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WARM, COLD AND MIXED SIGNALS : UNRAVELING THE SPECTRUM OF AUTOIMMUNE HEMOLYTIC ANEMIA

General Medicine

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ABSTRACT

Autoimmune hemolytic anemia (AIHA) is an uncommon but clinically significant hematological disorder characterized by the premature destruction of red blood cells (RBCs) due to the production of autoantibodies directed against self-erythrocyte antigens. Autoimmune hemolytic anemias have an estimated incidence in adults of 0.8-3 per 100,000/year, a prevalence of 17:100,000, and a mortality rate of 11%. Subclassification into warm, cold, mixed, and paroxysmal cold hemoglobinuria (PCH) guides diagnostic and therapeutic strategies. Here, we report three patients with distinct variants of AIHA: (i) mixed-type AIHA associated with autoimmune thyroid disease, (ii) warm AIHA with massive splenomegaly requiring extensive diagnostic evaluation, and (iii) cold agglutinin disease secondary to concomitant Mycoplasma pneumoniae and varicella infection. Each case carried a varied clinical presentation and required individualized therapy, thereby highlighting the diagnostic complexity and therapeutic challenges associated with AIHA.

KEYWORDS

Autoimmune Hemolytic Anemia, Direct Antiglobulin Test, Hematology, Hemolysis

CASE DETAILS

Case 1 - Mixed AIHA in a Patient with Autoimmune Thyroid Disease

A 32-year-old female presented with complaints of easy fatiguability, dyspnea on exertion, and yellowish discoloration of the eyes and urine for the past two weeks. There was no history of recent infections, drug use, or past Pregnancy losses.

Medical History

She had a history of hypothyroidism for the past 10 years and was on tablet levothyroxine 75 mcg once daily.

Examination Findings

On general examination, the patient had pallor and mild icterus. Mild bilateral pedal edema was noted. Abdominal examination revealed mild hepatosplenomegaly. Cardiovascular examination showed a pansystolic murmur best heard in the Aortic area. Respiratory and neurological systems were unremarkable. Table 1 depicts the course of her blood parameters and response to treatment during hospital stay.

Table 1- Laboratory Investigations

Date	Hb (g/dL)	MCV (fL)	LDH	Total/indirect bilirubin
Day 1	3.9	103.3	867	4.7/0.7
Day 2	5.0	126.9	968	4.2/0.7
Day 4	4.1	109.3		
Day 5	3.4	115.3		
Day 6	5.2	129.0		
Day 9	3.1	118.0		
Day 11	5.8	109.9		
Day 12	5.1	108.3		
Day 13	5.1	122.0	190	1.8/0.8
Day 14	5.9	138.6		
Day 18	6.0	115.9		

Additional Investigations

- Ultrasound Abdomen – Mild splenomegaly
- Direct Coombs Test (DCT): Positive
- Reticulocyte count: 14.3%
- Corrected Retic Count (CRC): 4.07%
- Reticulocyte Production Index (RPI): 2.35
- Peripheral Smear (Figure 1): Macrocytes seen, neutrophilic leukocytosis, fragmented RBCs, nucleated RBCs, polychromatophilia present → Suggestive of hemolytic anemia

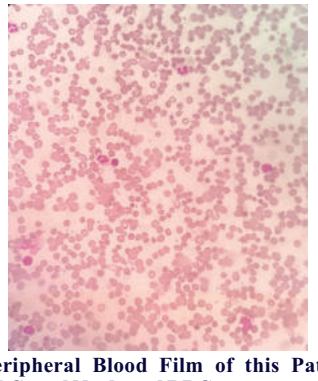

Figure 1 - Peripheral Blood Film of this Patient Showing Fragmented RBCs and Nucleated RBCs.

- Serum TSH: 30micIU/ml
- ANA: Negative
- Anti-TPO Antibody: Positive ; Anti – Thyroglobulin - Positive
- Immunohematology (IHC): Auto-control Positive @ 4°,22°,37°, DAT shows IgG and c3b/c3d antibodies, Cold agglutinin titres 1:28 ; Suggestive of Mixed Autoimmune Hemolytic Anemia

Diagnosis

Mixed-type Autoimmune Hemolytic Anemia (both warm and cold antibodies), associated with autoimmune hypothyroidism.

