

Treatment and clinical outcomes of pediatric autoimmune hemolytic anemia: real-world single-center data from Korea

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Background: Autoimmune hemolytic anemia (AIHA) is rare and characterized by hemolytic anemia with a positive direct antiglobulin test result after the exclusion of other causes. While adults often relapse within 1 year of first-line steroid therapy, children generally respond well. However, current treatment approaches lack substantial evidence and are primarily expert opinion-based.

Purpose: This study aimed to contribute our single-center experience to pediatric AIHA treatment guidelines.

Methods: Between January 2012 and June 2024, 475 children were diagnosed with anemia; of them, 18 had immune hemolytic anemia, including 6 with neonatal alloimmune hemolytic anemia, 2 who were treated at other centers, and 2 with transient bone marrow suppression due to a viral infection. Thus, this study retrospectively analyzed the treatment responses of 8 patients with AIHA.

Results: The median age at diagnosis was 5.2 years (range, 2.3–11.8 years); 62.5% (5 of 8) were male. Median hemoglobin (Hb) at diagnosis was 6.3 g/dL (range, 3.4–9.5 g/dL), median reticulocyte index was 6.53% (range, 1.64%–22.07%), median total bilirubin was 2.75 mg/dL (range, 0.98–7.23 mg/dL), and median lactate dehydrogenase was 1,662 U/L (range, 790–2,921 U/L). All haptoglobin levels were <10 mg/dL. Treatments included steroids (8 of 8), red blood cell transfusions (5 of 8), and intravenous immunoglobulins (2 of 8). Half of the steroid-treated patients received intravenous methylprednisolone for 1–5 days, while half received oral prednisolone (median, 1.78 [range, 0.79–3.39] mg/kg/day). The median time to age-adjusted normal Hb levels was 16.5 days (range, 9.0–22.0 days). Steroids were administered for a median 37.5 days (range, 14.0–119.0 days). Excluding one patient later diagnosed with systemic lupus erythematosus, no relapses occurred during the 3- to 19-month follow-up period.

Conclusion: Patients with pediatric AIHA showed relapse-free rapid hematological improvement and sustained steroid responses within 2 months, suggesting that systematic steroid treatment is feasible and highlighting the need for multicenter trials to establish standardized guidelines.

Key words: Anemia, Autoimmune hemolytic anemia, Pediatrics, Steroids

Key message

Question: Can pediatric autoimmune hemolytic anemia (AIHA) be effectively managed using first-line steroids?

Finding: In this single-center study, pediatric patients with AIHA achieved normal hemoglobin levels within 16.5 days (range, 9.0–22.0 days) of first-line steroid treatment and maintained effective responses for 2 months.

Meaning: These outcomes highlight the efficacy of steroid treatment in pediatric versus adult AIHA and underscore the need for multicenter trials to establish standardized treatment guidelines.

Introduction

Autoimmune hemolytic anemia (AIHA) is a rare disorder in both children and adults, characterized by hemolytic anemia caused by autoantibodies that destroy red blood cells (RBCs) and a positive direct antiglobulin test (DAT) after excluding other causes.^{1–3} The global incidence of AIHA is approximately 1–3 per 100,000 individuals. AIHA is classified into warm, cold, mixed, drug-induced, and atypical (DAT-negative) types.^{1,2,4} Warm antibody AIHA (wAIHA) represents more than 60%–80% of pediatric and adult AIHA cases.^{5–11} AIHA can be either primary

