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REVIEW ARTICLE

Neonatal Anaemia: Contemporary View of Primary Diagnosis and Management

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ABSTRACT

Neonatal anaemia affects preterm and term infants globally and is marked by reduced erythrocyte count or haemoglobin. It presents a significant clinical challenge, arising from physiological and pathological factors, demanding careful management to prevent increased mortality and neuro developmental risks. Diagnosis employs a tiered approach, starting with a complete blood count and progressing to focused investigations like Coombs tests and advanced testing like haemoglobin electrophoresis. Management strategies are tailored to etiology and severity, emphasizing minimal blood loss, erythropoiesis-stimulating agents, Delayed cord clamping, disease-specific treatments, and nutritional support, which play crucial roles. Along with strategies, regular monitoring and long-term neuro developmental assessments are essential for positive outcomes. Future directions focus on optimizing transfusions, reducing iatrogenic blood loss, and improving prenatal screening, targeted management, and preventive strategies, which are vital for improved outcomes.

KEYWORDS

• Neonates • Haemoglobin • Erythropoietin • Haematocrit • Prenatal Screening

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INTRODUCTION

Neonatal anaemia is a common haematological condition affecting newborns, characterized by a reduced erythrocyte count or haemoglobin levels. This condition is characterized by hemoglobin or hematocrit levels below age-specific reference ranges, resulting in diminished oxygen-carrying capacity.[1] It represents a significant clinical challenge in perinatal medicine, affecting preterm infants and term neonates. It arises from physiological processes (e.g., developmental erythropoietin changes) and pathological factors (E.g., blood loss, hemolysis). Maternal anemia during pregnancy reduces neonatal hemoglobin by 1.38g/dL on average, demonstrating the connections between generations health[2]. The unique physiological adaptations of the neonate to extrauterine life, coupled with developmental immaturity of hematopoietic processes, create a complex pathophysiological landscape that requires careful clinical management. Untreated cases correlate with increased mortality and neuro developmental risks. Understanding the mechanisms and risk factors associated with neonatal anemia is essential for early diagnosis and prevention[3].

Embryological Development of Erythrocytes

RBC creation, or erythropoiesis, starts early in the embryo's development. Erythropoiesis happens in three stages throughout fetal life. Mesoblastic Stage: The yolk sac's mesenchyme produces red blood cells throughout the first two months of intrauterine life. The liver is the primary organ responsible for producing red blood cells during the hepatic stage, which begins in the third month of intrauterine life. Additionally, engaged in erythropoiesis are the lymphoid and spleen organs. During the final three months of intrauterine life, known as the Myeloid Stage, the liver and red bone marrow create red blood cells.[4] The red bone marrow of all bones up to the age of 20 years produces red blood cells (RBCs) in newborns, children, and adults.[5]

Pathophysiology of Neonatal Anemia

The pathophysiology of neonatal anemia is influenced by multiple factors, such as inadequate erythropoiesis, increased red blood cell destruction, and blood loss. The condition can be classified based on the timing of onset: **Early-onset anemia**: This usually occurs within the first week of life and is often due to blood loss or hemolysis. **Late-onset anemia**: Develops after the neonatal period, commonly associated with prematurity and nutritional deficiencies.[6] All newborns experience a degree of physiological anemia during the first months of life. Hemoglobin concentration naturally decreases throughout this phenomenon, reaching its lowest point in term newborns at 8–12 weeks (nadir of 9–11 g/dL) and in preterm infants at 4–8 weeks (nadir of 7-9 g/dL).[7]

a. **Physiological causes:** It includes the following aspects of,
   - **Developmental erythropoietin (EPO) dynamics:** Preterm infants demonstrate significantly lower plasma erythropoietin levels, which can be attributed to shifts in production from the hepatic source to renal production, as well as an increase in the clearance rates of hormones from the bloodstream.[8]
   - **Oxygen set-point regulation:** Fetal hemoglobin's intrinsic high oxygen affinity is essential for inhibiting the signaling pathways linked to tissue hypoxia, which delays the production of erythropoietin.[9]
   - **Shorter RBC lifespan:** In preterm infants, the lifespan of erythrocytesis markedly shorter, averaging between 35 to 50 days, in contrast to the typical lifespan of 90 days observed in term infants.[10]
   - **Transition from fetal haemoglobin (HbF) to adult hemoglobin (HbA):** This transition is vital for enhancing oxygen delivery to tissues; however, it concurrently results in a delay in the production of new red blood cells.[11]

b. **Pathological Causes** It includes various causes like Blood loss, hemolysis, Impaired erythropoiesis, andanemia of prematurity, and other related causes are portrayed in Table 1.[12-,16]

