PLASU Journal of Health Sciences (PLASUJHS) Vol. 2, 2026
A Publication of the Faculty of Health Sciences, Plateau State University, Bokkos – Nigeria
ISSN: 3121-982 https://journals.plasu.edu.ng

PLATEAU STATE UNIVERSITY BOKKOS

JOURNAL OF HEALTH SCIENCES

VOL. 2, 2026

A publication of the Faculty of Health Sciences
Plateau State University, Bokkos, Nigeria

TO BE CITED AS PLASUJHS VOL. 2, 2026

PLATEAU STATE UNIVERSITY, JOURNAL OF HEALTH SCENCES (PLASUJHS)
A Publication of the Faculty of Health Sciences, Plateau State University, Bokkos — Nigeria

Vol. 2, 2026
ISSN: 3121-982
Website: https://journals.plasu.edu.ng

EDITORIAL INFORMATION

Publicity Committee
Mr. Jericho Bulus Ali- **Publicity Manager**
Mr. Biringmiap Koamnoe Linus- Member
Mr. Goyit Samson Stephen- Member
Mr. Maktu Dennis Mutdihin- Member
Mr. Ibrahim Yakubu – Member
Mr. Micheal Budaye -Member
Mrs. Leah Emmanuel Adangs - Member
Ms. Josephine Daniel Kakwi- Member
Ms. Nsere Bature- Member
Ms. Mercy Agada - Member

Contact:
PLASU Journal of Health Sciences
Faculty of Health Sciences
Plateau State University, Bokkos
Plateau State, Nigeria
Email: facultyofhealthsciences@plasu.edu.ng
Website: **https://journals.plasu.edu.ng**

Peer Review Statement
All submissions to the PLASU Journal of Health Sciences undergo a double-blind peer review process by at least two independent experts in the relevant field. The review process ensures originality, validity, and significance to the health sciences community.

Publication Ethics Statement
This journal adheres to international standards of publication ethics. The journal is committed to maintaining integrity in research and publication.

General guidelines for authors:

1. Manuscripts should be prepared in MS Word Format, Times New Roman, 12 font for text, 10 font for automatically generated footnotes, single line spacing, with 1.5 page margins.
2. Manuscripts should be accompanied with an abstract of about 300 words, and 4 to 6 keywords.
3. Inclusive of footnotes, the word count for articles is a minimum of 7,000 words and a maximum of 10,000 words.
4. The author's name, designation, affiliation, contact number and email should be provided on the cover page of the manuscript, for correspondence and future references.
5. Page numbers should be inserted at the bottom (middle) of pages.
6. The recommended style for this journal references and citations is the APA, format 7th edition.
7. Manuscript submissions to the Journal must be original, and not already published or under consideration for publication elsewhere.
8. A suitable format, for concept analysis of contemporary issues in health and illness of scientific paper is accepted e,g Walker and Avant methods of concept analysis or Beth L. Rogers Method of concept Analysis.
9. The final decision on the publication of an article will be made by the Editorial Board.
10. Articles are to be submitted in soft copy to facultyofhealthsciences@plasu.edu.ng

Manuscripts should follow this structure:

1. Article Title
2. Author's names and affiliations
3. Corresponding author's contact details.
4. Abstract (maximum of 300 words)
5. Keywords (4-6 words or phrase)
6. Introduction
7. Materials and methods
8. Results and discussion
9. Conclusion
10. Acknowledgment (if any)
11. Conflict of interest statement
12. Funding acknowledgment (if any)
13. References

Title

The title should be informative and concise

Authors and Affiliations

The surname of the authors should be written while other names should be abbreviated, institutional affiliations and country. The corresponding author should be indicated with an asterisk (*) and email address must be provided.

Abstract

A non-structured abstract of not more than 300 words summarizing the background, objectives, methods, findings and conclusions.

Keywords

Provide at least 4 – 5 keywords for indexing.

Introduction

The introduction should have the research problem, objectives and significance of the study clearly stated. Relevant literatures should be reviewed as part of the introduction and identify the knowledge gab.

Materials and methods

State the materials used including drugs and chemicals. Include the study design, materials, and procedures

Data Analysis

The method involved in the analysis of data should be clearly stated and the statistical software used.

Results and Discussion

The results should be clearly stated and concise. The discussion should interpret findings relating to the existing literatures.

Conclusion

The conclusion should summarize key findings and recommendations.

Tables and Figures

The tables and figures should be numbered consecutively with clear, self-explanatory captions.

References

The referencing style should be American Psychological Association (APA) 7th edition.

In-text citations:

i. One author: (Jatau, 2023)
ii. Two authors: (Olumide & Azi, 2025)
iii. Three or more authors: (Mallo et al., 2022)

In the reference section:

Journal article:

Ango, A. A., Toryila, J. E**., &** Mallo, M.J. (2026) A Study Of Body Mass Index (Bmi), Sex Hormones, And Lipid Profile Among Menopausal Women In Wukari Local Government Area Of Taraba State. *World Journal of Pharmaceutical Science and Research*, 5(2), 01-04. https://doi.org/10.5281/zenodo.18438257

Book

Thesis and dissertation
Miranda, C. (2019). Exploring the lived experiences of foster youth who obtained graduate level degrees: self-efficiency, resilience, and the impact on identity development (Publication No. 27542827) Doctoral dissertation, Pepperdine University.

Whole authored book
Sapolsky, R. M. (2017). *Behave: The biology of humans at our best and worst*. Penguin Books.