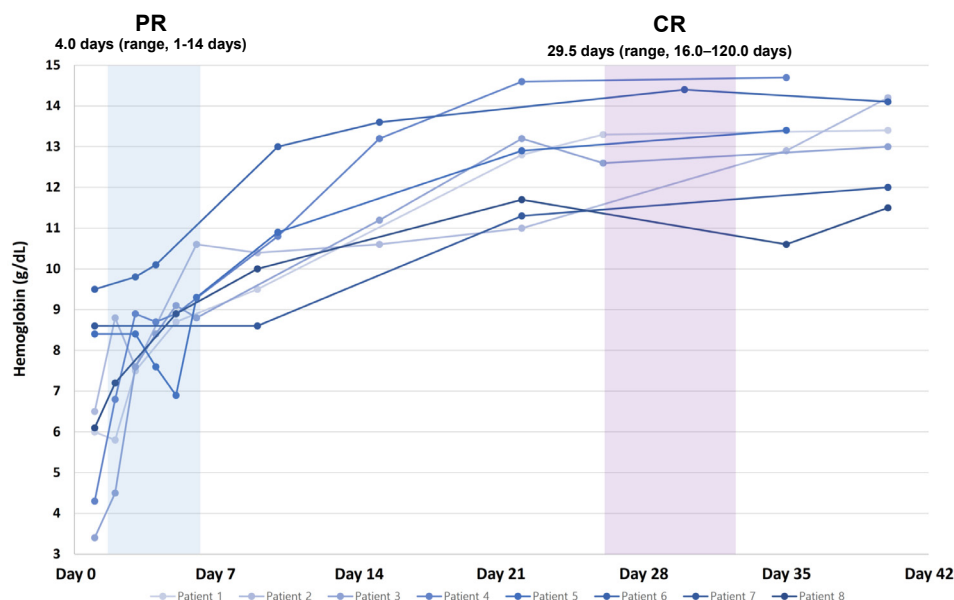
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Graphical abstract. Hemoglobin increase trends among pediatric patients with AIHA. The median time to achieve a CR was 29.5 days (range, 16.0–120.0 days). A PR was achieved within a median 4 days (range, 1–14 days). Steroid therapy continued for a median 37.5 days (range, 14.0–119.0). CR, complete response; PR, partial response.

(idiopathic) or secondary, with primary cases constituting 50% and secondary cases often linked to lymphoproliferative disorders, autoimmune diseases, infections, and tumors.^{6,10,12} Clinical manifestations vary widely, from gradual or compensated anemia to acute, life-threatening cases.^{4,6}

The diagnosis, prognosis, and treatment of AIHA present significant challenges, primarily because the pathophysiology of the disease is not fully understood. This complexity arises from intricate interactions between autoantigens, the complement system, and dysregulations in T- and B-cell functions.^{13,14} Current therapeutic approaches for AIHA lack a robust evidence base and predominantly depend on expert consensus and individual clinical expertise,^{15–18} with limited prospective therapeutic trials.^{19,20} Despite this, steroids remain the cornerstone of treatment, showing efficacy in 70%–80% of patients.^{4–9, 13,16,20–23} The choice and sequence of second-line treatments remain debated, and no definitive predictors of outcome exist. Second-line treatment options include: (1) steroids combined with intravenous immunoglobulin (IVIg); (2) immunosuppressive/cytotoxic drugs; (3) rituximab, and (4) splenectomy. Additionally, transfusion requirements, plasma exchange, and erythropoietin administration are considered.^{2,4–6,10–12,14–16}

Most guidelines and experts for adult AIHA recommend an initial dose of 1.0–1.5 mg/kg/day of prednisolone (PD),^{14,15,24,25} although the optimal starting dose and taper schedule have yet to be established. The initial dose is usually maintained for 1–3 weeks until hemoglobin (Hb)

levels exceed 10 g/dL. The response generally occurs by the second week. If there is minimal or no improvement by the third week, the therapy is deemed ineffective. After Hb stabilization, PD should be tapered gradually.¹² Patients with rapid hemolysis and severe anemia, or complex cases like Evans syndrome (ES), may require high-dose corticosteroid or intravenous (IV) methylprednisolone (MP).^{26,27} While there is no established consensus on the initiation and duration of steroid maintenance therapy, studies have reported that patients receiving low-dose corticosteroids for over 6 months demonstrate a lower relapse rate and prolonged remission compared to those who discontinue treatment within 6 months.²⁸ Furthermore, early initiation of steroid therapy is linked to a decreased risk of relapse. Accordingly, in adults, treatment typically requires over 6 months, with a high rate of refractoriness to first-line therapy or relapse within one year. In contrast, pediatric cases respond well to short-term steroid therapy in nearly 80% of patients.²³

At the onset of therapy, monitoring Hb and reticulocyte levels over the initial weeks is advisable. Routine monitoring of the DAT is standard, though a persistently positive result does not necessarily indicate uncontrolled disease. If hemolysis persists but is well compensated after PD tapering, second-line treatment may not be required.^{2,14}

This study aims to analyze the clinical characteristics and treatment outcomes of pediatric patients diagnosed with AIHA at Chungnam National University Hospital over the past decade. By reviewing medical records, we intend

to evaluate the efficacy of steroids as a first-line therapy for pediatric AIHA and establish a systematic approach to managing these patients based on our findings.