Table 1: Pathological causes of Neonatal Anaemia

1. Blood Loss
Prenatal/Perinatal Haemorrhage
Fetomaternal hemorrhage (occurs in 50-75% of all pregnancies)
Twin-to-twin transfusion syndrome
Placental abruption or previa
Cord accidents (velamentous cord insertion, vasa previa)
Traumatic delivery[12]
Iatrogenic Blood Loss
Phlebotomy for laboratory testing (primary cause in NICU patients)
Estimated blood loss of 0.8-3.1 mL/kg/day in critically ill neonates[13]
2. Hemolysis
Immune-mediated hemolysis
ABO incompatibility (most common cause of hemolytic disease of the newborn)
Rh incompatibility
Minor blood group incompatibilities (Kell, Duffy, Kidd)
Non-immune Hemolysis
Inherited red cell membrane defects (spherocytosis, elliptocytosis)
Enzymatic deficiencies (G6PD deficiency, pyruvate kinase deficiency)
Hemoglobinopathies (alpha thalassemia)
Microangiopathic processes (DIC, Kasabach-Merritt syndrome)
Infection-related hemolysis (bacterial, viral, protozoal)[14]
3. Impaired Erythropoiesis
Nutritional Deficiencies
Iron deficiency (late manifestation)
Vitamin E deficiency
Folate deficiency
Vitamin B12 deficiency[15]
Bone Marrow Failure
Congenital bone marrow failure syndromes (Diamond-Blackfananemia, Fanconi anemia)
Infection-related marrow suppression (parvovirus B19, CMV)
Leukemia and other infiltrative disorders
4. Anemia of Prematurity
A multifactorial condition affecting preterm infants, characterized by:
Inadequate erythropoietin production
Rapid postnatal growth with expanded blood volume
Shortened red cell lifespan
Increased phlebotomy losses
Immature bone marrow response[16]

Laboratory Diagnosis

A tiered approach for investigating neonatal anaemia is essential for accurate diagnosis and effective management. This method involves a systematic evaluation of several levels of investigation based on clinical presentation, gestational age, and other relevant factors: They are,

Tier 1: Initial assessment

The main indicator used to diagnose anemia is the quantity of the Hb protein present in RBCs. There are numerous excellent laboratory indicesavailable for these measures.

- **Complete Blood Count (CBC)**: This provides baseline hemoglobin levels and red blood cell indices.

- **Hemoglobin (Hb):**These are primary indices for diagnosing anemia. Low values indicate a decrease in RBC or their ability to carry oxygen.Diagnostic thresholds include: 14–22 g/dL for term newborns and 12–18 g/dL for Preterm newborns
- **RBC count:** The RBC is not a good indicator of the severity of anemia, as it can remain unchanged even when changes in other indices, such as the MCV or MCHC, can lower the total amount of Hb. Diagnostic thresholds include <4.5 million/µL for men and <4 million/µL for women.[17]. The abnormal values trigger further investigation into RBC indices and morphology depicted in Table 2.
- **Clinical Context**: assess the infant's clinical status, including symptoms such as pallor, tachycardia, or respiratory distress. The presence of these symptoms helps to determine the urgency of further investigations and potential interventions.

