Section: Medicine

Case Series

A CASE SERIES OF ADULT AUTOIMMUNE HEMOLYTIC ANEMIA MASQUERADING DIVERSE ETIOLOGIES

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ABSTRACT

Background: Autoimmune hemolytic anemia (AIHA) is a condition, characterized by an accelerated destruction of red blood cells (RBC's) due to presence of autoantibodies. The destruction predominantly occurs extravascularly particularly in spleen and liver, but may occasionally occur intravascularly. While the clinical course of AIHA is invariably divergent, early recognition and timely intervention can gain a lead point in time and can prevent severe complications.

Case report: The present case series describes four unlike cases of AIHA with pluralistic associations—AIH with AIHA, AIHA with Lupus with DVT (LAHPS), AIHA with SLE, idiopathic with no association. All were treated with steroids as the first and prime line agents and kept under regular follow up.

Conclusion: The present case series highlights the spectrum of AIHA and the enmeshed conditions, emphasizing their clinical presentations and tailored management strategies.

Keywords: SLE (Systemic Lupus Erythematosus), AIH (Auto Immune Hepatitis), LAHPS (Lupus anticoagulant Hypoprothrombinemia Syndrome). Idiopathic

INTRODUCTION

Autoimmune hemolytic anemia (AIHA) is a hemolytic disorder in which autoantibodies are targeted against red blood cells (RBC's) surface epitopes that lead to premature RBC destruction, and results in anemia.[1,2,3] AIHA is a kind of hemolytic anemia with an incidence rate of 1.8 pr. 100,000 person-years, and a prevalence of 9.5 per 100,000.[4] Females have proportionately higher preponderance in AIHA, explained by other coexistent autoimmune diseases like systemic lupus erythematosus (SLE).[5,6,7]

AIHA can be categorized into warm type (wAIHA), cold type (cold agglutinin disease (CAD), or mixed warm and cold type,[6,8] depending on the thermal amplitude of the antibodies involved. While warm AIHA autoantibody, is IgG mediated reacts best at 37degrees usually leads to extra vascular hemolysis, Cold agglutinin disease(CAD)autoantibody, is complement mediated leads to intravascular hemolysis and reacts strongly at lower temperatures (typically below 30 degrees).

AIHA may be seen in isolation (Idiopathic/ Primary) or in association (secondary) with other disorders such as Systemic Lupus Erythematosus, lymphoproliferative disorders, infections (parvovirus B19, HIV, HCV, EBV), drugs, vaccines, chronic inflammatory diseases, such as inflammatory bowel disease.[3,6,9,10] In addition to underlying diseases, organ transplantation can also cause secondary wAIHA.[7,11]

Direct antiglobulin test (DAT) is the test of choice that detects Immunoglobulin {wAIHA} and complement components {CAD} bound to surface of patients red cells and is diagnostic of AIHA. Presence of laboratory findings supporting hemolysis include, high LDH (Lactate Dehydrogenase), reticulocytosis, spherocytes in peripheral smear. For more specific and sensitive

AIHA, monospecific anti –IgA antisera, low ionic strength solutions (LISS), column Agglutination test, flow cytometry is being considered

In the present case series, the author presents four cases of AIHA with unpredictably capricious presentations. All were treated with steroids, which started to respond within two weeks of treatment and are still kept under follow up.

CASE SERIES

CASE 1.

A 44 years old female patient with no prior comorbidities presented with progressively increasing jaundice for last 3 months, low grade fever, intermittent in nature last 1 months, mild right hypochondriac region pain dull aching in nature last 1 months, abdominal distension last 20 days, bilateral pedal oedema last 15 days. She had easy fatigability, shortness of breath (NYHA2-3), last 15-20 days. There was no history of hematemesis, malena, altered sensorium. She was radiologically diagnosed to be a case of CLD. She received two units blood transfusions.

On examination, she had pallor, icterus, bilateral pedal oedema, raised JVP; yellowish discoloration of skin and eyes. There was no cyanosis, clubbing, no lymphadenopathy.

Her biochemical parameters were as follows:

Hb (4.5), TLC (7700), TPC (80k), urea (60), creat (0.9), Na (140), K (3.4), INR (1.5), total bilirubin (9.7) {direct (3.70), indirect (6.0)}.

RBS (120), BP (110/60), PR (89).

Peripheral smear was suggestive of high reticulocyte count 1.8%, microspherocytes.

HEV IGM Negative, HBSAG, anti HCV negative, HAV IGM positive.

