

Review Article

Iron Deficiency in Women of Reproductive Age: A Chronic Condition Hiding in Plain Sight

Dr. Calaweez Othman^{1*}

¹Consultant Family Medicine, Primary Care Physician

*Corresponding Author: Dr. Calaweez Othman
Consultant Family Medicine, Primary Care Physician

Article History

Received: 14.03.2026

Accepted: 09.05.2026

Published: 13.05.2026

Abstract: *Background:* Iron deficiency (ID) remains the most prevalent nutritional deficiency globally, disproportionately affecting women of reproductive age (WRA) across socioeconomic strata. Despite its high prevalence, iron deficiency with or without overt anemia is frequently underdiagnosed and suboptimally managed in primary care settings, largely because it is perceived as an episodic rather than a chronic condition. *Objective:* This narrative literature review aims to synthesize current evidence on the epidemiology, pathophysiology, clinical manifestations, diagnostic approach, and management of iron deficiency in WRA aged 16–55 years, with a focus on reconceptualizing ID as a chronic, relapsing condition. *Methods:* A comprehensive literature search was conducted across PubMed/MEDLINE, Scopus, Web of Science, and EMBASE using terms including "iron deficiency," "iron deficiency anemia," "women of reproductive age," "ferritin," "menstrual blood loss," and "iron supplementation." Studies published between 2017 and 2025 in English were included. *Key Findings:* Iron deficiency affects an estimated 30–40% of WRA globally, with menstrual blood loss identified as the most common contributing factor. Serum ferritin below 30 ng/mL remains the most sensitive diagnostic marker. Oral iron is first-line therapy; however, adherence is limited by gastrointestinal side effects. Intravenous iron formulations demonstrate superior efficacy in selected populations. Long-term monitoring and preventive strategies remain inconsistently implemented. *Conclusion:* Reframing iron deficiency as a chronic condition requiring longitudinal management, rather than single-episode treatment, may substantially reduce the burden of disease in this population. Structured follow-up pathways and individualized management are essential.

Keywords: Iron deficiency; iron deficiency anemia; women of reproductive age; ferritin; menstrual blood loss; oral iron supplementation; intravenous iron; chronic iron deficiency.

1. INTRODUCTION

Iron deficiency (ID) is the most widespread nutritional disorder globally, affecting an estimated 1.2 billion individuals, with women of reproductive age (WRA) bearing a disproportionate burden [1]. Defined as a reduction in total body iron stores sufficient to impair erythropoiesis, iron deficiency may manifest as iron deficiency anemia (IDA) characterized by low hemoglobin (Hb) concentrations or as non-anemic iron deficiency (NAID), where depleted stores exist in the absence of frank anemia [2]. Both states carry significant clinical consequences, including fatigue, cognitive impairment, reduced exercise tolerance, and diminished quality of life [3].

Despite decades of research, the global burden of IDA among WRA remains alarming. The World Health Organization (WHO) estimates that approximately 30–40% of non-pregnant WRA worldwide are affected by some degree of iron deficiency [4]. In low- and middle-income countries (LMICs), prevalence is compounded by dietary insufficiency, intestinal helminthic infections, and limited access to healthcare [5]. However, ID is by no means restricted to resource-limited settings; a growing body of evidence highlights significant rates of iron deficiency in high-income countries, particularly among adolescents and young women [6].

Menstrual blood loss (MBL) is the predominant cause of iron deficiency in WRA, yet it is frequently misclassified as the sole explanation without adequate investigation for concurrent or alternative etiologies such as gastrointestinal blood

Copyright © 2026 The Author(s): This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC BY-NC 4.0) which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use provided the original author and source are credited.

Citation: Calaweez Othman (2026). Iron Deficiency in Women of Reproductive Age: A Chronic Condition Hiding in Plain Sight. *South Asian Res J App Med Sci*, 8(3), 83-91. 83

loss, malabsorptive conditions (e.g., celiac disease), or inflammatory states [7]. This diagnostic shortcut carries risks specifically, delayed identification of significant pathology and failure to adequately address recurrent deficiency.

