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Clinical Presentation and Treatment Outcomes in Paediatric Autoimmune Hemolytic Anemia: A Case Series Analysis

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ABSTRACT

Background: Autoimmune hemolytic anemia (AIHA) is an acquired disorder characterized by immune-mediated destruction of red blood cells due to the production of autoantibodies against erythrocyte membrane antigens. In the pediatric population, the clinical spectrum is wide, ranging from mild, self-limiting illness to rapidly progressive and potentially life-threatening disease. Early identification, timely starting of therapy, and active monitoring are essential for optimal outcomes.

Purpose: The present case series aims to study the clinical presentation, course, and treatment outcomes of AIHA in children.

Methods: This case series was conducted over a period of two years (January 2024 to December 2025) in the Department of Paediatrics, Government Medical College, Palakkad. The present case series analysed the children admitted with characteristics of AIHA.

Results: We identified 4 children who presented with lethargy, severe pallor, jaundice, and hemoglobinuria. Direct Agglutination Test (DAT) was done for all 4 patients. DAT was positive in 3 patients but negative in 1 patient. Treatment with steroids was given to all 4 patients after ruling out alternate causes for DAT-negative haemolytic anemia. One child among the 4 cases had a fulminant course with cardiac decompensation but was managed successfully. One of our cases developed features of steroid toxicity, and we had to cut short the steroid course after 3 months. All children were followed up for one year, and none had any relapse.

Conclusion: Early diagnosis and early initiation of therapy have to be done in children with AIHA, and these children should be closely monitored for symptoms of steroid toxicity as they require a longer duration of steroid therapy.

1. Introduction

Autoimmune hemolytic anemia (AIHA) is an acquired hematological disorder in which the immune system of an individual starts destructing the red blood cells due to autoantibodies directed against erythrocyte membrane antigens. Although AIHA occurs across all age groups, it is uncommon in children, with an estimated annual incidence of about 0.8 per 100,000 individuals younger than 18 years (Aladjidi *et al.*, 2011; Sharma *et al.*, 2024). It accounts for a small proportion of pediatric anemias but can present with significant morbidity. AIHA may occur as a primary (idiopathic) condition or secondary to infections, autoimmune diseases, immunodeficiency, malignancy, or drugs. A slight male predominance is observed in younger children, while female predominance increases with age, particularly in association with systemic autoimmune disorders such as systemic lupus erythematosus. Evans syndrome (AIHA with immune thrombocytopenia) represents approximately 15–30% of pediatric cases (Chandra *et al.*, 2024).

AIHA results from immune dysregulation leading to the production of autoantibodies against red blood cell (RBC) membrane antigens. Mechanisms include breakdown of central and peripheral immune tolerance, autoreactive B- and T-cell activation, complement activation, and cytokine-mediated destruction. Molecular mimicry, commonly following viral infections, may alter RBC membranes or trigger cross-reactive immune responses. In warm AIHA, IgG-coated RBCs experience extravascular

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hemolysis mediated by splenic macrophages. In cold AIHA, IgM antibodies attach to complement and then get destroyed by the complement system. In extreme situations, membrane attack complexes lead to swift hemolysis (Chandra *et al.*, 2024; Maguire *et al.*, 2024).

AIHA can be classified according to the temperature at which the pathogenic antibodies are most active. It could be warm antibody–mediated, cold antibody–mediated, mixed-type AIHA, and paroxysmal cold hemoglobinuria (Loriamini *et al.*, 2024; Michalak *et al.*, 2020). Warm antibody–mediated AIHA is caused by immunoglobulin G (IgG) autoantibodies that are active at 37 °C or higher. Cold antibody–mediated AIHA involves immunoglobulin M (IgM) autoantibodies that react at lower temperatures. Mixed-type AIHA is defined by the coexistence of both warm and cold autoantibodies. Paroxysmal cold hemoglobinuria represents a rare form of immune-mediated hemolytic anemia and is observed more frequently in children than in adults. It is mediated by the Donath–Landsteiner antibody, which is an IgG cold-reactive antibody. The etiological classification of AIHA as primary (idiopathic) or secondary can be made based on the presence/absence of an underlying disease (such as immunodeficiency, infections, medications, connective tissue disorders, or malignancy) (Packman, 2006).

