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Platelet–Erythropoiesis–Inflammation Axis in Preterm Anemia: Clinical Mechanisms and Management Implications

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Abstract

Anemia of prematurity (AOP) is a common hematological disorder in neonatal intensive care units, driven by impaired erythropoietin response, shortened red cell survival, iatrogenic blood loss, and inflammatory stress. Beyond their traditional hemostatic role, platelets are increasingly recognized as modulators of hematopoiesis and immunity. They release cytokines and growth factors that directly influence erythroid progenitors, while inflammatory signaling further alters both platelet function and erythropoietic activity. This narrative review examines the interplay between platelets, erythropoiesis, and inflammation in preterm anemia, highlighting mechanistic pathways and the potential clinical utility of platelet indices such as mean platelet volume and immature platelet fraction. Understanding this triad provides new perspectives on the pathophysiology of AOP and offers opportunities for biomarker-guided diagnosis, optimized transfusion practices, and novel therapeutic strategies.

Keywords: Preterm anemia, Platelets, Erythropoiesis, Inflammation, Neonatology

Introduction

Anemia of prematurity (AOP) is one of the most frequent hematological complications in neonatal intensive care units (NICUs), affecting a substantial proportion of infants born before 32 weeks of gestation. It is characterized by a multifactorial pathogenesis, including inadequate endogenous erythropoietin (EPO) production, shortened red blood cell (RBC) lifespan, frequent phlebotomy losses, nutritional deficiencies, and systemic inflammatory stress. While the traditional understanding of AOP has largely centered on erythropoiesis and transfusion requirements, recent evidence points to a broader network of cellular and molecular interactions that extend beyond red cell production alone [1-2]. Platelets, once regarded solely as mediators of hemostasis, are now recognized as versatile immune and hematopoietic regulators. They secrete a wide array of cytokines, chemokines, and growth factors—including platelet factor-4 (PF4), transforming growth factor-β (TGF-β), and thrombopoietin (TPO)—which influence hematopoietic stem cell (HSC) function and erythroid progenitor activity. In addition, platelets actively interact with immune cells and endothelial cells, contributing to both the maintenance of vascular homeostasis and the propagation of inflammatory responses [3-4].

Inflammation, a frequent feature in preterm infants due to sepsis, necrotizing enterocolitis (NEC), or perinatal stress, further complicates the hematological picture. Pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) not only impair erythropoietic activity but also alter iron metabolism through hepcidin regulation. Activated platelets, in turn, amplify inflammatory cascades by releasing immune mediators and microparticles, creating a feedback loop that links thrombopoiesis, erythropoiesis, and inflammation [5-6]. This evolving perspective highlights the need to integrate platelet biology into our understanding of preterm anemia. By examining the interplay between platelets, erythropoiesis, and inflammation, this review aims to (1) synthesize mechanistic insights from experimental and clinical studies, (2) explore the diagnostic and prognostic potential of platelet indices, and (3) discuss therapeutic implications for optimizing the management of AOP.

Aim

The aim of this narrative review is to explore the interrelationship between platelets, erythropoiesis, and inflammation in the pathogenesis of anemia of prematurity.

Methods

This article was developed as a narrative review synthesizing current evidence on the interplay between platelets, erythropoiesis, and inflammation in preterm anemia. A comprehensive literature search was conducted using PubMed, Scopus, and Web of Science databases, covering studies published up to September 2025. The search terms included combinations of *"anemia of prematurity," "platelets," "erythropoiesis," "inflammation," "platelet indices,"* and *"neonates."* Original research articles, systematic reviews, meta-analyses, and relevant experimental studies were considered. Inclusion criteria focused on studies addressing the role of platelets in hematopoiesis, erythropoietic regulation, and inflammatory modulation in the context of neonatal or preterm populations. Exclusion criteria included case reports, conference abstracts without full texts, and studies not available in English.The selection process emphasized mechanistic insights, clinical associations, and translational implications. The included literature was critically appraised for relevance and synthesized thematically to construct an integrated understanding of how platelet biology intersects with erythropoiesis and inflammation in the pathophysiology and management of anemia of prematurity.