Methods

1. Patients and data collection

From January 2012 to June 2024, 475 pediatric anemia patients were diagnosed at Chungnam National University Hospital, of whom 18 had immune hemolytic anemia. 6 were diagnosed with neonatal alloimmune hemolytic anemia, 2 were treated at other centers, and 2 were excluded as the primary cause was transient bone marrow suppression due to an acute viral infection. This study focused on 8 pediatric AIHA patients who were diagnosed and followed up at our center. In all patients, hereditary hemolytic disease was ruled out (Fig. 1).

This retrospective study analyzed medical records, encompassing clinical information, laboratory results, and other relevant data. The collected data included patient demographics, laboratory findings, and therapeutic interventions such as medications and transfusions.

The study protocol was approved by the Institutional Review Board of Chungnam National University Hospital (IRB number: CNUH 2024-09-007). As the study involved only a review of existing medical records without any additional interventions, the requirement for written informed consent was waived.

2. Definitions

AIHA was defined as anemia accompanied by a positive DAT after excluding other causes. According to DAT results and the thermal characteristics of the autoantibody, patients were classified as wAIHA (DAT+ for IgG only or IgG plus C3d), cold agglutinin disease (CAD; DAT+ for C3d only, with cold agglutinins of I specificity at titer of 64 or higher), and mixed AIHA (DAT+ for IgG and C3d, with high-titer cold agglutinins).¹⁻⁴⁾

Anemia was identified when Hb levels fell below the 5th

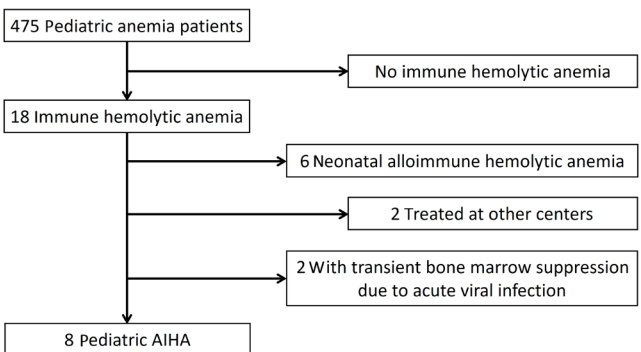


Fig. 1. Flow chart of study population enrollment. AIHA, autoimmune hemolytic anemia

percentile for each pediatric age group: less than 10.5 g/dL in children aged 6–23 months, less than 11 g/dL in children aged 24–59 months, less than 11.5 g/dL in children aged 5–11 years, less than 12 g/dL in nonpregnant female and male children aged 12–14 years, and less than 13 g/dL in males aged 15 years and older.²⁹⁾

In this study, severe AIHA was defined as cases where Hb levels corresponded to severe anemia based on age-specific criteria: less than 7.0 g/dL in children aged 6–59 months, less than 8.0 g/dL in children aged 5 years and older.²⁹⁾

Treatment responses in AIHA are categorized as complete response (CR), partial response (PR), or no response. CR was defined as the normalization of Hb levels and the resolution of hemolysis parameters, such as reticulocyte index (RI) and lactate dehydrogenase (LDH), with a negative DAT not being mandatory. The normal Hb indicates the absence of anemia, and standard RI was defined as 1.5% or lower, which remains consistent across all age groups except for neonates.³⁰⁾ The thresholds for normal LDH levels were defined as 900 U/L or lower for children aged 0–12 months, 700 U/L or lower for children aged 1–3 years, 640 U/L or lower for children aged 3–6 years, 600 U/L or lower for children aged 6–12 years, and 500 U/L or lower for adolescents aged 12–18 years.^{31,32)} PR is characterized by persistent anemia with at least a 2 g/dL increase in Hb from baseline, with or without ongoing signs of hemolysis, and no requirement for transfusion. Relapse was defined as a recurrence of AIHA after the patient had initially responded to treatment.

