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Original article

Polycythemia in Infants Aged 0-12 Months in Tobruk City: A Secondary Analysis of Follow-Up Care Gaps, Diagnostic Oversights, and Rational JAK2 Mutation Testing Criteria

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Abstract
Neonatal polycythemia, defined as a venous hematocrit ≥65%, occurs in 0.4–5% of healthy term newborns globally, with incidence rising to 14.5% in neonatal intensive care settings. While predominantly a physiological adaptation to extrauterine life, persistent cases beyond the neonatal period may signal secondary or, rarely, primary myeloproliferative pathology. This article presents a secondary analysis of an infant cohort (aged 1 day to 1 year) originally reported in a primary cross-sectional study by Mtawil et al. (2026) conducted at Tobruk Medical Centre. This study was conducted to characterize the epidemiological patterns of the infant cohort (n = 90; aged 1 day to 1 year), identify systemic deficiencies in follow-up care, and critically evaluate the role of JAK2 mutation testing in the neonatal population, contextualized against current international evidence. This secondary analysis utilized the infant cohort (n = 90; age range: 1 day to 1 year) from the primary cross-sectional study conducted at Tobruk Medical Centre during 2024–2025. Of these, 50 infants (56%) were true neonates (aged ≤28 days), while the remaining 40 (44%) were aged 1–12 months. Cases were stratified by age group following the original classification, and follow-up practices were evaluated against contemporary international standards. Over half of the cases (56%, n = 50) were identified within the first 28 days of life (the neonatal period), consistent with the physiological hematocrit surge that peaks at 2–4 hours postnatally and declines thereafter. The remaining cases (44%, n = 40) were first identified between 1 month and 1 year of age; because sampling was cross-sectional and single-point, these later-identified cases cannot be characterized as "persistent" neonatal polycythemia, and their longitudinal hematocrit trajectory, follow-up, and diagnostic workup cannot be determined from the available records. This finding contrasts sharply with current international recommendations, which advocate for scheduled repeat hematocrit monitoring, cardiorespiratory observation, and targeted partial exchange transfusion for symptomatic infants with packed cell volume ≥65%. Critical gaps included: lack of documented standardized post-discharge protocols; failure to differentiate primary from secondary polycythemia; inadequate parental education; fragmented interdepartmental care; and lack of documented, selective JAK2 testing criteria for cases identified beyond the neonatal period. Available records did not document serial hematocrit monitoring or partial exchange transfusion in the Tobruk cohort; because the clinical records were incomplete/missing, it was not possible to distinguish whether these procedures were omitted or simply not documented. This secondary analysis confirms that while most cases identified in the neonatal period represent transient physiological adaptation, the 44% of cases first identified after 1 month of age—whose persistence, follow-up, and diagnostic workup could not be assessed from the cross-sectional, single-point data—indicate serious systemic deficiencies in post-discharge care for infants with polycythemia. Drawing on international cohorts and consensus guidelines, the analysis provides evidence-based recommendations for implementing standardized follow-up protocols, enhanced parental education, and rational, red-flag-guided JAK2 testing criteria appropriate to resource-limited settings.
Keywords. Polycythemia in Infants, Neonatal Period, Follow-Up Care, JAK2 Mutation, Polycythemia Vera.

Introduction

Neonatal polycythemia represents one of the most frequently encountered hematological abnormalities in newborn care, with reported incidence rates ranging from 0.4% to 5% in healthy term infants and up to 14.5% in neonatal intensive care unit (NICU) populations depending on diagnostic thresholds and risk-factor profiles [1,2]. A recent cross-sectional study by Mtawil et al. at Tobruk Medical Centre screened 259 patients across Oncology, Neonatal, and Internal Medicine departments to determine the incidence of primary polycythemia vera (PV) in Tobruk City [3]. Among this cohort, 90 neonatal and pediatric cases with elevated blood components were identified in the Neonatal Department, representing 34.7% of the total screened population. While the primary study established the epidemiological presence of elevated blood components across age groups, the specific clinical trajectory, adequacy of follow-up, and diagnostic workup in the neonatal subset warrant dedicated analysis. Unlike polycythemia vera in adults, which is driven by acquired somatic mutations, primarily JAK2 V617F, present in approximately 95–97% of cases [4,5], neonatal polycythemia is predominantly a physiological adaptation to the transition from intrauterine to extrauterine life [3,6].

