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REVIEW

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Pediatric Acute Transfusion Reactions: Diagnostic Pitfalls and Emerging Mechanisms

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ABSTRACT

Acute transfusion reactions (ATRs) in children represent a heterogeneous group of allergic, immunologic, and non-immunologic events that can be life-threatening and clinically indistinguishable, particularly in immunocompromised cohorts. This case-based narrative review explores the spectrum of pediatric ATRs, focusing on pathophysiological mechanisms, diagnostic hurdles, and management strategies. The discussion is framed by two pediatric cases involving four distinct ATRs: first, a child with DOCK8 deficiency undergoing hematopoietic stem cell transplantation who experienced urticaria and subsequent anaphylaxis due to suspected passive allergen transfer; and second, a child with malignancy who developed febrile non-hemolytic and acute hemolytic reactions despite ABO compatibility. These scenarios are analyzed alongside critical entities such as transfusion-associated circulatory overload (TACO) and transfusion-related acute lung injury (TRALI). This review underscores the need for heightened clinical vigilance and highlights passive allergen transfer as an increasingly recognized mechanism essential to enhancing pediatric transfusion safety.

Keywords: Acute transfusion reaction, anaphylaxis, hematopoietic stem cell transplantation, hemolytic transfusion reaction, passive allergen transfer

INTRODUCTION

Blood transfusion remains a life-saving intervention across diverse clinical settings, including surgery, obstetrics, neonatal and intensive care, and hematology-oncology practice. Although generally safe, transfusions are associated with a spectrum of infectious and immunological complications, ranging from mild to potentially fatal reactions (1). Among pediatric patients, approximately 1% require transfusion, most commonly with erythrocyte suspensions or platelet concentrates (2). Despite its rarity, a severe transfusion reaction can be life-threatening, with mortality estimated between 1 in 200,000 and 1 in 420,000 transfused units (3).

Acute transfusion reactions (ATRs) may occur with any blood product—including red cell concentrates (RCCs), fresh frozen plasma (FFP), platelet concentrates (PCs), apheresis granulocytes, and cryoprecipitate—and encompass several clinical entities such as allergic and anaphylactic reactions, acute hemolytic transfusion reaction (AHTR), febrile nonhemolytic transfusion reaction (FNHTR), transfusion-related acute lung injury (TRALI), and transfusion-associated circulatory overload (TACO) (1,4). These events are defined as adverse reactions occurring within 24 hours of transfusion administration, the majority of reactions presenting during the transfusion or within the first four hours.

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While the general adverse event profile of transfusion is well documented, ATRs in the context of hematopoietic stem cell transplantation (HSCT) have been insufficiently characterized (5). HSCT recipients often present a unique vulnerability due to immune dysregulation, concomitant infections, and repeated exposure to blood products, making diagnosis and management particularly challenging.

Although the reported incidence of ATRs ranges from 0.5% to 4.2%, and may reach up to 13% in pediatric intensive care settings (6-8), these figures likely underestimate subclinical or misclassified reactions. Importantly, during our recent service as pediatric allergy consultants at a tertiary referral university hospital, we encountered four severe transfusion reactions in two pediatric patients within a single month.

MATERIALS and METHODS

A retrospective, case-based narrative review was conducted involving two pediatric patients who developed multiple ATRs during transfusion procedures, alongside a review of the relevant published literature. Clinical presentation, laboratory findings, transfusion details, and management were described and discussed. The informed consent was obtained from the patients and/or parents.