Abbreviations and Units

Use SI units and internationally accepted standards (IUPAC-IUB)

List of Reviewers

The editorial team acknowledges with gratitude the following reviewers who contributed to this edition:
Prof Caleb Mypet-College Provost. Plateau state University, Bokkos
Professor Matilda Edmund Banwat- University of Jos
Professor Joshua Istifanus Aneskoson – Federal University Wukari, Taraba State
Professor Lohya Ninzim – University of Jos
Dr Eunice Ari- University of Jos
Dr Aisha Salihu Abdullahi- Ahmadu Bello University, Zaria
Dr. Moforufu Olalekan Raimi- Federal University Otuoke, Bayelsa State
Dr Sarah Samson Jatau, Faculty coordinator, Plateau state University, Bokkos

ISSN: **3121-982**

TABLE OF CONTENTS

BLOOD TRANSFUSION SUPPORT AND BLOODLESS ALTERNATIVES IN CHEMOTHERAPY-INDUCED MYELOSUPPRESSION: A LINEAGE-BASED CRITICAL REVIE 10

ANTI-INFLAMMATORY EFFECTS OF LYCOPENE ON CARRAGEENAN INDUCED KNEE OSTEOARTHRITIS IN MALE WISTAR RATS 23

EFFECT OF HAEMOGLOBIN GENOTYPE ON EXERCISE TOLERANCE AMONG BINGHAM UNIVERSITY STUDENTS 31

ASSESSMENT OF THE UTILIZATION OF LONG-LASTING INSECTICIDE NETS AMONG PREGNANT WOMEN AND CAREGIVERS OF CHILDREN LESS THAN FIVE YEARS IN TWO URBAN LOCAL GOVERNMENT AREAS (JOS NORTH AND JOS SOUTH) IN PLATEAU STATE, NIGERIA 40

ASSOCIATION OF LIFESTYLE BEHAVIORS WITH SERUM URIC ACID LEVELS AMONG

NIGERIAN UNIVERSITY STUDENTS: A CROSS-SECTIONAL STUDY 50

DEMOGRAPHIC DETERMINANTS AND PREVALENCE OF ABNORMAL SERUM URIC ACID LEVELS AMONG APPARENTLY HEALTHY NIGERIAN UNIVERSITY STUDENTS 61

SOCIO-CULTURAL AND ECONOMIC DRIVERS OF CAESAREAN SECTION HESITANCY AMONG PREGNANT WOMEN ATTENDING A TERTIARY HOSPITAL IN SOUTHERN NIGERIA 68

KNOWLEDGE, ATTITUDE, AND PRACTICE OF NURSES REGARDING SCREENING AND EDUCATING WOMEN ON INTIMATE PARTNER VIOLENCE AND RECOURSE CHOICES AT NATIONAL HOSPITAL, ABUJA 79

PUNCTUALITY: CONCEPT ANALYSIS 90

FACTORS INFLUENCING STRESS AND BURNOUT EXPERIENCES AMONG NEWLY QUALIFIED NURSES IN THE UNIVERSITY OF BENIN TEACHING HOSPITAL, BENIN CITY: A CROSS-SECTIONAL STUDY 105

PERCEIVED INFLUENCE OF LOW-FIDELITY SIMULATION MANIKINS ON NURSING STUDENTS' SELF-CONFIDENCE AND PERCEIVED COMPETENCE: A CROSS-SECTIONAL STUDY AT THE UNIVERSITY OF BENIN 118

REVIEW ARTICLE;
AN OVERVIEW OF CERVICAL CANCER 129
DRUG ABUSE AND SCHOOL DROPOUT AMONG ADOLESCENTS IN MINING

COMMUNITIES OF JOS SOUTH LOCAL GOVERNMENT AREA, PLATEAU STATE, NIGERIA 143

BARRIERS TO THE PREVENTION OF POST-OPERATIVE INFECTION AMONG NURSES AT VOM CHRISTIAN HOSPITAL (VCH), JOS SOUTH LOCAL GOVERNMENT AREA, PLATEAU STATE 150

ALLERGIES AND HYPERSENSITIVITY 161

PREVALENCE AND MODEL PREDICTIONS OF *MYCOBACTERIUM TUBERCULOSIS* IN NIGER STATE, NIGERIA 168

KNOWLEDGE AND DISPOSITION TOWARDS CESAREAN SECTION AMONG EXPECTANT MOTHERS UTILIZING ANTENATAL SERVICES IN PLATEAU SPECIALIST HOSPITAL, JOS (PSSH) 176 of 190

BLOOD TRANSFUSION SUPPORT AND BLOODLESS ALTERNATIVES IN CHEMOTHERAPY-INDUCED MYELOSUPPRESSION: A LINEAGE-BASED CRITICAL REVIE

Akpoka, O. A.[1,2*]

[1] Department of Biological Science and Biotechnology, Caleb University, Lagos, Nigeria
[2] Department of Medical-Surgical Nursing, Faculty of Nursing Science, Caleb University, Lagos, Nigeria
*Corresponding author: Akpoka, O. A. ausbones@gmail.com

ORCID: 0000-0001-9190-9188

Abstract

Chemotherapy-induced myelosuppression produces cytopenias that have traditionally required supportive blood component therapy, but patient preference and transfusion-related risk have driven interest in transfusion-sparing and bloodless alternatives whose safe boundaries remain poorly defined. This critical narrative review synthesises current guideline statements and high-level evidence on transfusion support and transfusion-sparing strategies across the erythroid, megakaryocytic, and granulocytic compartments, and identifies the clinical contexts in which bloodless management is unsafe. Restrictive red-cell thresholds, prophylactic granulocyte colony-stimulating factor (G-CSF), and intravenous iron each have a defensible evidence base, with reported effect sizes drawn from existing meta-analyses (for example, G-CSF and febrile neutropenia, relative risk 0.54; intravenous iron and transfusion, relative risk 0.72). Erythropoiesis-stimulating agents raise haemoglobin but carry a mortality signal and are restricted to non-curative settings. Newer evidence supports more restrictive platelet transfusion thresholds and an emerging role for thrombopoietin receptor agonists in maintaining chemotherapy dose intensity, although the latter is not yet an approved indication. Synthetic oxygen carriers remain experimental in most high-income regulatory settings. Bloodless pathways can reduce transfusion exposure in selected patients, especially where anaemia and neutropenia can be anticipated and treated early, but remain unsafe or high-risk when cytopenias are rapid, profound, haemorrhagic, or complicated by limited physiological reserve. The review's distinct contribution is an integrated, compartment-based account of where bloodless management succeeds and fails, with explicit attention to nursing-led implementation.

Keywords: bloodless medicine; chemotherapy-induced myelosuppression; granulocyte colony-stimulating factor; oncology nursing; patient blood management; platelet transfusion threshold.

