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Pure Red Cell Aplasia with Overlapping Hemolytic Anemia in a Term Neonate: A Case Report

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Abstract

Pure Red Cell Aplasia (PRCA) is an infrequent hematological disorder marked by profound inhibition of erythroid production with preservation of other marrow cell lines. Its occurrence during the neonatal period is rare and often leads to delayed recognition. We describe a term neonate who presented with progressive pallor and severe anemia during the second week of life. Hematological evaluation revealed persistent normocytic normochromic anemia with marked reticulocytopenia. Bone marrow examination demonstrated selective erythroid hypoplasia with intact myeloid and megakaryocytic maturation, along with erythroblasts containing intranuclear inclusions, pointing toward Parvovirus B19–associated PRCA. Laboratory parameters also suggested a mild concomitant hemolytic process. The infant required repeated packed red blood cell transfusions and showed gradual hematologic recovery following intravenous immunoglobulin administration. This report emphasizes the need for heightened clinical suspicion of viral-induced PRCA in neonates presenting with unexplained severe anemia and highlights the benefit of early diagnostic evaluation and targeted therapy.

Keywords: Pure Red Cell Aplasia; Neonate; Anemia; Parvovirus B19

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Introduction

Pure red cell aplasia (PRCA) is an uncommon hematologic condition marked by anemia resulting from impaired erythroid production. It is defined by normocytic, normochromic anemia accompanied by reticulocytopenia and profound erythroid suppression, with erythroblasts accounting for less than 1% of nucleated cells within the bone marrow [1, 2, 3].

Reliable data on the overall incidence or prevalence of this disorder in the general population are lacking. In contrast, the congenital form of PRCA, known as Diamond–Blackfan syndrome, occurs at an estimated rate of about 5–7 cases per one million live births [4].

Pure red cell aplasia is a markedly heterogeneous disorder with wide clinical and pathological variability. It may occur in inherited or acquired forms, with Diamond–Blackfan syndrome (DBA) being the most extensively characterized congenital variant [5]. DBA is a sporadic, heterogeneous condition presenting soon after birth with red cell aplasia and skeletal defects. It is commonly associated with growth failure and anomalies involving the head, heart, and lungs.

Acquired PRCA can be primary with no identified underlying cause or secondary in association with a variable number of disorders. The primary PRCA is a disorder of an autoimmune-mediated mechanism that involves erythroid precursors most likely due to selective T-cell or natural killer NK-cells. The secondary PRCA may develop secondary to multiple conditions, including autoimmune or connective tissue disorders, leukemias, lymphoproliferative diseases, ABO-incompatible stem cell transplantation, solid malignancies, and viral infections such as HIV, parvovirus B19, HTLV-1, and EBV. There are no pathognomonic clinical features of PRCA; patients usually present with anemia-related manifestations, including fatigue, reduced exercise capacity, palpitations, and, in those with compromised cardiac function, presyncope or syncope [4].

Case Presentation

A term neonate weighing 2.6 kg, appropriate for gestational age, delivered by LSCS in the setting of chorioamnionitis (CIAB), developed progressively worsening pallor. The infant was admitted on day of life (DOL) 15 for evaluation of anemia. On examination the neonate was conscious, had severe pallor, with heart rate of 138/min and respiratory rate of 64/min. Chest retractions and pedal oedema was present. No lymphadenopathy, cyanosis or clubbing was noted. On investigation, his hemoglobin was 4.7g/dl, total leucocyte count was 5800/mm^3, DLC- Neutrophils-25%, Lymphocytes 60%, Monocytes-10%, Eosinophils-05%, Basophils-00%. Platelets were 5,60,000/mm3, RBC count – 2.8 million/mm^3, hematocrit 12%, reticulocyte count - 0.1%, ESR-18mm/hour. On peripheral blood film examination normocytic normochromic picture of RBCs was seen with no polychromasia (Figure 1). Both indirect and direct Coombs tests were positive, accompanied by mildly elevated lactate dehydrogenase levels and a slightly increased INR of 1.33, indicating a concurrent hemolytic component.

Bone marrow aspiration revealed slightly hypocellular marrow with increased myeloid to erythroid ratio of 19:1 and depression of erythroid series. Only 5% proerythroblasts were seen with absence of mature normoblasts (Figure 2a). Myeloid series showed normal differentiation and maturation. Megakaryocytes were adequate and functional. Differential count of non-erythroid series showed myeloblasts-00%, promyelocyte-10%, myelocyte-5%, metamyelocyte-02%, neutrophils including band forms- 43%, lymphocytes and their precursors-35%, eosinophils and their precursors-3% and plasma cells-2%, without any excess blasts, granulomatous inflammation, fungal elements or other abnormal infiltrates. Keeping in view the clinical history and examination, laboratory findings and bone marrow aspiration findings a diagnosis of acquired PRCA was made. In view of the presence of characteristic nuclear inclusions on bone marrow examination (Figure 2b), a diagnosis of parvovirus B19-associated pure red cell aplasia was considered. To further confirm parvovirus B19-associated PRCA, serological testing and molecular testing was advised; however, these investigations could not be performed due to financial constraints, and the sample could not be sent to a reference laboratory.

The infant received three packed red blood cell transfusions during the hospital course. On DOL 40, intravenous immunoglobulin therapy was administered, followed by vitamin K supplementation. Subsequently, hemoglobin levels showed a gradual upward trend, and no new clinical concerns were observed. The infant remained clinically stable and was discharged on DOL 57.