Diagnosis of pediatric AIHA relies on clinical features of hemolysis (pallor, jaundice, dark urine) and laboratory findings, including anemia, reticulocytosis, elevated lactate dehydrogenase, indirect hyperbilirubinemia, and low haptoglobin. The direct antiglobulin test (DAT) is the main test used to diagnose this condition, and it is positive in most cases. However, 5–10% of patients may not have a positive DAT because they have low-affinity antibodies, IgA autoantibodies, or low levels of RBC-bound IgG, which makes diagnosis difficult. In these situations, more sensitive methods or ruling out other hemolytic conditions are needed. Finding secondary causes is very important for prognosis and treatment (Chandra *et al.*, 2024).

Management of AIHA aims to control hemolysis, stabilize hemoglobin levels, and minimize transfusion requirements. Corticosteroids (prednisolone 1–2 mg/kg/day) are the first-line treatment for warm AIHA, and about 80% of people respond to them. In severe cases, methylprednisolone can be given through IV. Rituximab is the preferred second-line therapy for steroid-refractory or steroid-dependent disease and is also recommended in cold AIHA. In supportive care regimens, folic acid is prescribed, and severe cases are managed by blood transfusions. In chronic or recurrent cases, steroid-sparing drugs like azathioprine, mycophenolate mofetil, or cyclosporine may be used. Regular follow-up is essential to monitor treatment relapse and adverse effects (Chandra *et al.*, 2024).

Owing to the rarity of AIHA in children, standard management guidelines are not available. This case series highlights the diagnostic and therapeutic challenges of pediatric AIHA in a resource-limited tertiary care setting, where advanced immunohematological investigations may not be readily available. The expected outcome of the study is to increase awareness of the clinical presentation and management of this uncommon disease in the pediatric age group.

2. Case Details

Total 4 children admitted with characteristics of AIHA we analyzed in this series conducted at the department of Paediatrics, Government medical College, Palakkad over a period of 2 years (Figure 1).

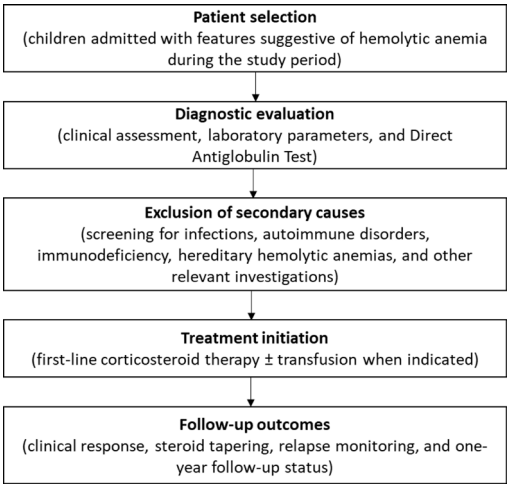


Figure 1: Flow Diagram Illustrating the Study Methodology, Including Patient Selection, Diagnostic Evaluation, Exclusion of Secondary Causes, Initiation of Corticosteroid Therapy with Supportive Management, and Assessment of Follow-Up Outcomes in Children with Autoimmune Hemolytic Anemia.

Case 1: A four-year-old male child presented with fever and cough for 5 days and dark-coloured urine for 1 day. He had a history of fever, running nose, and loose stools in the past month, for which he had received Paracetamol, Cetirizine, Zinc, and ORS. On examination, he was found to have pallor, jaundice, and hepatosplenomegaly. Investigations revealed severe anemia, indirect hyperbilirubinemia, elevated LDH, and peripheral smear with evidence of haemolysis and reticulocytosis. Direct agglutination test (DAT) was positive, and a diagnosis of AIHA was made. To rule out secondary causes, Cold agglutinin test, ANA-IFA, serum immunoglobulin profile, HIV, HBsAg, and HCV testing were done and were found to be negative. Important investigations of the child are depicted in Table 1.

By the second day of admission, the haemoglobin rapidly dropped to 2.8 mg/dL, and the child developed features of congestive cardiac failure. The patient was initiated on intravenous methylprednisolone at a dose of 1 mg/kg every 8 hours for two days, after which therapy was transitioned to oral prednisolone at 2 mg/kg/day administered in divided doses. He also required two PRBC transfusions (10 mL/kg each) and was empirically started on oral Azithromycin considering the possibility of infection-induced AIHA. The hemoglobin levels showed a steady rise, and the Direct Antiglobulin Test became negative by the ninth day of steroid therapy. The child was discharged on the eleventh day of hospitalization with a hemoglobin level of 11.8 g/dL. Oral prednisolone was maintained at 2 mg/kg/day for 14 days, followed by gradual tapering and discontinuation over a period of 4–6 months. During one year of follow-up, the child remained asymptomatic and did not require further blood transfusions.