Case 1

A three-year-old girl with DOCK8 deficiency, characterized by recurrent infections, eczema, and a known egg allergy, underwent HSCT from an HLA 5/10 haploidentical maternal donor. She was diagnosed with egg allergy due to the exacerbation of atopic dermatitis following the consumption of eggs at one year of age, corroborated by positive results from a skin prick test (egg white: 20x12 mm) and specific IgE testing (egg white specific IgE: 2.61 kU/L, Total IgE: 1306 kU/L). She did not exhibit any other food allergies. Following the implementation of an egg elimination diet, the exacerbation of atopic dermatitis improved, and she did not experience anaphylaxis. A myeloablative conditioning regimen including busulfan and fludarabine was administered to the patient and post-transplant cyclophosphamide, cyclosporine A, and mycophenolate mofetil were used as graft versus host disease prophylaxis. The source of the stem cells was bone marrow and the number of the CD34+ cells given to the recipient was $4x10^6$/kg. She received paracetamol, pheniramine, and methylprednisolone as premedication one hour before infusion. Approximately one hour into the HSCT infusion (after 100 mL had been administered), she developed generalized urticaria, pruritus, and erythema. The infusion was paused, and methylprednisolone was administered, leading to symptom resolution within two hours. Given the mild and non-life-threatening nature of the reaction, the transplantation was resumed at a reduced infusion rate with repeated antihistamine doses (cetirizine and desloratadine), and the remaining 470 mL of the stem cell product was successfully infused.

On day 9 post-HSCT, she underwent an apheresis-derived platelet transfusion. Despite premedication with methylprednisolone and pheniramine 20 minutes beforehand, she developed pruritus, generalized urticaria, eyelid angioedema, and tachycardia (190 beats/min) immediately after the 45-minute transfusion. Anaphylaxis was diagnosed. She received intramuscular (IM) epinephrine, methylprednisolone, antihistamines, and IV fluids. Five minutes later, a second IM epinephrine dose and epinephrine-salbutamol nebulization were administered due to hoarseness and cough. As symptoms persisted, a continuous IV epinephrine infusion was initiated, which resolved the respiratory and cardiovascular symptoms within minutes. During the follow up of the patient, neutrophil engraftment developed on day +17, and thrombocyte engraftment developed on day +21. Now the patient is in a good clinical condition with full donor chimerism on day + 32.

The patient's serum tryptase increased to 3.61 μg/L at the time of anaphylaxis, compared with a basal level of 1.13 μg/L. Differential testing for possible causes of transfusion-related anaphylaxis (including selective IgA deficiency, haptoglobin deficiency, latex or ethylene oxide allergy, and cytokine release syndrome) revealed serum IgA of 78 mg/dL, haptoglobin 1.44 g/L, total IgE 332 kU/L, latex sIgE 0.22 kU/L, ethylene oxide sIgE 0.01 kU/L, and interleukin-6 13.8 pg/mL (0-5.9 pg/mL). With no conventional explanation identified and as the patient was strictly following an egg elimination diet at the time of the reaction, further inquiry revealed that the donor had consumed eggs on the day of donation. In view of her history of egg allergy and reports of passive allergen transfer via blood products (9,10), subsequent transfusions were arranged from donors who had avoided egg consumption for at least 24 hours. The patient tolerated the next platelet and red cell transfusion uneventfully after a modified premedication regimen (methylprednisolone six hours prior and diphenhydramine two hours prior). Given the patient's continued hospitalization for post-HSCT, we have arranged for a reevaluation of the egg allergy after discharge.

This case illustrates two distinct allergic transfusion reactions in a highly atopic child: a generalized urticarial reaction during HSCT and life-threatening anaphylaxis following platelet transfusion, likely triggered by passively transferred food allergens from the donor.

Case 2

A 15-year-old girl with a history of diffuse hemispheric glioma had previously undergone tumor excision, followed by radiation therapy and chemotherapy. Due to anemia, she received an RBC transfusion after premedication with paracetamol and pheniramine. Two hours into the transfusion, she developed fever, chills, rigors, dyspnea, and pain. The transfusion was stopped, and she was treated with intravenous fluids (0.9% NaCl), paracetamol, and methylprednisolone, resulting in resolution of symptoms. The subsequent transfusion of the remained erythrocyte suspension was later tolerated without incident.

One month later, she presented with palpitations and severe anemia (hemoglobin: 5.5 g/dL). After an initial transfusion, the hemoglobin remained low (6.8 g/dL), prompting a second RBC transfusion following premedication with paracetamol and pheniramine. Fifteen minutes into the transfusion (after 20 mL had been administered), she developed shivering, rigors, back pain, and hypotension (80/40 mmHg). The transfusion was discontinued, and IV fluids and methylprednisolone were administered, leading to rapid clinical improvement and normalization of blood pressure.