Platelets Beyond Hemostasis: Functional Plasticity in Preterm Infants

Platelets have traditionally been regarded as small, anucleate cell fragments with a singular

purpose—maintaining hemostasis through clot formation. However, in recent years, this narrow view has shifted as accumulating evidence demonstrates that platelets are highly versatile cells with roles that extend far beyond coagulation. In preterm infants, whose hematological and immunological systems are immature, platelet biology exhibits distinctive features that influence not only vascular stability but also erythropoiesis and immune regulation [7]. Neonatal platelets, particularly in preterm infants, differ in both structure and function compared to those of term infants and adults. They are often fewer in number and show variable responsiveness to agonists, reflecting the immaturity of megakaryopoiesis and bone marrow regulation. Despite these limitations, they actively secrete an array of bioactive molecules, including platelet factor-4 (PF4), transforming growth factor-β (TGF-β), vascular endothelial growth factor (VEGF), and thrombopoietin (TPO). These factors serve as important signals within the bone marrow microenvironment, shaping hematopoietic stem cell behavior and influencing erythroid progenitor maturation [8-9]. Beyond their hematopoietic functions, platelets interact closely with immune cells and endothelial cells, positioning themselves as critical mediators at the intersection of inflammation and hematopoiesis. For instance, activated platelets can release microparticles that enhance immune responses, recruit leukocytes, and amplify inflammatory signaling. At the same time, they help preserve endothelial integrity, which is essential in ensuring adequate oxygen delivery and supporting the survival of developing red cells [10-11]. In the context of preterm anemia, this functional plasticity becomes particularly relevant. Platelets can both support and hinder erythropoiesis depending on the surrounding inflammatory milieu. While growth factors like TPO may indirectly promote red cell production by stimulating stem cell proliferation, molecules such as PF4 can exert inhibitory effects on erythroid colony formation. This delicate balance underscores the dual nature of platelet biology in preterm infants—acting as both protectors and potential suppressors of hematopoiesis [12].

Erythropoiesis in Preterm Infants: Challenges and Dysregulation

Erythropoiesis, the tightly regulated process of red blood cell production, is profoundly altered in preterm infants. Unlike term neonates and adults, preterm infants enter extrauterine life with an immature hematopoietic system that struggles to adapt to the rapid physiological changes of early neonatal life. This immaturity, combined with clinical stressors encountered in neonatal intensive care, sets the stage for the development of anemia of prematurity [13]. One of the most critical challenges lies in the reduced production of erythropoietin (EPO). In the fetus, the liver is the primary site of EPO synthesis, but it is less responsive to hypoxia compared with the kidney, which gradually assumes this role later in gestation. Preterm infants, therefore, experience a blunted EPO response to falling hemoglobin levels, limiting their ability to stimulate erythroid progenitor proliferation in the bone marrow. This inadequate hormonal drive, compounded by immature bone marrow activity, results in insufficient red cell production precisely when the infant's oxygen demands are rapidly increasing [14].

Another contributing factor is the shortened lifespan of neonatal red blood cells. Whereas adult erythrocytes circulate for approximately 120 days, those in preterm infants often survive only 35–50 days. This accelerated turnover exacerbates the decline in hemoglobin levels and places additional strain on the underdeveloped erythropoietic system. Frequent phlebotomy for diagnostic testing in NICUs further accelerates this decline, creating an iatrogenic contribution to anemia that is difficult to offset in such small and vulnerable patients [15-16]. Nutritional limitations also play a role in erythropoietic dysregulation. Preterm infants often experience deficiencies in iron, folate, and vitamin B12, essential cofactors for hemoglobin synthesis and red cell maturation. Iron stores, in particular, are accrued late in gestation; therefore, infants born prematurely begin life with lower reserves and are highly susceptible to iron-restricted erythropoiesis [17]. Emerging evidence suggests that platelet-

derived factors add another layer of complexity to erythropoietic regulation. Thrombopoietin (TPO), secreted by both hepatocytes and platelets, may support progenitor expansion, whereas platelet factor-4 (PF4) has been shown to inhibit erythroid colony formation under certain conditions. The balance between these stimulatory and inhibitory platelet-derived signals may influence the trajectory of anemia in preterm infants [18].