Table 1: Trend of Hematological and Biochemical Parameters During Hospitalization in a 4-Year-Old Male with AIHA, Demonstrating Severe Hemolysis at Presentation with Gradual Hematological Recovery Following Treatment. (DAT: Direct Antiglobulin Test; LDH: Lactate Dehydrogenase; PRBC: Packed Red Blood Cells; Hb: Hemoglobin)

Parameter	**D1**	**D2**	**D3**	**D4**	**D6**	**D8**	**D11**
Hemoglobin (g/dL)	7.0	5.5	2.8	5.0	8.4	9.8	11.8
Direct Agglutination Test (DAT)	—	Positive	—	—	—	—	Negative
Total / Direct Bilirubin (mg/dL)	2.6 / 0.5	—	—	3.0 / 0.5	1.0 / 0.3	—	—
Reticulocyte Count (%)	—	5.9	—	4.0	—	4.0	—
Lactate Dehydrogenase (U/L)	793	—	—	—	—	—	—
Peripheral Blood Smear	Normocytic normochromic anemia with evidence of accelerated hematopoiesis and occasional microspherocytes						

Case 2: A five-year-old male child with recent history of fever, increased frequency and urinary discolouration was evaluated and treated at a peripheral health care facility as urinary tract infection with some oral antibiotics (details of which were not available) and supportive measures. The child was referred to us on day 4 with pallor, jaundice, and cola coloured urine. At admission, haemoglobin was 4 g/dl and he had mild hepatosplenomegaly. Laboratory evaluation demonstrated a positive Direct Antiglobulin Test along with supportive features of hemolysis, including indirect hyperbilirubinemia, elevated lactate dehydrogenase levels, increased reticulocyte count, red cell agglutination, and the presence of occasional microspherocytes on peripheral blood smear. The diagnosis of AIHA was made and evaluation for secondary causes, ANA-IFA, screening for HIV, HCV, HbSAg, cold agglutination tests, and serum immunoglobulin profile was done, and all were negative. The child was started on oral prednisolone at 2 mg/kg/day in divided doses. The clinical course was uneventful with the lowest haemoglobin of 3.7 (without any features of CCF) and the haemoglobin levels showing an increasing trend after 2 days of oral prednisolone. At discharge (day 12 of prednisolone), the child had a haemoglobin of 7.9 g/dl. After 2 weeks of discharge, Hb became 10.8 g/dl and DAT was negative, hence prednisolone was continued in tapering doses. However, at later follow up visits the child developed features of steroid toxicity (cushingoid facies and hypertension @ prednisolone 1 mg/kg/day) and hence steroids were tapered and stopped within a total duration of 12 weeks. The child in subsequent follow up visits was closely monitored for relapse but remained asymptomatic.

Case 3: A diagnosis of DAT-negative AIHA was considered as a diagnosis of exclusion in a seven-year-old boy who presented with fever, pallor, icterus, and laboratory features of hemolysis. The boy was referred from the periphery in view of low haemoglobin levels (4.8 mg/dl). Investigations showed other supportive evidence of hemolytic anemia. DAT was done three times and was negative. No

significant perinatal, past, or family history was noted. Also, no significant recent drug history was noted. In view of hepatomegaly and Coombs negative hemolysis, Wilsons was ruled out (S. Copper and Ceruloplasmin levels were normal). Since DAT was negative, investigations for hereditary hemolytic anaemias were done (including peripheral smear, osmotic fragility, G6PD levels, sickling test, and Hb electrophoresis) and ruled out. Since the child was not improving, the possibility of DAT negative AIHA was considered and he was started on oral prednisolone at 2 mg/kg/day in divided doses. Hb improved gradually over the subsequent days, and the child was discharged after 1 week with a haemoglobin of 9.2 g/dl. The child was kept under follow up and was asymptomatic and transfusion free. Steroids were tapered and stopped in 5 months. G6PD levels were repeated after 3 months, and it was found to be normal.

Case 4: A five-year-old girl with history of fever and cough for 4 days. On examination she was found to have pallor, mild icterus, and splenomegaly. No significant recent drug history was noted. Haemoglobin at admission was 6.2 g/dl. Other investigations revealed evidence of hemolysis (increased indirect hyperbilirubinemia, elevated LDH, and peripheral smear with evidence of haemolysis and reticulocytosis) and DAT was positive. The haemoglobin levels in subsequent days dropped till 4.5 g/dl and plateaued. She was started on oral Prednisolone considering the diagnosis of AIHA. Over the subsequent days the patient's course was steady, without features of deterioration. The child responded to the steroid therapy and was discharged after 12 days with a haemoglobin of 10.2 g/dl. The child showed no relapse during the subsequent follow up. Prednisolone was tapered and stopped in around 4.5 months.

