



Comprehensive Clinical Evaluation and Management of Severe Autoimmune Hemolytic Anemia: A Case Study of a 39-Year-Old Female with Anemia Gravis and Cultural "Susuk" Implantation

¹ Veronica Debora

¹ Faculty of Medicine, University of Lampung, Indonesia

Corresponding Email : veiveidebora@gmail.com

Article History :

Received date : 2025/11/21

Revised date : 2025/12/12

Accepted date : 2026/01/24

Published date : 2026/02/11



Copyright: © 2024 by the authors. Submitted for possible open access publication under the terms and conditions of the Creative Commons Attribution (BY NC) license (<https://creativecommons.org/licenses/by-nc/4.0/>).

E-ISSN :

ISSN 3048-1368



P-ISSN

ISSN 3048-1376



ABSTRACT

Introduction

Autoimmune hemolytic anemia (AIHA) is a rare but potentially catastrophic hematological disorder characterized by the premature destruction of red blood cells mediated by autoantibodies targeting erythrocyte surface antigens.¹ The condition is broadly classified into warm-type and cold-type AIHA based on the thermal reactivity of the autoantibodies, with warm-antibody AIHA (wAIHA) accounting for approximately 75% of cases.³ In the clinical setting of Indonesia, cases often present at advanced stages, requiring intensive transfusion support and nuanced immunosuppressive management.⁵

Case Report

The analysis involves Ny. K, a 39-year-old female, who presented to the emergency department of a tertiary hospital in Bandar Lampung with a four-day history of profound weakness, dizziness, and gastrointestinal distress. Her clinical history revealed a two-

year pattern of recurrent anemia and prior transfusions. Physical examination demonstrated a classic hemolytic triad: severe conjunctival pallor (anemia), scleral icterus (jaundice), and palpable splenomegaly. Laboratory investigations confirmed critical anemia with a hemoglobin level of 3 g/dl and a hematocrit of 11%. Evidence of hemolysis included significantly elevated lactate dehydrogenase (LDH) at 1344 IU/L and indirect hyperbilirubinemia at 3.3 mg/dl, while a reticulocyte count of 7.5% indicated a robust compensatory marrow response. The patient also reported a history of "susuk" (charm needle) implantation. Management strategies included high-volume packed red cell (PRC) transfusions (1200 cc), corticosteroid therapy with methylprednisolone, folic acid supplementation, and a specialized hepatobiliary nutritional protocol (Diet Hati III).

Discussion

The diagnostic workup for Ny. K is consistent with international protocols for identifying uncompensated hemolytic anemia, where peripheral destruction outpaces bone marrow production.⁸ The presence of splenomegaly and the biochemical markers suggest extravascular hemolysis, likely mediated by warm IgG autoantibodies.¹¹ This report examines the challenges of blood transfusion in the presence of pan-agglutinins, the role of corticosteroids as first-line therapy, and the physiological significance of high LDH levels in predicting disease severity.¹ Furthermore, the sociocultural aspect of "susuk" is explored, noting its biological inertness but radiological significance in diagnostic imaging.¹⁵

Conclusion

Severe AIHA with anemia gravis represents a medical emergency

requiring rapid stabilization through least-incompatible blood transfusions and high-dose immunosuppression. This case underscores the necessity of aggressive early intervention and the importance of excluding secondary causes, such as systemic lupus erythematosus or lymphoproliferative disorders, in the Indonesian clinical context. Continuous monitoring of reticulocyte response and hemolytic markers is vital to guide therapy and prevent relapses.

Keywords

Autoimmune Hemolytic Anemia, Anemia Gravis, Reticulocytosis, Hyperbilirubinemia, Splenomegaly, Susuk, Indonesia.