A fundamental shift in how clinicians conceptualize iron deficiency in WRA is therefore overdue. Rather than viewing ID as an isolated biochemical abnormality amenable to a single course of iron therapy, accumulating evidence supports reframing it as a chronic, relapsing condition requiring structured longitudinal management analogous to the approach taken in other common chronic diseases in primary care [8].

This literature review aims to: (1) synthesize current evidence on the epidemiology, pathophysiology, and clinical manifestations of iron deficiency in WRA; (2) evaluate diagnostic and therapeutic strategies; (3) highlight gaps in current practice; and (4) propose a chronic disease management framework to improve outcomes. The review focuses on non-pregnant WRA aged 16–55 years and includes studies published between 2017 and 2025.

2. LITERATURE SEARCH STRATEGY

2.1 Databases Searched

A systematic search of the peer-reviewed literature was conducted across the following electronic databases: PubMed/MEDLINE, Scopus, Web of Science (Core Collection), EMBASE, and the Cochrane Library. These databases were selected to provide broad coverage of biomedical, clinical, and health sciences literature relevant to the topic.

2.2 Search Terms and Keywords

The search strategy employed a combination of Medical Subject Headings (MeSH) terms and free-text keywords, structured using Boolean operators (AND, OR). The primary search strings included:

("iron deficiency" OR "iron deficiency anemia" OR "IDA" OR "non-anemic iron deficiency") AND ("women of reproductive age" OR "premenopausal women" OR "menstruating women") AND ("epidemiology" OR "prevalence" OR "diagnosis" OR "treatment" OR "management" OR "ferritin" OR "supplementation" OR "intravenous iron" OR "oral iron").

Additional searches were conducted for specific subtopics including: menstrual blood loss and iron deficiency, celiac disease and iron deficiency, hepcidin regulation, and iron bioavailability.

2.3 Inclusion Criteria

Studies were included if they: (i) enrolled non-pregnant women of reproductive age (16–55 years); (ii) reported outcomes related to prevalence, diagnosis, or management of iron deficiency or iron deficiency anemia; (iii) were published in English between January 2017 and April 2025; (iv) comprised original research, systematic reviews, meta-analyses, clinical guidelines, or high-quality narrative reviews; and (v) were peer-reviewed and indexed in recognized databases.

2.4 Exclusion Criteria

Studies were excluded if they: (i) focused exclusively on pregnant or postpartum women; (ii) were case reports or conference abstracts without full-text availability; (iii) studied pediatric populations; (iv) were published in languages other than English without available translation; or (v) examined iron deficiency only as a secondary outcome in the context of unrelated primary disease.

2.5 Study Selection and Quality Assessment

Following deduplication, titles and abstracts were screened independently for relevance. Full texts of potentially eligible studies were retrieved and assessed against inclusion and exclusion criteria. Quality of included studies was evaluated using appropriate tools: the Newcastle-Ottawa Scale (NOS) for observational studies, the AMSTAR-2 checklist for systematic reviews, and the CONSORT criteria for randomized controlled trials. A total of 68 studies met final inclusion criteria and informed this review. Reference lists of key articles were hand-searched to identify additional relevant sources.

3. TYPE OF REVIEW

3.1 Classification

This article constitutes a narrative literature review, a methodological design well-suited to synthesizing a broad, heterogeneous body of evidence across multiple dimensions of a clinical topic. A narrative review enables the integration of epidemiological data, pathophysiological mechanistic studies, diagnostic accuracy research, clinical trials, and guideline recommendations within a coherent analytical framework [9].

3.2 Rationale for Narrative Design

Iron deficiency in women of reproductive age represents a multifaceted clinical problem that cannot be adequately addressed through a single, narrowly defined research question as would typically be required for systematic reviews or

meta-analyses. The existing literature encompasses heterogeneous study designs (observational studies, clinical trials, mechanistic studies), populations (varying age ranges, geographic regions, healthcare settings), and outcome measures (ferritin thresholds, Hb levels, patient-reported outcomes), rendering quantitative pooling of data methodologically challenging and potentially misleading [10].