Table 2: Comparison of RBC indices and morphology with interpretation[18]

Parameter	Calculation	Normal Range (Adults)	Normal Range (Infants)	Interpretations
MCV	MCV=PCV %×10 Red cell count	80–100 fL	69.5–86.7 fL (4–6 months)	Decreasediron deficiency anemia; increased in megaloblastic anemia.
MCH	MCH=Hb (g/dL)×100 Red cell count in mm^3	27–32 pg	32–34 pg (newborns)	Decreased iron deficiency anemia; increased in megaloblastic anemia.
MCHC	MCHC = Hb (g/dL)×100 PCV (%)	32–36 g/dL	320–360 g/L	Decreased iron deficiency anemia; increased in hereditary spherocytosis and megaloblastic anemia.
PCV or Hematocrit	The ratio of RBC to serum expressed in percentage	Men: 39–45%, Women: 33–43%	Birth: 47–75%, 2 weeks: 41–65%	Decreased in anemia (<30%); increased in severe dehydration or polycythemia.
RDW	Index to measure degree of anisocytosis of RBCs	11.5–14.5%	Birth: 15.5–20%	Increased in sideropenicanemia, megaloblastic anemia, immune hemolyticanemia; normal in thalassemia.
ESR	Measurement of sedimentation of RBC after one hour	Men: 4–14 mm/hr, Women: 6–20 mm/hr	Newborns: 0–2 mm/hr, Children: 3–13 mm/hr	Very high (0-1 mm/hr): TB, multiple myeloma, rheumatoid arthritis; Very low (0 - 1 mm/hr): polycythemia, cardiac failure, hypo or afibrinogenaemia. Low : Sickle cell anaemia.

Abbreviations: MCV - Mean Corpuscular Volume, **MCH** - Mean Corpuscular Hemoglobin, **MCHC**- Mean Corpuscular Hemoglobin Concentration**, PCV** - Packed Cell Volume-, **RDW -** Red Cell Distribution Width, **ESR** - Erythrocyte Sedimentation Rate, **RBC** - Red Blood Cells, **g/dl** – Gram per Deciliter, **g/l** – Gram per liter, **pg**- picogram.

Tier 2: Focused Investigations

If initial assessments suggest the presence of anemia, additional investigations may encompass the following procedures:

- **Blood Type and Coombs Test:** Neonatal anemia can occur due to blood group incompatibility between the mother and the fetus. This incompatibility often involves Rh or ABO blood groups. When a mother's immune system detects the fetus's red blood cells as foreign, it may produce antibodies against them. These antibodies can cross the placenta and attach to the fetus's red blood cells, leading to their destruction (hemolysis) after birth. This test is conducted to determine the risk of hemolytic anemia that may arise due to incompatibilities in ABO or Rh blood types. Immune-mediated hemolysis is identified by the Direct Coombs Test (DCT), which looks for complement proteins or antibodies adhered to the surface of red blood cells. A positive DCT is a sign of Hemolytic Disease of the Newborn (HDN), which is caused by maternal antibodies that break down the newborn's red blood cells. Finding Rh-positive antibodies during pregnancy is crucial for the Indirect Coombs Test (ICT), which may have an impact on subsequent pregnancies. A newborn with a positive DCT is more likely to experience severe anemia and jaundice, which calls for treatment and monitoring, including phototherapy or exchange blood transfusions.[19]
- **Reticulocyte Count:** This count assists in evaluating the responsiveness of the bone marrow to the presence of anemia. It measures the output of the bone marrow in producing red blood cells. A low reticulocyte count indicates potential production issues, such as in cases of aplastic anemia. A high reticulocyte count suggests ongoing bleeding or the occurrence of hemolysis.
- **Iron Studies&Vitamin Levels:** These assessments include measuring serum ferritin levels, which are typically low in instances of iron deficiency, along with transferrin saturation and total iron-binding capacity. Deficiencies in Vitamin B12 and folate are known to result in macrocytic anemia.[20]
- **Hemolysis Markers:** The presence of elevated levels of lactate dehydrogenase (LDH), increased indirect bilirubin, and reduced haptoglobin levels collectively indicates the destruction of erythrocytes.
- **Peripheral Blood Smear:** This examination is performed to assess the morphology of erythrocytes, which can provide insights into specific haematological conditions, as portrayed in Table 3.