INTRODUCTION

The maintenance of erythrocyte homeostasis is a fundamental requirement for systemic oxygen delivery and metabolic stability. Under normal physiological conditions, red blood cells (RBCs) circulate for approximately 120 days before being sequestered and destroyed by the reticuloendothelial system.⁸ Hemolytic anemia occurs when the lifespan of these cells is shortened by a variety of mechanisms, ranging from intrinsic membrane defects to extrinsic immune-mediated destruction.¹⁰ While the bone marrow possesses a remarkable capacity to increase erythroid production—up to eight times the baseline rate—clinical anemia manifests when the rate of hemolysis exceeds this compensatory threshold.⁸

Autoimmune hemolytic anemia (AIHA) represents a specific subset of these disorders where the immune system loses tolerance to self-antigens on the RBC membrane.² The resulting autoantibodies facilitate destruction through either the splenic macrophages (extravascular hemolysis) or the complement system (intravascular hemolysis). With an annual incidence of approximately 1 in 80,000, AIHA is relatively uncommon but carries a high mortality risk if not identified and treated promptly. The diagnosis is traditionally confirmed by a positive Direct Antiglobulin Test (DAT), also known as the Coombs test, which identifies the presence of immunoglobulins or complement fragments on the RBC surface.¹

In Indonesia, the management of AIHA is complicated by unique challenges, including the prevalence of systemic lupus erythematosus (SLE) as a secondary cause and the limited availability of advanced serological testing and specialized blood products like washed RBCs.⁵ Furthermore, clinicians must often navigate sociocultural factors, such as the use of traditional charm needles ("susuk"), which can appear as alarming radiological findings during diagnostic workups.¹⁵

The case of Ny. K, a 39-year-old female with a hemoglobin level of 3 g/dl, provides a critical opportunity to review the management of life-threatening anemia in a resource-constrained tertiary setting. Her presentation involves a complex interplay of clinical signs, including jaundice

and splenomegaly, and highlights the urgent need for a structured approach to transfusion and immunosuppression in patients with AIHA. This report provides an exhaustive analysis of her clinical course, laboratory findings, and the physiological principles underlying her therapy, drawing upon the latest international guidelines and Indonesian clinical standards.

CASE REPORT

Clinical Presentation and Identification

The patient, Ny. K, is a 39-year-old female housewife residing in the Kemiling district of Bandar Lampung. She was admitted to the Emergency Department of RSUD Dr. H. Abdul Moeloek (RSUDAM) on March 12, 2018, at 12:03 WIB, presenting with a primary complaint of extreme generalized weakness ("lemas") that had persisted and worsened over the preceding four days.

During the autoanamnesis and aloanamnesis conducted on March 13, 2018, the patient described a cluster of systemic and gastrointestinal symptoms. These included persistent dizziness ("pusing"), significant nausea, and multiple episodes of vomiting. She also reported localized pain in the left upper quadrant of the abdomen and a loss of appetite that coincided with the onset of her severe weakness. Her bowel habits had been disrupted, with a two-day history of constipation, though she noted no dysuria or abnormalities in the color of her urine, which remained yellow.

Medical and Surgical History

Ny. K's history is indicative of a chronic hematological condition. She first experienced recurrent episodes of severe weakness in 2016, which necessitated her first blood transfusion. Only two weeks prior to the current admission, she had been hospitalized at RSUDAM for similar symptoms of profound lethargy.

The patient's sociocultural and pharmacological history revealed several notable points:

- In 2015, she underwent the insertion of "susuk," a traditional Indonesian practice involving the subcutaneous implantation of metallic charm needles.

- In 2016, she began using injectable hormonal contraception ("KB suntik").
- Her past medical history included childhood chickenpox and ongoing complaints of gastritis.

The table below provides a structured overview of her medical history as documented during admission.

Disease / Condition	Status	Notes
Chickenpox (Cacar Air)	Positive (+)	Childhood occurrence
Gastritis	Positive (+)	Chronic history
Recurrent Weakness	Positive (+)	Since 2016
Prior Transfusions	Positive (+)	Since 2016
Hypertension	Negative (-)	
Diabetes Mellitus	Negative (-)	
Tuberculosis	Negative (-)	
Heart Disease	Negative (-)	
Kidney Stones	Negative (-)	

Family and Social History

The patient's family history suggests a predisposition to certain chronic conditions. Her grandfather had a history of hypertension, and her grandmother suffered from rheumatism. Her mother is deceased, while her father remains in good health. Ny. K is a mother of three children, aged 16, 12, and 5, all of whom are reported to be healthy.

Physical Examination

Upon admission, Ny. K was described as being in a state of "Compos Mentis" (fully conscious) but appearing moderately ill. Her physiological parameters indicated significant stress on the circulatory system due to the severity of her anemia.