Introduction

Cytotoxic chemotherapy acts preferentially on rapidly dividing cells and therefore commonly impairs marrow haematopoiesis, producing anaemia, thrombocytopenia, and neutropenia. Transfusion of red blood cells (RBCs) and platelets remains the most rapid means of correcting these deficits and has long been a cornerstone of supportive oncology care (Carson et al., 2016; Stanworth et al., 2013). Patient preferences, notably among some Jehovah's Witnesses, together with awareness of transfusion-related risk, have nonetheless driven the development of patient blood management (PBM), now codified in international consensus recommendations (Mueller et al., 2019), and of bloodless-medicine programmes that prioritise conservation, endogenous haematopoiesis, and non-blood haemostasis.

What remains underdeveloped is not whether individual transfusion-sparing interventions work, but how far they can safely replace blood component therapy across different cytopenic compartments and oncological risk contexts. This review addresses that gap. Four concepts are frequently conflated and are distinguished here.

Transfusion avoidance is the reduction of unnecessary transfusion in patients who may still accept blood. Transfusion refusal is the declining of red cells, platelets, plasma, or whole blood by an informed patient. Patient blood management is a system-level programme to optimise the use and conservation of a patient's own blood. Bloodless medicine is a pathway designed around non-blood or patient-acceptable fraction strategies for those who will not accept allogeneic components. Restrictive transfusion practice is a feature of good PBM; it is not, in itself, bloodless care, and the two should not be conflated.

This review addresses adults receiving chemotherapy for solid and haematological malignancies, in curative and palliative settings. Its contribution is twofold: a compartment-based synthesis (erythroid, megakaryocytic, granulocytic) pairing each conventional support with its bloodless alternative, time to effect, and quantified effect; and a graded delineation of contraindications intended to support realistic consent and patient selection, including a nursing-led implementation framework.

Materials and Methods

Study design. This work was conducted as a critical, interpretive narrative review of transfusion support and transfusion-sparing strategies in chemotherapy-induced myelosuppression. It was designed to integrate and appraise existing high-level evidence and guideline statements rather than to generate pooled estimates, and no statistical synthesis was undertaken.

Data sources and search strategy. This was a deliberate interpretive and critical-narrative design rather than a systematic protocol. An exhaustive, iterative search of PubMed, Embase, and the Cochrane Library was used to capture foundational consensus guidelines alongside recent paradigm-shifting trials, for example the 2025 AABB and ICTMG platelet guideline (Metcalf et al., 2025) and the 2026 RECITE trial of romiplostim (Al-Samkari et al., 2026), ensuring the clinical currency of the synthesis. Search strings, date limits, and eligibility criteria were not formally pre-specified, so the review is not reproducible in the systematic-review sense and makes no claim to have independently quantified comparative effects; all quantitative estimates are attributed to their source meta-analyses.

Evidence synthesis. Sources were organised around three haematopoietic compartments (erythroid, megakaryocytic, and granulocytic), and the evidence was appraised narratively for quality, directness to the chemotherapy setting, and applicability, with particular attention to the boundary conditions under which transfusion-sparing management becomes unsafe.

Results and Discussion

Pathophysiology of chemotherapy-induced myelosuppression

Chemotherapeutic agents differ in marrow toxicity and temporal course. Most conventional regimens produce haematologic nadirs at approximately one to two weeks post-dose, with subsequent recovery, although certain agents, notably the nitrosoureas, are characteristically associated with delayed or prolonged suppression. The depth and duration of cytopenia, and the consequent risk of febrile neutropenia and its complications, are modified by cumulative dose and dose intensity, baseline marrow reserve, prior radiotherapy or alkylator exposure, marrow infiltration by tumour, advancing age, performance status, and nutritional status (Crawford et al., 2004). Several mechanisms compound the cytopenia beyond direct cytotoxicity. Renal dysfunction reduces endogenous erythropoietin production; systemic inflammation, which is common in malignancy, raises hepcidin and restricts iron availability, producing an iron-restricted, normocytic anaemia of inflammation that blunts the marrow response and limits the efficacy of oral iron and, in part, of erythropoiesis-stimulating agents (Ganz, 2019); and intercurrent infection further suppresses and consumes marrow output. The erythroid, megakaryocytic, and granulocytic compartments differ in the speed at which each deficit can be corrected

endogenously, which frames the transfusion-versus-bloodless debate and motivates the compartment-based structure below. The term compartment is preferred to lineage, since erythroid and megakaryocytic precursors are themselves of myeloid origin and three haematopoietic compartments (erythroid, megakaryocytic, and granulocytic) are intended.

Conventional transfusion support

Contemporary guidelines favour a restrictive RBC strategy for most stable hospitalised adults, with a threshold near haemoglobin 7 g/dL; a higher threshold of 7.5 to 8 g/dL applies to patients undergoing orthopaedic or cardiac surgery and those with pre-existing cardiovascular disease, while symptomatic anaemia is itself an indication to transfuse irrespective of a numerical trigger (Carson et al., 2016, 2023). Much of this evidence is extrapolated when applied to chemotherapy-induced anaemia, where dedicated randomised data are limited. The 2025 AABB and ICTMG international guideline makes a strong recommendation to transfuse platelets below 10×10^9/L in non-bleeding patients with hypoproliferative thrombocytopenia who are actively receiving chemotherapy or undergoing allogeneic stem cell transplantation, and, conditionally, recommends against prophylactic transfusion in autologous transplant or aplastic anaemia (Metcalf et al., 2025). This prophylactic strategy is supported by the TOPPS trial, in which prophylaxis reduced bleeding versus no prophylaxis (Stanworth et al., 2013); dose is a separate lever, and the PLADO trial showed that lower prophylactic doses do not increase grade 2 or higher bleeding (Slichter et al., 2010). Higher thresholds apply procedurally, near 20×10^9/L for lumbar puncture and near 50×10^9/L for active bleeding or major procedures (Metcalf et al., 2025; Schiffer et al., 2018). In haematological malignancy and transplantation, leucoreduced and, where indicated, irradiated and cytomegalovirus-safe components are used, with HLA-matched or crossmatched platelets reserved for alloimmune refractoriness. Granulocyte transfusion is reserved for refractory life-threatening infection during persistent neutropenia, its routine use limited by sparse randomised evidence, short cell half-life, and immunological risk (Estcourt et al., 2016) (Table 1).