The transfused unit was returned to the blood bank; ABO compatibility was confirmed, and direct and indirect Coombs tests were negative, excluding clerical error or alloimmune hemolysis. Serum tryptase (3.18 µg/L) was within normal limits, ruling out anaphylaxis. However, an incompatibility was detected for subgroups. After preparation of subgroup-compatible RBCs, transfusion proceeded uneventfully, with hemoglobin rising to 9.6 g/dL. This patient, affected by malignancy and recurrent severe anemia, experienced two distinct transfusion reactions: the first consistent with an FNHTR and the second representing a suspected AHTR, both requiring prompt recognition and supportive management.

Given that severe ATRs often present with overlapping clinical manifestations—including hypotension, fever, dyspnea, rash, and respiratory compromise—a multidisciplinary approach is essential. Allergists and immunologists play a pivotal role in differential diagnosis, identification of immunologic mechanisms, and prevention of recurrence. A practical approach for differential diagnosis of ATRs is presented in Figure 1.

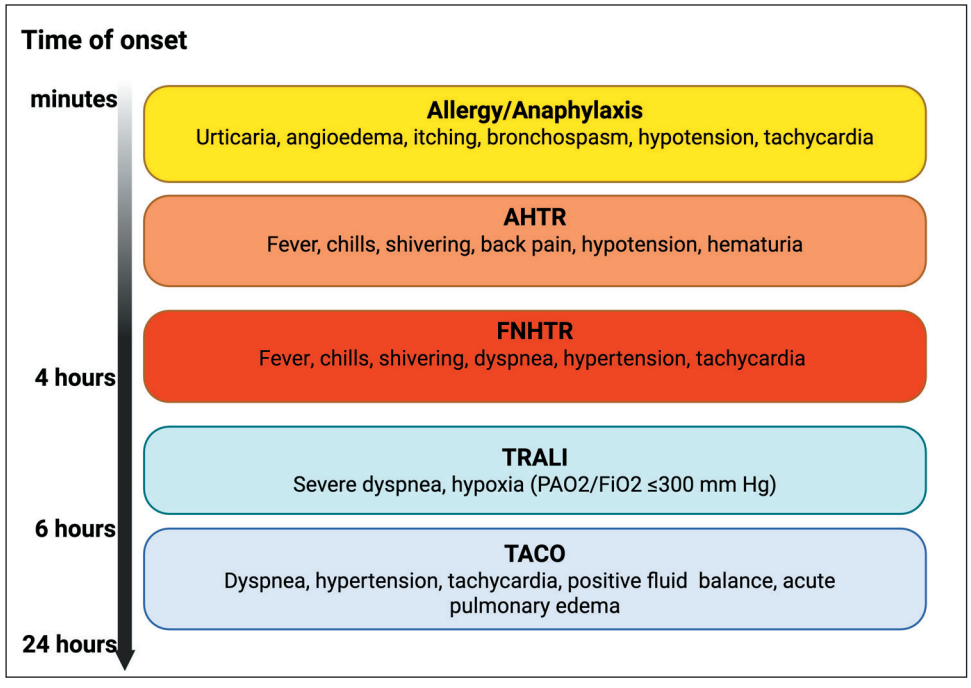

Figure 1: A practical approach for the differential diagnosis of acute transfusion reactions

AHTR: Acute hemolytic transfusion reactions, **FNHTR:** Febrile non-hemolytic transfusion reactions, **TRALI:** Transfusion-related acute lung injury, **TACO:** Transfusion associated circulatory overload

DISCUSSION

We presented four distinct acute transfusion reactions in two pediatric patients, illustrating the diagnostic complexity and management challenges, particularly in the settings of immune dysregulation during the HSCT period.

Blood transfusion reactions arise predominantly through two principal mechanisms (1). The first involves an immune response directed against transfused allogeneic cells with incompatible blood group or through repeated transfusions, which promote the development of recipient alloantibodies against donor antigens—most notably human leukocyte antigens (HLA) expressed on donor leukocytes. The resulting alloimmune recognition triggers complement activation, subsequently amplifying the coagulation cascade, and thereby contributing to a spectrum of immune-mediated tissue reactions. The second mechanism is attributable to the passive transfer of soluble inflammatory mediators present in stored blood products. These include complement fragments such as C3a and C3b (the latter often generated through contact activation upon exposure to plastic blood bag surfaces), as well as proinflammatory cytokines (e.g., IL-1β, IL-6, TNF-α), kinin system mediators (e.g., bradykinin), histamine, and prostaglandins. Collectively, these bioactive molecules can precipitate non-immune transfusion reactions through direct inflammatory and vasoactive effects.