3. Discussion

In our series, 4 cases of AIHA were included, including 3 boys and 1 girl. The median age at diagnosis was 5 years. All four cases gave a history of antecedent fever. Two out of four showed symptoms of a preceding acute respiratory infection. Laboratory evaluation demonstrated a positive Direct Antiglobulin Test along with supporting features of hemolysis, including indirect hyperbilirubinemia, elevated lactate dehydrogenase levels, increased reticulocyte count, red cell agglutination, and the presence of occasional microspherocytes on peripheral blood smear. None had associated thrombocytopenia or neutropenia. Three out of our four patients had positive DAT, and one was DAT negative. Clinical and laboratory parameters of the children are given in Table 2.

A total of four children were included in the study. The mean age at diagnosis was 5.25 years (median 5 years; range 4–7 years) with a male predominance (male: female = 3:1). The mean hemoglobin at admission was 5.5 g/dL (range 4.0–7.0 g/dL), while the mean lowest documented hemoglobin was 3.75 g/dL (range 2.8–4.5 g/dL). The mean total leukocyte count was 14,800/μL and the mean platelet count was 4.9×10^5/μL. Direct Antiglobulin Test was positive in three children (75%) and negative in one child (25%). One child (25%) required packed red blood cell transfusion. The mean duration of steroid therapy was 4.6 months (range 3–6 months) (Table 2).

Table 2: Clinical characteristics, laboratory findings, and treatment details of children with AIHA. *(DAT: Direct Antiglobulin Test; LDH: Lactate Dehydrogenase; PRBC: Packed Red Blood Cells; Hb: Hemoglobin)*

Age at onset (years) / Sex	Gender	Clinical presentation	Hb (g/dL)	PCV	Lowest Hb documented (g/dL)	Total WBC count (/μL)	Platelet count ($\times 10^5$/μL)	Poly-specific DAT	Treatment given	Blood transfusion	Total duration of therapy
4 yrs	Male	Fever and cough (5 days), dark-coloured urine	7	21	2.8	12,300	6.6	Positive	IV methylpre-dnisolone followed by oral prednisolone	Yes (2 units PRBC, 10 mL/kg each)	6 months
5 yrs	Male	Fever, increased frequency of micturition (4 days), dark-coloured urine	4	11.7	3.7	17,300	3.9	Positive	Oral prednisolone	No	3 months

7 yrs	Male	Fever (3 days), giddiness, dark-coloured urine	4.8	11.9	4	14,600	4.7	Negative	Oral prednisolone	No	5 months
5 yrs	Female	Fever and cough (4 days)	6.2	18.6	4.5	15,000	5	Positive	Oral prednisolone	No	4.5 months
Mean age: 5.25 years	Male: Female ratio: 3:1	Mean duration: 4 days	Mean: 5.5	Mean: 15.8	Mean: 3.75	Mean: 14,800	Mean: 5.05	Positive: 75%	All received steroids	25% required transfusion	Mean duration of therapy: 4.6 months
Median age: 5 years	Median: 4 days Median: 5.5 Median: 3.85							Negative: 25%			
Range: 4–7 years		Range: 3–5 days	Range: 4.0–7.0	Range: 11.7–21	Range: 2.8–4.5	Range: 12,300–17,300	Range: 3.9–6.6				

All our patients received first line corticosteroid therapy. In three out of four cases, the treatment was started with oral Prednisolone at 2 mg/kg/day in divided doses. In case 1, as the child had a rapidly progressive course, IV methyl prednisolone was given for the initial 2 days and then changed to oral prednisolone. The response to steroids was apparent (rising haemoglobin values) in all four cases within 24 to 72 hours and haemoglobin levels returned to near normal values in an average of 10 to 14 days. All children also showed a negative DAT by this time. After normalization of haemoglobin, the steroids were tapered slowly. Among the four children in our series, one child developed features of steroid toxicity (steroid induced hypertension) and required rapid dose reduction and discontinuation of prednisolone in a total of 12 weeks. In the remaining patients, prednisolone was tapered gradually over a period of approximately six months, as rapid dose reduction or abrupt cessation has been associated with an increased risk of disease relapse (Naithani *et al*., 2007). In the study conducted by Arora *et al*. (2021) prednisolone at a dose of 2 mg/kg/day was administered as first line therapy to ten children. Among those who showed inadequate response or relapse, second line treatment was initiated: two patients received oral cyclosporine (5 to 6 mg/kg/day in divided doses), while three were treated with rituximab at 375 mg/m² once weekly for four weeks. Rituximab was well tolerated, and no significant infectious or other adverse effects were reported during therapy (Arora *et al*., 2021).