Inflammation as a Mediator of Preterm Anemia

Inflammation is a critical, though often underappreciated, factor in the pathogenesis of anemia of prematurity. Preterm infants are uniquely vulnerable to inflammatory insults due to immature immune defenses, fragile intestinal and respiratory barriers, and the high risk of exposure to infections and invasive procedures in neonatal intensive care units. Conditions such as neonatal sepsis, necrotizing enterocolitis (NEC), and bronchopulmonary dysplasia frequently create a systemic inflammatory environment that profoundly disrupts normal hematopoietic processes [19-20]. At the core of this disruption is the impact of pro-inflammatory cytokines on erythropoiesis. Interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interferon-gamma (IFN-γ) exert direct inhibitory effects on erythroid progenitor cells, reducing their proliferation and differentiation. These cytokines also alter the hormonal regulation of red cell production by suppressing erythropoietin (EPO) synthesis, further limiting the capacity of the bone marrow to mount an adequate response to anemia [21].

In addition to suppressing erythropoiesis, inflammation profoundly affects iron metabolism. IL-6 induces the production of hepcidin, a key regulator of iron homeostasis, which restricts intestinal iron absorption and promotes sequestration of iron within macrophages. This leads to functional iron deficiency, even when systemic iron stores appear adequate, depriving developing erythroid cells of the substrate necessary for hemoglobin synthesis. Such inflammatory blockade of iron utilization mirrors the pathophysiology of anemia of chronic disease and contributes significantly to the severity of anemia in preterm infants [22]. Platelets play an amplifying role in this inflammatory milieu. Activated platelets release pro-inflammatory mediators, such as interleukin-1β and platelet-derived microparticles, which intensify leukocyte recruitment and endothelial activation. Rather than simply reflecting inflammation, platelets act as active participants, perpetuating a feedback loop in which inflammation impairs erythropoiesis while simultaneously driving platelet activation, which in turn sustains and magnifies the inflammatory response [23-24]. The combined effects of cytokine-mediated marrow suppression, iron restriction, and platelet-driven immune amplification explain why inflammation often transforms a mild physiological anemia of prematurity into a clinically significant disorder requiring transfusion support. This interplay highlights the importance of early recognition and management of inflammatory conditions in preterm infants, not only to reduce morbidity but also to alleviate hematological consequences [25].

Platelet Indices as Biomarkers in Preterm Anemia

In recent years, platelet indices have gained increasing attention as potential biomarkers for monitoring hematological disturbances in preterm infants. Traditionally, platelet counts have been assessed mainly for the detection of thrombocytopenia, but advances in hematology analyzers now allow clinicians to evaluate a wider spectrum of platelet parameters that may reflect bone marrow activity, inflammatory status, and erythropoietic drive. In the setting of anemia of prematurity, these indices provide valuable insights into the complex interplay between thrombopoiesis, erythropoiesis, and systemic inflammation [26-27]. One of the most widely studied indices is the **mean platelet volume (MPV)**, which reflects average platelet size. Larger platelets are generally more metabolically and functionally active, and in neonates, an elevated MPV may signal heightened platelet turnover or inflammatory activation. In preterm infants with anemia, changes in MPV have been

associated with systemic inflammatory conditions, suggesting that this index could help identify infants in whom anemia is compounded by inflammation [28].