Table 1. Conventional transfusion thresholds in chemotherapy-associated cytopenias. SCT, stem cell transplantation; Hb, haemoglobin.

Component	Context	Threshold	Source	Caveat
Red cells	Stable hospitalised adult	Hb ~7 g/dL	Carson et al. (2016, 2023)	Extrapolated from non-oncology trials
Red cells	Cardiac/orthopaedic surgery; cardiovascular disease	Hb ~7.5 to 8 g/dL	Carson et al. (2016, 2023)	Symptomatic anaemia transfused regardless
Platelets	Non-bleeding, chemotherapy or allogeneic SCT	$<10 \times 10^9$/L	Metcalf et al. (2025)	Strong recommendation
Platelets	Autologous SCT or aplastic anaemia	No prophylaxis	Metcalf et al. (2025)	Conditional recommendation
Platelets	Procedures / active bleeding	~20 to 50 $\times 10^9$/L	Metcalf et al. (2025); Schiffer et al. (2018)	Procedure-dependent

Component	Context	Threshold	Source	Caveat
Granulocytes	Refractory infection in neutropenia	Case-by-case	Estcourt et al. (2016)	Limited evidence

Sources: (Slichter et al., 2010; Carson et al., 2016; Estcourt et al., 2016; Schiffer et al., 2018; Carson et al., 2023; Metcalf et al., 2025).

Bloodless strategies by haematopoietic compartment

Time to effect is a defining constraint and is summarised in Table 2. RBC and platelet transfusion act immediately; G-CSF acts within days; intravenous iron and erythropoiesis-stimulating agents act over one to several weeks; and thrombopoietin receptor agonists act over days to weeks. None of the pharmacological options is an immediate rescue therapy, which is central to understanding where bloodless management fails. Across all compartments, phlebotomy minimisation reduces iatrogenic loss, since every 100 mL of diagnostic phlebotomy is associated with a fall in haemoglobin of approximately 0.7 g/dL (Thavendiranathan et al., 2005); chemotherapy modification and intraoperative blood-conservation techniques further reduce need.

Erythroid compartment. In functional iron deficiency, intravenous iron raises haemoglobin and reduces transfusion: a meta-analysis of randomised trials found it reduced the proportion requiring RBC transfusion compared with oral iron (relative risk 0.72, 95% CI 0.55 to 0.95; number needed to treat 20) and increased haematopoietic response (relative risk 1.23, 95% CI 1.01 to 1.50), with no excess adverse events (Buchrits et al., 2022). Erythropoiesis-stimulating agents (ESAs) raise haemoglobin but carry a mortality signal: a patient-level meta-analysis of 53 trials found increased on-study mortality in the whole cohort (hazard ratio 1.17, 95% CI 1.06 to 1.30) and an attenuated, non-significant effect in the chemotherapy-only subgroup (hazard ratio 1.10, 95% CI 0.98 to 1.24) (Bohlius et al., 2009). Current ASCO and ASH guidance restricts ESAs to non-curative intent with haemoglobin below 10 g/dL (Bohlius et al., 2019). By contrast, a study-level meta-analysis found no significant effect on mortality or disease progression when ESAs were used within label, although venous thromboembolism increased (Glaspy et al., 2010), so the magnitude of harm specific to the chemotherapy setting remains debated. The guideline restriction to non-curative intent is grounded partly in a concern that ESAs might promote tumour progression (Bohlius et al., 2019); one proposed but unproven mechanism is the expression of erythropoietin receptors on some tumour cells, a hypothesis weakened by evidence that the antibodies used to detect such receptors are non-specific, so it should be read as contested rather than established, whereas the thromboembolic hazard is well documented (Glaspy et al., 2010). Haemoglobin-based oxygen carriers (HBOCs) are not approved for routine use in most high-income regulatory settings; HBOC-201 holds jurisdiction-specific approval in South Africa and Russia and is available in the United States only through expanded or compassionate-access pathways (Mackenzie et al., 2010). A meta-analysis of 16 trials found a significant increase in death (relative risk 1.30, 95% CI 1.05 to 1.61) and myocardial infarction (relative risk 2.71, 95% CI 1.67 to 4.40) (Natanson et al., 2008), and safety concerns and limited availability preclude routine use, although some authors argue that development was constrained by a regulatory equivalence requirement (Mackenzie et al., 2015).

Megakaryocytic compartment. Thrombopoietin receptor agonists (TPO-RAs) were long investigational for chemotherapy-induced thrombocytopenia, but the phase 3 RECITE trial changed the evidence base: in 165 patients receiving oxaliplatin-based regimens for gastrointestinal cancers, romiplostim allowed 84.4 percent to avoid chemotherapy dose modification versus 35.7 percent with placebo, with higher platelet nadirs and a 2 percent rate of thromboembolic events (Al-Samkari et al., 2026). This is emerging rather than established, since the evidence is confined to gastrointestinal and oxaliplatin-based regimens and regulatory labelling does not yet list the indication. Because romiplostim acts by stimulating a residual pool of megakaryocytic

progenitors, its effect is delayed and may be blunted entirely where that pool is depleted, as after cumulative alkylator exposure or in a tumour-infiltrated marrow; it cannot stabilise acute haemorrhage or be relied upon during a rapid nadir. Although tranexamic acid reduces bleeding and transfusion in surgery (relative risk 0.62, 95% CI 0.58 to 0.65) (Ker et al., 2012), that benefit has not carried over to this setting: two randomised, placebo-controlled trials of prophylactic tranexamic acid in haematological-malignancy patients with hypoproliferative thrombocytopenia, A-TREAT and TREATT, found no reduction in WHO grade 2 or higher bleeding or in platelet transfusion, with no clear excess of thrombosis (Gernsheimer et al., 2022; Estcourt et al., 2025). Tranexamic acid is therefore not a substitute for platelet transfusion in chemotherapy-induced thrombocytopenic bleeding, for which no rapid substitute exists.