Table 3: Red blood cell morphology and its abnormalities[18]

Size Abnormalities (Anisocytosis)	Shape Abnormalities (Poikilocytosis)	Color Abnormalities	Inclusions	Arrangement Abnormalities
Microcytes	Spherocytes	Hypochromia	Basophilic stippling	Rouleaux formation
Macrocytes	Elliptocytes/Ovalocytes	Hyperchromia	Howell - jolly bodies	Autoagglutination
Normocytes	Schistocytes(Fragmented cells)	Polychromasia	cabot rings	
	Target cells		Pappenheimer bodies/ siderotic granules	
	Teardropcells(Dacrocytes)		Hemoglobin H inclusions	
	Burr cells (Echinocytes)		Heinz bodies	
	Acanthocytes (Spur cells)		Hemoglobin C crystal	
	Sickle cells			
	Bite cells			
	Pencil - shaped cells			
	Teardrop and pear-shaped cells			
	Stomatocytes			

Tier 3: Advanced Testing

In instances where preliminary investigations fail to provide a definitive diagnosis, the implementation of more specialized tests may become necessary:

- **Hemoglobin Electrophoresis:** Hemoglobin electrophoresis is a vital diagnostic tool for neonatal anemia,

especially those caused by inherited hemoglobin disorders. It helps to identify abnormal hemoglobin variants that may cause hemolytic anemia or other red blood cell abnormalities in newborns. This test helps identify conditions like sickle cell anemia and thalassemia by separating hemoglobin types based on their electrical charge. Early diagnosis allows for timely interventions, such as prophylactic antibiotics for sickle cell disease or monitoring for thalassemia complications.[21]

- **Genetic Testing & Bone Marrow Biopsy:** This examination is performed for hereditary conditions, particularly in instances where there exists a recorded family history of blood disorders. While this procedure is occasionally necessary, it may be considered in exceptional cases.

MANAGEMENT

- Neonatal anemiarequires tailored management strategies based on etiology, gestational age, and clinical severity. Evidence-based management strategies and key approaches include monitoring, transfusion protocols, nutritional support, and targeted therapies for underlying causes.

General Management Principles

- **Diagnostic Evaluation:** Confirm the presence of anemia by measuring hemoglobin (Hb) levels that have been adjusted for gestational age and postnatal day. Identify the underlying causes by gathering maternal history, conducting a physical examination, and performing laboratory tests, which include the Coombs test, reticulocyte count, and peripheral smear. Exclude the possibility of hemolysis, which can be indicated by elevated bilirubin, elevated lactate dehydrogenase (LDH), and low haptoglobin levels, as well as to rule out blood loss due to factors such as placental abnormalities or twin-twin transfusion syndrome.
- **Minimize Iatrogenic Blood Loss:** Limit the frequency and volume of phlebotomy by utilizing micro-sampling techniques in conjunction with closed blood-conservation systems.[22]
- **Erythropoiesis-Stimulating Agents (ESAs) - Recombinant erythropoietin (r-HuEPO):**Decreases the necessity for blood transfusions in preterm infants when initiated after the first week of life.Standard dosage: 200–400 IU/kg/week administered subcutaneously. Frequently administered alongside iron supplementation (2–6 mg/kg/day).[23]
- **Supplements:** Iron is an essential micronutrient required for hemoglobin synthesis and oxygen transfer. For preterm infants, initiate supplementation at a dose of 2–4 mg/kg/day by the age of 2 weeks to prevent iron depletion, and for term infants, provide 1–2 mg/kg/day in instances where maternal iron stores are found to be deficient. Administer Folate/Vitamin B12 supplements in cases where deficiency is suspected, such as in the context of maternal malnutrition[24]
- **Delayed Cord Clamping (DCC):**Delayed Cord Clamping (DCC) is a procedure that allows for the transfusion of iron-rich blood from the placenta to the newborn, improving iron stores and reducing the risk of iron deficiency anemia in infancy. It also results in higher hemoglobin levels at birth and during the first few months of life, helping prevent neonatal anemia. DCC is associated with a lower need for blood transfusions in preterm infants due to increased blood volume and improved hematocrit levels.DCC is also linked to reduced risks of intraventricular hemorrhage and necrotizing enterocolitis in preterm infants.For both term and preterm infants, the American College of Obstetricians and Gynecologists advises postponing cord clamping for at least 30 to 60 seconds. Monitoring for jaundice is crucial as DCC may increase the risk of jaundice requiring phototherapy.[25]

Disease-Specific Treatments

1. **Hemolytic Anemia** caused by Alloimmune hemolysis (such as Rh or ABO incompatibility) can be treated by phototherapy for the management of hyperbilirubinemia and administering intravenous immunoglobulin (IVIG). Hereditary spherocytosis or pyruvate kinase (PK) deficiency can be treated

by performing a splenectomy in severe instances, typically at a later stage in the individual's life.