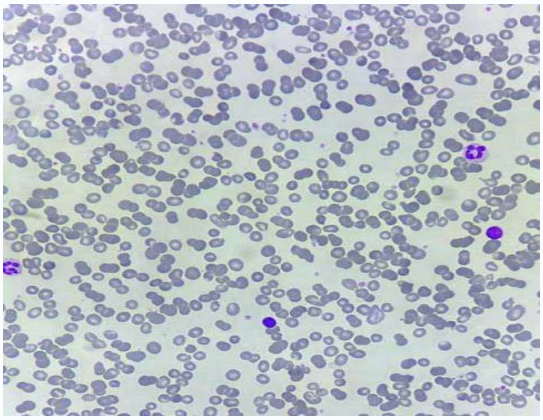

Figure 1: Microphotograph of peripheral blood smear displaying predominantly normocytic normochromic red blood cells. (Leishman stain;400x).

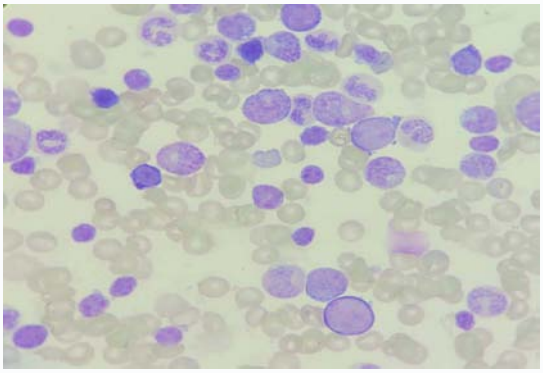

Figure 2a: Bone marrow aspirate smear demonstrates selective suppression of erythropoiesis with scattered few erythroblasts.Other non-erythroid hematopoietic lineages shows normal morphology and maturation (Leishman stain; 400x).

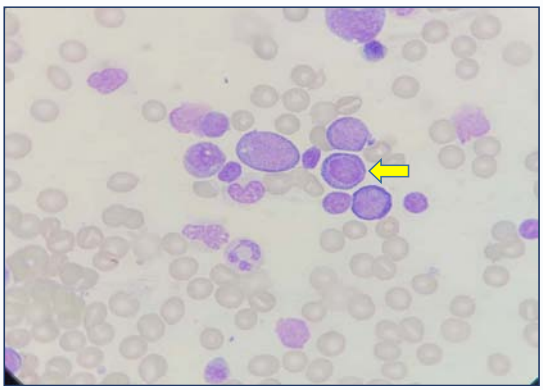

Figure 2b: Bone marrow aspirate smear demonstrates erythroblast displaying conspicuous intranuclear inclusion (highlighted with arrow) supporting a viral-associated pure red cell aplasia (Leishman stain; 400x).

Discussion

Pure red cell aplasia (PRCA) is a hematologic condition defined by profound suppression of erythroid production with preservation of other hematopoietic lineages. It can occur at any age. The disorder presents with severe anemia, marked reticulocytopenia, and approximately 0.5% late erythroblasts. In some cases, the marrow is dominated by early proerythroblasts with complete absence of late erythroid forms. Overall marrow cellularity is typically normal, with no abnormalities in non-erythroid lineages [6].

Earlier studies have indicated that approximately 55% of adult cases represent secondary pure red cell aplasia, whereas in the pediatric population, most patients—about 59%—have idiopathic disease [7]. In most instances, diagnosing PRCA is relatively straightforward, with bone marrow evaluation typically revealing fewer than 0.5% late erythroblasts. However, in certain cases, erythroblast counts in marrow aspirates and biopsies may appear normal or elevated, accompanied by maturation arrest, increased hematogones, and lymphocyte infiltration [8]. However, similar erythroid maturation arrest can occur in regenerating marrow, making it essential to conduct a thorough evaluation to exclude other potential causes

when such bone marrow findings are observed [9].

In all cases, secondary PRCA should be excluded by evaluating potential underlying causes, as this has important implications for both management and prognosis.

The overarching goal in PRCA management is to achieve normal hemoglobin levels without transfusion support.

Our study highlights Parvovirus B19–associated pure red cell aplasia as an important and potentially underrecognized cause of severe anemia in term neonates. The coexistence of positive direct and indirect Coombs tests with mild biochemical evidence of hemolysis suggests a possible overlapping immune-mediated component. Timely supportive care with packed red blood cell transfusions and targeted therapy with intravenous immunoglobulin resulted in progressive hematologic recovery, underscoring the importance of early recognition and appropriate management to achieve favorable outcomes in affected neonates.

Conclusion

This case illustrates that acquired pure red cell aplasia, although rare in the neonatal period, may present with overlapping features of hemolytic anemia. Awareness of this atypical presentation and meticulous hematological assessment are essential for accurate diagnosis and optimal patient management. This case underscores the critical role of meticulous microscopic examination in establishing the diagnosis of viral associated pure red cell aplasia, particularly in resource-limited settings where advanced diagnostic facilities and health insurance coverage are often lacking.

Data availability: The data sets used during the current study are available from the corresponding author per reasonable request.

Conflicts of interest: The author declares that they have no conflicts of interest.

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Consent: Informed/written consent was obtained from the patient's parents for publication of this case report and accompanying images.

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