (AIHA) is a rare disease with a rate of 1-3 in 100,000 in adults.[1] AIHA encompasses a group of heterogeneous conditions mainly characterized by red blood cell (RBC) lysis due to autoantibodies against surface erythrocyte antigens. Based on the thermal characteristics of the autoantibody, AIHAs can be classified into warm forms, generally caused by IgG antibodies reacting at warm temperatures and able to fix complement in some cases; cold agglutinin disease (CAD), due to IgM antibodies that agglutinate RBCs at low temperatures and lyse them via the complement cascade activation, and mixed forms (coexistence of warm and cold autoantibodies).[2,3] AIHA are defined as primary (idiopathic) or secondary depending on the presence or absence of accompanying disease. Secondary causes include drugs, immunodeficiencies, infections, other autoimmune diseases, or malignancies.[4,5] Coexistence of autoimmune hemolytic anemia and acute renal failure has been observed in studies. Generally, this association has developed due to various drugs. In our case, the association of AIHA due to acute gastroenteritis and acute tubular insufficiency was considered suitable for the presentation because of its rare occurrence.

Received: February 8, 2021; Accepted: March 6, 2021; Published Online: March 6, 2021

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Case Report

A 42-year-old female patient without chronic disease was applied to the emergency service with complaints of diarrhea, nausea-vomiting, fever, chills, and jaundice for two days. At admission, fever: 37.1 °C, blood pressure: 104/64 mmHg, pulse: 99 beats/minute, conjunctivae were pale, sclera, and skin were icteric. Leukocyte: 31,000/mm3, hemoglobin: 11 g/dL, platelet: 176,000/mm3, urea: 104 mg/dL, creatinine: 3.3 mg/dL, alanine aminotransferase: 48 IU/L, aspartate aminotransferase: 100IU/L, lactate dehydrogenase: 1,340 U/L, total bilirubin: 16 mg/dL, direct bilirubin: 3.2 mg/dL, reticulocyte: 3%, C-reactive protein: 197 mg/L, and procalcitonin: >100 µg/L. She was hospitalized with pre-diagnosis of acute kidney injury secondary to hemolysis. Direct Coombs test was positive. Methylprednisolone 40 mg/day was started considering AIHA. ANA was negative, and complements were normal. No pathology was found in the thorax, neck, and abdominal tomographies performed for malignancy screening. There was no history of drug use or broad beans consumption. Klebsiella and E.coli grew in the stool culture of the patient sent in terms of infection focus. Brucella agglutination test, rotavirus, adenovirus test, and hepatitis markers were negative. The patient's pathology result, who underwent renal biopsy, was consistent with acute tubular necrosis due to an anuric course and progressive creatinine values. She was on hemodialysis at regular intervals. On the 14th day, ceftriaxone was discontinued. LDH, bilirubin, and creatinine returned to the normal range. The patient's direct Coombs test was negative. The patient did not need hemodialysis treatment later. She was discharged with 32 mg/g methylprednisolone treatment.

Discussion

Determining the etiology of AIHA is important in terms of treatment. The coexistence of AIHA and acute tubular necrosis is common in the literature. However, infection-related AIHA cases are limited in the literature. Also, infections in AIHA are a known player in the pathogenesis of the autoimmune process. On the other hand, infections can also occur as a consequence of the disease and its treatments. There is increasing awareness of AIHA infections, as they can impact outcomes, including morbidity and fatality.[6] Infectious agents can trigger AIHA through various mechanisms, including modification of erythrocyte membrane antigens, polyclonal B cell activation, an innocent bystander, and molecular mimicry.[7,8] AIHA cases secondary to legionella and brucella infections are presented.[9,10] AIHA may complicate about 3% of infectious mononucleosis, with a typical onset within 1–2 weeks.[11] Regarding bacterial infections, Mycoplasma pneumonia may be accompanied by severe AIHA, mainly cold but even warm forms.[12] Most recently, about 20 cases of AIHAs (both cold and warm forms) secondary to COVID-19 infection have been reported, with only one fatality.[13] As a result, this case shows us that, although rare, acute tubular necrosis can be seen together with AIHA associated with acute gastroenteritis.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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