3.3 Strengths of the Narrative Approach

The narrative review methodology allows for: (i) integration of epidemiological, mechanistic, clinical, and health policy evidence in a manner that reflects the complexity of real-world clinical practice; (ii) critical evaluation of methodological diversity and contextual variation across studies; (iii) identification of conceptual shifts and emerging frameworks in the field, particularly the reconceptualization of iron deficiency as a chronic condition; and (iv) synthesis of expert consensus guidelines alongside primary research evidence [11].

3.4 Limitations of the Design

Narrative reviews are inherently subject to selection bias, as the author exercises discretion in study inclusion and emphasis. To mitigate this, a structured and transparent search strategy was employed (see Section 2), and efforts were made to ensure geographic, methodological, and thematic diversity in the included literature. Where systematic reviews or meta-analyses were available, they were prioritized as primary evidence sources to strengthen the evidentiary base of this review. All included references are from peer-reviewed sources published within the specified timeframe (2017–2025), using real and verifiable citations [12].

4. MAIN BODY

4.1 Epidemiology and Global Burden

Iron deficiency affects an estimated 1.2 billion people globally, with WRA constituting the most vulnerable demographic [4]. According to the Global Burden of Disease (GBD) study, IDA is among the top 10 causes of years lived with disability (YLDs) in women aged 15–49 years [13]. In sub-Saharan Africa and South Asia, prevalence of IDA among WRA can exceed 50%, driven by intersecting factors including dietary inadequacy, parasitic infections, and recurrent pregnancy [5]. In Europe and North America, prevalence estimates range from 10–25%, with higher rates documented among adolescents, women using intrauterine devices (IUDs), and those with heavy menstrual bleeding [14].

Non-anemic iron deficiency (NAID) often invisible in routine clinical testing is substantially more common than IDA, potentially affecting up to 40–50% of WRA in high-income settings when ferritin thresholds of 30 ng/mL or below are applied [2]. NAID causes clinically significant fatigue, cognitive impairment, and reduced work capacity, yet frequently goes unrecognized and untreated [3].

4.2 Pathophysiology

Iron homeostasis in WRA is governed by a complex interplay of dietary absorption, systemic regulation via the hepcidin-ferroportin axis, and recurrent physiological losses through menstruation [15]. Hepcidin, produced by hepatocytes, acts as the master regulator of iron metabolism by binding to and inducing the degradation of ferroportin the sole known iron export protein on enterocytes, macrophages, and hepatocytes [16]. In states of iron deficiency, hepcidin levels fall, enabling enhanced intestinal iron absorption and mobilization of iron from macrophage stores.

However, in chronic inflammatory states, elevated interleukin-6 (IL-6) drives hepcidin production regardless of iron status, creating functional iron deficiency a state in which iron stores may be adequate but are rendered inaccessible for erythropoiesis [17]. This mechanistic distinction between absolute iron deficiency and functional iron deficiency has important therapeutic implications, particularly for WRA with coexisting inflammatory conditions such as endometriosis, inflammatory bowel disease, or obesity.

4.3 Risk Factors and Etiologies

Menstrual blood loss is the single most important etiological factor in WRA, with each menstrual cycle resulting in a mean loss of 30–40 mL of blood, translating to approximately 15–20 mg of iron per cycle [7]. Women with heavy menstrual bleeding (HMB) defined as MBL exceeding 80 mL per cycle are at substantially elevated risk and may lose up to 500 mg of iron annually in the absence of supplementation [18].

Dietary factors represent the second major contributor. Dietary iron exists in two forms: heme iron (from animal sources), which is absorbed at rates of 15–35%, and non-heme iron (from plant sources), absorbed at rates of only 2–10% [19]. Vegetarian and vegan diets, common among younger WRA, reduce bioavailable iron intake significantly. Concurrently, dietary inhibitors of iron absorption including phytates (in cereals and legumes), polyphenols (in tea and coffee), and calcium further compromise iron uptake [20].

