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Case Report

Acute autoimmune hemolytic anemia with transient aplastic crisis likely triggered by Parvovirus B19 infection in an adolescent: a case report

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ABSTRACT

Autoimmune hemolytic anemia (AIHA) is an uncommon but potentially life-threatening disorder characterized by immune-mediated destruction of red blood cells. Parvovirus B19 infection is a well-recognized cause of transient aplastic crisis due to suppression of erythroid precursors, and the simultaneous occurrence of immune hemolysis with marrow suppression presents a diagnostic challenge. We report the case of a 13-year-old boy who presented with fever, jaundice, and dark-colored urine. Laboratory evaluation revealed severe anemia (hemoglobin 5.5 g/dl), indirect hyperbilirubinemia, and a positive direct antiglobulin test confirming AIHA. Notably, the reticulocyte count was inappropriately low for the degree of hemolysis, suggesting transient marrow suppression. Extensive autoimmune and infectious investigations were largely negative except for equivocal parvovirus B19 IgM serology. The patient showed significant clinical and hematological improvement following packed red blood cell transfusion and corticosteroid therapy. This case highlights the importance of considering Parvovirus B19 infection in AIHA patients presenting with reticulocytopenia, as early recognition and appropriate supportive management are essential to prevent serious complications.

Keywords: Autoimmune hemolytic anemia, Parvovirus B19, Aplastic crisis, Pediatric hemolysis

INTRODUCTION

Autoimmune hemolytic anemia (AIHA) is an acquired disorder characterized by premature destruction of erythrocytes driven by autoantibodies directed against red cell membrane antigens. It is clinically and immunologically heterogeneous and is commonly classified by the thermal characteristics of the implicated antibodies into warm AIHA (wAIHA), cold agglutinin disease (CAD), mixed AIHA, and other variants.[1,2] Warm antibody AIHA, the most frequent form in many cohorts, is typically mediated by IgG autoantibodies that promote extravascular hemolysis, predominantly in the spleen, and is especially notable in pediatric populations where it can present acutely with anemia and reticulocytosis.[1-3] Laboratory hallmarks of AIHA include anemia with hemolysis (elevated indirect bilirubin and LDH, reduced haptoglobin), reticulocytosis, and a positive direct antiglobulin (Coombs) test, which remains a cornerstone for diagnosis in most patients.[1,2]

Parvovirus B19 is a single-stranded DNA virus with a well-established tropism for erythroid progenitor cells via the P antigen receptor, leading to a transient arrest of erythropoiesis that lasts approximately one week in immunocompetent hosts.[4,5] In individuals with high red cell turnover or chronic hemolysis (as seen in AIHA or other hemolytic conditions), this transient erythropoietic pause can precipitate a profound and clinically significant anemia, termed transient aplastic crisis, due to the sudden insufficiency of erythroid production.[4,5]

Although AIHA and Parvovirus B19–induced erythroid aplasia are both well described in the literature, their

coexistence remains rare but clinically relevant. Viral infections have been proposed to trigger autoimmunity through mechanisms including immune dysregulation and molecular mimicry, which may culminate in AIHA or in immune-mediated cytopenias during parvoviral infection, or both in susceptible patients.[4,5] Several reports have highlighted cases in which Parvovirus B19 infection has interacted with autoimmune hematologic processes, including AIHA, enhancing the complexity of management in affected children and adults.[4-9]

In pediatric patients, AIHA typically presents with insidious or acute onset of pallor, jaundice, and fatigue, and the diagnostic approach integrates clinical assessment with laboratory evidence of hemolysis and a DAT-based classification to guide therapy, which commonly includes corticosteroids as first-line therapy and rituximab for refractory disease in selected cases.[2,6,9] When Parvovirus B19 coinfection or post-infectious aplasia accompanies AIHA, careful interpretation of hematologic indices and bone marrow studies is essential to distinguish isolated AIHA from parvovirus-induced aplasia and to tailor treatment accordingly, while ensuring vigilance for potential immune-mediated relapse or escalation of hemolysis.[4-8]