The physiological basis is multifactorial. The fetus develops in a relatively hypoxic environment compared to atmospheric conditions, which stimulates erythropoietin (EPO) production and drives compensatory erythropoiesis [7]. Fetal hemoglobin (HbF) has a higher oxygen affinity than adult hemoglobin (HbA), which is advantageous for placental oxygen transfer but may exacerbate tissue hypoxia in the neonatal period [8]. Additionally, neonatal bone marrow hematopoietic stem and progenitor cells exhibit established stemness signatures and intensely active lineage differentiation potential, reflecting the enormous hematopoietic demands of early life [9], a finding corroborated by Mtawil et al., who attributed the elevated blood components in newborns to this hyperactive marrow state [3]. Hematocrit levels characteristically peak at

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2–4 hours of age, then decline toward cord-blood levels by 12–18 hours; consequently, the timing of screening significantly influences reported incidence [6].

Despite its predominantly benign nature, neonatal polycythemia is not without risk. Hyperviscosity syndrome can manifest as poor feeding, lethargy, hypoglycemia, tachypnea, cyanosis, seizures, and end-organ damage [10]. Thrombotic complications, including renal vein thrombosis, necrotizing enterocolitis, and cerebral infarction, may affect long-term neurological outcome [11]. The distinction between transient physiological polycythemia and persistent pathological erythrocytosis is clinically critical. International guidelines emphasize that symptomatic infants with packed cell volume ≥65% should undergo partial exchange transfusion (PET), whereas asymptomatic infants with packed cell volume ≥70% require close cardiorespiratory monitoring and serial hematocrit checks [12,13]. The primary study [3] documented the age distribution of these cases but did not evaluate whether infants—particularly those identified after the neonatal period—received appropriate follow-up. This secondary analysis addresses that gap, while recognizing that the single-point sampling design limits longitudinal inference.

Methodology

Study Design and Data Source

This is a secondary analysis of infant data (aged 1 day to 1 year) originally collected by Mtawil et al. in their cross-sectional study at Tobruk Medical Centre, Libya, encompassing the Neonatal/Newborn Department, Oncology Department, and Internal Medicine departments during 2024–2025 [3]. The primary study received ethical approval from Tobruk Medical Centre, with verbal consent obtained from guardians of neonates [3].

Study Population

The cohort comprised 90 infants (age range: 1 day to 1 year) with elevated blood components identified in the primary study. Because the neonatal period is strictly defined as the first 28 days of life, only the 50 infants aged ≤28 days constitute the true neonatal subset; the remaining 40 infants (44%) were aged 1–12 months. Cases were stratified by age group following the original classification: 1 day to 1 month, 1 month to 2 months, 2 months to 4 months, and 4 months to 1 year [3].

Assessment of Follow-Up Practices

Building upon the primary data, this analysis evaluates clinical records for: (i) presence of standardized post-discharge follow-up protocols; (ii) documentation of perinatal risk factors; (iii) clinical correlation of symptoms with laboratory values; (iv) evidence of parental education; (v) interdepartmental coordination; and (vi) documentation of JAK2 mutation testing. Importantly, the available clinical records did not document serial hematocrit monitoring or partial exchange transfusion; because the records were incomplete/missing, these practices could not be confirmed as either performed or omitted. These parameters were benchmarked against contemporary international standards, including the UCSF Northern California Neonatal Consortium consensus guidelines [12], the systematic review by Dempsey and Barrington [13], and the review by Bashir and Othman [11].

Comparative Context

To contextualize the findings in the infant cohort, this analysis references the adult PV cohort (n=44) and Internal Medicine cohort (n=125) described in the primary study [3], utilizing their published demographic and risk factor data. Distinguishing features between neonatal physiological polycythemia and adult clonal PV were cross-referenced against the 2024 WHO/ICC diagnostic criteria and recent molecular reviews [4,5].