Allergic and Anaphylactic Transfusion Reactions

Allergic and anaphylactic transfusion reactions are among the most common ATRs, with reported incidences ranging from 0.3% to 3.7%. Clinical manifestations vary from urticaria, pruritus, erythema, and angioedema to life-threatening anaphylaxis characterized by bronchospasm and hypotension. Allergic reactions occur more frequently in pediatric patients (11). In one study of 4,913 children, allergic reactions occurred in 2.4%, and one-fifth of these progressed to anaphylaxis (3). Although severe anaphylaxis remains rare—estimated at 1 in 20,000-50,000 transfusions (1)—its clinical impact can be profound. The incidence of allergic reactions is generally higher with FFP and PCs than with RCCs (12), and PCs are particularly associated with anaphylactic rather than mild allergic reactions (3). Mechanistically, allergic ATRs are type I hypersensitivity reactions, typically triggered by plasma proteins such as IgA or haptoglobin (1). Severe IgA-mediated anaphylaxis is most often seen in IgA-deficient individuals (IgA < 0.5 mg/L) who have developed anti-IgA antibodies, though reactions have also been reported in patients with normal IgA levels possessing IgA subclass- or allotype-specific antibodies (13). Another important but underrecognized mechanism is passive allergen transfer that is reported extremely rarely in the literature. Anaphylaxis has been reported in a peanut-allergic child after PC transfusion from donors who had consumed peanuts prior to donation. The digestion-resistant peptide of Ara h 2 (DRP-Ara h 2) has been identified and can be detected in circulation for up to 24 hours post-ingestion. This peptide, with a molecular size of approximately 10 kDa, retains the ability to bind IgE antibodies, potentially triggering allergic reactions. IgE antibodies reactive to both Ara h 2 and DRP-Ara h 2 have been identified in the patient with peanut allergy (10). Also a potential peanut related acute allergic transfusion reaction was described in a woman with sickle cell anemia had history of peanut allergy, during RCC transfusion. Moreover a peanut allergen Ara h 6 was detected in blood transfusion products obtained from donors (14,15). Similarly, our egg-allergic patient (Case 1) experienced anaphylaxis after receiving a platelet transfusion from a donor who had eaten eggs earlier that day and the subsequent transfusions were successfully done from donors who avoided egg consumption for at least 24 hours. This observation supports passive allergen transfer as a hypothetical and clinically inferred mechanism, based on exclusion of alternative causes, despite the absence of direct laboratory confirmation (e.g., testing the specific donor product for egg protein). The patient exhibited elevated interleukin 6 levels, which have been associated with anaphylaxis (16) and may also occur during cyclosporine treatment (17). She received cyclosporine treatment for growth versus host disease prophylaxis.

Transfusion Reactions During HSCT

Allergic or other adverse events may also occur during HSCT. When cryopreserved grafts are used, such reactions are often attributed to dimethyl sulfoxide (DMSO) or necrotic cell debris (18). However, limited data exist regarding reactions to fresh (non-cryopreserved) grafts. In one series of 331 allogeneic bone marrow transplantations, allergic reactions occurred in 3.9% of cases despite the absence of cryoprotectants (5). The etiology of urticarial reactions during HSCT remains uncertain; trace amounts of egg proteins or other unidentified factors may contribute. Potential mechanisms include infusion-product constitu-

