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Diagnosis and Treatment of Immune Heamolytic Aneamia

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ABSTRACT

Immune hemolytic anemia (IHA) stands as a multifaceted hematologic disorder characterized by accelerated destruction of red blood cells due to immune system dysregulation. This paper aims to elucidate the nuanced landscape of IHA, focusing on its diverse diagnostic approaches and evolving treatment strategies. Diagnostic considerations encompass a range of laboratory tests, including direct and indirect Coombs tests, hemolysis markers, and autoimmune antibody panels, elucidating their roles in confirming IHA etiology and subtype classification. Additionally, advancements in imaging modalities aid in identifying underlying causative factors contributing to hemolysis. The paper further scrutinizes therapeutic interventions, spanning corticosteroids, immunosuppressive agents, and monoclonal antibodies, while also appraising emerging modalities like rituximab and splenectomy. Moreover, the role of supportive care and transfusion strategies in managing IHA-associated complications is delineated. Challenges in IHA management, including refractory cases and adverse effects of therapies, are deliberated alongside the potential of future advancements in precision medicine and targeted therapies. By assimilating the latest diagnostic methodologies and treatment paradigms, this review provides a comprehensive outlook on navigating the complexities of immune hemolytic anemia to optimize patient care and outcomes.

Keywords: haemolytic anaemia, anaemia, immunity, diagnosis, treatment

INTRODUCTION

Anaemia is a clinical condition in which the body does not have enough healthy red blood cells that provide oxygen to the body's tissues. Red blood cells last for about 120 days before the body gets rid of them [1]. The immune haemolytic anaemia comprise a set of diseases characterized by shortened red blood cell survival (haemolysis) mediated by the action of antibodies and serum complement. The haemolysis is evidenced by a raised reticulocyte count. Immune haemolytic anaemia occurs when antibodies form against the body's own red

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blood cells and destroy them. This happens because the immune system recognizes these blood cells as foreign [2]. The great majority of acquired haemolytic anaemias are mediated by autoantibodies. In contrast, congenital haemolytic anaemia, such as hereditary spherocytosis are due to an intrinsic defect in red blood cell (RBC) structure. During the first half of the twentieth century, clinicians experienced difficulty in distinguishing acquired from congenital haemolytic anaemia. Elegant cross-transfusion experiments using the Ashby differential agglutination technique to determine RBC lifespan in-vivo permitted the first distinction between these two disorders reviewed by [3]. In these experiments, RBCs from normal individuals exhibited shortened survival in the circulation of patients with acquired haemolytic anaemia, suggesting that an extrinsic defect was responsible for the haemolysis. In contrast, RBCs from patients with congenital haemolytic anaemia exhibited shortened survival in the circulation of normal individuals, suggesting the presence of a defect intrinsic to the RBC. The development of the antiglobulin test [4] and its application to patients in the 1940s [5] provided the defining distinction between acquired and congenital haemolytic anaemia, namely the presence of antibody or complement fragments on RBCs in the former, and their absence in the latter. History of immunology autoimmune haemolytic anaemia is classified in two complementary ways: by the temperature (warm or cold) at which autoantibodies bind most efficiently to patients' RBCs and according to the absence or presence of a related underlying disease (primary or secondary). A third category, drug-induced immune hemolytic anaemia, may involve autoantibodies directed against patient RBC antigens, or antibodies directed against drugs or their metabolites [6].

Mechanism of Red Blood Cells Destruction

Normal human red blood cells have an average life span of about 120 days in the circulation after which they are engulfed by macrophages. The immune destruction of red cells can occur as a complement mediated intravascular process or extravascularly where the red cells are destroyed by macrophages following interaction with cell-bound IgG1,IgG3, and/or C3b. Red cells can be destroyed in vivo by humoral mechanism or by a combined humoral and cellular immune mechanism [7].

Three (3) basic mechanisms explain the immune destruction of red blood cells.

- Extravascular hemolysis. It begins as macrophages in the liver or spleen capture red blood cells by attaching to antibodies or to complement C3b molecules or both, and damage the red blood cell membrane. Macrophages capture and phagocytize red blood cells that are coated with antibodies or complement C3b molecules or both. Red blood cells coated with IgG are destroyed primarily in the spleen, and IgM-coated cells are destroyed primarily in the live [8].
- Intravascular haemolysis. Occurs when complement proteins C5 through C9 attach to red blood cells, forming pores that allow the cell contents to leak out. Since IgM and IgA antibodies are efficient at binding and activating complement, both intravascular and extravascular haemolysis can occur when these antibodies are involved.
- Complement System. This may become activated and complement molecules may attach to red blood cells if the antibody is an IgM, IgA, IgGI, or lgG3, but not lgG2 or lgG4 [9].