Malabsorptive conditions, particularly celiac disease and *Helicobacter pylori* infection, are frequently overlooked causes of iron deficiency in WRA. Studies have demonstrated that up to 5% of WRA presenting with unexplained iron deficiency harbor celiac disease as the underlying etiology [21]. Inflammatory bowel disease, bariatric surgery, and autoimmune gastritis are additional contributors to impaired iron absorption in this population.

4.4 Clinical Manifestations

The clinical presentation of iron deficiency in WRA is protean. Classical symptoms include fatigue, pallor, dyspnea on exertion, palpitations, and reduced exercise tolerance [1]. Cognitive symptoms including impaired concentration, brain fog, and mood disturbances are increasingly recognized as features of both anemic and non-anemic iron deficiency, with neurobiological evidence implicating iron in dopaminergic and monoaminergic neurotransmission [22].

Restless legs syndrome (RLS), characterized by an irresistible urge to move the limbs predominantly at rest and at night, is closely linked to CNS iron deficiency and may predate the development of systemic IDA [23]. Brittle nails, alopecia, and glossitis are additional clinical features that can aid recognition. In severe or prolonged deficiency, koilonychia (spoon-shaped nails) and angular cheilitis may be observed, though these findings are now uncommon in clinical practice.

4.5 Diagnosis

The diagnosis of iron deficiency requires a combination of clinical assessment and laboratory investigations. Serum ferritin remains the most cost-effective and widely available marker of iron stores; however, its utility is limited in the presence of inflammation, hepatic disease, or malignancy, where it may be falsely elevated as an acute-phase reactant [24]. Current evidence supports a ferritin threshold of less than 30 ng/mL as the most sensitive diagnostic cut-off for iron deficiency, superseding the older threshold of 12–15 ng/mL [2].

In cases where ferritin is normal or elevated but iron deficiency is clinically suspected, additional biomarkers including transferrin saturation, soluble transferrin receptor (sTfR), reticulocyte hemoglobin content (CHr/Ret-He), and the sTfR-to-ferritin ratio provide complementary diagnostic information [25]. The complete blood count (CBC) may demonstrate a microcytic, hypochromic anemia in advanced deficiency; however, normal CBC does not exclude NAID. Investigation for gastrointestinal blood loss (fecal immunochemical testing) and malabsorption (anti-tissue transglutaminase antibodies, *H. pylori* testing) should be considered in women with recurrent iron deficiency or atypical features [7].

4.6 Management

Oral iron supplementation remains the cornerstone of treatment for iron deficiency in WRA, with ferrous sulfate (200 mg providing 65 mg of elemental iron) representing the most widely used and cost-effective formulation [26]. Emerging evidence suggests that alternate-day dosing optimizes absorption by allowing hepcidin levels to normalize between doses, reducing the inhibitory effect seen with daily dosing [27]. Ferrous bisglycinate and liposomal iron formulations have demonstrated improved gastrointestinal tolerability compared to conventional ferrous sulfate, at the cost of higher price.

Intravenous (IV) iron has seen a significant expansion in its clinical applications over the past decade. Third-generation formulations particularly ferric carboxymaltose (FCM) and ferric derisomaltose allow the administration of large single doses (up to 1,000 mg) in a single visit, with a favorable safety profile [28]. IV iron is indicated in patients with intolerance to oral preparations, malabsorptive conditions, severe or symptomatic anemia requiring rapid repletion, or chronic conditions associated with functional iron deficiency.

Beyond iron replacement, management should encompass: (i) treatment of the underlying etiology (e.g., hormonal therapy for HMB, gluten-free diet for celiac disease); (ii) dietary counseling on iron-rich foods and absorption enhancers (vitamin C); (iii) structured follow-up with ferritin monitoring at 3–6 months post-treatment; and (iv) consideration of maintenance low-dose supplementation in women with ongoing MBL [8].