Herein, we present a pediatric case illustrating the dual pathology of AIHA and Parvovirus B19–associated erythroid aplasia. The report emphasizes on the diagnostic interplay between AIHA and transient parvoviral erythroid suppression, the clinical implications of coexisting processes on transfusion requirements and hematologic recovery, and the therapeutic considerations when immune-mediated hemolysis occurs concomitantly with a parvoviral–induced cytopenic phase. This synthesis draws on contemporary evidence detailing the pathophysiology, diagnostic strategies, and management paradigms for AIHA and Parvovirus B19 infection, including their potential intersection in pediatric hematology.[1,2,4-9]

CASE REPORT

A 13-year-old previously healthy male presented with low-grade fever and upper respiratory symptoms for ten days, followed by abdominal discomfort, dark urine, and scleral icterus. There was no prior chronic illness. Family history revealed parental consanguinity. On examination, he was pale, icteric, and tachycardic (109/min). Blood pressure was 110/50 mmHg. There was no hepatosplenomegaly. Cardiovascular and respiratory examinations were unremarkable.

Initial laboratory evaluation showed hemoglobin 5.5 g/dl, red blood cell count 1.75 million/mm^3, and packed cell volume 16%. Total bilirubin was 4.67 mg/dl with predominantly indirect fraction, consistent with hemolysis. Leukocytosis was present while platelet count remained normal. Direct antiglobulin test was positive, confirming immune-mediated hemolysis. Reticulocyte count was 1%, unexpectedly low for severe hemolysis, raising suspicion of marrow suppression. By day six, reticulocyte count increased to 6%, indicating recovery of erythropoiesis.

Autoimmune screening including antinuclear antibody profile was negative. Complement levels were normal. Infectious workup including blood cultures, malaria, dengue, influenza, and enteric fever tests was negative. Parvovirus B19 IgM was equivocal. Given clinical context and reticulocytopenia, Parvovirus-related aplastic crisis was strongly suspected. Peripheral smear showed features of hemolysis. Sickling test and hemoglobin electrophoresis were normal.

Due to severe anemia and risk of cardiovascular compromise, two units of cross-matched packed red blood cells were transfused. Intravenous methylprednisolone was administered as first-line therapy for warm AIHA. Supportive management including folic acid supplementation and close monitoring was provided. Hemoglobin improved to 9 g/dl by discharge. Bilirubin levels normalized gradually. Reticulocyte count rose to 6%, reflecting marrow recovery. The patient stabilized clinically with resolution of tachycardia and jaundice.

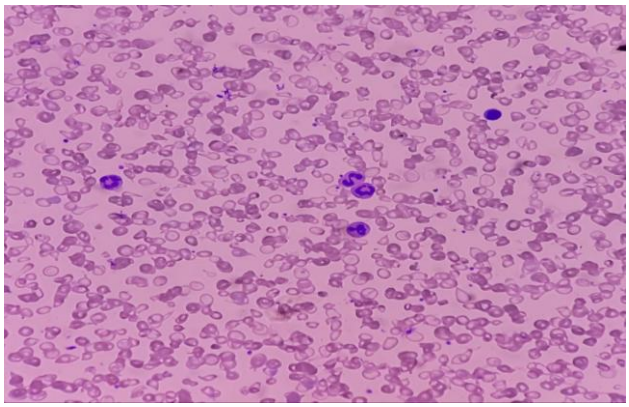

Figure 1: Peripheral blood smear demonstrating spherocytes and polychromasia consistent with immune-mediated hemolysis.