Another useful measure is the **immature platelet fraction (IPF)**, which indicates the proportion of newly released platelets in circulation. A rising IPF suggests active bone marrow response, while a low IPF may point to impaired thrombopoietic or hematopoietic activity. In preterm infants, IPF has been proposed as an indirect marker of marrow responsiveness to both erythropoietic and thrombopoietic stress. Since erythropoiesis and thrombopoiesis share common progenitors, changes in IPF may parallel suppressed or stimulated erythropoietic activity, making it a potential adjunct in evaluating anemia severity [29]. Composite markers such as the **platelet-to-lymphocyte ratio (PLR)** are also being explored. This index integrates platelet activity and immune response, offering a window into the inflammatory burden carried by preterm infants. A high PLR has been correlated with inflammatory conditions such as sepsis and necrotizing enterocolitis, which are themselves significant contributors to the aggravation of anemia (Table 1) [30-31].

Table 1: Key Platelet Indices and Their Clinical Relevance in Preterm Anemia

Platelet Index	Biological Significance	Clinical Relevance in Preterm Anemia	Limitations / Notes
Mean Platelet Volume (MPV)	Reflects average platelet size; larger platelets are metabolically active	Elevated MPV may indicate increased platelet turnover or systemic inflammation; correlates with anemia severity	Affected by pre-analytical factors; gestational age-dependent reference ranges required
Immature Platelet Fraction (IPF)	Proportion of newly released platelets; marker of bone marrow activity	Higher IPF reflects active thrombopoiesis and potential erythropoietic drive	Limited availability in some NICUs; interpretation may vary with sepsis or inflammation
Platelet Count (PLT)	Absolute number of circulating platelets	Thrombocytopenia may signal marrow suppression or sepsis; complements anemia monitoring	Non-specific; does not provide functional information
Platelet-to-Lymphocyte Ratio (PLR)	Integrates platelet and lymphocyte counts; reflects inflammatory burden	Elevated PLR may indicate systemic inflammation contributing to anemia	Influenced by infection, stress, and steroid exposure
Plateletcrit (PCT)	Total platelet mass in circulation	May reflect overall platelet contribution to hematopoiesis	Less studied in neonates; limited normative data

Clinical Implications and Therapeutic Perspectives

Understanding the interplay between platelets, erythropoiesis, and inflammation in preterm anemia has significant clinical implications for neonatal care. Traditionally, management strategies have focused on red blood cell transfusions and nutritional supplementation. While these remain essential, emerging insights suggest that a more nuanced approach—one that considers platelet function and inflammatory status—may improve outcomes and reduce the risks associated with repeated transfusions. Platelet indices, such as mean platelet volume (MPV) and immature platelet fraction (IPF), can serve as adjunctive biomarkers to identify infants at higher risk of severe anemia or those whose marrow is actively responding to hematopoietic stress. Integrating these measures into routine

monitoring may allow clinicians to tailor transfusion thresholds more precisely, potentially avoiding unnecessary interventions while ensuring timely support for those who truly need it [32].

Erythropoiesis-stimulating agents (ESAs), including recombinant erythropoietin, represent another therapeutic avenue. The efficacy of ESAs in preterm infants can be influenced by inflammatory mediators and platelet-derived signals, suggesting that optimizing the timing and dosing of these agents in the context of systemic inflammation could enhance their effectiveness. Addressing underlying inflammatory triggers—through prompt management of sepsis, necrotizing enterocolitis, or other inflammatory conditions—may further support erythropoietic response and reduce the severity of anemia [33]. Nutritional optimization remains a cornerstone of therapy. Adequate provision of iron, folate, and vitamin B12 is essential to support both erythropoiesis and platelet production. Iron supplementation strategies should be individualized, considering the infant's gestational age, baseline iron stores, and inflammatory status, as inflammation-induced hepcidin upregulation can impair iron utilization [34].Mechanistic insights into platelet-mediated modulation of erythropoiesis and inflammation may yield novel therapeutic targets. Interventions aimed at modulating platelet-derived cytokines, or at dampening excessive inflammatory signaling without compromising host defense, could offer a precision medicine approach to managing anemia in preterm infants. Such strategies have the potential to reduce transfusion dependence, minimize associated risks, and improve long-term developmental outcomes (Table 2 and Table 3) [35-36].