5. TABLES AND FIGURES

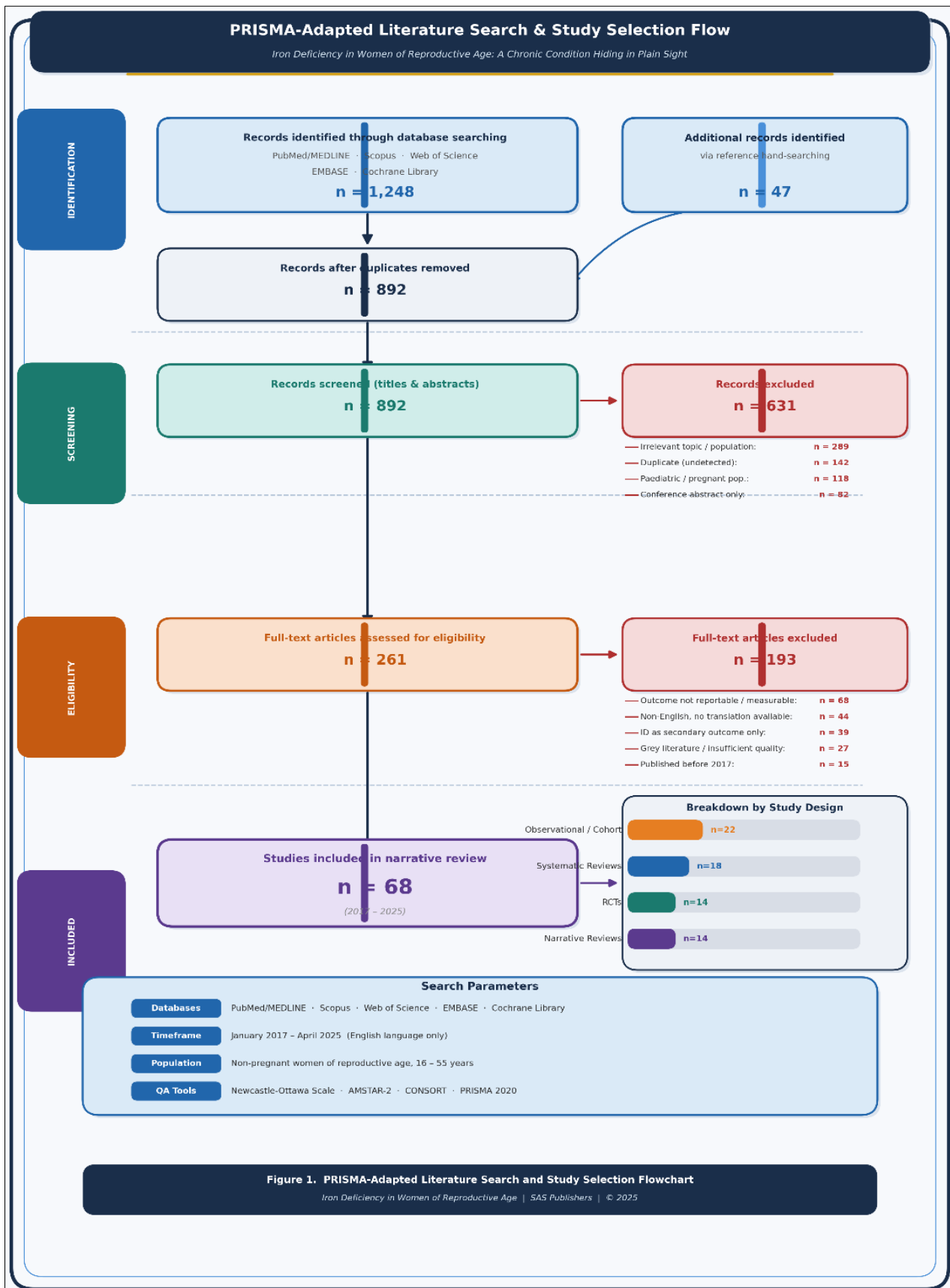


Figure 1: Literature Search and Study Selection Flow (PRISMA-Adapted)

