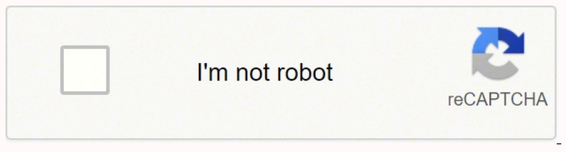

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Published in final edited form as:
Pediatr Transplant. 2015 June ; 19(4): 391–398. doi:10.1111/petr.12455.

The incidence of autoimmune hemolytic anemia in pediatric hematopoietic stem cell recipients post first and second hematopoietic stem cell transplant

Ibrahim Ahmed, M.D.[1], **Jun Teruya, M.D., D.Sc.**[2,4], **Cristina Murray-Krezan, M.S.**[3], and **Robert Krance, M.D.**[2]

[1]Department of Pediatric Hematology/Oncology and BMT, Children's Mercy Hospital; 2401 Gillham Road, Kansas City, Missouri 64108

[2]Department of Pediatrics, Center for Cell and Gene Therapy, Baylor College of Medicine, 1102 Bates Street, Suite 1770, Houston, TX 77030, USA

[3]Department of Internal Medicine, Division of Epidemiology, Biostatistics, and Preventive Medicine, University of New Mexico Health Sciences Center, Albuquerque, New Mexico

[4]Departments of Pathology & Immunology, Pediatrics, and Medicine, Baylor College of Medicine, Houston, TX 77030, USA

Abstract

The reported incidence of post allogeneic hematopoietic stem cell transplant (HSCT) auto-immune hemolytic anemia (AIHA) was between 4.4% and 6% following a single transplant. Cord blood transplantation, T-cell depletion and chronic GvHD are significantly associated with post-transplant AIHA. During an 11 year period, data for 500 pediatric HSCT recipients were eligible for evaluation of the incidence of AIHA post first and second transplants. Demographic, transplant, and post-transplant related variables were analyzed. Twelve/500 (2.4%) recipients at a median of 273 days and 7/72 (9.7%) recipients at a median of 157 days developed AIHA post first and second HSCT respectively. Post first HSCT, none of the matched related donor recipients developed AIHA (0/175 MRD vs. 12/325 other donors, p=0.04). Four/12 required a second HSCT to control the AIHA. Post the second HSCT, matched unrelated donor was significantly associated with the development of AIHA. No other variables were associated with the post-second transplant AIHA.

The incidence of AIHA post first and second HSCT was less than reported. The increased incidence of AIHA among recipients of second HSCT is most likely due to the profound immune dysregulation. A much larger, prospective study would be needed to evaluate the incidence, complications and management of post-transplant AIHA.

Correspondence Author: Ibrahim Ahmed, MD, Department of Pediatric Hematology/Oncology and BMT, Children's Mercy Hospital, 2401 Gillham Road, Kansas City, Missouri 64108, (816) 302-6808 (Office), (816) 302-9894 (Fax), iaahmed@cmh.edu

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Review

Autoimmune Hemolytic Anemia in the Pediatric Setting

Aikaterini Voulgaridou [1] and Theodosia A. Kalfa [1,2,*]

[1] Division of Hematology, Cancer and Blood Diseases Institute, Cincinnati Children's Hospital Medical Center, Cincinnati, OH 45229, USA; Aikaterini.Voulgaridou@cchmc.org
[2] Department of Pediatrics, University of Cincinnati College of Medicine, Cincinnati, OH 45267, USA
* Correspondence: Theodosia.Kalfa@cchmc.org; Tel.: +1-513-636-0989

Abstract: Autoimmune hemolytic anemia (AIHA) is a rare disease in children, presenting with variable severity. Most commonly, warm-reactive IgG antibodies bind erythrocytes at 37 °C and induce opsonization and phagocytosis mainly by the splenic macrophages, causing warm AIHA (w-AIHA). Post-infectious cold-reactive antibodies can also lead to hemolysis following the patient's exposure to cold temperatures, causing cold agglutinin syndrome (CAS)-due to IgM autoantibodies, or paroxysmal cold hemoglobinuria (PCH) due to atypical IgG autoantibodies which bind their target RBC antigen and fix complement at 4 °C. Cold-reactive antibodies mainly induce intravascular hemolysis after complement activation. Direct antiglobulin test (DAT) is the gold standard for AIHA diagnosis; however, DAT negative results are seen in up to 11% of warm AIHA, highlighting the need to pursue further evaluation in cases with a phenotype compatible with immune-mediated hemolytic anemia despite negative DAT. Prompt supportive care, initiation of treatment with steroids for w-AIHA, and transfusion if necessary for symptomatic or fast-evolving anemia is crucial for a positive outcome. w-AIHA in children is often secondary to underlying immune dysregulation syndromes and thus, screening for such disorders is recommended at presentation, before initiating treatment with immunosuppressants, to determine prognosis and optimize long-term management potentially with novel targeted medications.