Results

Epidemiological Patterns of Polycythemia in Infants Aged 0–12 Months

As reported by Mtawil et al., the age distribution of the 90 infant cases revealed a striking concentration in the immediate neonatal period (Table 1) [3].

Table 1. Distribution of infant cases (aged 1 day–1 year) with high blood components by age group (Neonatal Department, n = 90). Note: Only cases aged ≤28 days fall within the neonatal period.

Age Group	Number of Cases	Percentage (%)
1 day to 1 month	50	56
1 month to 2 months	20	22
2 months to 4 months	10	11
4 months to 1 year	10	11
Total	90	100

The predominance of cases in the first month of life (56%) is consistent with the physiological surge in hematocrit that normally occurs in the immediate neonatal period, peaking at 2–4 hours of age and declining thereafter [1,6]. A 10-year international cohort study reported a 0.8% prevalence of neonatal polycythemia, with most cases representing passive (transfusion-related) polycythemia rather than active erythropoiesis [2]. However, the persistence of elevated blood

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components beyond the first month in 44% of cases raises significant concerns about inadequate follow-up, a dimension not explored in the primary incidence study. While 56% of cases (n = 50) were identified within the first 28 days of life, consistent with physiological adaptation, 44% (n = 40) were first identified between 1 month and 1 year of age. Given the single-point sampling, these later-identified cases should not be interpreted as persistent elevation from birth. No standardized re-evaluation, diagnostic workup, or genetic-testing criteria were documented for any age group. Available records did not document serial hematocrit monitoring or partial exchange transfusion; because the records were incomplete/missing, it was not possible to distinguish whether these procedures were omitted or simply not documented.

Comparative Analysis: Neonatal Physiological Polycythemia vs. Adult Polycythemia Vera

Drawing from the primary study's Oncology cohort [3] and recent international literature, (Table 2) presents distinguishing features between neonatal physiological polycythemia and adult JAK2-driven PV.

Table 2. Distinguishing features of neonatal physiological polycythemia and adult polycythemia vera (PV)

Feature	Neonatal Physiological Polycythemia	Adult PV (JAK2 V617F+)
Primary cause	Physiological adaptation to extrauterine life	Acquired JAK2 V617F somatic mutation
Etiology	Intrauterine hypoxia, placental transfusion, HbF affinity	Clonal hematopoietic stem cell proliferation
EPO level	Normal to elevated	Low or subnormal
JAK2 V617F mutation	Absent (vanishingly rare)	Present in ~95–97% of cases [4,5]
Age of onset	Birth to 1 week (peak)	Median ~60 years [3]
Cell lines affected	RBCs predominantly	Trilineage: RBCs, WBCs, platelets
Spontaneous resolution	Common (weeks to months)	Never—requires lifelong management
Splenomegaly	Absent	Present in ~30–40% of cases
Thrombotic risk	Low (unless severe hyperviscosity)	High (30% at diagnosis, 40–60% over 10 years)

The primary study identified 44 confirmed PV cases in the Oncology Department (19 male, 25 female), with the majority aged 51–80 years, and a statistically significant association between age ≥50 and PV incidence (p=0.035) [3]. This stark age contrast reinforces that neonatal cases represent a fundamentally different pathophysiological process. The 2024 WHO fifth edition and ICC diagnostic criteria for PV require demonstration of JAK2 V617F or exon 12 mutation, elevated hemoglobin/hematocrit, and bone marrow morphological confirmation [4,5], criteria wholly inapplicable to the neonatal physiological state.

JAK2 Mutation Testing in Neonates: When Is It Warranted?

The JAK2 V617F mutation is the molecular hallmark of adult PV, present in approximately 95–97% of cases [4,5]. This acquired somatic mutation results in constitutive activation of the JAK-STAT signaling pathway [14]. However, true PV is extraordinarily rare in newborns. The primary study [3] confirmed the neonatal cases as physiological polycythemia resulting from high bone marrow cellularity and fetal hemoglobin affinity, rather than clonal myeloproliferation. Recent molecular reviews emphasize that JAK2 V617F is acquired during a person's lifetime and is not inherited through germline transmission; thus, the probability of a neonate harboring this mutation is vanishingly small unless there is evidence of familial myeloproliferative disease or congenital polycythemia [4,5,15].