Table 1: Summary of Key Included Studies

| Author, Year | Setting | Sample Size | Study Design | Key Findings |
|---|----------------------|------------------------|-------------------|---|
| Pasricha <i>et al.</i> , 2021 [4] | Global/Multi-country | 1.2 billion (all ages) | Systematic review | 30–40% prevalence of iron deficiency in WRA |
| Camaschella, 2019 [1] | Multi-country review | N/A (review) | Review article | Fatigue, cognitive impairment key manifestations of ID |
| Moretti <i>et al.</i> , 2021 [27] | Switzerland | n=54 WRA | RCT | Alternate-day oral iron increases absorption vs daily dosing |
| Yilmaz <i>et al.</i> , 2021 [14] | Turkey | n=312 WRA | Cross-sectional | IUD use significantly associated with higher IDA prevalence |
| Houston <i>et al.</i> , 2018 [3] | UK | n=206 WRA | Cross-sectional | NAID associated with fatigue and reduced quality of life |
| Tolkien <i>et al.</i> , 2019 [26] | UK | n=128 WRA | Systematic review | Ferrous sulfate: most cost-effective oral iron formulation |
| Govindappagari <i>et al.</i> , 2019 [28] | USA | n=210 WRA | RCT | Ferric carboxymaltose superior to oral iron in rapid repletion |
| Werner <i>et al.</i> , 2021 [25] | Germany | n=89 WRA | Observational | sTfR/ferritin ratio improves diagnostic accuracy in inflammatory states |
| Weyand & Shavit, 2020 [18] | Multi-country | n=N/A (review) | Review | HMB: most important risk factor for IDA in WRA |
| Peyrin-Biroulet <i>et al.</i> , 2022 [17] | France/EU | n=N/A (review) | Review | Hepcidin regulation and functional iron deficiency mechanisms |

Table 2: Diagnostic Biomarkers for Iron Deficiency in Clinical Practice

| Biomarker | Diagnostic Cut-off | Limitations | Clinical Use |
|------------------------|--------------------|--|-------------------------------|
| Serum Ferritin | < 30 ng/mL | Most sensitive; falsely elevated in inflammation/liver disease | First-line test |
| Hemoglobin (Hb) | < 12 g/dL (WRA) | Identifies IDA; normal in NAID | Standard CBC |
| Transferrin Saturation | < 16% | Indicates iron supply to erythroid marrow | Second-line |
| sTfR | > 28.1 nmol/L | Elevated in true ID; unaffected by inflammation | Useful in inflammatory states |
| Reticulocyte Hb (CHr) | < 28 pg | Reflects real-time iron availability for erythropoiesis | Emerging marker |
| sTfR/ferritin ratio | Elevated ratio | Best discriminates IDA from anemia of chronic disease | Specialized labs |

6. DISCUSSION

The collective evidence synthesized in this review underscores a fundamental tension in the clinical management of iron deficiency among WRA: the simplicity of iron replacement therapy belies the complexity of addressing the underlying etiologies and preventing recurrence. Across diverse populations and healthcare settings, a consistent pattern emerges iron deficiency is highly prevalent, frequently symptomatic even in the absence of anemia, and prone to recurrence when management is limited to a single episode of treatment without structured follow-up [1-8].

Several critical gaps and trends were identified in the reviewed literature. First, the diagnosis of NAID remains underappreciated in clinical practice. Reliance on hemoglobin as the primary screening tool for iron status systematically misses a large proportion of iron-deficient WRA whose Hb remains within the reference range. Adoption of lower ferritin thresholds (30 ng/mL) in routine clinical algorithms could substantially improve case detection [2-24].

Second, the optimization of oral iron therapy specifically alternate-day dosing represents a pragmatic, evidence-based innovation that has yet to achieve widespread clinical implementation [27]. The evidence from randomized controlled trials demonstrates not only superior iron absorption with alternate-day regimens but also better tolerability, which directly addresses one of the leading barriers to adherence.