Treatment and Clinical Course

The patient was initiated on intravenous methylprednisolone 1 gram daily for 3 days, followed by intravenous dexamethasone 8 mg once daily for 7 days. The least incompatible Blood group-matched RBC was transfused in view of severe anemia She was then transitioned to oral prednisolone 40 mg once daily. Her Thyroxine dosage was uptitrated. The patient showed a gradual improvement in hemoglobin levels and resolution of icterus over the subsequent weeks.

Case 2- Warm Autoimmune Hemolytic Anemia with Massive Splenomegaly

Clinical Presentation

A 30-year-old female presented with complaints of easy fatiguability, exertional breathlessness, abdominal distention, and yellowish discoloration of the eyes and urine for two weeks. There was no history of fever, weight loss, joint pains, or recent drug intake.

Examination Findings
On physical examination, the patient appeared pale and icteric. Mild bilateral pedal edema was noted. Abdominal examination revealed massive splenomegaly. Cardiovascular examination was normal with no audible murmurs. No lymphadenopathy was appreciated. Table 2 depicts the laboratory investigations done during hospital stay.

Table 2- Laboratory Investigations

Timeline	Hb (g/dL)	MCV (fL)	LDH	Total/indirect bilirubin
Day 1	2.8	90	1123	3.1/1.6
Day 2	3.69	89.7	1228	
Day 5	4.2	90.3		4.1/0.9
Day 7	4.5	95.3		
Day 9	6.6	92.4		
Day 12	8.4	101.0	190	1.9/0.9

- Hemoglobin: Low (serial values from 2.8 g/dL to 8.4 g/dL; see Table 2)
- MCV: Normocytic (range: 89-101 fL)
- LDH: 1228 IU/L (elevated)
- Reticulocyte Count: 12.3%
- Reticulocyte Production Index : 2.05
- Direct Coombs Test (DCT): Positive
- Peripheral Smear: Normocytic normochromic anemia with polychromatophils, fragmented RBCs, and nucleated RBCs (nRBCs) - suggestive of hemolytic anemia
- Autoimmune Workup: ANA negative
- EBV/CMV Serology: Positive IgM for both
- Immunohematology (IHC):
  - o Autocontrol: Positive at 4°C and 37°C
  - o Cold agglutinin titre: 1:2 (insignificant)
  - o Direct Antiglobulin Test (DAT): Positive for IgG and C3b/C3d
    → Suggestive of warm autoimmune hemolytic anemia (AIHA)

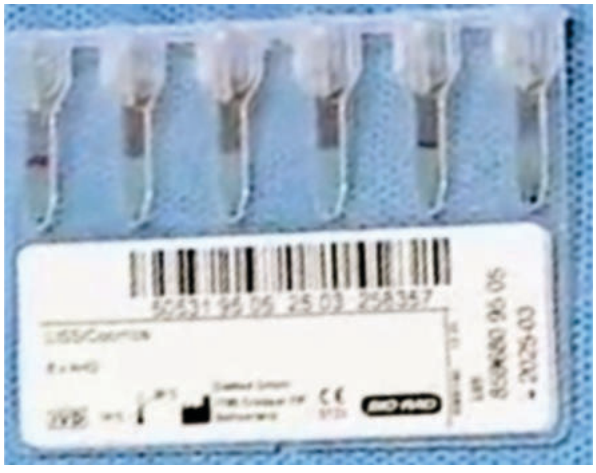

Figure 2 - Immunohematology Panel Showing Agglutination Positive for IgG and C3b/C3d.

Bone Marrow Aspiration
Performed in view of massive splenomegaly to rule out hematologic malignancy. Findings revealed trilineage hematopoiesis with adequate megakaryocytes, ruling out marrow failure or infiltration.