Keywords: warm autoimmune hemolytic anemia; cold agglutinin syndrome; paroxysmal cold hemoglobinuria; direct antiglobulin test

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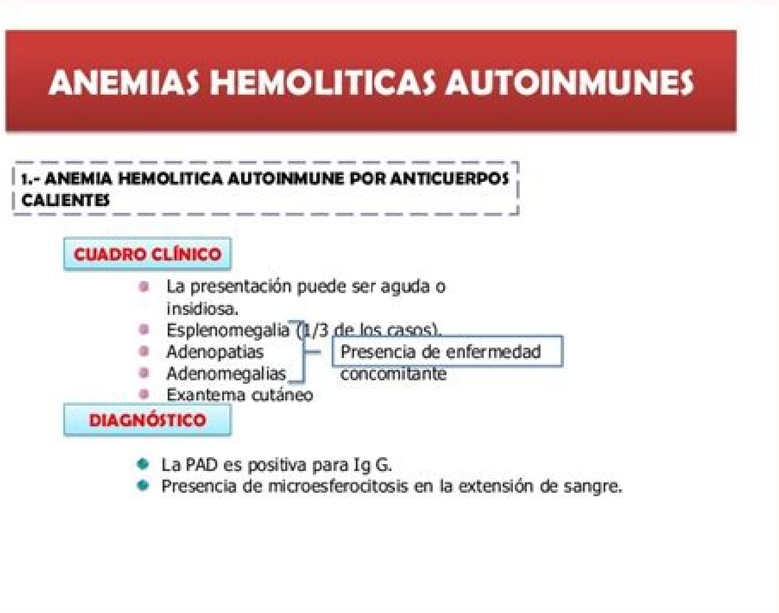
Citation: Voulgaridou, A.; Kalfa, T.A. Autoimmune Hemolytic Anemia in the Pediatric Setting. *J. Clin. Med.* **2021**, *10*, 216. https://doi.org/10.3390/jcm10020216

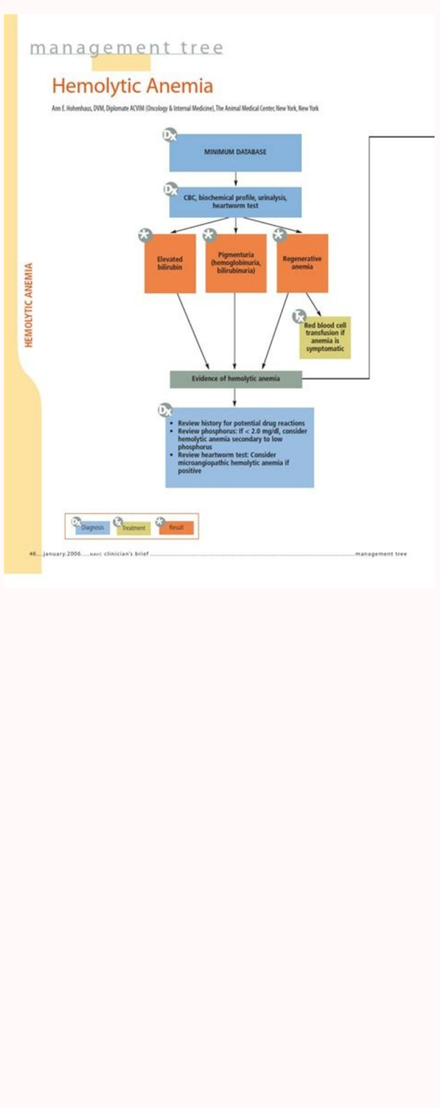
Received: 16 December 2020
Accepted: 5 January 2021
Published: 9 January 2021

1. Introduction

Autoimmune hemolytic anemia (AIHA) is an acquired form of hemolytic anemia in which autoantibodies target red blood cell (RBC) membrane antigens, inducing cell rupture (lysis). Hemolysis triggers compensatory RBC production by increasing erythropoietin levels; however, this response is typically insufficient to redress normal hemoglobin blood levels leading to anemia. AIHA is characterized as "extrinsic" because the autoantibodies affect otherwise normal RBCs. A recent systematic review assessing AIHA terminology concluded that there is significant heterogeneity in the definition and diagnostic criteria for the disease [1]. In most of the reviewed studies, AIHA was defined as hemolytic anemia with a positive direct antiglobulin test (DAT) and concurrent exclusion of alternative diagnoses. However, there are limitations in the use of that definition since it does not include DAT-negative cases. AIHA is classified as "warm" or "cold" based on the optimal temperature at which the antibodies present maximal reactivity and as primary or secondary depending on the presence of a recognized underlying cause, such as immunodeficiency, infections, medications, or malignancy [2]. It affects both pediatric and adult populations, although its presentation in childhood is relatively rare, with the annual incidence estimated to be approximately 0.8 per 100,000 individuals under 18 years old [3].