DISCUSSION

This case illustrates a complex hematologic and infectious milieu in a previously healthy 13-year-old boy, in whom an immune-mediated hemolytic process (Coombs-positive AIHA) occurred in the setting of Parvovirus B19–related transient erythroblastopenia with reticulocytopenia, followed by hematologic recovery. The clinical trajectory—initial ten days of fever and upper respiratory symptoms, indirect hyperbilirubinemia with marked anemia, reticulocytopenia that later evolved toward reticulocytosis by day six—parallels the well-described pattern of Parvovirus B19–driven marrow suppression on a background of active hemolysis. Reticulocytopenia in a patient with ongoing hemolysis should thus prompt consideration of an accompanying Parvovirus B19

infection or marrow suppression even when serologies are equivocal, with molecular testing (e.g., Parvovirus B19 DNA PCR) providing critical diagnostic support when IgM results are uncertain.[10-12]

The autoimmune component is underscored by a positive direct antiglobulin test, confirming AIHA; this process may coexist with or be unmasked by viral-induced marrow suppression, necessitating a dual-focused therapeutic approach that addresses both immune-mediated hemolysis and transient erythroid failure.[10,13] The patient's peripheral smear, along with negation of sickle cell disease and hemoglobinopathies by negative sickling tests and normal electrophoresis, helped delineate the hemolytic process from inherited RBC disorders, reinforcing the value of a comprehensive differential in such presentations.[10,14] Supportive transfusion for severe anemia two units of cross-matched packed red blood cells was appropriately employed to avert cardiovascular compromise in the face of tachycardia and reduced oxygen-carrying capacity, aligning with pediatric AIHA and parvovirus-driven aplastic crisis management principles described in the literature.[11,15] The initial use of high-dose corticosteroids for warm AIHA is consistent with standard first-line therapy for autoimmune hemolysis in this age group, even though Parvovirus B19–associated marrow suppression can necessitate extended monitoring and, in refractory cases, the use of IVIG; while IVIG was not documented in this case, its consideration is supported in mixed AIHA/parvovirus contexts, particularly when rapid correction of hemolysis or marrow recovery is desired.[10-12]

Folic acid supplementation served as an important adjunct given the increased erythropoietic turnover and the transient marrow suppression, a detail consistently emphasized in reviews of hemolytic and aplastic processes.[10,11] Patient's favorable outcome, with hematologic recovery and resolution of jaundice, mirrors reported experiences where careful supportive care and corticosteroid therapy yield positive trajectories in concurrent AIHA and Parvovirus B19–related marrow suppression, though the precise sequence and interplay of pathogenic mechanisms can vary among cases.[10,13,14] Taken together, this case reinforces a pragmatic diagnostic and therapeutic framework: in children and adolescents with reticulocytopenic anemia superimposed on ongoing hemolysis, clinicians should pursue Parvovirus B19 testing (preferably PCR-based) and maintain readiness to treat AIHA with steroids while providing robust supportive care and transfusion as needed until marrow recovery occurs; acknowledgement of potential serologic limitations and the possibility of concurrent autoimmune phenomena is essential to avoid misattribution to a single etiologic process.[10-13] Finally, the familial context of consanguinity, while not explanatory for this acute presentation, warrants consideration of underlying hereditary hemolytic disorders in future care planning, given the known associations between hereditary RBC defects and parvovirus-triggered aplastic events.[13,16]

CONCLUSION

This case highlights the simultaneous occurrence of immune-mediated hemolytic anemia and Parvovirus B19–related marrow suppression in a previously healthy adolescent. Key lessons include the importance of recognizing reticulocytopenia amid ongoing hemolysis as a potential sign of concurrent parvoviral infection, the utility of Coombs testing to define AIHA, and the role of supportive transfusion and corticosteroid therapy in achieving rapid hematologic stabilization. Early, integrates diagnostic workup emphasizing PCR-based Parvovirus B19 testing when serology is equivocal facilitates timely management and favorable recovery, with marrow recovery typically paralleling clinical improvement. Awareness of this dual pathology can prevent misattribution to a single etiology and informs a balanced treatment approach that combines immune modulation with supportive care.

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