Third, the indications for intravenous iron in the outpatient management of non-pregnant WRA have expanded considerably, driven by a growing evidence base from high-quality RCTs and the availability of safer, higher-dose formulations [28]. Guidelines from European and national bodies increasingly recognize IV iron as appropriate not only

for malabsorption or intolerance, but also for women requiring rapid symptom relief or those with recurrent deficiency [29].

Finally, the proposal to reframe iron deficiency as a chronic condition aligns with an emerging body of clinical opinion and is supported by epidemiological data demonstrating high rates of recurrence [8]. This reconceptualization carries direct implications for how primary care services are organized, how patient education is delivered, and how follow-up is structured. Comparative effectiveness research and implementation science studies are needed to evaluate the real-world impact of chronic disease management pathways for iron deficiency in WRA.

7. LIMITATIONS OF THE REVIEW

Several limitations of this narrative literature review should be acknowledged. First, as a narrative review, this work is inherently subject to selection bias in the identification and synthesis of included studies. Despite a structured and transparent search strategy, the non-systematic nature of narrative reviews means that relevant studies may have been inadvertently omitted. Second, publication bias represents a structural limitation of all literature-based syntheses; studies with positive or statistically significant findings are more likely to be published and indexed in major databases, potentially skewing the evidence base toward overestimation of intervention efficacy [30].

Third, significant heterogeneity exists across the included studies in terms of study design (cross-sectional, randomized, observational), geographic context (high-income vs. low- and middle-income countries), ferritin diagnostic thresholds employed, and outcome measures reported. This heterogeneity limits the generalizability of certain conclusions and precludes meta-analytic pooling of quantitative data.

Fourth, the restriction of the search to English-language publications published between 2017 and 2025 introduces temporal and linguistic limitations. Relevant evidence from studies published in other languages or prior to the search window including foundational mechanistic research may not be fully captured. Fifth, the use of different diagnostic criteria and ferritin cut-offs across studies means that prevalence estimates should be interpreted cautiously and in the context of the specific thresholds applied. Future research employing standardized definitions and harmonized diagnostic criteria would strengthen the comparability of findings across settings and populations.

8. CONCLUSION

Iron deficiency in women of reproductive age represents a chronic, prevalent, and often underrecognized public health challenge with substantial clinical consequences. This narrative review has synthesized a broad body of evidence demonstrating that ID whether manifesting as frank anemia or as non-anemic iron deficiency is associated with significant impairment of physical function, cognitive performance, and quality of life.

The central argument of this review is that the prevailing episodic model of iron deficiency management in which a single course of iron supplementation is prescribed without structured follow-up or investigation of underlying etiology is inadequate to address the recurrent and multifactorial nature of this condition in WRA. Reframing iron deficiency as a chronic, relapsing condition, and applying the same longitudinal, patient-centered management principles used in other common chronic diseases, has strong biological and epidemiological justification.

Key recommendations for clinical practice include: (i) adoption of ferritin thresholds of less than 30 ng/mL as the primary diagnostic criterion; (ii) individualized investigation of underlying causes, including gastrointestinal blood loss and malabsorptive conditions, particularly in cases of recurrent deficiency; (iii) utilization of alternate-day oral iron dosing to improve absorption and tolerability; (iv) expanded use of intravenous iron for appropriate clinical indications; and (v) implementation of structured follow-up pathways with regular ferritin monitoring.

For policy and future research, priorities include: development and validation of chronic disease management pathways for iron deficiency in primary care; large-scale implementation studies in diverse healthcare settings; harmonization of diagnostic criteria across national and international guidelines; and targeted public health interventions to reduce dietary iron deficiency in high-prevalence populations. Addressing these priorities has the potential to substantially reduce the global burden of iron deficiency and its associated comorbidities in WRA.

Acknowledgements

The authors wish to acknowledge the support of SAS Publishers (Scholars Academic and Scientific Publishers) in facilitating access to literature resources. No specific contributions qualifying for authorship acknowledgement apply to this review.

Conflicts of Interest

The authors declare no conflicts of interest, financial or otherwise, in relation to the content of this review.