CLINICAL FEATURES

The symptoms experienced by patients with autoimmune haemolytic anaemia are influenced largely by the rapidity of the haemolysis. The signs and symptoms of immune haemolytic anaemia can vary considerably, depending on the rapidity of the haemolysis, the degree of anaemia, and the presence of any underlying diseases. Most patients with warm autoantibody mediated haemolysis, some patients with cold autoantibody-mediated haemolysis and those with drug-immune haemolysis of the drug adsorption or true autoantibody types experience a gradual on set of anaemia, and thus their symptoms appear gradually. In these patients, RBC destruction occurs primarily in the spleen (extravascular haemolysis). Such patients may complain of easy fatigue, dyspnoea on exertion, rapid heartbeat, malaise, pale skin, Shortness of breath, fatigue, dizziness, angina, pallor, and jaundice may be present in immune haemolytic anaemia [10-11].

- Immune haemolysis immune cytopenia (such as, autoimmune thrombocytopenia).
- Underlying autoimmune disorders (such as, systemic lupus erythematosus).
- Lymphoproliferative disorders (such as, chronic lymphocytic leukemia).
- Pregnancy complicated by haemolytic disease of the newborn.
- Transfusions reactions.
- Viral, parasitic or bacterial infection.

Laboratory Diagnosis/Investigations of Immune Haemolytic Anaemia

The most common laboratory investigations of immune haemolytic anaemia include:

- Reticulocytes count: In mature red blood cells are called reticulocytes. One thing the medical team will be looking for is the percentage of immature red blood cells in the blood circulation using reticulocte count, if the body has a lot of them, it means that your bone marrow is working overtime

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to keep pace with a high rate of red blood cell destruction that is associated with haemolytic anaemia. Raised reticulocyte count is common to patients with immune or autoimmune haemolytic anaemia to help the body system to adjust to the lower oxygen levels at high altitudes [12].

- Direct antiglobulin or Direct Coombs' test: Is a test used to identifies anti RBCs antibodies or complement adhered to RBCs, it looks for antibodies against the red blood cells. If your body is making these antibodies, you will be confirm coomb's positive. The single most important test for establishing and characterizing an immune haemolytic anaemia process is the direct antiglobulin test (DAT or Coombs' test) [13]. Originally developed to be used before transfusion to detect red cell alloantibodies to minor blood group antigens (eg, Kell, Duffy, Rh) and thus provide an increased margin of safety in cross matching blood, the DAT has proved valuable for other purposes as well, such as detecting, characterizing, and investigating the red cell autoantibodies and drug-related antibodies associated with red cell haemolysis [14]. The DAT uses standardized preparations of antihuman globulin to detect IgG or complement (the C3d component) coating the red cell. It can detect as few as 100 to 200 molecules of IgG per red cell, but in rare cases of immune haemolysis the DAT can be negative if there are fewer than 100 to 150 IgG molecules per red cell. In cases of autoimmune hemolytic anemia due to IgM or IgA, the DAT is still often positive owing to the presence of complement on the red cell. In cases in which the DAT is positive with anti-IgG antihuman globulin, the antibody can be eluted off the red cell and its characteristics further examined. The eluate can be tested against reagent red cells of known antigenic phenotype to determine the specificity of the IgG antibody (e.g., anti-D, panagglutinating antibody). An elution study can also provide clinically useful information in cases of known or suspected red blood cell alloimmunization secondary to transfusion or pregnancy [4].
- Peripheral Blood Film: Red Cell Morphology in peripheral blood film shows fragmentation, spherocytes and blister cells in which haemoglobin appears to have separated from the cell membrane. Spherocytes may be seen on blood smears. This is small RBC with a loss of central pallor produced by incomplete destruction of RBCs by macrophages. They form when macrophages remove a segment of red blood cells without the loss of intracellular content. Spherocytosis is a very suggestive of immune haemolytic anaemia [14]. The mean corpuscular volume may be elevated because of reticulocytosis. The reticulocyte count is usually elevated in haemolytic anaemia (sometimes as high as 30%), but it is normal or low in as many as 25% of cases. The peripheral blood smear may demonstrate increased spherocytes, polychromatophilia, and occasionally, nucleated red cells. The spherocytosis can be high enough to be confused with hereditary spherocytosis. Red cell agglutination or rouleaux formation may be visually and grossly evident in cold antibody autoimmune haemolytic anaemia.
- Serum bilirubin: Common serum biochemistry changes in immune haemolytic anaemic patients is hyperbilirunemia with accelerated red blood cells destruction, increased bilirubin production by marcophages can overwhelm hepatic processing capacity resulting in hyperbilirunemia. However, it may also be due to concurrent hepatobiliary disease. Normal bilirubin level is often seen in mind or chronic cases of immune haemolytic anaemia because healthy liver can still handle the extra bilirun [14].
- Lactate dehydrogenase: Lactate dehydrogenase is an enzyme that's present in red blood cells. When red blood cells are destroyed, the level will rise especially in cold autoimmune haemolytic anaemia.
- Haptoglobin test: Haptoglobin is a protein that eliminate debris produced by damaged red blood cells. If the body is using up a lot of haptoglobin, the level will be low.
- Osmotic Fragility Test: The most noticeable abnormality of red cells of patients with autoimmune haemolytic aneamia with warm autoantibodies is the increased osmotic fragility of the red cells. This appeared to be most pronounced in patients with incomplete IgG warm autoantibody with occur invivo. It is caused by damage done to the sensitized red blood cells [15].
- Bone marrow erythroid hyperplasia
- Serum ferritin: Ferrintin is a blood protein that stores iron. A ferrintin blood test can indicate if iron levels are high or low
- Examination of the bone marrow is generally shows erythroid hyperplasia