3. Diagnosis and treatment

For patients suspected of AIHA, the primary evaluation included a complete blood count, blood smear, bilirubin, and hemolysis parameters such as reticulocytes, LDH, DAT, haptoglobin, plasma Hb, and urinalysis. Antinuclear antibody (Ab) and anti-double-stranded DNA tests were performed to investigate possible underlying disorders. A viral workup was also conducted, including tests for cytomegalovirus, Epstein-Barr virus (EBV), parvovirus B19, influenza virus, coronavirus disease 2019, and other respiratory viruses. Mycoplasma infection was additionally investigated.

IV MP was given to patients with the poor condition or difficulty with oral intake, while those able to take orally received PD. RBC transfusions were performed if severe anemia was present. Steroid treatment began at 1–3 mg/kg/day of PD equivalent, with higher doses of MP at 7–8 mg/kg/day used in cases of severe anemia with high fever. The initial dosage was maintained for 3–7 days, with the option to extend if PR was not achieved.

After achieving PR, the steroid dosage was tapered by

25%–50% every 5–14 days and then reduced to below 1 mg/kg/day, maintaining this for 2–6 weeks before discontinuation.

4. Statistical analysis

Statistical analyses were performed using R 4.4.0 (The R Foundation for Statistical Computing). Mann-Whitney *U* tests were used to compare continuous variables between the severe AIHA and nonsevere AIHA subgroups, while Fisher exact tests were applied to categorical variables. *P* values <0.05 were considered statistically significant. Kendall's rank correlation was used to analyze factors affecting steroid response and duration due to the small sample size and nonlinearity of the data.

Results

1. Clinical characteristics of patients

Among the 8 patients included in the study, 5 (62.5%) were male, with a mean age of 5.2 years (range 2.3–11.8 years). None had a family history of hematologic or immunologic disorders. Initial symptoms included pallor (8 of 8), fever (3 of 8), dark urine (3 of 8), and skin rash (1 of 8). While some patients experienced transient tachycardia associated with fever, there were no instances of vital instability, such as hypotension or decreased oxygen saturation. Regarding predisposing factors, Kawasaki disease (KD) (n=1), febrile illness (n=5), and idiopathic cases (n=2) were identified. Of the 2 idiopathic cases, one patient was diagnosed with systemic lupus erythematosus (SLE) 9 months after initially being diagnosed with AIHA with thrombocytopenia. The patient presented with left knee pain and skin rash and had a positive antinuclear antibody (Ab) (speckled, 1:320); anti-dsDNA Ab at 28.35 IU/mL (reference <10.0); and anti-Smith Ab at 12.67 U/mL (reference <7.0); elevated anti-cardiolipin IgG (169.5 phospholipid units [PL-U]/mL; reference <10.0) and IgM (15.3 PL-U/mL; reference <10.0); anti- β_2 -glycoprotein I IgG (40.6 U/mL; reference <7.0) and IgM (37.2 U/mL; reference <20.0); reduced complement levels (C3: 27 mg/dL, reference 86–160, and C4: 2 mg/dL, reference 17–45), confirming the diagnosis of SLE.

At the time of diagnosis, the median hematologic parameters were as follows: Hb 6.3 g/dL (range 3.4–9.5 g/dL), RI 6.53% (range 1.64%–22.07%), total bilirubin 2.75 mg/dL (range 0.98–7.23 mg/dL), direct bilirubin 0.49 mg/dL (range 0.22–0.63 mg/dL), LDH 1662.0 U/L (range 790–2,921 U/L). Haptoglobin levels were <10 mg/dL in all patients. None met the criteria for autoimmune disease, and no active viral or mycoplasma infections were confirmed in any patients. All patients received steroids as the first-line

treatment. Among these, 5 patients also underwent RBC transfusions at 10 mL/kg for 1–2 sessions, and 2 patients received IVIg: one at 0.5 g/kg/day for 4 days, and the other at 1.0 g/kg/day for 5 days. Of the steroid-treated patients, half (4 of 8) were initially administered IV MP for 1–5 days, while the other half (4 of 8) started with oral PD. The median initial PD dose was 1.78 mg/kg/day (range 0.79–3.39 mg/kg/day) (Table 1).