In a study by Weli *et al*. (2020) involving thirteen patients, complete remission was achieved in seven cases, while four patients required additional immunosuppressive therapy, including cyclosporine A, azathioprine, and mycophenolate mofetil (Weli *et al*., 2020). Another study by Arora *et al*. (2021) including 11 children with AIHA reported that seven responded to the first line Prednisolone therapy, but the remaining four needed additional treatment including Rituximab, Cyclosporine, MMF, and 6 MP (Arora *et al*., 2021). In our study, complete remission was obtained in all 4 cases with the first line steroid therapy alone. None required any second line or additional therapy. Also, during our period of study and follow up, all four remained asymptomatic and transfusion free, with no relapse. In the study conducted by Fan *et al*. (2016) 29 of 45 patients (64.4%) maintained sustained remission throughout the follow up period. However, 35.6% experienced relapse after achieving their initial complete remission. Among those who relapsed, 56.3% responded favourably to reintroduction of glucocorticoid therapy, indicating that a substantial proportion retained steroid responsiveness despite recurrence (Fan *et al*., 2016).

The gold standard for diagnosis of AIHA is DAT. However, up to 11% of patients with warm AIHA exhibit clinical and laboratory features of immune mediated hemolysis despite a negative Direct Antiglobulin Test, a condition referred to as DAT negative warm AIHA (Kalfa,

2016; Naithani *et al.*, 2007). Possible explanations for DAT negative AIHA include hemolysis mediated by IgA autoantibodies that are not detected by standard testing reagents, the presence of low affinity IgG antibodies, or red blood cell bound IgG levels below the detection threshold of routine assays (Garratty, 2005). In such situations, the use of monospecific antisera directed against IgA, along with techniques such as low ionic strength saline (LISS) enhancement or cold washing of red cells, may help detect otherwise DAT negative cases. Nevertheless, despite the availability of advanced testing methods, approximately 5% of AIHA cases remain DAT negative, and the diagnosis is ultimately established after excluding alternative causes of hemolysis and observing a favourable clinical response to treatment (Hill & Hill, 2018).

In a two-year study conducted by Arora *et al.* (2021) at a super specialty pediatric hospital and postgraduate teaching institute in Delhi, India, one out of eleven children were diagnosed with DAT negative autoimmune hemolytic anemia. In this patient, a high burden of IgA autoantibodies bound to red blood cells was detected, showing a strong (4+) reaction on monospecific DAT gel card testing, and the child was successfully treated with a short course of prednisolone (Arora *et al.*, 2021). In our case series we had 1 patient in whom we diagnosed DAT negative AIHA. This child had fever and running nose for 2 days followed by generalised tiredness, pallor and brownish urine. Alternate causes of hemolysis causing similar clinical presentation were ruled out, like G6PD deficiency and Hemolytic Uremic Syndrome (HUS). Since we could not do further testing for DAT negative AIHA in our resource poor setting, we treated the child with steroids and the child improved very well.

4. Conclusion

Children with AIHA sometimes follow a fast progressive and life-threatening course. So prompt diagnosis and early initiation of treatment is of paramount importance. In a small percentage of AIHA, DAT is negative. The diagnosis of DAT negative AIHA is established after ruling out other causes of hemolysis and is supported by a favourable clinical response to therapy. Since children with AIHA require a longer duration of treatment with steroids, they should be closely monitored for symptoms of steroid toxicity. Long term follow up is essential to monitor for disease relapse and to identify steroid related adverse effects in children requiring prolonged therapy.

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Authorship Contribution

All authors contributed equally to the conception, design, data acquisition, analysis, manuscript drafting, and final approval of the manuscript.

Ethical Approval

The study was conducted in accordance with institutional ethical standards and the Declaration of Helsinki. The study has been approved by the institute ethics committee of Government Medical College, Palakkad (IEC/GMCPKD/16/2025/166).

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Conflict of Interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Data Availability Statement

Authors declare that the data supporting the conclusions of this study can be obtained upon request from the corresponding author, [AM]. The data are not publicly accessible due to information that may compromise the privacy of research participants.

Declarations

The authors declare that the manuscript is original, has not been published previously, and is not under consideration for publication elsewhere. All sources of information used in this study have been appropriately acknowledged and cited.

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