Laboratory Abnormalities Associated with Immune Heamolytic Anaemia (Red Cell Hemolysis):

Hyperbilirubinemia (unconjugated/indirect).
Decreased serum haptoglobin
Decreased serum hemopexin
Increased serum methemalbumin
Increased serum lactate dehydrogenase
Increased (mild) serum aspartate aminotransferase
Increased serum free hemoglobin (hemoglobinemia)

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Increased urine hemoglobin (hemoglobinuria)

TREATMENT

- Corticosteroids therapy: Treatment with corticosteroids is usually the first line of treatment for autoimmune disorders of the red cell, particularly in cases of warm autoimmune haemolytic anaemia. A total daily dose of 60 to 100mg of prednisonlone produces a clinical response in approximately 80% to 90% of patients with warm autoimmune hemolytic anaemia. After 2 or 3 days of therapy, the first indication of a response may be a transient increase in the reticulocyte count, followed by increases in the hemoglobin and hematocrit levels. The DAT (direct Coombs' test) may continue to be positive despite improvement in other clinical and laboratory measures. Most patients show a response to corticosteroids within 10 to 14 days, but the response may not be maximal until after 3 weeks of therapy. Patients with life-threatening hemolysis may benefit from larger doses of corticosteroids, such as 100 to 200 mg of methyl prednisolone or an equivalent drug, given intravenously in divided doses over 24 hours. Once the patient has responded to prednisolone, the dose can be gradually reduced over the ensuing weeks and months until the lowest possible dose is achieved. Alternate-day prednisone therapy should be started as soon as clinically feasible after the patient is stable. Approximately 20% to 30% of patients achieve a lasting remission with prednisone therapy, 50% continue to require some form of low-dose maintenance prednisone therapy for months, and another 10% to 20% either do not respond to prednisone therapy or require unacceptably high doses. Corticosteroid therapy has generally not shown clinical efficacy in patients with uncomplicated cold agglutinin autoimmune hemolytic anaemia, paroxysmal cold hemoglobinuria, or drug-induced immune hemolysis.
- Cytotoxic drugs: If corticosteroid therapy does not produce a remission or if the patient has a relapse, additional immunosuppressive therapy may be needed. Cytotoxic drugs such as azathioprine and cyclophosphamide have been used with some success. In adults, cyclophosphamide 60 mg/m2 or azathioprine 80 mg/m2 per day can be considered. Some investigators consider cytotoxic drugs to be a third- or fourth-line treatment option.
- Splenectomy: Splenectomy may be the first choice of treatment in some types of hemolytic anaemia, such as hereditary spherocytosis if other measures have failed [16].