USG: coarse echotexture of liver, dilated portal vein(15mm), gross ascitis, splenomegaly, suggestive of Chronic Liver Disease (CLD).

CECT Whole abdomen: suggestive of hepatosplenomegaly, gross ascites, with shrunken liver, suggestive of CLD and no SOL (Space occupying lesion).

Special tests

Direct Coombs Test (Direct Antiglobulin test): positive

Indirect Coombs Test: positive.

Total IgG = 5000mg/ dl

LDH= 1488

ENA= Anti Ku Ab 2+ (systemic sclerosis, myositis other Connective Tissue Disorders.)

G6PD negative, ANA/ ASMA/ LKM, ANCA: negative

RK 39: negative, Malaria, scrub negative.

On the basis of above investigations and clinical findings, patient was diagnosed as a case of AIHA with AIH.

She received two BT under supervision after two days of hospitalization, but her Hb didn't rise and total bilirubin rose to 50 mg/dl.

Ascitic tap was done suggestive of candida tropicalis, sensitive to caspofungin, fluconazole, miconazole, voriconazole.

Blood culture developed Enterococcus sensitive to vancomycin, linezolid. She was treated with culture sensitive antibiotics and antifungals.

She was subjected to Transjugular Liver (Biopsy TJLB) by gastroenterologist and histological report showed presence of interface hepatitis, plasma cell predominance in porta, rosettes formation, which was suggestive of autoimmune hepatitis+ autoimmune cholangitis> chronic Alcoholic.

Subsequently she was started on oral steroids (20mg), her Hb rose to 8.3 in 7 days, and total bilirubin reduced to 20 mg/dl, ascitis and pedal oedema started to resolve. There was no hemoglobin drop in hospital and her bilirubin improved to 10 mg/dl. The patient was finally discharged in hemodynamically stable condition and is kept under regular follow up.

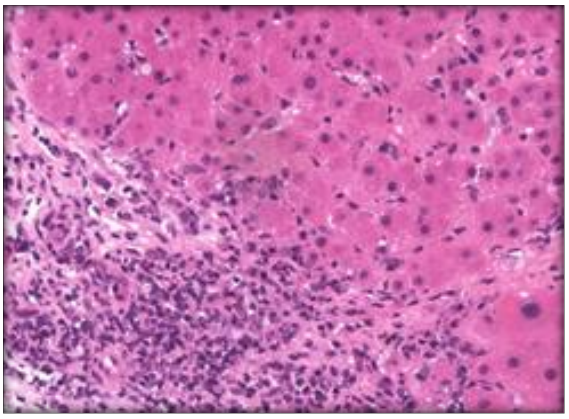

Figure 1: Moderate portal lymphoplasmacytic infiltrate with interface hepatitis

Table 1

Date	2/09/2024	10/09/2024	16/09/2023	21/07/2024	24/07/2024
Hb	4.5	8.3	10.1	10.2	10.9
TLC	7700	6700	8000	5000	5600
TPC	80k	82k	90k	1lac	1.1 lac
T.Bil.	9.7	50	29.9	18.3	9.2
SGOT/PT	74/29	70/82	77/67	98/94	56/67
ALP	117	102	101	121	123
Urea	60	47	50	48	45
Creat	0.6	0.8	0.5	0.7	0.4
Alb	2.8	2.4	2.6	2.2	2.1
Na/K	140/3.5	141/4.2	137/3.8	142/4.1	1344.0
INR	1.7	1.6	1.5	1.5	1.3

CASE 2

A 19 years old female patient, with no prior comorbidities, student, presented with chief complaints of easy fatigability last 20-25 days, mouth ulcers last 1 week, menorrhagia last 1 week, Right lower limb edema last 5 days. There was no history of fever, no history of pain abdomen, no history of cough, corrhyza. There is no history of hematemesis, malena, bleeding from any site. She visited a nearby hospital, got symptomatic treatment and came to our hospital for further evaluation.

On examination, there was no icterus, no lymphadenopathy, no clubbing, no cyanosis, JVP not raised. She had pallor, right lower limb edema with Homans positive, Moses positive sign.

On systemic examination, she was conscious oriented to time, place and person. Respiratory examination was inconclusive; no added sounds. Cardiac examination suggested of systolic murmur. The was no focal neurological deficit. Per abdomen examination revealed no organomegaly.