Diagnosis
Warm Autoimmune Hemolytic Anemia associated with viral seropositivity (EBV/CMV), in the absence of underlying autoimmune or malignant pathology.

Management and Outcome
The patient was managed with supportive transfusions and corticosteroids. Immunosuppressive therapy was initiated in the form of Tablet Azathioprine. Serial monitoring showed clinical and hematological improvement. Subsequently, Steroids were tapered.

Case 3: Cold Agglutinin Disease Secondary to Mixed Mycoplasma Pneumonia and Varicella Infection
Clinical Presentation
A 28-year-old male presented with high-grade fever, productive cough, breathlessness for 1 week. 2 days after admission, he developed vesicular rash over the trunk and upper extremities. The rash appeared 2 days after the onset of fever. Over the course of his hospital stay, he developed progressive yellowish discoloration of the eyes and complaints of passing dark-colored urine.

Examination
On general examination, the patient had pallor, icterus, and low-grade fever. Respiratory examination revealed bilateral crepitations. Skin examination showed multiple vesicular lesions consistent with varicella zoster infection in different stages of healing. There was no hepatosplenomegaly or lymphadenopathy.

Table 3 depicts the course of his blood parameters and response to treatment during hospital stay.

Table 3- Laboratory Investigations

Date	Hb (g/dL)	MCV (fL)	LDH	Total/indirect bilirubin
Day 1	14.4	100		2.2/1.0
Day 4	10.8	101.7	1087	7.8/5.3
Day 6	8.1	109.3	999	23/9.5
Day 8	7.7	102.3		
Day 11	8.0	100.4		
Day 15	9.5(post IVIG)	88.8	212	5.4/1.4
Day 17	10.1	94.7	190	1.9/0.9

- Reticulocyte count: 8.5%
- Reticulocyte Production Index : 3.12
- Peripheral smear (done after incubation at 37° to prevent agglutination) – Figure 3 : Severe anemia, macrocytes seen. polychromasia, and occasional spherocytes seen. Neutrophilic leukocytosis. Fragmented RBCs seen. In view of Agglutination at Cold temperatures and disappearance on Incubation at 37°C – possibility of Cold Agglutinin considered.

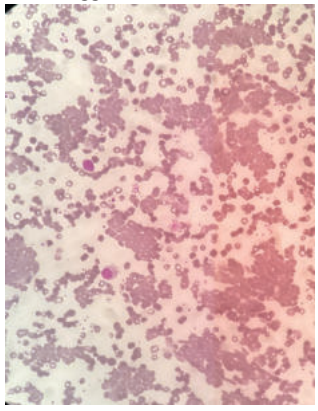

Figure 3 – Peripheral Blood Film of this Patient at 37C Showing Extensive Fragmented RBCs, Numerous Spherocytes and Macrocytes.

- Direct Coombs Test (DCT) after incubation: Positive for C3d, negative for IgG
- Cold agglutinin titre: 1:2048 at 4°C (highly significant). Repeat titres after Steroid therapy and IVIG – 1:256. (3 weeks apart)
- Mycoplasma IgM: Positive
- Varicella IgM: Positive
- Bone marrow examination: Normocellular marrow with erythroid hyperplasia (secondary to hemolysis)

Diagnosis
Cold Agglutinin Disease (secondary AIHA) triggered by co-infection with Mycoplasma pneumoniae and Varicella virus.

Management and Outcome
The patient was initially managed with antibiotics for the Pneumonia (azithromycin and Ceftriaxone), and acyclovir for varicella infection. Given worsening Hemolysis, the patient was initiated on Methylprednisolone pulse therapy for 3 days. The patient was advised to avoid cold exposure. Due to a lack of adequate response, the patient was given Intravenous Immunoglobulin at 0.4g/kg for 4 days. Over 3 weeks, there was clinical improvement with stabilization of hemoglobin (rise to 10.1 g/dL), fall in titres of Cold agglutinin antibodies and resolution of respiratory and skin symptoms. The patient was switched onto oral Steroids that were tapered over a course of 6-8weeks.