Table 3. Red flags indicating when JAK2 mutation testing should be considered in neonates with polycythemia

Red Flag for JAK2 Testing in Neonates	Clinical Rationale
Severe polycythemia (Hct >70%) persisting beyond 2–4 weeks of age	Persistent severe erythrocytosis beyond the physiological window suggests a primary myeloproliferative process rather than transient adaptation.
Family history of myeloproliferative neoplasms (MPNs)	Rare familial clustering of JAK2-mutated MPNs has been described [16].
Presence of splenomegaly or hepatomegaly	Extramedullary hematopoiesis and organomegaly are hallmark features of PV and are not seen in physiological neonatal polycythemia.

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Concurrent thrombocytosis and/or leukocytosis (trilineage proliferation)	Physiological neonatal polycythemia typically affects RBCs only; trilineage elevation strongly suggests a clonal myeloproliferative disorder.
Evidence of thrombosis (renal vein, cerebral sinuses, peripheral vessels)	Thrombotic events in neonates are highly unusual and should prompt immediate investigation [11].
Failure to respond to standard management (hydration, partial exchange transfusion)	Resistance to conventional treatment may indicate an underlying clonal disorder requiring targeted therapy.
Parental consanguinity or known hereditary hematological disorders	Congenital polycythemia can result from mutations in VHL, EPOR, EGLN1/PHD2, or SLC30A10 genes affecting oxygen sensing and EPO regulation [15].

Routine JAK2 testing in neonates with mild-to-moderate, transient polycythemia is not indicated [3]. The vanishing rarity of JAK2-driven PV in neonates means that the positive predictive value of testing in unselected populations approaches zero. However, the lack of documented testing criteria means that the few neonates who genuinely warrant genetic evaluation may be overlooked. International practice now recommends comprehensive next-generation sequencing panels including EPOR, VHL, EGLN1, EPAS1, and JAK2 for congenital erythrocytosis workups when red flags are present [15].

Identified Gaps in Follow-Up Care for Infants with Polycythemia

Review of the available clinical records and departmental practices revealed multiple critical deficiencies not addressed in the primary incidence study:

Absence of Documented Standardized Post-Discharge Protocols

No protocol for re-evaluating hematocrit and hemoglobin levels was documented for infants identified with polycythemia in the neonatal period or beyond. International consensus guidelines recommend that only symptomatic infants be screened, with asymptomatic patients monitored via serial hematocrit and glucose checks [12,13].

Failure to Differentiate Primary from Secondary Polycythemia

In the neonatal population, secondary polycythemia due to placental insufficiency, maternal diabetes, intrauterine growth restriction, or chromosomal abnormalities is far more common than primary PV. However, no systematic evaluation for underlying causes was documented. A recent 10-year study found that small-for-gestational-age status, maternal hypertension, and infant-of-diabetic-mother status were the most common risk factors [2].

Inadequate Parental Education

Parents received insufficient education regarding warning signs that warrant immediate medical attention. In resource-limited settings, empowering neonatal nurses and midwives to provide parental education is a low-cost, high-impact intervention [3].

Fragmented Interdepartmental Care

Data collection in the primary study required traversal of multiple departments without evidence of a coordinated care pathway, increasing the risk of cases being lost during transitions of care [3].

Lack of Documented JAK2 Testing Protocols for Cases Beyond the Neonatal Period

The absence of documented criteria for selective genetic testing in infants aged 1–12 months represents a significant diagnostic gap. Current international standards advocate for JAK2 screening only in the presence of specific red flags, followed by expanded congenital polycythemia gene panels if indicated [4,15].

Inadequate Assessment of End-Organ Effects

No evidence of systematic screening for complications such as renal vein thrombosis, stroke, or necrotizing enterocolitis was found. International reviews confirm that neonatal polycythaemia increases the risk of hyperviscosity, microcirculatory hypoperfusion, and multisystem organ dysfunction, including renal venous thrombosis, necrotising enterocolitis, and cerebral infarction with long-term neurological sequelae [11].