Her biochemical parameters were

Hb (5.2), TLC (3.52), MCV (88fl), TPC (1.52), Urea (62), creat (0.4), Ca (8.5), T.bil (0.6), SGOT (32), SGOT (21), PT, INR (1.92), uric acid (8.5), RBS (115), BP (110/70), PR (78)

Iron studies, suggestive of normal picture. Peripheral smear suggestive of moderate anisopoikilocytosis having marked degree of hypochromia. Fair number of microcytes and macrocytes were also seen.

Ultrasound whole abdomen suggested of normal study.

In view of right lower limb edema, and positive signs of DVT, USG venous and arterial Doppler was done which was suggestive of right leg DVT (non compressible venous segment, loss of phasic flow).

2D Echo done suggestive of normal study.

Special tests

ICT positive, DCT positive.

LDH 1400

Positive APLA panel {beta2 glycoprotein, cardiolipin}

Antibody typing by Gel card technique, detected antibodies {IgG, IgM, C3d}.

DVT panel, raised D Dimer (0.9), PT, APTT raised.

ANA (negative).

She was planned for blood transfusion; her blood was found incompatible, due to the presence of antibodies. It was finally decided not to transfuse blood and diagnosed to be a case of mixed AIHA. Finally, she was diagnosed to be case of AIHA with Lupus with DVT.

She was started on oral prednisolone (40mg) per day.

After 1 week, Hb rose (6.8), TLC (4000), TPC (1.8), Urea (60), creat (0.5), uric acid (8.1), t.bil (1.0).

The venous thrombosis began to resolve with negative signs of thrombosis. The patient was then disharged to follow up in further visits in OPD.

Table 2

Hb	**5.2**	**5.6**	**5.7**	**6.0**	**6.2**
Tlc	3.52	3.56	4.2	4.5	4.9
Tpc	1.14	1.00	1.20	1.78	1.56
T.bil	0.6	0.7	0.8	0.4	1.2
Na	134	135	131	136	135
K	4.0	4.0	4.8	4.2	4.9
PT/INR	1.92	1.95	1.95	1.90	1.90
SGOT	45	45	56	47	46
SGPT	35	35	45	46	49
Uric acid	8.5	8.9	8.9	8.9	8.9
D dimer				0.9	
PT			1.90		

CASE 3

A 46-year-old female presented with chief complaints of low grade fever, for last 3 months, easy fatigability last 2 months, and dyspnea on exertion last 1 month. These symptoms were gradually increasing in intensity last 2 weeks. There was no history of orthopnea, paroxysmal nocturnal dyspnea (PND), chest pain, dizziness, bleeding from any sites, vomiting, swelling, arthralgias. She was labeled as anemic and was transfused with 2 units of blood within these 2 months.

On general examination, she had pallor, icterus, bilateral pedal edema, normal JVP. BP (110/80), PR(89).

Systemic examination revealed respiratory examination to be normal with no added sounds, cardiovascular system (CVS) examination showed hemic murmur and per abdomen examination revealed a palpable liver, Central Nervous System (CNS) examination revealed no focal neurological deficit.

Biochemical parameters were

Hb 2.8 gm%, TLC 7100, platelets 1.38 lakhs, RBS 120 mg%, S. Creat 0.9 mg%, Bl. Urea 19 mg%, T Bil 5.9 mg/dl, DBil 0.8 mg/dl, IBil 5.1 mg/dl, AST 119 U, ALT 30 U, T Prot/Alb 7.1/3.3 mg/dl, ESR 150 mm, MCV 119, MCH 34.9, MCHC 29.7, high reticulocyte count 44.2%.

Peripheral smear was suggestive of anisocytosis, poikilocytosis, microcytic hypochromic red cells, target cells, tear drop cells, microovalocytes and normal leucocytes.

Vitamin B12 level was 221 ng/L, S Folate level 10.3 µg/L, S. Iron 107 µg/dL, and S. Ferritin 750 ng/mL.

HBsAg, Anti HCV, HIV negative.
She was tested and found malaria parasite to be negative, dengue negative, typhidot negative, widal showed insignificant titre. Stool for occult blood was negative. Urine routine and culture was normal.
USG whole abdomen showed hepatosplenomegaly and mild bilateral hydronephrosis. CECT Whole abdomen revealed hepatosplenomegaly with no SOL. Chest X-ray was normal.
Special tests;
Direct Coombs test positive.
Indirect coombs positive.
Reticulocyte count 44.2%.
LDH 455 U/L
ANA positive, anti ds DNA positive.
Complement C4 was 26 CAE units (normal)
The patient was thence diagnosed to be a case of systemic lupus erythematosus (SLE) with AIHA.
Patient was transfused with two units of packed red cells. She was started on prednisolone at 1 mg/kg body weight once daily. The hemoglobin level increased and patient started to feel better symptomatically. Her Hb gradually rose to 10.4 gm% and Tbil/AST/ALT were 1.3 mg%/53 U/36 U while reticulocyte count was 12.09%. Patient was discharged on a tapering course of oral steroid for follow up in OPD on a regular basis.