Children with AIHA can present with a variable degree of severity. The most common form of AIHA in the pediatric population is due to warm-reactive autoantibodies. Warm antibody AIHA (w-AIHA) is diagnosed in more than half of autoimmune hemolytic





Conclusion: Combination chelation is well tolerated and more efficacious in children with elevated serum ferritin or cardiac-siderosis. This is the first study to demonstrate that when oral iron chelators are delivered in lower doses at regular intervals to provide round-the-clock chelation, it results in increased efficacy, reduced toxicity and adequate compliance.

RIC-1.6
IVIG INDUCED HEMOLYTIC ANEMIA – A REPORT OF TWO CASES

Putun Patel, Sandip Bartakke, Vibha Balbu, Bhakti Sarangi, Kavita Srivastava, Jitendra Oswal, Vijay Kalras, Sanjay Lalwani. *Department of Pediatrics, Bharati Vidyapeeth Medical College (DU), Pune, India*

Introduction: Intravenous immunoglobulin (IVIG) is an established treatment for deficiency states, immune disorders and neurologic diseases. That it can rarely cause hemolytic anemia, due to passive transfusion of anti-A and B antibodies; is not widely known. We report here two such cases.
Case 1: A 12-year-old, A-positive male child, with Guillain Barre syndrome was treated with IVIG at 2g/kg over four days, following which his hemoglobin dropped from 14 to 7g/dl. His lactate dehydrogenase (LDH) and indirect bilirubin (IB) were raised and direct coombs test (DCT) was positive. Immune hemolytic anemia (IHA) was diagnosed and was treated with steroids. At present the child is well and off all medications.
Case 2: An 8-year-old, A-positive male child, with Burkitt lymphoma, was treated with IVIG for pure red-cell aplasia due to Parvovirus B19 infection. On the next day he developed hemoglobinuria. His hemoglobin dropped from 9 to 5 g/dl, DCT was positive, LDH and IB was raised. So considering IHA, he was treated with steroids and IVIG was stopped.
Discussion: Hemolytic anemia is a potentially serious but lesser known complication of IVIG and is currently not listed in product inserts. The passive transfer of anti-A and B antibodies found in IVIG due to pooling from O blood group donors are proposed as mechanism for hemolysis. If anemia occurs, IHA should be suspected, quickly confirmed by doing laboratory investigations and treated speedily with steroids. In addition, a crossmatch could be performed with serum and IVIG. Both patients were A-positive, generated within one month of each other and received the same brand of IVIG.
Conclusion: Physicians should be aware of this rare and serious sequelae of IVIG so that it will be adequately managed. Manufacturers should follow more stringent industry standards and label anti-A and anti-B titers on their product.

RIC-1.7
NEXT GENERATION SEQUENCING TO AID IN UNDIAGNOSED HEMOLYTIC ANEMIA

Ajit Pratap Singh[1], Richa Singh[1], Jyotsna Madan[2], Nita Radhakrishnan[1]. [1]*Department of Pediatric Hematology Oncology, Super Speciality Pediatric Hospital and PG Teaching Institute, India.* [2]*Department of Pathology, Super Speciality Pediatric Hospital and PG Teaching Institute, Noida, India.* [3]*Super Speciality Pediatric Hospital and PG Teaching Institute, Noida, India*