Granulocytic compartment. For regimens with febrile-neutropenia risk of at least 20 percent, prophylactic G-CSF is recommended (Smith et al., 2015; Klastersky et al., 2016). A meta-analysis of 17 randomised trials found that primary prophylaxis reduced febrile neutropenia (relative risk 0.54, 95% CI 0.43 to 0.67), infection-related mortality (relative risk 0.55), and early all-cause mortality (relative risk 0.60) (Kuderer et al., 2007); pegfilgrastim reduces febrile neutropenia to a greater extent than daily filgrastim (Cooper et al., 2011). Timing matters: G-CSF should not be given concurrently with, or within 24 hours of, cytotoxic chemotherapy, since the progenitors it mobilises are then maximally vulnerable to cytotoxic damage and myelosuppression may paradoxically worsen; it is conventionally begun 24 to 72 hours after chemotherapy (Smith et al., 2015). G-CSF acts within days, making this compartment the most amenable to bloodless support (Table 2).

Table 2. Transfusion-sparing interventions by haematopoietic compartment.

Compartment	Intervention	Mechanism	Time to effect	Effect size	Evidence	Limitations
Erythroid	IV iron	Repletes functional deficiency	1 to several weeks	Transfusion RR 0.72; NNT 20	Moderate	Small trials; withhold in infection
Erythroid	ESA	Stimulates erythropoiesis	Weeks	Mortality HR 1.17 (whole cohort)	High harm signal	Non-curative, Hb <10; tumour-progression and VTE risk
Erythroid	HBOC	Acellular oxygen carrier	Immediate	Death RR 1.30; MI RR 2.71	Experimental	Not approved in most settings
Megakaryocytic	Romiplostim	TPO-RA, raises platelets	Days to weeks	84% vs 36% avoided dose modification	Phase 3 (RECITE)	GI/oxaliplatin only; needs progenitor pool; not for acute haemorrhage
Megakaryocytic	Tranexamic acid	Antifibrinolytic	Hours	No bleeding reduction (haematology RCTs)	Phase 3 (A-TREAT, TREATT)	No prophylactic benefit in thrombocytopenia; not a platelet substitute
Granulocytic	G-CSF	Accelerates neutrophil recovery	Days	FN RR 0.54; early mortality 0.60	High	Bone pain; acts over days (not rescue)
Cross-cutting	Phlebotomy minimisation	Reduces iatrogenic loss	Continuous	~0.7 g/dL per 100 mL drawn	Moderate	Observational

IV, intravenous; ESA, erythropoiesis-stimulating agent; HBOC, haemoglobin-based oxygen carrier; TPO-RA, thrombopoietin receptor agonist; RR, relative risk; NNT, number needed to treat; HR, hazard ratio; MI, myocardial infarction; VTE, venous thromboembolism; FN, febrile neutropenia; G-CSF, granulocyte colony-stimulating factor; GI, gastrointestinal.

Sources: (Thavendiranathan et al., 2005; Kuderer et al., 2007; Natanson et al., 2008; Bohlius et al., 2009; Glaspy et al., 2010; Ker et al., 2012; Smith et al., 2015; Buchrits et al., 2022; Gernsheimer et al., 2022; Estcourt et al., 2025; Al-Samkari et al., 2026).

Critical appraisal of the evidence

The evidence base is uneven. The strongest support exists for restrictive RBC thresholds (Carson et al., 2016, 2023), the 2025 platelet thresholds (Metcalf et al., 2025), and G-CSF prophylaxis (Kuderer et al., 2007). Intravenous iron is supported by randomised evidence, though trials are small and the transfusion estimate

imprecise (Buchrits et al., 2022). The ESA harm signal is robust in the whole cohort (Bohlius et al., 2009) but equivocal in the chemotherapy subgroup and when agents are used within label (Glaspy et al., 2010). The RECITE trial is high quality but narrow, confined to gastrointestinal oxaliplatin regimens, so its result should not be generalised to all chemotherapy-induced thrombocytopenia (Al-Samkari et al., 2026). Antifibrinolytic prophylaxis, despite its benefit in surgery (Ker et al., 2012), showed no benefit in two randomised haematology trials (Gernsheimer et al., 2022; Estcourt et al., 2025), and the evidence for complete bloodless pathways in oncology is largely observational, single-centre, and subject to selection bias. Few studies report patient-important outcomes, and almost none randomise transfusion refusal, which is ethically infeasible.

When bloodless management is unsafe: a graded assessment

Usually unsafe (absolute high-risk): catastrophic bleeding with haemodynamic instability; acute leukaemia induction in patients who will not accept transfusion; peri-transplant aplasia; concurrent disseminated intravascular coagulation or hyperfibrinolysis, in which clotting factors and platelets are actively consumed; and severe symptomatic anaemia with cardiac ischaemia. In these settings the speed and depth of cytopenia or blood loss outpace any endogenous or pharmacological response, and immediate component therapy is standard of care; therapeutic platelet transfusion for active bleeding in thrombocytopenic cancer patients is itself guideline-supported (Schiffer et al., 2018; Metcalf et al., 2025). The principle that early, balanced component therapy improves haemostasis is drawn from trauma resuscitation, where the pivotal trial found that a 1:1:1 ratio improved early haemostasis without an overall mortality difference versus 1:1:2 (Holcomb et al., 2015) and where it is embedded in major-haemorrhage guidelines (Rossaint et al., 2023). That trauma evidence is not directly equivalent to chemotherapy-induced thrombocytopenic bleeding, so its application here is an extrapolation rather than a directly evidenced recommendation.

Feasible only in specialised centres: selected lower-intensity regimens, stable solid tumours, and mild to moderate anticipated cytopenias may be managed without transfusion where a pre-planned bloodless pathway, intensive monitoring, and immediate access to growth factors and intravenous iron exist. Dose-dense schedules improve survival when delivered with G-CSF support (Citron et al., 2003) but intensify cumulative myelosuppression and raise transfusion requirements beyond what bloodless strategies can usually manage.