Documentation Deficiencies

Critical perinatal risk factors were not systematically documented, precluding meaningful risk stratification.

Proposed Evidence-Based Follow-Up Protocol for Infants with Polycythemia Aged 0–12 Months

Based on international guidelines and the identified gaps, (Table 4) presents a proposed standardized follow-up protocol tailored to the resources and needs of Tobruk Medical Centre.

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Table 4. Proposed evidence-based follow-up protocol for infants (aged 1 day–1 year) with polycythemia at Tobruk Medical Centre

Hematocrit at Birth / Diagnosis	Clinical Status	Recommended Action	Follow-Up Schedule
<60%	Asymptomatic	Observation, ensure adequate hydration	Repeat CBC at 1 week; discharge if normalized
60–65%	Asymptomatic	Observation, intravenous hydration	Repeat CBC at 48 hours, 1 week, and 2 weeks
60–65%	Symptomatic*	Partial exchange transfusion + hydration	Daily CBC until stable; then weekly for 1 month
>65%	Asymptomatic	Consider partial exchange transfusion	Daily CBC for 3 days; then every 3 days for 2 weeks
>65%	Symptomatic*	Urgent partial exchange transfusion	Intensive monitoring; CBC every 12 hours initially
Any level	Persistent beyond 2–4 weeks	Refer to pediatric hematology; evaluate for secondary causes	Comprehensive workup including EPO level, genetic testing if red flags present

**Symptomatic = poor feeding, lethargy, tachypnea, cyanosis, seizures, hypoglycemia.*

This protocol aligns with current international recommendations: asymptomatic infants with packed cell volume ≥70% should receive cardiorespiratory monitoring and serial hematocrit checks [13]; symptomatic infants with packed cell volume ≥65% should undergo PET [13]. Fluid boluses alone are ineffective in reducing subsequent hematocrit values or PET requirements [11].

Discussion

Differentiating Physiological from Pathological Polycythemia in Infants Aged 0–12 Months

The primary study by Mtawil et al. confirmed that the vast majority of neonatal cases at Tobruk Medical Centre represent physiological adaptation rather than pathological myeloproliferation, attributing this to the unstable and hyperactive nature of neonatal bone marrow [3]. The concentration of 56% of cases within the first month of life aligns precisely with the expected timeline of physiological hematocrit elevation, which peaks at 2–4 hours postnatally and declines toward baseline by 12–18 hours [1,6]. However, the persistence of elevated blood components beyond one month in 44% of the cohort (n = 40) is a concerning finding reported but not interpreted in the primary study. In well-resourced healthcare systems, such persistence would trigger a structured diagnostic algorithm: repeat complete blood count, peripheral blood smear examination, serum EPO level measurement, evaluation for secondary causes, and, only in the presence of red flags, JAK2 mutation analysis and bone marrow biopsy [4,14]. The absence of any documented evidence of such systematic evaluation at Tobruk Medical Centre indicates that post-discharge care coordination could not be verified from the available records. International NICU studies have demonstrated that noninvasive interventions or observation alone are often ineffective in reducing hematocrit in polycythemic neonates, underscoring the need for protocol-driven management [1].

The JAK2 Mutation in Neonates: Rational Testing vs. Overinvestigation

The primary study identified JAK2 V617F as the principal driver of adult PV cases in their Oncology cohort, consistent with global literature [3,4,5]. However, the study did not establish protocols for when this testing might be appropriate in the neonatal population. In adults, the JAK2 V617F mutation is acquired somatically during a person's lifetime, not inherited through germline transmission. The probability of a neonate harboring this mutation is vanishingly small unless there is evidence of familial myeloproliferative disease or congenital polycythemia [4,16]. Our secondary analysis reveals no documented JAK2 testing protocols for neonates at Tobruk Medical Centre. While this may seem appropriate given the rarity of the mutation in newborns, the lack of documented testing criteria means that the few neonates who genuinely warrant genetic evaluation those with severe persistent polycythemia, trilineage proliferation, splenomegaly, thrombosis, or a strong family history may be overlooked. Rational JAK2 testing in neonates should be guided by the red flags outlined in (Table 3). When testing is indicated, a positive result has profound implications: it necessitates comprehensive family screening, genetic counseling, long-term hematological surveillance, and potential early cytoreductive therapy [17]. A negative result should redirect diagnostic efforts toward congenital polycythemia genes (VHL, EPOR, EGLN1/PHD2, SLC30A10) and secondary causes [15]. Recent case reports highlight the importance of excluding PV through JAK2 testing before investigating rare congenital erythrocytosis variants, using expanded next-generation sequencing panels when indicated [15].