Introduction: Diagnosis of hemolytic anemia involves a step-wise approach that incorporates clinical information, peripheral smear, electrophoresis and specific tests. In certain situations, a diagnosis is still not obvious and many patients are labelled 'transfusion dependent anemia'. We describe an 18-year-old girl who presented to us with anemia since childhood with no definitive etiology and was subsequently was diagnosed followed next-generation sequencing.
Case: 18-year-old girl presented to us with history of pallor and jaundice since 4-years of age. She was advised blood transfusions earlier which the family refused. She was also diagnosed to have gall stones and advised surgery. She is the second-born to non-consanguineous parents and her younger sister had similar complaints. She was pale, icteric with short stature and splenohepatomegaly. CBC showed anemia (Hb: 5.2gm/dl), normal MCV and mild leukopenia. Indirect hyperbilirubinemia was present. Peripheral smear showed anisopoikilocytosis, elliptocytes and spherocytosis. Corrected reticulocyte count: 2.3%. Osmotic fragility test showed increased resistance compared to normal control. HPLC, alpha thalassemia evaluation, DCT, G6PD and pyruvate kinase levels done earlier were normal. At our center, she was counseled for regular transfusions and genetic evaluation was advised as extensive evaluation so far not yielded definitive results.
Clinical exome sequencing was done (Courtesy: Medgenome Laboratories, Bangalore) with focus on hemolytic anemia genes. A novel heterozygous missense variation in exon 4 of the PKLR gene (c.403C>G[7]) which was predicted damaging and a known heterozygous silent splice site proximal variation in exon 8 of the PKLR gene (c.1200G>G[A]) were detected. A definitive diagnosis of pyruvate kinase deficiency was thus established and the family has been counselled regarding further treatment.
Conclusion: It is well known that patients with PK deficiency may display normal or even increased enzyme activity due to various reasons. Early institutes of genetic evaluation would help such patients to achieve at a definitive diagnosis.

SCT-1.1
PEDIATRIC HAPLOIDENTICAL TRANSPLANT: SINGLE CENTRE EXPERIENCE FROM NORTH INDIA

Neha Singh, Faizuddin Ansari, Vikas Dua. *Department of Pediatric Hemato-Oncology and Bone marrow transplantation, Fortis Memorial Research Institute, Gurgaon, Haryana, India*
E-mail address: drnehasingh1508@outlook.com (N. Singh).

Background: Out of an estimated 20,000 patients requiring an Allogeneic HSCT in India, only 500 odd patients receive one. Keeping in cognizance the resource constraints, the major reason for the discordance lies in the lack of alternate donors. Whilst unrelated donor marrow and cord suffice for the ethnic majorities in Europe and the USA, such registries provide for less than 10% of HSCT recipients due to both cost and an available match.
Aims and Objectives: To initiate a Pediatric Haploidentical Family Donor HSCT program in our Institute.
Material and Methods: We chose a Post-transplant Cyclophosphamide (PTCY) based approach in all except 4 in which TCR alpha beta CD 19 depletion was used with PBSC as the graft source. We have carried out 29 transplants in 27 patients over the last 3 years (AML-7; ALL-6; CML-BC-2; Fanconi Anemia-1; Thalassemia-3, Wiskott Aldrich Syndrome-1, Aplastic anemia – 2, Chediak Higashi Syndrome – 1, Neuroblastoma – 1, JMML- 1). All received PBSC with PTCY on days 3 and 4 followed by cyclosporine/tacrolimus and MMF except four. The conditioning regimen comprised of Fludarabine/ Busulphan/ Melphalan (AML), Fludarabine/ Thiotepa/ Melphalan (ALL), Fludarabine/ Cyclo/ TBI (IA and Aplastic Anemia), Fludarabine / Busulphan/ ATG (Thalassemia), ATG/ Treosulpha/ Fludarabine/ Melphalan (Neuroblastoma), ATG/ treosulpha/ Fludarabine/ Melphalan (Chediak Higashi Syndrome) and Fludarabine/ Busulphan/ Melphalan/ ATG (JMML).
Results: 24/27 patients surviving beyond 21 days had successful engraftment with full donor Chimerism, 2 patients undergoing a second Haploidentical HCT engrafted as well. Acute GVHD grade 2-4 developed in 9/ 27 patients. Day 100 NRM (Non relapse mortality) was 8/27 (29.6%) and the overall survival at 1 year was 58%. The major cause of NRM was infection with Gram Negative Bacilli accounting for 70% of the deaths. Chronic GVHD occurred in 14% of the evaluable patients. With stringent monitoring for CMV and pre-emptive therapy, none succumbed to CMV disease.
Conclusion: Haploidentical HCT based on PTCY and PBSC graft is feasible and probably the most viable form of alternate donor HCT in resource constrained settings.

SCT-1.2
PNEUMATOSIS INTESTINALIS IN TWO PEDIATRIC PATIENTS AFTER HAPLOIDENTICAL STEM CELL TRANSPLANTATION

Faizuddin Ansari, Neha Singh, Vikas Dua. *Department of Paediatric Hemato-Oncology and Bone marrow transplantation, Fortis Memorial Research Institute, Gurgaon, Haryana, India*
E-mail address: drfaizuddinansari@outlook.com (F. Ansari).

Background and objective: Pneumatosis intestinalis (PI) is the accumulation of sub mucosal or sub serosal gas-filled cystic lesions in the

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