Table 3

Hb	2.8	6.4	7.7	10.4
TLC	7100	6600	7000	7800
TPC	1.38	1.34	1.32	1.55
T.Bil	5.9	4.04	3.08	1.3
I.Bil	5.1	3.2	2.6	0.9
Retic Count	44.2	36	20	12.09
LDH	455	400	340	250
ESR	150	100	100	80

CASE 4
A 31-year-old female patient presented to emergency with chief complaints of fever past 3 weeks, increasing pallor last 2 weeks. There was no history of cough, bleeding manifestations, no yellowish discoloration of skin, no loose stools, no headache, no edema.
On general examination, she had pallor, platynychia. There was no icterus, no cyanosis, no clubbing, no edema, no raised JVP.
Systemic examination revealed heapatosplenomegaly on abdominal examination. Rest, respiratory, neurological and musculoskeletal systemic examination were inconclusive.
Biochemical parameters:
Hb (3.2), TLC (4500), TPC (1.3), LFT (0.9/56/57/112), KFT (20/0,8), LDH (600), Chest Xray normal. BP=110/70, RBS =120, PR=110.
Peripheral smear suggestive of high reticulocyte count (2.9%) with no atypical cells.
USG suggestive of splenomegaly. CXRAY was normal.
CECT whole abdomen normal.
HIV, Anti HCV, HbS Ag, negative.
Special tests:
Direct cooms test positive
Indirect coombs test positive.
LDH (600)
ANA, ENA, Lupus anticoagulant, beta 2 glycoprotien were negative.
Antibody typing, demonstrated presence of IgG, C3d antibodies.
The patient was diagnosed to be a case of idiopathic AIHA and was started on oral prednisolone (40mg) per day during hospitalization. Within 10 days, Hb began to rise and patient started to improve symptomatically and discharged to follow up on OPD basis.

Table 4

Hb	3.2	5.2	6.2	7.9
TLC	4500	3400	4600	5000
TPC	1.30	2.00	1.60	1.89
LFT	1.1/34/45	1.3/54/45	1.2/23/23	1.8/34/24
KFT	30/0.9	34/0.5	23/0.5	39/0.7

DISCUSSION

Autoimmune hemolytic anemia (AIHA) is a condition in which the immune system produces autoantibodies that target and destroy body's own red cells, leading to premature destruction (hemolysis). This results in anemia, pallor, jaundice and sometimes splenomegaly. It is often associated with other autoimmune diseases due to underlying dysregulated immune system, like SLE, Rheumatoid Arthritis. It is this association with autoimmune diseases that highlights the importance of evaluating patients with AIHA for underlying systemic conditions to ensure comprehensive management. In this case series, the author would like to throw some light on the associated conditions like AIHA with AIH, AIHA with Lupus, AIHA with SLE.
AIH is a type of chronic hepatitis which is characterized by immune mediated attack on hepatocytes, leading to progressive liver damage. It is diagnosed by serum autoantibody,

hypergammaglobulinemia, in liver biopsy by interface hepatitis, and periportal plasma cell infiltration. There are two types of AIH: types 1 and 2. Type 1 is commonly found in adults, while type 2 is common in children and young women. Type 1 is associated with serum antinuclear antibodies (ANA) or/and anti-smooth muscle antibodies (SMA), while type 2 is associated with serum antibody to liver/kidney microsome (Anti-LKM) or antibody to liver cytosol-1 (Anti-LC1). In diagnosing AIHA, the sensitivity and specificity of ANA are 65.0% and 75.1%, respectively, and the sensitivity and specificity of SMA are 59.3% and 92.6%, respectively.[12] There are case studies that summarized that AIH-associated AIHA can be stimulated by viral infections such as Parvovirus B19 and HAV infection.[13,14] In our case, patient tested hepatitis A infection positive prior testing positive for AIHA and AIHA. The cause of AIH following HAV remains unclear, but more studies are into the way.