Systemic Deficiencies in Follow-Up Care: A Call for Structural Reform

The gaps identified in this secondary analysis are not merely clinical oversights but reflect systemic deficiencies in healthcare infrastructure, training, and resource allocation. The primary study [3] successfully documented the incidence of elevated blood components across departments but did not evaluate care quality or follow-up adequacy. The lack of documented standardized post-discharge protocols means that clinical decisions appear to be ad hoc, dependent on

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individual physician awareness rather than institutional policy. This variability inevitably leads to inconsistent care. International consensus guidelines now recommend that asymptomatic patients not be routinely screened regardless of risk factors; instead, only symptomatic infants should undergo hematocrit evaluation, with treatment decisions based on venous (not capillary) samples [12]. The lack of parental education is particularly concerning—parents are the first line of defense against complications after discharge. Simple, low-cost interventions could dramatically improve outcomes. Interdepartmental fragmentation compounds these problems. The primary study required data collection across Neonatal, Oncology, and Internal Medicine departments [3], yet no coordinated care pathway exists. The development of defined triggers for referral and shared electronic health records would ensure continuity of care.

Regional Context and Resource-Limited Settings

It must be emphasized that serial hematocrit monitoring and partial exchange transfusion were not evaluable from the available Tobruk records; this analysis therefore benchmarks documented practice against international standards rather than retrospectively assessing the performance of these specific procedures. Tobruk City faces healthcare challenges common to many resource-limited settings. In this context, the implementation of complex follow-up protocols may seem impractical. However, the proposed protocol in (Table 4) is designed to be feasible: it prioritizes clinical observation and hydration for mild cases, reserves exchange transfusion for symptomatic or severe cases, and defers specialized testing to clearly defined red flags. The key intervention, standardized documentation and scheduled follow-up, requires minimal additional resources but offers substantial clinical benefit. Empowering neonatal nurses and midwives to provide parental education, establishing a simple appointment scheduling system, and creating a basic registry using existing administrative infrastructure would address the most critical gaps [3]. A 10-year European study demonstrated that even in settings with limited resources, systematic documentation of risk factors and scheduled monitoring significantly improved identification of infants requiring intervention [2].

Conclusion

The primary study by Mtawil et al. identified 90 neonatal and pediatric cases with elevated blood components at Tobruk Medical Centre, establishing the epidemiological presence of this hematological finding in the region [3]. This secondary analysis demonstrates that while the vast majority of these cases represent normal physiological adaptation to extrauterine life, with 56% occurring within the first month, consistent with the well-documented neonatal hematocrit surge that peaks at 2–4 hours postnatally [1,6], the persistence of elevated blood components beyond this window in 44% of cases demands systematic re-evaluation that is currently not occurring. The role of JAK2 mutation testing in neonatal polycythemia is limited to rare congenital or familial cases characterized by specific red flags. Routine JAK2 testing in neonates with transient, mild-to-moderate polycythemia is unnecessary, consistent with the primary study's finding that neonatal cases represent physiological rather than clonal processes [3], and with international evidence that JAK2 V617F is an acquired somatic mutation with negligible prevalence in newborns [4,5]. However, the complete absence of selective testing protocols means that the few neonates who genuinely require genetic evaluation may be overlooked. The current follow-up system in Tobruk City contains serious deficiencies correctable through low-cost, high-impact interventions: implementation of standardized post-discharge protocols aligned with international consensus guidelines [12,13], enhanced parental education, establishment of clear referral pathways, creation of a neonatal hematology registry, and development of rational JAK2 testing criteria. These changes do not require substantial capital investment but demand administrative commitment, staff training, and interdepartmental coordination. Tobruk Medical Centre and affiliated healthcare facilities must prioritize the restructuring of neonatal hematological follow-up care. The current gaps represent not merely clinical inconvenience but a genuine risk to neonatal health—one that is entirely preventable through systematic, evidence-based care. Future research should build upon the Mtawil et al. [3] dataset through longitudinal cohort studies to determine the natural history of polycythemia in infants in the Tobruk population, evaluate the effectiveness of implemented protocols, and investigate the prevalence of JAK2 and other driver mutations in persistent or familial cases.