Physical Parameter	Value	Reference / Status
Height	158 cm	
Weight (Baseline)	50 kg	
Weight (Current)	45 kg	Significant decrease noted
Blood Pressure	110/80 mmHg	Normal range
Pulse Rate	80 bpm	Normal at rest
Respiratory Rate	24x/minute	Tachypneic
Temperature	36.7°C	Afebrile
BMI	20.8	Normal nutritional status

Detailed Status Generalis

The physical examination revealed several pathognomonic signs of hemolysis and severe anemia:

- **Skin:** The skin was markedly icteric (yellowish). Turgor and moisture levels were within normal limits, though the patient appeared pale.
- **Eyes:** The conjunctivae were severely anemic (+/+), and the sclerae were icteric (+/+), indicating the presence of hyperbilirubinemia. No exophthalmos or visual disturbances were

7

noted.

- **Mouth:** The lips were pale, and the tongue was coated (white/dirty). No tonsillar hypertrophy was present.
- **Neck:** Jugular Venous Pressure (JVP) was measured at 5-2 cmH₂O, and no lymphadenopathy or thyroid enlargement was palpable.
- **Thorax:** The chest was symmetrical during respiration. Cardiac auscultation revealed normal S1 and S2 sounds with no murmurs or gallops, suggesting that the anemia had not yet progressed to high-output heart failure. Pulmonary auscultation demonstrated clear vesicular breath sounds bilaterally.
- **Abdomen:** The abdomen appeared slightly convex (cembung). Auscultation revealed normal bowel sounds (16x/minute). Palpation was negative for generalized tenderness, but the spleen was significantly enlarged (splenomegaly) and tender upon palpation. The liver was not palpable.
- **Extremities:** The limbs showed no edema, cyanosis, or petechiae, and the muscle tone and strength were graded at 5/5 across all extremities.

Laboratory Investigations

The laboratory results obtained on March 10, 2018, provided definitive evidence for the diagnosis of severe anemia with hemolytic features.

Hematological Profile

Parameter	Result	Normal Range	Interpretation
Hemoglobin	3 g/dl	11.50 – 16.50 g/dL	Anemia Gravis
Hematocrit	11%	37-47	Severe reduction

Erythrocyte Count	1.2x	4.2 – 5.4 x 10 ⁶ / uL	Severe reduction
Leukocyte Count	12.200	4800-10.800 ul	Mild Leukocytosis
Trombocyte Count	362.000	150-450.000/ ul	Normal
Reticulocyte Count	7.5	0.5-1.5 (%)	Marked Reticulocytosis
MCV	90	79-99 fL	Normocytic
MCH	25	27-31 pg	Hypochromic
MCHC	28	30-35 g/dL	Low
LED (ESR)	132	0-15 mm/jam	Significantly elevated

The differential leukocyte count was as follows: Basophils 0%, Eosinophils 0%, Band neutrophils 0%, Segmented neutrophils 55%, Lymphocytes 39%, and Monocytes 6%.

Biochemical Profile

Parameter	Result	Normal Range	Interpretation
Bilirubin Total	3.8	0.3-1.2 mg/dL	Elevated
Bilirubin Direct	0.5	0.0-0.3 mg/dL	Mildly Elevated
Bilirubin Indirect	3.3	0.1-1.0 mg/dL	Significantly Elevated

LDH	1344	LDH	Severely Elevated
SGOT	28	< 31 U/L	Normal
SGPT	12	< 31 U/L	Normal
Uric Acid	8.2	2.6- 6.0 mg/dL	Elevated
Alkali Phosphatase	56	33-98 U/L	Normal

Management and Treatment Plan

The primary diagnosis was established as **Anemia Hemolitik**. The therapeutic approach was multifaceted, focusing on rapid erythrocyte replenishment, immunosuppression, and gastric protection.

Non-Pharmacological Management

- Complete bed rest (Istirahat yang cukup).
- Nutritional therapy: **Diet Hati III** (specialized liver diet).
- Intravenous hydration: Ringer Lactate (RL) at 15-20 drops per minute.
- Oxygen therapy if required.