Generally feasible: prophylactic G-CSF, phlebotomy minimisation, iron optimisation, and careful restrictive thresholds in stable patients are appropriate across most settings. Concurrent sepsis, profound baseline cytopenia, poor functional status, malnutrition, advanced age, and renal or hepatic dysfunction all shift a patient towards the higher-risk tiers (Table 3).

Table 3. Graded risk of bloodless management. G-CSF, granulocyte colony-stimulating factor.

Tier	Settings
Usually unsafe (absolute high-risk)	Catastrophic bleeding with instability; acute leukaemia induction without transfusion acceptance; peri-transplant aplasia; concurrent disseminated intravascular coagulation or hyperfibrinolysis; severe symptomatic anaemia with cardiac ischaemia
Feasible only in specialised centres	Selected lower-intensity regimens; stable solid tumours; mild to moderate cytopenias with a pre-planned bloodless pathway and intensive monitoring

Tier	Settings
Generally feasible	Prophylactic G-CSF; phlebotomy minimisation; iron optimisation; careful restrictive thresholds in stable patients

Sources: (Citron et al., 2003; Kuderer et al., 2007; Holcomb et al., 2015; Schiffer et al., 2018; Rossaint et al., 2023; Metcalf et al., 2025; Al-Samkari et al., 2026), and evidence cited in the text.

Nursing implications for bloodless supportive oncology care

Because bloodless pathways succeed or fail at the point of anticipation and monitoring, nursing practice is central rather than ancillary. Key contributions include early identification of transfusion refusal or transfusion anxiety before myelosuppressive therapy begins; clear consent documentation and pre-agreed escalation plans; structured symptom surveillance during the nadir; infection surveillance and febrile-neutropenia triage; bleeding-risk education; and coordination across oncology, haematology, transfusion medicine, pharmacy, ethics, and spiritual care.

Phlebotomy stewardship should be operationalised through paediatric or microtainer collection tubes for adult patients, batched and consolidated test ordering, and elimination of redundant daily laboratory draws, since cumulative diagnostic sampling is a measurable and modifiable contributor to hospital-acquired anaemia (Thavendiranathan et al., 2005). Validated instruments support surveillance and triage: the MASCC risk index stratifies the risk of complications in febrile neutropenia and identifies patients who may be managed as lower risk (Klastersky et al., 2000), complementing guideline-based febrile-neutropenia pathways (Klastersky et al., 2016), while the WHO bleeding scale grades clinical bleeding (Miller et al., 1981), where alert surveillance for grade 1 signs such as petechiae or ecchymoses, not overt epistaxis or haematuria alone, can prompt an early change to the mobility plan and proactive local antifibrinolytic or mechanical haemostatic measures. Patient-reported outcomes, including fatigue, anxiety, and quality of life, should be assessed systematically through the nadir, since they shape the acceptability of a bloodless pathway. This coordinating and surveillance role is the discipline's distinctive advantage, is under-recognised in the transfusion-medicine literature, and is most effective when embedded within a formal patient blood management programme of the kind set out in international consensus recommendations (Mueller et al., 2019).

Ethical and legal considerations

Adults with decision-making capacity generally retain the right to refuse transfusion, even where refusal raises the risk of serious harm, and caring for such patients raises intertwined ethical, legal, and clinical challenges that are sharpest in the emergency setting (Scharman et al., 2017); honest prognostic discussion and careful documentation are essential. Acceptance is not all-or-none: individual patients differ in whether they will accept specific fractions or autologous techniques such as albumin, clotting-factor concentrates, or intraoperative cell salvage, so the precise boundaries should be established and recorded in advance rather than assumed (Scharman et al., 2017). Laws and the handling of incapacity differ by jurisdiction, so local legal and institutional guidance should be sought rather than presumed. Where bloodless approaches are unlikely to succeed, options include modification to less intensive regimens with reduced curative potential, referral to specialised centres, or deferral of treatment, each with full disclosure, and advance care planning that records acceptable emergency interventions.

Clinical implementation framework

A practical pathway is summarised in Table 4 and comprises early identification and documentation of transfusion preferences; baseline haematologic, nutritional, and cardiopulmonary-reserve assessment with regimen risk stratification; pretreatment optimisation through iron repletion and phlebotomy minimisation;

prophylactic growth-factor support and individualised regimen modification; intensive nadir-period monitoring with pre-agreed escalation thresholds; and documented contingency planning for haemorrhage or deterioration, including the point at which deferral becomes the safer course. These elements mirror the core components of established institutional bloodless-medicine programmes, which combine pretreatment anaemia optimisation, meticulous blood conservation including phlebotomy minimisation, and pharmacological support within a single coordinated pathway (Resar & Frank, 2014).

Table 4. Practical implementation pathway.

Stage	Actions
Assessment	Identify and document transfusion preferences; baseline iron studies, B12/folate, cardiopulmonary reserve; regimen risk stratification
Optimisation	Intravenous iron for functional deficiency; correct deficiencies; phlebotomy stewardship
Treatment	Prophylactic G-CSF where indicated; consider regimen modification; ESA only if non-curative and Hb <10
Monitoring	Nadir-period surveillance; febrile-neutropenia triage; bleeding-risk review at defined thresholds
Escalation	Pre-agreed thresholds; contingency plan for haemorrhage or deterioration; point of deferral defined
Documentation	Consent, acceptable emergency interventions, multidisciplinary and ethics input recorded

G-CSF, granulocyte colony-stimulating factor; ESA, erythropoiesis-stimulating agent; Hb, haemoglobin. Compiled by the author from cited evidence and consensus recommendations (Klastersky et al., 2000; Resar & Frank, 2014; Smith et al., 2015; Mueller et al., 2019; Buchrits et al., 2022; Metcalf et al., 2025).