Conflict of interest. Nil

References

1. Alsafadi TRM, Alsharif KF, Alsharif AF, Alotaibi ST, Alhazmi AF. Polycythemia in Neonatal Intensive Care Unit, Risk Factors, Symptoms, Pattern, and Management Controversy. J Clin Neonatol. 2014;3(3):163-7.
2. Jerković Raguž M. A decade of neonatal polycythaemia – has anything changed in this field? Dev Period Med. 2024;28(2):127-34.
3. Mtawil RA, Aboubaker S, Yousif EF, Abdelqader EA, Salem ME, Saleh ABA, et al. Incidence of Primary Polycythemia Vera in Tobruk City. Derna Acad J Appl Sci. 2026;2(2):69-74.
4. Tefferi A. Polycythemia vera: 2024 update on diagnosis, risk-stratification, and management. Am J Hematol. 2023;98(8):1303-16.
5. Bellosillo B, Doubek M, Tomuleasa C, Drobek J, Sili U, Kiladjian JJ, et al. JAK2 mutations in polycythemia vera: from molecular origins to inflammatory pathways and clinical implications. memo. 2025;17(Suppl 4):79-93.
6. Neonatal Polycythemia: A Review. Clin Med Rev Case Rep. 2024;11:142.
7. Hussain SM. Physiology of Blood: Red Blood Cells (RBC). In: Human Physiology [lecture series]. 2020.

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8. Mohan H. Textbook of Pathology. 7th ed. New Delhi: Jaypee Brothers Medical Publishers; 2015.
9. Xie X, Gou F, Zheng Z, Zhang Y, Zhang Y, Dong F, et al. Decoding human bone marrow hematopoietic stem and progenitor cells from fetal to birth. iScience. 2024;27(8):110445.
10. Lu X, Chang R. Polycythemia Vera. [Updated 2023 Apr 24]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan–. Available from: https://www.ncbi.nlm.nih.gov/books/NBK557660/
11. Bashir BA, Othman SA. Neonatal polycythaemia. Sudan J Paediatr. 2019;19(2):81-83.
12. UCSF Northern California Neonatal Consortium (UCSF NCNC). Consensus Guidelines for Partial Exchange Transfusion for Polycythemia in Neonates. San Francisco, CA: UCSF Benioff Children's Hospital; July 5, 2023. Available from: https://medconnection.ucsfbenioffchildrens.org/polycythemia-guidelines
13. Dempsey EM, Barrington K. Short and long term outcomes following partial exchange transfusion in the polycythaemic newborn: a systematic review. Arch Dis Child Fetal Neonatal Ed. 2006;91(1):F2-6.
14. Arber DA, Orazi A, Hasserjian R, Thiele J, Borowitz MJ, Le Beau MM, et al. The 2016 revision to the World Health Organization classification of myeloid neoplasms and acute leukemia. Blood. 2016;127(20):2391-405.
15. Giannini R, Donati M, Biagi C, Dondi A, Pession A, Lanari M. Case Report: A rare form of congenital erythrocytosis due to SLC30A10 biallelic variants—differential diagnosis and recommendation for biochemical and genetic screening. Front Pediatr. 2024;12:1319885.
16. Rumi E, Passamonti F, Pietra D, Della Porta MG, Arcaini L, Boggi S, et al. JAK2(V617F) as an acquired somatic mutation and a secondary genetic event associated with disease progression in familial myeloproliferative disorders. Cancer. 2006;107(9):2206-11.
17. Tefferi A, Barbui T. Polycythemia vera and essential thrombocythemia: 2017 update on diagnosis, risk-stratification, and management. Am J Hematol. 2017;92(1):94-108.

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