Table 4. Summary of Clinical and Laboratory Characteristics of the 3 Patients with Autoimmune Hemolytic Anemia (AIHA)

Parameter	Case 1: Mixed AIHA + Autoimmune Thyroiditis	Case 2: Warm AIHA with Massive Splenomegaly	Case 3: Cold Agglutinin Disease (Mycoplasma + Varicella)
Age / Sex	45 / Female	38 / Female	27 / Male
Presentation	Fatigue, exertional dyspnea, jaundice, dark urine	Fatigue, jaundice, dark urine	Productive cough, Anemia, jaundice post-infection
Comorbidities	Hypothyroidism (on levothyroxine, 10 years)	None	Recent Mycoplasma pneumonia & varicella infection
Examination	Pallor, icterus, mild pedal edema, hepatosplenomegaly, pansystolic murmur	Pallor, icterus, pedal edema, massive splenomegaly, no murmur	Pallor, icterus, acral cyanosis, mild splenomegaly
Hemoglobin (g/dL)	Low (severe anemia)	6.2 g/dL	Markedly reduced
Reticulocyte Count	Elevated	12.3%	Elevated
LDH (U/L)	↑	1228	↑
Bilirubin	Indirect hyperbilirubinemia	Indirect hyperbilirubinemia	Indirect hyperbilirubinemia
Peripheral Smear	Spherocytes, polychromasia	Normocytic anemia, polychromasia, fragmented RBCs, nRBCs	RBC agglutination
DAT (Direct Coombs Test)	Positive (IgG & complement)	Positive (IgG + C3d)	Positive (C3d only)
Other Tests	ANA: negative, anti-TPO: positive	EBV & CMV IgM: positive, ANA: negative, bone marrow: trilineage hematopoiesis	Cold agglutinin titer Positive, Mycoplasma & varicella serology positive
Final Diagnosis	Mixed AIHA associated with autoimmune thyroiditis	Warm AIHA (secondary to viral infection)	Cold agglutinin disease secondary to infection
Treatment	Methylpred pulse → Oral prednisolone tapered	Corticosteroids	Supportive (warmed transfusion, antimicrobials, cold avoidance) Methylpred Pulse → IVIG → Oral Prednisolone tapered
Outcome	Clinical and hematologic improvement	Good response to steroids	Hemolysis subsided with infection resolution

DISCUSSION

Autoimmune hemolytic anemia (AIHA) is a rare disorder caused by autoantibodies directed against self-red blood cells (RBC). This autoimmune process leads to hemolysis, resulting in anemia of varying severity, reticulocytosis, and elevated markers of hemolysis such as indirect bilirubin and lactate dehydrogenase (LDH).

The condition may be idiopathic (primary AIHA) or secondary to underlying disorders such as autoimmune diseases, lympho-proliferative malignancies, infections, or drug exposure. AIHA has been classified based on the thermal reactivity of the autoantibodies into warm AIHA, cold agglutinin disease (CAD), and mixed-type AIHA [1]. Among these, warm AIHA is the most prevalent form, accounting for nearly 70–80% of cases.

The disease carries considerable morbidity and mortality if unrecognized or inadequately treated. Early diagnosis, classification, and appropriate therapeutic strategies are essential to improve outcomes. This chapter explores the epidemiology, pathogenesis, classification, clinical features, diagnostic modalities, treatment approaches, complications, and recent advances in AIHA.

Epidemiology

AIHA is a rare hematological condition, with an estimated annual incidence of 1 to 3 cases per 100,000 individuals [2]. The prevalence varies according to geographic location and the underlying associated conditions. Warm AIHA accounts for the majority of cases, followed by cold agglutinin disease and mixed AIHA.

Age distribution: AIHA can occur at any age but is more frequently diagnosed in adults. Warm AIHA commonly presents in the fourth to sixth decade, whereas cold agglutinin disease often occurs in elderly individuals. Pediatric AIHA is uncommon but tends to have an acute and self-limiting course, often post-infectious.

Sex distribution: There is a female predominance, especially in warm AIHA, reflecting the autoimmune predisposition in women.