2. Treatment outcomes

Among the 8 patients who received steroid treatment, 7 were initially hospitalized. Patients were discharged once PR was achieved after starting treatment.

All 8 patients achieved CR with first-line steroid therapy. The median time to achieve CR was 29.5 days (range, 16–120 days). PR was reached in a median of 4.0 days (range, 1–14 days) (Graphical abstract). One patient was treated on an outpatient basis as their anemia and dark urine were already improving at the initial consultation, which led to a delayed response evaluation and likely an underestimation of the actual response timing. The

Table 1. Patients' clinical characteristics (N=8)

Characteristic	Value
Male sex	5 (62.5)
Age (yr)	5.2 (2.3–11.8)
Body weight (kg)	17.7 (11.1–47.5)
Presenting symptoms at diagnosis	
Pallor	8 (100)
Fever	3 (37.5)
Dark urine	3 (37.5)
Skin rash	1 (12.5)
Initial laboratory findings	
Hemoglobin (g/dL)	6.3 (3.4–9.5)
White blood cell (/ μ L)	11,570 (4,130–35,400)
Platelet ($\times 10^3$ / μ L)	345 (9–735)
MCV (fL)	85.55 (74.5–109.7)
MCH (pg)	29.35 (26.4–42.0)
Reticulocyte index (%)	6.53 (1.64–22.07)
Lactate dehydrogenase (U/L)	1,662.0 (790–2,921)
Total bilirubin (mg/dL)	2.75 (0.98–7.23)
Direct bilirubin (mg/dL)	0.49 (0.22–0.63)
Patients with haptoglobin <30 mg/dL	8 (100.0)
Plasma hemoglobin (mg/dL) ^a	29.93 (14.96–162.69)
Initial treatments	
Patients with steroids	8 (100)
Oral prednisolone	4 (50.0)
Initial prednisolone dose (mg/kg/day)	1.78 (0.79–3.39)
Intravenous methylprednisolone	4 (50.0)
Initial methylprednisolone dose (mg/kg/day)	5.30 (2.95–8.2)
Patients with intravenous immunoglobulin	2 (25.0)
Patients with red blood cell transfusion	5 (62.5)

Values are presented as number (%) or median (range).
MCV, mean corpuscular volume; MCH, mean corpuscular hemoglobin.
^aReference range (all ages): 0–5.0 mg/dL.

median time to normalize Hb levels was 16.5 days (range, 9–22 days). Hemolysis parameters normalized at a median of 27.5 days (range, 16.0–75.0 days) for the RI and 22 days (range, 9–120 days) for LDH. The median time for DAT negativity was 47.0 days (range, 22–395 days), though one patient receiving alternate-day steroid therapy remains DAT positive. Steroid therapy lasted a median of 37.5 days (range, 14.0–119.0 days) in all 8 patients (Table 2) and 36.0 days (range, 14.0–54.0 days) in 7 patients, excluding the one later diagnosed with SLE. Except for this patient, no relapses occurred during the 3- to 19-month follow-up period.

Among the 8 patients with AIHA, 5 were classified as severe, with 3 receiving initial IV MP and 4 undergoing RBC transfusions. In the nonsevere group of 3 patients, 1 received initial IV MP, and 1 underwent RBC transfusion. In the severe AIHA group, 4 patients received RBC transfusions (mean, 17 mL/kg; range, 10–20 mL/kg) within the first 1–2 days of treatment, while 1 patient in the nonsevere group received 10 mL/kg on the first day of treatment. In group with severe AIHA, the time to achieve PR appeared shorter, likely due to the higher rate of RBC transfusions in this group. For the 2 patients who received IVIG, the median time to PR was 3 days (range, 2–4 days) and to CR 17 days (range, 12–22 days), while in the 6 patients who did not receive IVIG, the median time to PR and CR was 4.5 days (range, 1.0–14.0 days) and 16.5 days (range, 9.0–35.0 days), respectively. Steroid therapy lasted a median of 49 days (range, 31–54 days) for severe AIHA and 34 days (range, 14–119 days) for nonsevere AIHA (Table 2). However, the small sample size limited the ability to demonstrate statistical significance (Supplementary Table 1).