Gaps and future directions

Priority gaps include the absence of prospective registries of transfusion-declining oncology patients; the lack of validated risk scores for bloodless-chemotherapy feasibility; the scarcity of evidence from African and other low-resource oncology settings, where the TRACT trial in African children with severe anaemia from infectious causes, although not an oncology population, nonetheless showed that conservative management of uncomplicated severe anaemia was safe (Maitland et al., 2019), and, more directly, a prospective study at the Uganda Cancer Institute found that adopting the lower prophylactic platelet-transfusion threshold used in higher-income settings reduced transfusions without increasing clinically significant bleeding or mortality in patients with haematological malignancy (Ddungu et al., 2019); cost and access constraints for intravenous iron, G-CSF, ESAs, and TPO-RAs; undefined safe lower bounds for permissive anaemia in ambulatory chemotherapy; and a near-total absence of patient-reported outcomes and of data on clinician moral distress. In many low- and middle-income settings, bloodless management is not a preference driven by religious conviction or advanced PBM infrastructure but an uncontrollable reality imposed by chronic blood-bank shortages, limited screening reagents, and the fiscal toxicity of transfusion; at one sub-Saharan cancer institute, for example, demand for platelet units markedly exceeded the supply that could be provided (Ddungu et al., 2020). There, mastering the thresholds of permissive anaemia and applying low-cost phlebotomy stewardship become survival mechanisms rather than refinements, which makes the graded risk assessment in Table 3

globally, not only locally, relevant. Future research should prioritise prospective registries, validated risk scores, evidence from low-resource settings, and patient-reported outcomes to refine the safe boundaries of bloodless oncology care.

Conclusion

Bloodless pathways can reduce transfusion exposure in selected chemotherapy patients, especially where anaemia and neutropenia can be anticipated and treated early, with the firmest support for restrictive thresholds, G-CSF prophylaxis, and intravenous iron in functional deficiency. They remain unsafe or high-risk when cytopenias are rapid, profound, haemorrhagic, or complicated by limited physiological reserve. The distinct contribution of this review is an integrated, compartment-based account of where bloodless management succeeds and where it fails, with explicit attention to nursing-led implementation, which is frequently absent from the transfusion-medicine literature.

Acknowledgements

The author received no specific assistance in the preparation of this review.

Conflict of Interest

The author declares no conflict of interest.

References

Al-Samkari, H., Munoz, C., Geredeli, C., et al. (2026). Romiplostim versus placebo for chemotherapy-induced thrombocytopenia. *N Engl J Med, 394*(11), 1061-1073. https://doi.org/10.1056/NEJMoa2511882

Bohlius, J., Schmidlin, K., Brillant, C., et al. (2009). Recombinant human erythropoiesis-stimulating agents and mortality in patients with cancer: a meta-analysis of randomised trials. *Lancet, 373*, 1532-1542. https://doi.org/10.1016/S0140-6736(09)60502-X

Bohlius, J., Bohlke, K., Castelli, R., et al. (2019). Management of cancer-associated anemia with erythropoiesis-stimulating agents: ASCO/ASH clinical practice guideline update. *J Clin Oncol, 37*(15), 1336-1351. https://doi.org/10.1200/JCO.18.02142

Buchrits, S., Itzhaki, O., Avni, T., et al. (2022). Intravenous iron supplementation for the treatment of chemotherapy-induced anemia: a systematic review and meta-analysis. *J Clin Med, 11*, 4156. https://doi.org/10.3390/jcm11144156

Carson, J. L., Guyatt, G., Heddle, N. M., et al. (2016). Clinical practice guidelines from the AABB: red blood cell transfusion thresholds and storage. *JAMA, 316*, 2025-2035. https://doi.org/10.1001/jama.2016.9185

Carson, J. L., Stanworth, S. J., Guyatt, G., et al. (2023). Red blood cell transfusion: 2023 AABB international guidelines. *JAMA, 330*, 1892-1902. https://doi.org/10.1001/jama.2023.12914

Citron, M. L., Berry, D. A., Cirrincione, C., et al. (2003). Randomized trial of dose-dense versus conventionally scheduled and sequential versus concurrent combination chemotherapy as postoperative adjuvant treatment of node-positive primary breast cancer: first report of Intergroup Trial C9741/CALGB 9741. *J Clin Oncol, 21*, 1431-1439. https://doi.org/10.1200/JCO.2003.09.081

Cooper, K. L., Madan, J., Whyte, S., Stevenson, M. D., & Akehurst, R. L. (2011). Granulocyte colony-stimulating factors for febrile neutropenia prophylaxis following chemotherapy: systematic review and meta-analysis. *BMC Cancer, 11*, 404. https://doi.org/10.1186/1471-2407-11-404

Crawford, J., Dale, D. C., & Lyman, G. H. (2004). Chemotherapy-induced neutropenia: risks, consequences, and new directions for its management. *Cancer, 100*, 228-237. https://doi.org/10.1002/cncr.11882

Ddungu, H., Krantz, E. M., Kajja, I., et al. (2019). How low can you go: what is the safe threshold for platelet transfusions in patients with hematologic malignancy in sub-Saharan Africa. *PLoS One, 14*, e0211648. https://doi.org/10.1371/journal.pone.0211648

Ddungu, H., Krantz, E. M., Kajja, I., et al. (2020). Transfusion challenges in patients with hematological malignancies in sub-Saharan Africa: a prospective observational study from the Uganda Cancer Institute. *Sci Rep, 10*, 2825. https://doi.org/10.1038/s41598-020-59773-y

Estcourt, L. J., Stanworth, S. J., Hopewell, S., et al. (2016). Granulocyte transfusions for treating infections in people with neutropenia or neutrophil dysfunction. *Cochrane Database Syst Rev, 4*, CD005339. https://doi.org/10.1002/14651858.CD005339.pub2

Estcourt, L. J., McQuilten, Z., Stanworth, S. J., et al. (2025). Tranexamic acid versus placebo to prevent bleeding in patients with haematological malignancies and severe thrombocytopenia (TREATT): a randomised, double-blind, parallel, phase 3 superiority trial. *Lancet Haematol, 12*, e14-e22. https://doi.org/10.1016/S2352-3026(24)00317-X

Ganz, T. (2019). Anemia of inflammation. *N Engl J Med, 381*, 1148-1157. https://doi.org/10.1056/NEJMra1804281

