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Case Study

AUTOIMMUNE HAEMOLYTIC ANEMIA IN 40 Y FEMALE: A CASE REPORT

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ABSTRACT

The uncommon disorder referred to as autoimmune hemolyticanemia (AIHA) occurs when the immune system mistakenly attacks red blood cells, leading to the production of autoantibodies by the patient's body that destroy erythrocytes. A 40 y old female presented with complaint of shortness of breath aggravated on exertion, chest pain, palpitation over chest for 15 d. At the time of admission, her Hb was 5.5 g/dl, She is a case of autoimmune hemolyticanemia, ANA immunoblot shows Ro 60 was positive.

Keywords: Autoimmune hemolyticanemia anemia

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INTRODUCTION

An autoimmune attack on red blood cells causes autoimmune hemolyticanemia (AIHA), an uncommon illness characterized by the body's production of autoantibodies that kill erythrocytes. The disease is classified as either warm (wAIHA, which accounts for 48–70% of cases) or cool (cAIHA, which accounts for 15–25% of cases) based on the optimal temperature of activity of the autoantibodies and their isotypes. The remaining cases are of mixed illnesses [1, 2]. The disease is not uncommon, despite its rarity. Approximately 70% of all new cases seen each year are in patients over 40. With a peak occurrence between the ages of 60 and 70, the illness frequently affects more women than males. The ratio of males to women is 40:60, and the death rate is approximately 11% [3]. Secondary causes of AIHA may include lymphoproliferative disorders, including chronic lymphocytic leukemia (5–10%), and systemic autoimmune diseases (10% of cases of systemic lupus erythematosus) [4]. While some unusual kinds may be straightforward to diagnose, others may be difficult, which could cause a detrimental delay in receiving the right treatment [5]. AIHAs have historically been classified into warm forms, cold agglutinin disease (CAD), and more uncommon types (mixed, atypical, and paroxysmal cold hemoglobinuria, PCH) based on the isotype and thermal properties of the autoantibody.

CASE REPORT

A 40 y old female presented with complaint of shortness of breath aggravated on exertion, chest pain, palpitation over chest for 15 d. She also complaint of headaches, lost appetite, and abdominal pain. She complains of dryness of mouth and dry eyes. There was no history of fever, cough, or bleeding from any site. She denied a history of blood transfusions or any other major illness in past. The patient had no past history of coronary heart disease, hypertension and diabetes mellitus. The patient had a bladder, bowel, and a normal sleep pattern unaltered. Examination revealed a thin-built, pallor present with pedal edema and vital parameters were normal except blood pressure was 140/90 mm of Hg with normal systemic examination, Lungs were clear to auscultation and the abdomen was soft, tender on right hypochondrium, and mild hepatomegaly with normal bowel sounds. No rash was identified.

She was admitted and routine investigation was carried out. Hb was 5.5 g/dl, ESR was 18 mm in 1 hr, iron was 26.4, ferritin 972 ng/ml, TSH was 0.2085 mIU/l, T4 was 3.61 ng/dl, T3 was 3.94 pg/ml, Albumin 2.6 g/dl, globulin was 4.0 g/dl, A/G ratio was 0.6, AST was 32.5 U/l, ALT WAS 35.3 U/l, ALP was 291.0 U/l. Her RBS was 97.3 mg/dl, urea 128.0 mg/dl, creatinine was 1.9 mg/dl, sodium 134.7 mmol/l, potassium was 4.4 mmol/l, TBIL was 0.4 mg/dl, DBIL was 0.4 mg/dl, delta bilirubin was 0.4 mg/dl. Anti – CCP was 1.30, ANA was 363.92. HIV, hepatitis B surface antigen, and Hep C antibody were non-reactive. Her USG abdomen showed mild hepatomegaly with hepatic haemangioma with B/l raised renal echotexture. Her PT was 17 sec. 2 D echo was normal. Her stool examination was normal. CRP was 200.3 mg/l. Urine examination showed pus cell 3-4/hpf, epithelial cell-4-5/hpf, and calcium oxalate crystals present. Ro60 was positive (SS-A antigen found), suggestive of Sjogren syndrome with the positive laboratory findings, a diagnosis of autoimmune haemolytic anemia was confirmed. The patient reported improvement of her symptoms with steroid therapy. Her haemoglobin improved up to 9.5 g/dl.

Table 1: Investigation profile of patient

Laboratory parameters		At the time of admission
CBC	Hb	5.5 g/dl
	WBC	6.38 x 10^3/µl
	RBC	2.32 x 10^6/µl
	Platelet	0.32 x 10^3/µl
	ESR	18 mm in 1 h
Renal function test	Urea	128.0 mg/dl
	Creatinine	1.9 mg/dl
Liver function test	Total protein	6.6 g/dl
	Albumin	2.6 g/dl
	Globulin	4.0 g/dl
	A/G ratio,	0.6
	AST	32.5 U/l
	ALT	35.3 U/l
	ALP	291.0 U/l

Laboratory parameters		At the time of admission
RBS		97.3 mg/dl
Thyroid function test	TSH	0.2085 mIU/l
	T4	3.61 ng/dl
	T3	3.94 pg/ml
Serum electrolytes	Sodium	134.7 mmol/l
	Potassium	4.4 mmol/l
HIV		non-reactive
Hepatitis B surface antigen		non-reactive
Hep C antibody		non-reactive
Iron		26.4 mg/dl
Ferritin		972 ng/ml
TBIL		0.4 mg/dl
Delta bilirubin		0.4 mg/dl
DBIL		0.4 mg/dl
stool examination		norma
CRP.		200.3 mg/l
Urine examination	Pus cell	3-4/hpf
	Epithelial cell	4-5/hpf
		Calcium oxalate crystals
Ro60		Positive (SS-A antigen found
. Anti – CCP		1.30
ANA		363.92 (positive)
PT		17 sec

DISCUSSION

The acquired, diverse collection of disorders known as autoimmune hemolytic anemia (AIHA) is caused by complement and/or autoantibodies, as well as activated macrophages, T lymphocytes, and cytokines that target red blood cells and cause premature erythrocyte destruction [6]. The accelerated destruction of RBC in the presence of complement and/or anti-RBC autoantibodies is known as AIHA. The spleen and other reticuloendothelial tissues experience most of the extravascular hemolysis in AIHA. Primary autoimmune illnesses or those brought on by infections or stressful situations are also possible. A number of factors, such as genetics, infections, cancer, or other autoimmune diseases, can cause AIHA. AIHA is more common in women compared to men [7]. The patient undergoes an investigation to find out the cause of AIHA. On ANA screening, find to be positive patient underwent immunoblot ANA and was found to be Serum Ro60 positive. AIHA treatment choices are influenced by a number of variables. If the anemia is slight, it usually goes away on its own. Seventy to eighty percent of persons require little to no intervention or no treatment. On the other hand, some people will require blood transfusions, medicines, or surgery. Patient was started on steroid and anemia was treated. Patient improved and discharged. Treating the illness or altering the drug may lessen the risk of an underlying cause, such as cancer, an infection, or the usage of certain medications [8].

CONCLUSION

AIHA could be a problem for diagnosis and treatment. The differential diagnosis is made more difficult by the presence of numerous confounders, including malignancies, chronic liver or kidney illnesses, nutritional deficits, and infections, in addition to the usual presentation of anemia and hemolysis. The treatment of AIHA patients with warm and cold autoantibodies is challenging and should therefore be provided by following the recommended protocol.

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AUTHORS CONTRIBUTIONS

All authors have contributed equally

CONFLICT OF INTERESTS

Declared none

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