We performed a correlation analysis between the response to and duration of steroid treatment and the initial clinical parameters, but no significant results were found due to limitations such as the small sample size (Supplementary Table 2).

Discussion

AIHA is a rare condition, usually with a favorable clinical course, and is often viewed as benign, leading hematologists to consider it less critical. Consequently, large-scale randomized controlled trials and clinical studies are scarce, and observational studies on biochemical profiles and treatment responses are limited. This has resulted in a lack of established clinical guidelines for the evaluation, diagnosis, and treatment of AIHA in both pediatric and adult populations. Nonetheless, a small subset of patients presents a significant medical challenge due to resistance to conventional treatments or the occurrence of life-threatening acute complications.^{4,6} The largest study to date, conducted by the GIMEMA group across 8 Italian hematologic centers, involved 308 patients with a median age of 58 years, predominantly adults but also including 10 pediatric patients. Nearly all patients received first-line steroid therapy, with most not requiring second-line treatment. However, thrombotic events occurred in approximately 10% of patients. Predictors of fatal outcomes included severe infections, ES, and the need for 4 or more lines of treatment.⁴ The largest observational study in pediatric AIHA, conducted by 26 national pediatric CEREVANCE units with 265 patients, found that 60% presented with fever, 80% presented with dark urine, and severe complications occurred in 3% of patients. ES was diagnosed in 37% of patients. AIHA was postinfectious in 10% of cases, immunological in 53%, and primary in 37%. First-line steroid therapy achieved complete remission in 58% of patients within the first month, though 45% required prolonged therapies. Overall, 90% reached CR, with 39% maintaining continuous remission. Positive IgG and IgG+C3d tests were linked to a lower chance of continuous remission.⁶

There is ongoing controversy regarding the definition of severe AIHA. Some studies classify it based on the need for blood transfusion due to critically low initial Hb levels.^{4,33}

Table 2. Treatment outcomes of severe versus nonsevere AIHA groups

Variable	Total (N=8)	Group with severe AIHA (N=5)	Group with nonsevere AIHA (N=3)
Patients with Intravenous methylprednisolone	4 (50.0)	3 (60.0)	1 (33.3)
Patients with red blood cell transfusion	5 (62.5)	4 (80.0)	1 (33.3)
Days to partial response	4 (1–14)	4 (1–5)	9 (3–14)
Days to normal hemoglobin	16.5 (9.0–22.0)	21 (12–22)	14 (9–19)
Days to normal reticulocyte index	27.5 (16.0–75.0)	22 (19–75)	33 (16–51)
Days to normal lactate dehydrogenase	22 (9–120)	24 (14–120)	14 (9–33)
Days to complete response	29.5 (16.0–120.0)	26 (21–120)	33 (16–51)
Days to negative DAT	47 (22–390)	47 (22–390)	42 (33–51)
Duration of steroids (days)	37.5 (14.0–119.0)	39 (31–54)	34 (14–119)

Values are presented as median (range) or number (%).
AIHA, autoimmune hemolytic anemia; DAT, direct antiglobulin test.

Similarly, in our study, we divided patients into severe and nonsevere AIHA groups using Hb thresholds of 7 or 8, depending on age, as the criteria for transfusion necessity.

The DAT, using both polyspecific and monospecific anti-human globulins (AHG), is the primary serological test for diagnosing AIHA. Initial testing should involve a polyspecific AHG containing anti-IgG and anti-C3d, followed by monospecific AHG testing to separately detect IgG and C3d if the initial DAT is positive. However, in our study, AIHA subtypes could not be definitively classified, as only polyspecific AHG was available. Diagnosing DAT-negative AIHA, which requires testing for low-affinity IgG and IgA/IgM autoantibodies, was also not possible due to the unavailability of these tests at our institution. Nonetheless, wAIHA is the most common subtype, accounting for 60%–80% of pediatric AIHA cases.⁵⁻⁹⁾ One study reported that 20% of wAIHA cases exhibit IgG+ C3d positivity, while 13% show only C3d positivity,³⁴⁾ suggesting that serologic findings do not always clearly distinguish AIHA subtypes. Excluding the patient later diagnosed with SLE, 5 patients had a preceding or concurrent febrile illness, and one classified as idiopathic was 2.3 years old at diagnosis, suggesting a prior acute infection. Based on 2 case reports of AIHA associated with KD,^{35,36)} our patient, diagnosed with KD 6 days before AIHA, was also considered to have KD as a predisposing factor. However, the underlying pathophysiology of KD in relation to AIHA remains unclear.