Gernsheimer, T. B., Brown, S. P., Triulzi, D. J., et al. (2022). Prophylactic tranexamic acid in patients with hematologic malignancy: a placebo-controlled, randomized clinical trial. *Blood, 140*, 1254-1262. https://doi.org/10.1182/blood.2022016308

Glaspy, J., Crawford, J., Vansteenkiste, J., et al. (2010). Erythropoiesis-stimulating agents in oncology: a study-level meta-analysis of survival and other safety outcomes. *Br J Cancer, 102*, 301-315. https://doi.org/10.1038/sj.bjc.6605498

Holcomb, J. B., Tilley, B. C., Baraniuk, S., et al. (2015). Transfusion of plasma, platelets, and red blood cells in a 1:1:1 vs a 1:1:2 ratio and mortality in patients with severe trauma: the PROPPR randomized clinical trial. *JAMA, 313*, 471-482. https://doi.org/10.1001/jama.2015.12

Ker, K., Edwards, P., Perel, P., Shakur, H., & Roberts, I. (2012). Effect of tranexamic acid on surgical bleeding: systematic review and cumulative meta-analysis. *BMJ, 344*, e3054. https://doi.org/10.1136/bmj.e3054

Klastersky, J., Paesmans, M., Rubenstein, E. B., et al. (2000). The Multinational Association for Supportive Care in Cancer risk index: a multinational scoring system for identifying low-risk febrile neutropenic cancer patients. *J Clin Oncol, 18*, 3038-3051. https://doi.org/10.1200/JCO.2000.18.16.3038

Klastersky, J., de Naurois, J., Rolston, K., et al. (2016). Management of febrile neutropaenia: ESMO clinical practice guidelines. *Ann Oncol, 27*, v111-v118. https://doi.org/10.1093/annonc/mdw325

Kuderer, N. M., Dale, D. C., Crawford, J., & Lyman, G. H. (2007). Impact of primary prophylaxis with granulocyte colony-stimulating factor on febrile neutropenia and mortality in adult cancer patients receiving chemotherapy. *J Clin Oncol, 25*, 3158-3167. https://doi.org/10.1200/JCO.2006.08.8823

Mackenzie, C. F., Moon-Massat, P. F., Shander, A., et al. (2010). When blood is not an option: factors affecting survival after the use of a hemoglobin-based oxygen carrier in 54 patients with life-threatening anemia. *Anesth Analg, 110*, 685-693. https://doi.org/10.1213/ANE.0b013e3181cd473b

Mackenzie, C. F., Pitman, A. N., Hodgson, R. E., et al. (2015). Are hemoglobin-based oxygen carriers being withheld because of regulatory requirement for equivalence to packed red blood cells? *Am J Ther, 22*, e115-e121. https://doi.org/10.1097/MJT.0000000000000009

Maitland, K., Kiguli, S., Olupot-Olupot, P., et al. (2019). Immediate transfusion in African children with uncomplicated severe anemia. *N Engl J Med, 381*, 407-419. https://doi.org/10.1056/NEJMoa1900105

Metcalf, R. A., Nahirniak, S., Guyatt, G., et al. (2025). Platelet transfusion: 2025 AABB and ICTMG international clinical practice guidelines. *JAMA, 334*, 606-617. https://doi.org/10.1001/jama.2025.7529

Miller, A. B., Hoogstraten, B., Staquet, M., & Winkler, A. (1981). Reporting results of cancer treatment. *Cancer, 47*, 207-214. https://doi.org/10.1002/1097-0142(19810101)47:1<207::AID-CNCR2820470134>3.0.CO;2-6

Mueller, M. M., Van Remoortel, H., Meybohm, P., et al. (2019). Patient blood management: recommendations from the 2018 Frankfurt Consensus Conference. *JAMA, 321*, 983-997. https://doi.org/10.1001/jama.2019.0554

Natanson, C., Kern, S. J., Lurie, P., Banks, S. M., & Wolfe, S. M. (2008). Cell-free hemoglobin-based blood substitutes and risk of myocardial infarction and death: a meta-analysis. *JAMA, 299*, 2304-2312. https://doi.org/10.1001/jama.299.19.jrv80007

Resar, L. M., & Frank, S. M. (2014). Bloodless medicine: what to do when you can't transfuse. *Hematology Am Soc Hematol Educ Program, 2014*, 553-558. https://doi.org/10.1182/asheducation-2014.1.553

Rossaint, R., Afshari, A., Bouillon, B., et al. (2023). The European guideline on management of major bleeding and coagulopathy following trauma: sixth edition. *Crit Care, 27*, 80. https://doi.org/10.1186/s13054-023-04327-7

Scharman, C. D., Burger, D., Shatzel, J. J., Kim, E., & DeLoughery, T. G. (2017). Treatment of individuals who cannot receive blood products for religious or other reasons. *Am J Hematol, 92*, 1370-1381. https://doi.org/10.1002/ajh.24889

Schiffer, C. A., Bohlke, K., Delaney, M., et al. (2018). Platelet transfusion for patients with cancer: ASCO clinical practice guideline update. *J Clin Oncol, 36*, 283-299. https://doi.org/10.1200/JCO.2017.76.1734

Slichter, S. J., Kaufman, R. M., Assmann, S. F., et al. (2010). Dose of prophylactic platelet transfusions and prevention of hemorrhage. *N Engl J Med, 362*, 600-613. https://doi.org/10.1056/NEJMoa0904084

Smith, T. J., Bohlke, K., Lyman, G. H., et al. (2015). Recommendations for the use of WBC growth factors: ASCO clinical practice guideline update. *J Clin Oncol, 33*, 3199-3212. https://doi.org/10.1200/JCO.2015.62.3488

Stanworth, S. J., Estcourt, L. J., Powter, G., et al. (2013). A no-prophylaxis platelet-transfusion strategy for hematologic cancers. *N Engl J Med, 368*, 1771-1780. https://doi.org/10.1056/NEJMoa1212772

Thavendiranathan, P., Bagai, A., Ebidia, A., et al. (2005). Do blood tests cause anemia in hospitalized patients? *J Gen Intern Med, 20*, 520-524. https://doi.org/10.1111/j.1525-1497.2005.0094.x