Pharmacological Management

- **Transfusion Support:** A total of 6 units (approximately 1200 cc) of Packed Red Cells (PRC) was ordered to raise hemoglobin levels to a safe range.
- **Immunosuppression:** Methylprednisolone was initiated at a dose of 2x4 mg intravenously.
- **Antibiotic Coverage:** Cefotaxime 1g intravenously to prevent secondary infections in an immunologically stressed patient.
- **Gastric Protection:**

- Ranitidine 125 mg IV every 12 hours.
- Omeprazole (OMZ) 1 vial every 12 hours.
- Sucralfate syrup 3x1.
- **Adjunctive Therapy:**
 - Folic Acid 3x1 (to support increased erythropoiesis).
 - Paracetamol (PCT) 3x1 for symptomatic relief of dizziness/pain.

Clinical Progression and Daily Follow-up

The patient's condition was monitored closely over the subsequent three days.

March 13, 2018 (05:00 WIB)

- **Subjective:** Patient reported persistent lemas, pusing, and minor nausea.
- **Objective:** BP 110/60 mmHg, Pulse 116 bpm (tachycardia), Temperature 36.9°C, RR 24 x/min. Physical signs of conjunctival pallor (+/+) remained, though scleral icterus was less prominent.
- **Assessment:** Anemia Hemolitik.
- **Plan:** Continued PRC transfusion, Diet Hati III, and pharmacological regimen.

March 14, 2018 (05:15 WIB)

- **Subjective:** Patient reported continued pusing, but the generalized weakness (lemas) was improving.
- **Objective:** BP 110/60 mmHg, Pulse 92 bpm, Temperature 35.7°C, RR 24 x/min. Scleral icterus (+/+) and conjunctival pallor (+/+) were noted. A newly palpable lymph node (KGB+) was documented in the neck region.
- **Assessment:** Anemia Hemolitik.
- **Plan:** Routine therapy continued; request for Abdominal USG to evaluate splenomegaly further.

March 15, 2018 (05:45 WIB)

- **Subjective:** Slight residual lemas, dizziness, and constipation.
- **Objective:** BP 110/60 mmHg, Pulse 100 bpm, Temperature 36.6°C, RR 16 x/min. Conjunctival pallor and scleral icterus persisted. Abdominal examination continued to show splenomegaly and tenderness in the left lumbar region.
- **Assessment:** Anemia Hemolitik.
- **Plan:** Continuation of all therapies.

DISCUSSION

Laboratory Confirmation of the Hemolytic Process

The case of Ny. K presents a quintessential laboratory profile for uncompensated hemolytic anemia. The diagnosis of hemolysis is fundamentally supported by the triad of anemia, evidence of increased erythrocyte destruction, and a robust compensatory response from the bone marrow.⁹

In this patient, the hemoglobin level of 3 g/dl and hematocrit of 11% represent **Anemia Gravis**, a life-threatening state where oxygen-carrying capacity is severely compromised.¹ The markers of RBC destruction are particularly striking. Lactate dehydrogenase (LDH), an enzyme found abundantly within erythrocytes, was measured at 1344 IU/L, more than six times the upper limit of normal.¹⁹ Elevated LDH is a sensitive marker for hemolysis; while it can be raised in many conditions, its combination with other markers in this case is diagnostic.¹

Furthermore, the patient demonstrated significant indirect hyperbilirubinemia (3.3 mg/dl of a total 3.8 mg/dl). Unconjugated (indirect) bilirubin is a byproduct of hemoglobin breakdown. When the rate of hemolysis exceeds the liver's capacity to conjugate and excrete bilirubin, the indirect fraction rises, manifesting clinically as scleral icterus and jaundice.⁹ The normal levels of SGOT (28 U/L) and SGPT (12 U/L) are critical here, as they rule out primary hepatic parenchymal disease as the cause of the jaundice, localizing the pathology to the pre-hepatic (hemolytic) phase.