In patients with warm autoimmune hemolytic anaemia who do not respond to corticosteroid therapy, splenectomy is often the second line of therapy. However, only patients with IgG autoantibody (whose red cells are destroyed primarily in the spleen) would be expected to benefit from splenectomy. Not all patients respond to splenectomy, and those who show an initial response may relapse and require additional immunosuppressive therapy. The need to give pneumococcal vaccine before the splenectomy, as well as the need for any long-term prophylactic antibiotic therapy, should be assessed in the individual patient.

- Intravenous immunoglobulin: A number of studies have examined the efficacy of intravenous immunoglobulin in treating autoimmune hemolytic anaemia, primarily of the warm antibody type. However, the success rate has been variable and not as encouraging as in other immune disorders.
- Folic acid: (1 mg per day) is usually recommended as long as there is ongoing hemolysis, because this nutrient is necessary for red blood cell maturation.

Alternate therapy for refractory cases Cyclosporine shows promise in autoimmune hemolytic anaemia refractory to conventional therapy, or when an alternative to splenectomy is being considered, according to several papers, mostly case reports. The dosage was usually 4 to 6 mg/kg/day by mouth, and side effects were minimal. Plasma exchange has been used in a few cases, often as an adjunct to other therapies in refractory cases. Vincristine, danazol, and other therapies have been used in small numbers of refractory cases. Treating specific hemolytic diseases Underlying diseases and infections associated with autoimmune hemolytic anaemia or paroxysmal cold hemoglobinuria should be treated, and any drugs implicated or proven as a cause for the patient's immune hemolysis should be stopped. In cold agglutinin disease, avoiding cold ambient temperatures is often all that is necessary to provide symptomatic relief. In addition, in hospitalized patients, intravenous solutions may need to be warmed before infusion. Corticosteroids and splenectomy have not generally been shown to be effective in patients with cold antibody autoimmune hemolytic anaemia. Transfusion reactions when treating proven or suspected alloimmune hemolysis secondary to transfusion it is critical to discontinue the transfusion, as there is a direct relationship between morbidity and mortality and alloimmune hemolysis. The primary complications with transfusion-associated alloimmune hemolysis are acute renal failure and disseminated intravascular coagulation. Treatment is primarily supportive. The efficacy of corticosteroids or intravenous immunoglobulin in such cases has not been validated and cannot be recommended as standard therapy. Additional reviews on the treatment of hemolytic transfusion reactions should also be consulted. Transfusion Consultation with a transfusion medicine specialist is extremely important in hemolysis. Finding compatible blood for patients with autoimmune

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hemolytic anaemia can often be difficult and challenging. The antibody in patients with warm autoimmune hemolytic anaemia typically demonstrates activity against all red cells (except Rh-null red cells); therefore, all crossmatch will be "incompatible." Transfused red cells will also become coated with antibody and undergo immune hemolysis at the same rate as the patient's own red cells. Thus, the beneficial effect of transfusion in immune haemolysis will be temporary. There is always a risk of an acute or delayed hemolytic transfusion reaction after any transfusion, and an immune hemolytic process may make such reactions more difficult to detect and prevent. However, in patients clearly in need of transfusion because of cardiovascular, central nervous system, or pulmonary decompensation, red cells should not be withheld, even if crossmatch compatibility cannot be confirmed or guaranteed. Other important supportive measures in such patients include maintenance of blood volume with intravenous fluids and volume expanders, oxygen therapy, and maintenance of renal function. Although transfusion can precipitate warm-antibody autoimmune hemolytic anemia in otherwise-normal patients, it is less clear whether transfusion alone increases the severity of autoimmune hemolytic anaemia in patients who already have ongoing immune haemolysis [17-25].

- Transfusion Therapy. One should avoid transfusion unless absolutely necessary. However, transfusion may be essential for patients with angina or a several compromised cardiopulmonary status. It is best to administer packed red cells slowly to avoid cardiac stress. In antoimmune hemolytic aneamia (AIHA), typing and cross-matching may be difficult. One should use the least incompatible blood if transfusion is indicated. The risk of destruction of transfused blood is high, but the degree of hemolysis depends on the rate of infusion. Therefore, one should slowly transfuse half units of packed red cells to prevent rapid destruction of transfused blood and iron overload due to multiple transfusions [17-25].

CONCLUSION

The diagnosis and treatment of immune haemolytic anaemia study is aimed to identify the causes and stepwise approach to maintain the effective immune system and increase the number of healthy and normal red cells to carry oxygenated blood to the body tissues for general wellbeing of the body.

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