Given that all patients in this study were pediatric and considering the identified predisposing factors, we initiated first-line steroid treatment in the absence of a clear etiology for CAD, such as bacterial or viral infections (e.g., *Mycoplasma pneumoniae* or EBV), malignancies, or other autoimmune disorders.⁵⁻⁹⁾

All 8 patients requiring therapeutic intervention achieved CR with first-line steroid therapy, as evidenced by the normalization of Hb and hemolysis parameters. None required second-line treatment. Notably, CR was achieved within approximately one month, and except for the patient later diagnosed with SLE, the response was sustained without relapse despite the relatively short treatment duration of less than 2 months. This outcome contrasts with the typical adult AIHA treatment course, where at least 3–4 months of steroid therapy is often necessary to achieve similar results.^{12,14,15,22,24,25)}

The efficacy of IVIg in AIHA is debated, but some studies suggest it may be effective in severe cases with pretreatment Hb levels below 6–7 g/dL,^{37,38)} and it is particularly effective in pediatric patients with low toxicity.³⁹⁾ In our study, 2 patients with initial Hb levels of 3.4 and 6.1 g/dL received IVIg alongside steroids.

RBC transfusion is often needed to maintain adequate

Hb levels in rapidly progressing anemia until specific treatments take effect. The decision to transfuse should be based on the patient's overall clinical status and comorbidities rather than Hb levels alone, though it is generally advised when Hb drops below 6 g/dL. Effective management of transfusions in AIHA requires close coordination between clinicians and transfusion centers, with early steroid administration and careful monitoring during transfusion to reduce the risk of transfusion reactions.^{5,15,25)} In our study, the median Hb level at the time of RBC transfusion was 6.0 g/dL (range, 3.4–8.4 g/dL).

Among the 8 patients who received steroid therapy, one initially presented with thrombocytopenia and positive antinuclear and anti-dsDNA Ab but was not diagnosed with another autoimmune disease. Given the potential for ES and the higher relapse rate associated with early steroid tapering in ES,^{40,41)} this patient remained on prolonged low-dose PD therapy compared to others. The patient achieved CR within 16 days of treatment and maintained normal Hb levels thereafter. However, after tapering steroids, the RI increased, DAT remained strongly positive, and haptoglobin levels remained low. Despite receiving steroids for more than 119 days, there was no improvement in hemolysis parameters aside from Hb levels. 9 months after the initial diagnosis of AIHA with thrombocytopenia, the patient was ultimately diagnosed with SLE, and hemolytic anemia recurred. This case highlights the need for regular monitoring for underlying autoimmune diseases in patients with AIHA and thrombocytopenia or those experiencing worsening hemolysis parameters, such as an increasing RI, during steroid tapering or discontinuation.

Although we analyzed data collected over a relatively long period, the single-institution setting and lack of specialized personnel resulted in a small cohort size, limiting our ability to draw statistically significant trends. In more severe AIHA cases, aggressive early interventions such as IV MP and RBC transfusions were employed, but the lack of systematic treatment guidelines often necessitated reliance on clinician experience and judgment. Additionally, conclusions from retrospective or observational studies on treatment efficacy are inherently less definitive than those from prospective studies.

In conclusion, pediatric AIHA patients showed rapid hematologic improvement, maintaining effective steroid responses within 2 months without any relapses—a clear contrast to typical adult responses. These findings highlight the potential for a systematic approach to managing pediatric AIHA and developing tailored treatment strategies. With the advancement of prospective multicenter research and clinical trials, there is hope that standardized treatment guidelines for pediatric AIHA will be realized.

Footnotes

Supplementary materials: Supplementary Tables 1-2 are available at 10.3345/cep.2024.02026.

Conflicts of interest: No potential conflict of interest relevant to this article was reported.

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