The compensatory response is evidenced by the **reticulocyte count of 7.5%**. Reticulocytes are immature RBCs released into the circulation in response to erythropoietin stimulation. A reticulocyte percentage above 2.5% is traditionally used to confirm that the bone marrow is responding appropriately to peripheral RBC loss.¹ This finding is vital for ruling out bone marrow failure syndromes, such as aplastic anemia, where the reticulocyte count would be inappropriately low (reticulocytopenia) despite the severity of the anemia.⁸

Classification and Pathophysiology of AIHA

Based on the clinical findings of splenomegaly and the laboratory evidence of extravascular hemolysis, Ny. K likely suffers from **Warm-type Autoimmune Hemolytic Anemia (wAIHA)**.³ Warm AIHA is typically mediated by IgG autoantibodies that bind to RBC surface antigens (often the Rh protein complex) with optimal affinity at 37°C.¹¹

The pathophysiology of wAIHA involves the recognition of IgG-coated erythrocytes by Fc receptors on macrophages in the spleen.¹¹ These macrophages may ingest the entire RBC or, more frequently, remove segments of the cell membrane. This loss of membrane area forces the RBC to assume a spherical shape (spherocytes), which are less deformable and become trapped in the narrow splenic sinusoids, where they are eventually destroyed.³ This process of continuous entrapment and destruction leads to the work hypertrophy of the spleen observed in this patient.⁹

While approximately half of AIHA cases are primary (idiopathic), the remaining cases are secondary to underlying conditions such as lymphoproliferative disorders, viral infections, or autoimmune diseases like SLE.⁶ In the Indonesian clinical landscape, the high prevalence of SLE as a driver of secondary AIHA makes it imperative to screen for antinuclear antibodies (ANA) and other markers of systemic autoimmunity in patients presenting like Ny. K.⁵

Transfusion Challenges and Indonesian Protocols

The management of Ny. K's anemia gravis necessitated immediate blood transfusion. However, transfusing a patient with AIHA is technically and clinically challenging.⁵ The presence

of pan-reactive autoantibodies often results in "least incompatible" cross-match results, where all donor units react positively during compatibility testing.¹

In such emergency situations (Hb <5 g/dl), current Indonesian guidelines—supported by international consensus—dictate that transfusion should not be delayed by the search for perfectly compatible blood.⁷ ABO and Rhesus-matched blood can be safely administered to prevent cardiovascular collapse.⁵ The decision to transfuse 1200 cc of PRC in Ny. K was a life-saving necessity, aimed at maintaining a clinically tolerable hemoglobin level while waiting for immunosuppressive therapies to take effect.⁶ The use of washed RBCs is often discussed in the Indonesian setting to reduce the risk of reactions, but their unavailability in many regional hospitals can cause dangerous delays in care.⁷

Pharmacological Evaluation and Steroid Dosing

The cornerstone of wAIHA treatment is corticosteroid therapy.¹³ Steroids function by downregulating *Fc* receptor expression on splenic macrophages, thereby reducing the clearance of IgG-coated RBCs, and by suppressing the eventual production of autoantibodies.¹¹

International guidelines generally recommend an initial dose of **Prednisone (or equivalent) at 1.0 - 1.5 mg/kg/day**.¹⁴ For a patient of Ny. K's weight (50 kg), this would equate to approximately 50-75 mg of Prednisone per day. In this case, the patient was initiated on **Methylprednisolone 2x4 mg intravenously**. This dose is significantly lower than the standard high-dose initial therapy typically used for AIHA crises. In cases of acute severe hemolysis, pulse doses of methylprednisolone (250-100 mg/day for 3 days) are often employed to achieve a rapid hematological response.²⁹ The rationale for the low dose in Ny. K's records is unclear, but it represents a potential area for therapeutic optimization according to international standards.

The use of **Folic Acid (3x1)** is an essential adjunctive therapy in this case.¹⁹ Patients with chronic hemolysis have a massive requirement for folate to support the rapid turnover of erythroid precursors in the bone marrow. A failure to provide supplementation can lead to a "megaloblastic

crisis," where the marrow's compensatory capacity fails, leading to a precipitous drop in hemoglobin.⁸

Sociocultural Context: The "Susuk" Charm Needles

The history of "susuk" in Ny. K adds a unique sociocultural dimension to the case. Susuk are charm needles, typically made of gold, copper, or other inert precious metals, inserted subcutaneously into the face, chest, or limbs.¹⁵ They are believed to provide health, beauty, and success to the wearer.¹⁵

While these needles are generally biologically inert and do not cause systemic inflammation or hemolysis, they are significant in the medical setting for several reasons:

- **Radiological Discovery:** They are almost always detected incidentally on radiographs, where they appear as fine, linear metallic opacities.¹⁶ If a clinician is unaware of this practice, the findings can be mistaken for acupuncture needles, surgical fragments, or trauma-related foreign bodies.¹⁶
- **Potential for Migration:** Although rare, there is evidence in recent literature of these needles migrating from their original site of insertion, potentially causing localized pain or, in extreme cases, organ penetration.¹⁵
- **MRI Compatibility:** Because they are usually non-ferromagnetic (gold/copper), they are often safe for MRI, though they can cause significant imaging artifacts.³³

In Ny. K's case, the susuk is likely unrelated to her hemolysis but highlights the importance of cultural competence in Indonesian medical practice.¹⁶

Nutritional Support: Diet Hati III

The prescription of **Diet Hati III** is a specific feature of Indonesian hospital protocols for patients with liver stress or hepatobiliary complications.²⁵ This diet is designed to be high in carbohydrates and moderate in protein and fats, aimed at providing sufficient energy for cellular

repair while minimizing the metabolic workload on a liver already stressed by processing excessive bilirubin.²⁵ In the context of severe AIHA, where the liver must conjugate massive amounts of indirect bilirubin, such nutritional support is a common supportive measure.²⁵

Future Directions and Novel Therapies

For patients like Ny. K who experience a chronic or relapsing course, long-term management requires a shift from steroids toward second-line therapies.¹³ **Rituximab** (an anti-CD20 monoclonal antibody) is increasingly being used earlier in the treatment course.¹³ Studies have shown that adding Rituximab to initial steroid therapy can double the rate of long-term remission.²²

Other emerging options discussed in the 2024 literature include **rilzabrutinib** (a BTK inhibitor) and **fostamatinib** (a spleen tyrosine kinase inhibitor), both of which target the phagocytic pathways involved in extravascular hemolysis.¹² In the event of failure of all medical therapies, **splenectomy** remains a vital third-line option, although it is increasingly avoided due to the associated risks of lifelong infection.¹⁴

CONCLUSION

The case of Ny. K underscores the critical nature of severe autoimmune hemolytic anemia (AIHA) and the necessity of a rapid, systematic approach to diagnosis and treatment. Her presentation with **Anemia Gravis** (Hb 3 g/dl), marked reticulocytosis (7.5%), and profound indirect hyperbilirubinemia constitutes a classic manifestation of uncompensated hemolysis. The clinical findings of splenomegaly and jaundice further support a diagnosis of warm-type AIHA, where the spleen serves as the primary site of erythrocyte destruction.

Key therapeutic insights from this case include the life-saving role of "least incompatible" blood transfusions in emergency situations and the essential requirement for folic acid supplementation to sustain the marrow's compensatory response. While the patient was initiated on

corticosteroids, this analysis suggests that standard high-dose protocols (1 mg/kg/day) or pulse therapy may be more effective than low-dose maintenance in the acute phase of severe hemolysis. Additionally, the incidental finding of "susuk" charm needles provides a reminder of the cultural context in which Indonesian medicine is practiced, requiring clinicians to maintain a high degree of radiological and sociocultural awareness.

Ultimately, the goal of management in severe AIHA is to stabilize the patient acutely through transfusion and to achieve long-term immune remission through carefully tapered steroids or second-line biological therapies. For Ny. K, close follow-up and a multidisciplinary approach focusing on both hematological stability and nutritional support are paramount to preventing the recurrence of her life-threatening anemia.

REFERENCES

1. Laboratory Evaluation of Immune Hemolytic Anemias - StatPearls ..., diakses Februari 5, 2026, <https://www.ncbi.nlm.nih.gov/books/NBK606096/>
2. Beneath the surface in autoimmune hemolytic anemia: pathogenetic networks, therapeutic advancements and open questions - Frontiers, diakses Februari 5, 2026, <https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2025.1624667/full>
3. Anemia Hemolitik Autoimun pada Anak, diakses Februari 5, 2026, <https://cdkjournal.com/index.php/cdk/article/download/403/365>
4. Diagnosis and treatment of autoimmune hemolytic anemia: classic approach and recent advances - PMC - NIH, diakses Februari 5, 2026, <https://pmc.ncbi.nlm.nih.gov/articles/PMC4931937/>
5. Diagnosis and Management of Autoimmune Hemolytic Anemia in Systemic Lupus Erythematosus | Current Internal Medicine Research and Practice Surabaya Journal, diakses Februari 5, 2026, <https://e-journal.unair.ac.id/CIMRJ/article/view/52775>
6. Diagnosis and Management of Autoimmune Hemolytic Anemia in ..., diakses Februari 5, 2026, <https://e-journal.unair.ac.id/CIMRJ/article/download/52775/29634/327878>