Secondary causes: Approximately 50% of AIHA cases are secondary to systemic lupus erythematosus (SLE), chronic lymphocytic leukemia (CLL), non-Hodgkin lymphoma, infections (such as Mycoplasma pneumoniae or Epstein-Barr virus), or drug-induced immune hemolysis [3].

Despite its rarity, AIHA remains an important cause of acquired hemolytic anemia, and its diagnosis requires careful exclusion of other hemolytic conditions.

Pathogenesis

The pathogenesis of AIHA involves a breakdown of immune tolerance, leading to the production of autoantibodies directed against RBC antigens. These autoantibodies cause premature destruction of red cells via extravascular or intravascular hemolysis.

Mechanisms of Hemolysis

1. Warm AIHA:

- Caused by IgG autoantibodies that bind optimally at 37°C.
- Opsonized red cells are recognized by Fc receptors on splenic macrophages, leading to extravascular hemolysis.
- Complement activation is usually incomplete, and hemolysis is predominantly splenic [4].

2. Cold Agglutinin Disease (CAD):

- Mediated by IgM autoantibodies, which bind to RBCs at lower temperatures (typically 4–30°C).
- These antibodies fix complement efficiently, leading to C3b deposition and phagocytosis in the liver.
- Severe complement activation can result in intravascular hemolysis.

3. Mixed AIHA:

- Involves both warm IgG and cold IgM autoantibodies.
- Hemolysis may be severe and resistant to therapy.

4. Drug-Induced AIHA:

Can occur by several mechanisms [5,6]:

- Hapten mechanism (e.g., penicillin) where drugs bind covalently to RBC membranes, eliciting antibody formation.
- Immune complex mechanism (e.g., quinidine) where drug-antibody complexes activate complement.
- Autoantibody induction (e.g., methyldopa) where the drug alters immune tolerance, leading to true autoantibody formation.

Classification

Autoimmune hemolytic anemia (AIHA) represents a heterogeneous group of disorders characterized by premature red blood cell (RBC) destruction mediated by autoantibodies. Classification is based on the thermal amplitude of autoantibody activity and the underlying etiology.

1. Based on Thermal Reactivity [7,8,9]

- Warm AIHA (wAIHA):

Constitutes approximately 70–80% of cases. The predominant antibodies are IgG immunoglobulins that react optimally at 37 °C. Hemolysis is typically extravascular, occurring within the splenic reticuloendothelial system. Warm AIHA is frequently secondary to autoimmune diseases (e.g., systemic lupus erythematosus), lymphoproliferative malignancies (e.g., chronic lymphocytic

leukemia), or may present as idiopathic.

- Cold Agglutinin Disease (CAD):

Accounts for 15–20% of cases and is mediated by IgM autoantibodies active between 4–30 °C. Hemolysis results from complement activation, often within the hepatic sinusoids. Secondary causes include Mycoplasma pneumoniae, Epstein–Barr virus, and indolent B-cell lymphomas.

- Mixed AIHA:

A less frequent but clinically severe subtype where both IgG and IgM autoantibodies are detected. Mixed AIHA is often refractory to first-line therapy and follows a chronic, relapsing course [10].

- Paroxysmal Cold Hemoglobinuria (PCH):

An uncommon variant, primarily seen in children following viral illnesses. It is mediated by the Donath–Landsteiner biphasic hemolysin (IgG), leading to acute intravascular hemolysis.

2. Based on Etiology

- Primary (Idiopathic) AIHA: No underlying trigger identified.
- Secondary AIHA: Associated with systemic autoimmunity (SLE, rheumatoid arthritis, Sjögren's syndrome), hematological malignancies (CLL, lymphoma), chronic infections (HIV, hepatitis viruses, EBV, Mycoplasma), or drug-induced immune responses (e.g., penicillin, cephalosporins, methyldopa).

Clinical Manifestations

The clinical spectrum ranges from compensated hemolysis with minimal symptoms to fulminant, life-threatening anemia.