2. **Anemia of Prematurity (AOP)** assess weekly monitoring of Hb levels; initiate transfusions when levels exceed established thresholds, and also enhance protein and caloric intake to promote optimal erythropoiesis[16]. Blood transfusions are recommended for infants experiencing acute blood loss, which is defined as a loss of 20% or 10% of blood volume, and those with decreased oxygen delivery symptoms after volume resuscitation. In chronic blood loss, blood transfusions are considered based on the infant's respiratory support needs, which are dependent on an HCT or hemoglobin value measured from a central venous or arterial sample detailed in table 4[1,26]. Exchange transfusion is reserved for severe hemolytic anemia with hyperbilirubinemia. Risks include volume overload, infection, and necrotizing enterocolitis.

Table 4: Based on 2024 guidelines provided by AABB (Association for the Advancement of Blood & Biotherapies) for preterm infants <30 weeks' gestation[26]

Postnatal Week	On Respiratory Support	Without Respiratory Support
Week 1	≤11 g/dL	≤10 g/dL
Week 2	≤10 g/dL	≤8.5–9 g/dL
≥Week 3	≤9 g/dL	≤7–8 g/dL

3. **Infection-Related Anemia:** Neonatal anemia is primarily caused by infections, including bacterial infections like sepsis and malaria, which can lead to hematological abnormalities like anemia, leukocytosis, thrombocytopenia, and coagulation disorders. Common pathogens include Coagulase-Negative Staphylococci, Klebsiella pneumoniae, and Staphylococcus aureus. Malaria is a significant cause, especially in endemic regions, and other viral infections like cytomegalovirus and herpes simplex can also contribute to anemia.[27]

Follow-Up And Prognosis

Regular monitoring of hemoglobin levels is crucial, especially in preterm infants or those with a history of significant blood loss or hemolysis. Long-term neuro developmental assessments are necessary for infants with severe anemia, as iron deficiency during infancy can have lasting effects on cognitive and motor development. Chronic anemia management involves assessing for complications like iron overload and ensuring adequate growth and development milestones. In complex cases, consultation with a hematologist is recommended for tailored management strategies. With preventive strategies and regular follow-up, the prognosis for neonatal anemia is generally favourable.[28]

Future Directions

It focuses on optimizing red blood cell (RBC) transfusions through evidence-based guidelines and neonatal-specific blood banking practices. Reducing iatrogenic anemia is vital, with innovations like point-of-care testing and micro-collection techniques minimizing phlebotomy losses. Promoting delayed cord clamping (DCC) as a standard practice can enhance iron stores and reduce anemia risk. Early detection of iron deficiency using advanced markers and personalized nutrition strategies tailored to individual needs are standard approaches reducing neonatal mortality rate. Improved prenatal screening and management of maternal anemia can prevent hemolytic disease of the newborn (HDN). Long-term follow-up studies are essential to understand the neuro developmental impacts of neonatal anemia. Emerging technologies, such as biomarker discovery, collaborative databases for transfusion practices, and international research collaboration, aim to standardize guidelines and improve positive outcomes of neonates globally.[29]

CONCLUSION

Mild anemia in healthy term or preterm infants usually resolves without intervention, as erythropoiesis resumes naturally by 4-6 months of age. Severe anemia, caused by conditions like hemolytic disease, may require transfusions or exchange transfusions but often has favourable outcomes if managed according to appropriate standards. Severe or untreated anemia can lead to neuro developmental delays and cognitive and motor impairments, which may persist into adolescence or adulthood if not addressed early. Severe cases with acute complications may have higher mortality rates.

Hence, health care professionals, especially in the neonatology team, address the neonates with a standardized approach of appropriate treatment and regular follow-up, which makes the neonates recover well and directly reduces consequences of outcomes.

Conflict of Interest: There is no conflict of interest among all the authors involved in the study.

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