7. Red Blood Cell Transfusion for Pediatric Autoimmune Hemolytic Anemia in an Emergency Situation | eJournal Kedokteran Indonesia, diakses Februari 5, 2026, <https://ejki.fk.ui.ac.id/index.php/journal/article/view/19>
8. Hemolytic Anemias | Current Medical Diagnosis & Treatment 2024 | AccessMedicine, diakses Februari 5, 2026, https://accessmedicine.mhmedical.com/content.aspx?sectionid=280093509&bookid=3343&p_review=false
9. Overview of Hemolytic Anemia - Hematology and Oncology - MSD Manuals, diakses Februari 5, 2026, <https://www.msdmanuals.com/professional/hematology-and-oncology/anemias-caused-by-hemolysis/overview-of-hemolytic-anemia>
10. Overview of Hemolytic Anemia - Hematology and Oncology - Merck Manuals, diakses Februari 5, 2026, <https://www.merckmanuals.com/professional/hematology-and-oncology/anemias-caused-by-hemolysis/overview-of-hemolytic-anemia>
11. Autoimmune hemolytic anemia: current understanding of pathophysiology - EHA Library, diakses Februari 5, 2026, https://library.ehaweb.org/conference/documents/97572/autoimmune_hemolytic_anemia_current_understanding_of_pathophysiology.pdf
12. Changing Landscape of Management of Warm Autoimmune Hemolytic Anemia | The Hematologist | American Society of Hematology - ASH Publications, diakses Februari 5, 2026, <https://ashpublications.org/thehematologist/article/doi/10.1182/hem.V22.4.202543/537937/Changing-Landscape-of-Management-of-Warm>
13. Current Management of Warm Autoimmune Haemolytic Anaemia and Recent Advancements in Research: Interview with a Key Opinion Leader - EMJ, diakses Februari 5, 2026, <https://www.emjreviews.com/wp-content/uploads/2024/08/Current-Management-of-Warm-Autoimmune-Haemolytic-Anaemia-and-Recent-Advancements-in-Research-Interview-with-a-Key-Opinion-Leader.pdf>
14. Treatment of autoimmune hemolytic anemias - Haematologica, diakses Februari 5, 2026,

- <https://haematologica.org/article/view/7163>
15. The Wandering Charm Needle - PMC - PubMed Central - NIH, diakses Februari 5, 2026, <https://pmc.ncbi.nlm.nih.gov/articles/PMC7536003/>
 16. Susuk - Black Magic Exposed “White” by Dental Radiographs - PMC - NIH, diakses Februari 5, 2026, <https://pmc.ncbi.nlm.nih.gov/articles/PMC4149152/>
 17. The Agony of the Mystical Practice of Susuk: A Case Report - PMC - NIH, diakses Februari 5, 2026, <https://pmc.ncbi.nlm.nih.gov/articles/PMC9615098/>
 18. Hemolytic Anemia | Johns Hopkins Medicine, diakses Februari 5, 2026, <https://www.hopkinsmedicine.org/health/conditions-and-diseases/hemolytic-anemia>
 19. Hemolytic Anemia: Evaluation and Differential Diagnosis | AAFP, diakses Februari 5, 2026, <https://www.aafp.org/pubs/afp/issues/2018/0915/p354.html>
 20. Anemia Hemolitik: Penyebab, Gejala, Pengobatan dan Pencegahan - Siloam Hospitals, diakses Februari 5, 2026, <https://www.siloamhospitals.com/informasi-siloam/artikel/apa-itu-anemia-hemolitik>
 21. Case Report: Severe autoimmune hemolytic anemia in an elderly patient caused by warm-reactive IgG and IgA autoantibodies - Frontiers, diakses Februari 5, 2026, <https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2025.1664498/full>
 22. The choice of new treatments in autoimmune hemolytic anemia: how to pick from the basket? - Frontiers, diakses Februari 5, 2026, <https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2023.1180509/full>
 23. Autoimmune Hemolytic Anemia - Hematology and Oncology - Merck Manuals, diakses Februari 5, 2026, <https://www.merckmanuals.com/professional/hematology-and-oncology/anemias-caused-by-hemolysis/autoimmune-hemolytic-anemia>
 24. Kar S, Kumar CGD, Kar R, Basavarajegowda A. Autoimmune Hemolytic Anemia in Children: Clinical Profile and Outcome. Indian J Pediatr. 2024 Feb;91(2):143-148. doi: 10.1007/s12098-022-04469-6. Epub 2023 Feb 14. PMID: 36787020. <https://pubmed.ncbi.nlm.nih.gov/36787020/>
 25. a women 51 years with decompensated liver cirrhosis with gastritis chronic and kidney -