- General features of anemia: Fatigue, exertional dyspnea, tachycardia, and pallor.
- Features of hemolysis: Icterus due to unconjugated hyperbilirubinemia, dark urine (hemoglobinuria), splenomegaly, and occasionally hepatomegaly.
- Cold antibody–related symptoms (CAD/PCH): Acrocyanosis, peripheral ischemia resembling Raynaud's phenomenon, and episodic hemoglobinuria following cold exposure.
- Severe presentations: Hemolytic crisis with abrupt hemoglobin fall, jaundice, hypotension, and in chronic cases, high-output cardiac failure or thromboembolic complications (particularly in CAD) [11].

Diagnostic Approach

Diagnosis requires demonstration of hemolysis and confirmation of autoantibody-mediated RBC destruction.

- Laboratory evidence of hemolysis:

Normocytic or macrocytic anemia with reticulocytosis, peripheral smear showing spherocytes (wAIHA) or RBC agglutination (CAD), elevated LDH and indirect bilirubin, and reduced serum haptoglobin. Urinalysis may reveal hemoglobinuria or hemosiderinuria.

- Direct Antiglobulin Test (DAT):

Diagnostic cornerstone.

- Warm AIHA: DAT positive for IgG ± C3
- Cold AIHA: DAT positive for C3 only
- Mixed AIHA: DAT positive for both IgG and C3
- PCH: Donath–Landsteiner test positive
- Additional tests: Cold agglutinin titers, complement fixation assays, autoimmune serologies (e.g., ANA), viral serologies, and malignancy work-up.

Management

Therapeutic strategies are individualized according to the AIHA subtype, severity, and underlying etiology.

- Supportive measures: Folic acid supplementation, avoidance of cold exposure (CAD/PCH), and transfusion of "least incompatible" packed RBCs in severe anemia.
- Warm AIHA:
- First-line: Corticosteroids (prednisone 1 mg/kg/day), with response rates of 70–85%.
- Second-line: Splenectomy or rituximab (anti-CD20 therapy).
- Rescue options: Immunosuppressants (azathioprine, cyclophosphamide, cyclosporine), and emerging biologics such as FcRn inhibitors or BTK inhibitor [12].
- Cold Agglutinin Disease:
- Corticosteroids are generally ineffective.
- Preferred therapy: Rituximab (alone or with bendamustine).
- Complement-directed therapies (sutimlimab, eculizumab) are promising, with sutimlimab already FDA-approved [13].
- Mixed AIHA: Requires combination therapy with corticosteroids and rituximab, often necessitating additional immuno-suppressants. Amongst the major subtypes of Autoimmune Hemolytic Anemias, Mixed type is commonly Resistant to 1st and 2nd line therapeutic agents [14].
- Paroxysmal Cold Hemoglobinuria: Predominantly self-limiting in children; managed with supportive care and cold avoidance.

Complications

Untreated AIHA can result in severe anemia with cardiac decompensation, recurrent infections (from immunosuppression or splenectomy), thrombosis (especially in CAD), and secondary iron overload in transfusion-dependent patients.

Recent Therapeutic Advances

- Rituximab has emerged as a mainstay in refractory AIHA.
- Sutimlimab, a humanized anti-C1s monoclonal antibody, has been recently approved for CAD, significantly reducing transfusion requirements.
- Complement inhibitors (eculizumab, pegcetacoplan) and FcRn inhibitors (nipocalimab) are under active clinical investigation.
- BTK inhibitors (ibrutinib, rilzabrutinib) represent another potential class targeting B-cell signaling pathways [15].

CONCLUSION

AIHA constitutes a rare but clinically significant cause of acquired hemolysis. Subclassification into warm, cold, mixed, and paroxysmal variants is essential for guiding diagnosis and therapy. While the direct antiglobulin test remains the diagnostic gold standard, newer immunological and molecular tools are expanding our understanding of pathophysiology. The therapeutic landscape is rapidly evolving, with biologic and complement-directed therapies offering novel, targeted options beyond conventional steroids and splenectomy. Early recognition and personalized management remain key to reducing morbidity and improving survival in patients with AIHA.

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