Table 2: Mechanistic Pathways Linking Platelets, Erythropoiesis, and Inflammation in Preterm Anemia

Pathway / Factor	Mechanism	Impact on Erythropoiesis	Impact on Platelets	Clinical Relevance
Thrombopoietin (TPO)	Stimulates hematopoietic stem cell proliferation	Indirectly enhances erythroid progenitor expansion	Promotes megakaryocyte maturation and platelet production	May support recovery from anemia; potential therapeutic target
Platelet Factor-4 (PF4)	Released from alpha granules; binds to erythroid progenitors	Can inhibit erythroid colony formation under inflammatory conditions	Marker of platelet activation	Elevated PF4 may exacerbate anemia during systemic inflammation
Interleukin-6 (IL-6)	Cytokine released during infection/inflammation	Suppresses erythropoietin production; inhibits progenitor proliferation	Activates platelets; contributes to pro-inflammatory feedback	Links systemic inflammation to worsened anemia
Hepcidin	Liver-derived peptide regulated by IL-6	Sequesters iron in macrophages; reduces iron availability for erythropoiesis	Indirect effect via reduced erythropoietic demand	Explains functional iron deficiency in inflamed preterm infants
Platelet Microparticles	Vesicles released from activated platelets	May modulate bone marrow microenvironment	Reflect platelet activation and turnover	Elevated levels indicate inflammation-driven suppression of erythropoiesis

Table 3: Therapeutic and Management Strategies Targeting Platelet-Erythropoiesis-Inflammation Interplay in Preterm Anemia

Strategy	Mechanism / Rationale	Evidence / Observations	Potential Limitations
Red Blood Cell Transfusion	Directly corrects anemia	Widely used; improves oxygen delivery	Risk of transfusion reactions, iron overload, and infection; may not address underlying pathology
Erythropoiesis-Stimulating Agents (ESAs)	Stimulate RBC production by mimicking erythropoietin	Can reduce transfusion needs; effectiveness influenced by inflammation	Blunted response in high cytokine states; risk of thrombosis
Iron Supplementation	Provides substrate for hemoglobin synthesis	Essential in infants with low iron stores; improves erythropoiesis	Functional iron deficiency if hepcidin elevated; risk of oxidative stress if overdosed
Anti-inflammatory Interventions	Target sepsis, NEC, or cytokine-driven suppression	Reduces inflammatory suppression of erythropoiesis; may improve platelet function	Limited specific anti-inflammatory agents; timing critical
Platelet Monitoring (MPV, IPF, PLR)	Biomarkers to guide transfusion and therapy	Helps identify infants at risk; may predict response to ESAs	Reference ranges vary with gestational age; not universally available
Nutritional Optimization (Folate, Vitamin B12)	Supports DNA synthesis and RBC maturation	Essential for effective erythropoiesis	Deficiencies may persist if absorption impaired; requires careful dosing

Conclusion

Anemia of prematurity is a multifactorial condition shaped by the combined effects of immature erythropoiesis, shortened red cell lifespan, nutritional limitations, and systemic inflammation. Emerging evidence highlights the central role of platelets not only in hemostasis but also as active regulators of erythropoiesis and modulators of inflammatory responses. The dynamic interplay between platelets, erythroid progenitors, and cytokine-mediated pathways creates a complex feedback loop that influences the onset, severity, and progression of anemia in preterm infants. Platelet indices, including mean platelet volume and immature platelet fraction, offer promising avenues for early detection, risk stratification, and monitoring of anemia, while understanding the impact of inflammation provides insights into potential therapeutic interventions. Integrating these perspectives with optimized transfusion practices, targeted use of erythropoiesis-stimulating agents, and nutritional support can enhance the management of preterm anemia and potentially improve short- and long-term neonatal outcomes.Future research should focus on elucidating the molecular mechanisms by which platelets influence erythropoiesis, defining clinically relevant biomarker thresholds, and exploring novel therapeutic strategies that modulate platelet function and inflammatory pathways. Such efforts will advance a more precise, individualized approach to the prevention and treatment of anemia in preterm infants.

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