Table I: Overview of acute transfusion reactions

Reaction Type	Definition	Clinical Features / Mechanism	Incidence / Additional Notes	Ref
Allergic/anaphylactic transfusion reactions	Type I hypersensitivity reactions, typically triggered by plasma proteins	Rapid onset Urticaria, pruritus, erythema, angioedema, life-threatening anaphylaxis characterized by bronchospasm and hypotension	Severe anaphylaxis is rare Platelet concentrates are more commonly associated with anaphylaxis	(1,3)
Febrile Non-Hemolytic Transfusion Reaction (FNHTR)*	Temperature rise of ≥1°C to ≥38°C during or within 4 hours post-transfusion	Immune response to donor leukocytes or cytokines; usually self-limiting	Incidence has declined with routine leukoreduction	(24)
Acute Hemolytic Transfusion Reaction (AHTR)**	Caused by IgM-mediated complement activation following ABO incompatibility	Rapid onset (within minutes); most severe and life-threatening transfusion complication; accounts for 80% of clerical errors	Low frequency (≈1 in 70,000 units)	(25,26)
Transfusion-Related Acute Lung Injury (TRALI)	Acute hypoxemia ($PaO_2/FiO_2 \leq 300$ mmHg) within 6 hours of transfusion	Often associated with anti-HLA antibodies in donor plasma Symptoms may mimic other causes of respiratory distress	Less frequently reported in children, differentiation from TACO or infection is essential	(27,28)
Transfusion-Associated Circulatory Overload (TACO)	Volume overload occurring within 6 hours of transfusion	Pulmonary edema, tachycardia, hypertension, and positive fluid balance	Preventable with careful fluid management and risk assessment, particularly in pediatric and cardiac-compromised recipients	(29)

* Despite routine use of leukocyte-reduction filters, Case 2 experienced FNHTR.
** In Case 2, AHTR occurred without ABO incompatibility, but subgroup incompatibility was identified.

ents, donor immune transfer, drug hypersensitivity, and the passive transfer of allergenic food proteins—such as egg protein—from donor to recipient. HSCT may both attenuate allergen-specific IgE responses and transfer donor-derived immune memory, including allergen-specific IgE or sensitized lymphocytes (19).

Beyond HSCT, similar donor-to-recipient allergy transfer has been observed after solid organ transplantation, particularly liver transplants from allergic donors to previously non-allergic children (20). These observations highlight the need for further research into immune transfer mechanisms in transplantation-related allergic phenomena.

Routine premedication with antihistamines (e.g., administered one hour before transfusion) is commonly used in patients with prior allergic reactions (1). However, several studies have questioned the efficacy of paracetamol and antihistamines in preventing mild reactions when compared with placebo in both adult and pediatric populations (21).

For IgA-deficient patients, transfusions must be sourced exclusively from IgA-deficient donors (22). Preventive measures for passive allergen transfer—particularly in highly allergic recipients—may include donor dietary restrictions before donation or enhanced allergen labeling systems for blood components. The basophil activation test (BAT) could serve as a supportive in vitro tool to identify high-risk individuals, such as hemato-oncology patients, although its availability is still limited (23).

Febrile Non-Hemolytic Transfusion Reaction

Febrile non-hemolytic transfusion reaction is defined by a temperature rise of ≥1°C to ≥38°C during or within 4 hours post-transfusion. It is usually self-limiting and results from immune responses to donor leukocytes or cytokines. The incidence has declined with routine leukoreduction (24). Leukocyte-reduction filters are routinely used in our practice during the transfusion of RCC to reduce the risk of alloimmunization and febrile non-hemolytic transfusion reactions; however, case 2 experienced FNHTR.

Acute Hemolytic Transfusion Reaction

Acute hemolytic transfusion reaction is caused by IgM-mediated complement activation following ABO incompatibility, accounting for 80% of clerical transfusion errors. Despite a low frequency (≈1 in 70,000 units), AHTR remains the most severe and life-threatening transfusion complication (25,26) starting within minutes. In Case 2, AHTR occurred despite the absence of ABO incompatibility; however, subgroup incompatibility was identified.

Given the symptom overlap in severe ATRs, the differential diagnosis should include other major transfusion-related complications. Table I presents an overview of acute transfusion reactions.

In conclusion, these two pediatric cases illustrate the diverse mechanisms and clinical presentations of acute transfusion reactions, emphasizing the critical role of allergy and immunology specialists in their diagnosis, management, and prevention. Recognition of passive allergen transfer and consideration of individualized preventive measures—including donor screening and dietary guidance—are essential to improve transfusion safety, particularly in high-risk or allergic recipients.

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Conflict of Interest

The authors have no conflicts of interest to declare.

Author Contributions

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