AIHA, is characterized by hemolysis of red cells, and hemolysis is an independent risk factor for thrombosis. The studies conducted in 1960's suggested the occurrence of pulmonary embolism in 11% of patients,[15] and a hypercoagulable state in the patients,[16] of AIHA. There are many reports that depict thrombotic manifestations are hallmark of AIHA, and about 10–20% of patients experience a thrombotic event in their lifetime.[17] Study conducted by Hendrick et al,[18] also suggested that auto-immune haemolysis increases the risk of thromboembolism. In our case of AIHA, this young patient developed, right leg thrombosis. The thrombosis began to resolve spontaneously after she was taken on steroids post one week of treatment. She is kept under follow up to keep a check on systemic progression of thrombosis if comes up in future.

Lupus anticoagulant (LA) is associated with thrombotic events rather than bleeding events. Bleeding symptoms in a patient with LA may be caused by coexisting specific antibodies directed against prothrombin (factor II). The occurrence of factor II deficiency, suspected on the basis of prolonged PT and APTT, along with LA positivity, is known as LAHPS (Lupus anticoagulant Hyperprothrombinemia Syndrome). It is a rare entity with just 100 cases reported till date.[19] This anti-prothrombin antibody causes increased clearance of prothrombin, leading to hypoprothrombinemia.[20] Thus lupus patients present with varying degrees of bleed, ranging from mild mucocutaneous bleed to severe life-threatening hemorrhages, like pulmonary and adrenal hemorrhage,specifically if the factor II level falls below 10%.[21]

Our young patient was also a case of lupus with AIHA with suspected LAHPS. We did not perform an anti-prothrombin antibody test because it was not available at our hospital. Based on the coagulation profile (raised PT, APTT, raised D dimer), the patient was provisionally kept to be a case of AIHA WITH LUPUS WITH DVT, LAHPS.

SLE is a chronic autoimmune disease with unclear etiology that affects multiple organs. The unexpected presentation and lack of definitive pathognomonic features or investigations makes the diagnosis of SLE really challenging. Distinct symptoms presentation makes the diagnosis much tougher untill differentials are discussed and a thorough work-up is done.

Hemolytic anemia, leucopenia, lymphopenia, and immune-mediated thrombocytopenia are frequently seen in patients with SLE as it affects all call lines. AIHA is a clinical diagnostic criterion for SLE. Hemolytic anemia can occur years before or after the diagnosis of SLA is made, and is rarely an initial presentation. In our case, the patient tested to be a case of SLE with AIHA, presented to Emergency department with signs of failure due to low Hb. The initial presentation was anemia which was longstanding with cause unkown. There are many case reports that had AIHA as initial and solitary manifestation of SLE.[22] Cases of Idiopathic AIHA have also been demonstrated.

The drug of choice for AIHA is steroids as seen in successful trials.[23] Corticosteroids are given at the initial dose of 1.0–1.5 mg/kg/day for 1–3 weeks until hemoglobin levels reach about 10 g/dL. After stabilization of hemoglobin, steroid should be gradually tapered at 10–15 mg weekly to a daily dose of 20–30 mg, then by 5 mg every 1–2 weeks until a dose of 15 mg, and subsequently by 2.5 mg every two weeks with the aim of taking off of the drug. Response usually occurs in the second week of treatment, and if none or minimal improvement is seen in third week, the therapy is labeled to be ineffective

Corticosteroids act by reducing hemolysis by reducing rate of hemolysis or decreasing titre of autoantibodies. The patients need to be kept on long duration of steroids, a checkup on the side effects of steroids must be kept like weight gain, osteoporosis, increased tendency to catch infections. Usually oral prednisolone is preffered but intravenous methyl prednisolone is also chosen, followed by tapering dose of oral steroids. Novel methods of treatment incorporating biologicals have also come into play

CONCLUSION

AIHA frequently coexists with other autoimmune diseases, reflecting the interconnected nature of the immune dysregulation. There are so many conditions, in which the initial presentation is hemolytic anemia, such conditions need to be worked up thoroughly highlighting the complexity of clinical spectrum of AIHA. Henceforth, recognizing these initials and keeping in mind, the associations, it is crucial to keep regular follow up as the presentation may be a part of spectrum of autoimmune diseases. Regular eye of the symptoms

is of utmost importance as they are significant markers of underlying disorder and it may influence disease progression, treatment decisions, and outcomes. Comprehensive evaluation and multidisciplinary approach are essential to optimize management, reduce complications and improve quality of life of the affected individuals.

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