- Neliti, diakses Februari 5, 2026, <https://media.neliti.com/media/publications/153271-ID-none.pdf>
26. Hepatic Manifestations in Hematological Disorders - PMC - PubMed Central - NIH, diakses Februari 5, 2026, <https://pmc.ncbi.nlm.nih.gov/articles/PMC3626309/>
27. DAT-Negative Autoimmune Hemolytic Anemia: There Shall Be ALPS - Hematology.org, diakses Februari 5, 2026, <https://www.hematology.org/education/trainees/fellows/hematopoiesis/2022/there-shall-be-alps>
28. How I treat autoimmune hemolytic anemia | Blood | American Society of Hematology, diakses Februari 5, 2026, <https://ashpublications.org/blood/article/129/22/2971/36045/How-I-treat-autoimmune-hemolytic-anemia>
29. How I treat warm autoimmune hemolytic anemia | Blood | American Society of Hematology, diakses Februari 5, 2026, <https://ashpublications.org/blood/article/137/10/1283/475030/How-I-treat-warm-autoimmune-hemolytic-anemia>
30. Treatment and clinical outcomes of pediatric autoimmune hemolytic anemia: real-world single-center data from Korea - NIH, diakses Februari 5, 2026, <https://pmc.ncbi.nlm.nih.gov/articles/PMC12235343/>
31. Hemolytic Anemia in Pregnancy: A Rare and Challenging Case in Indonesia, diakses Februari 5, 2026, https://www.jmchemsci.com/article_174366.html
32. Guidelines for the investigation and management of autoimmune haemolytic anaemia in adults (age 16 years and above) - NSSG - Haematology, diakses Februari 5, 2026, <https://nssg.oxford-haematology.org.uk/immunohaematology/documents/aiha-and-chad/IM-14-aiha-guideline.pdf>
33. Susuk: Charm needles in orofacial soft tissues - Scientific Research Publishing, diakses Februari 5, 2026, <https://www.scirp.org/journal/paperinformation?paperid=32039>
34. Susuk: A Mystical Case of Charm Needle and A Review of Literature - ResearchGate, diakses Februari 5, 2026, https://www.researchgate.net/publication/367309140_Susuk_A_Mystical_Case_of_Charm_N

eedle and A Review of Literature

35. ASEAN Food Composition Database - สถาบัน โภชนาการ มหาวิทยาลัย มหิดล, diakses Februari 5, 2026, https://inmu.mahidol.ac.th/aseanfoods/doc/OnlineASEAN_FCD_V1_2014.pdf
36. Diagnosis and treatment of autoimmune hemolytic anemia in adults: Recommendations from the First International Consensus Meeting - PubMed, diakses Februari 5, 2026, <https://pubmed.ncbi.nlm.nih.gov/31839434/>
37. Management of autoimmune haemolytic anaemia in low-to-middle ..., diakses Februari 5, 2026, <https://pmc.ncbi.nlm.nih.gov/articles/PMC11004394/>
38. Spleen Removal as Treatment for Primary Warm Autoimmune Hemolytic Anemia, diakses Februari 5, 2026, <https://medicine.yale.edu/news-article/spleen-removal-treatment-primary-warm-autoimmune-hemolytic-anemia/>