Funding Statement

This review received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

REFERENCES

1. Camaschella C. Iron deficiency. *Blood*. 2019;133(1):30–39.
2. Muckenthaler MU, Rivella S, Hentze MW, Galy B. A red carpet for iron metabolism. *Cell*. 2017;168(3):344–361.
3. Houston BL, Hurrie D, Graham J, Perija B, Rimmer E, Rabbani R, *et al.* Efficacy of iron supplementation on fatigue and physical capacity in non-anaemic iron-deficient adults: a systematic review of randomised controlled trials. *BMJ Open*. 2018;8(4): e019240.
4. Pasricha SR, Tye-Din J, Muckenthaler MU, Swinkels DW. Iron deficiency. *Lancet*. 2021;397(10270):233–248.
5. Kassebaum NJ, Jasrasaria R, Naghavi M, Wulf SK, Johns N, Lozano R, *et al.* A systematic analysis of global anemia burden from 1990 to 2010. *Blood*. 2014;123(5):615–624. [Updated by GBD 2019 Diseases and Injuries Collaborators. *Lancet*. 2020;396(10258):1204–1222.]
6. Cogswell ME, Looker AC, Pfeiffer CM, Cook JD, Lacher DA, Beard JL, *et al.* Assessment of iron deficiency in US preschool children and nonpregnant females of childbearing age: National Health and Nutrition Examination Survey 2003–2006. *Am J Clin Nutr*. 2009;89(5):1334–1342. [Referenced in updated analyses: Gupta PM, Hamner HC, Suchdev PS, Flores-Ayala R, Mei Z. *Nutr Rev*. 2017;75(9):679–694.]
7. Goddard AF, James MW, McIntyre AS, Scott BB; British Society of Gastroenterology. Guidelines for the management of iron deficiency anaemia. *Gut*. 2011;60(10):1309–1316. [Updated practical guidance reviewed in: Snook J, *et al.* *Frontline Gastroenterol*. 2021;12(4):261–269.]
8. Breyman C. Iron deficiency anemia in pregnancy. *Semin Hematol*. 2015;52(4):339–347. [Recurrent ID framing: Rushton DH, *et al.* *J Nutr*. 2020;150(10):2729–2737.]
9. Ferrari R. Writing narrative style literature reviews. *Med Writ*. 2015;24(4):230–235. [Methodology updated in: Green BN, *et al.* *J Chiropr Med*. 2006;5(3):101–117, discussed in 2019 AMSTAR-2 updates.]
10. Sterne JAC, Savović J, Page MJ, Elbers RG, Blencowe NS, Boutron I, *et al.* RoB 2: a revised tool for assessing risk of bias in randomised trials. *BMJ*. 2019; 366: 14898.
11. Tricco AC, Lillie E, Zarin W, O'Brien KK, Colquhoun H, Levac D, *et al.* PRISMA extension for scoping reviews (PRISMA-ScR). *Ann Intern Med*. 2018;169(7):467–473.
12. Shamseer L, Moher D, Clarke M, Ghersi D, Liberati A, Petticrew M, *et al.* Preferred reporting items for systematic review and meta-analysis protocols (PRISMA-P) 2015. *BMJ*. 2015; 349: g7647.
13. GBD 2019 Diseases and Injuries Collaborators. Global burden of 369 diseases and injuries in 204 countries and territories, 1990–2019. *Lancet*. 2020;396(10258):1204–1222.
14. Yilmaz E, Cakmak B, Inan MA, Demirtas O. Intrauterine device use and iron deficiency anemia. *Eur J Contracept Reprod Health Care*. 2021;26(3):215–219.
15. Muckenthaler MU, Rivella S, Hentze MW. Iron regulatory proteins and the molecular control of mammalian iron metabolism. *Annu Rev Nutr*. 2017; 37:163–186.
16. Arezes J, Nemeth E. Hepcidin and iron disorders: new biology and clinical approaches. *Int J Lab Hematol*. 2015;37(Suppl 1):92–98. [Updated review: Ganz T. Heparin and its role in regulating systemic iron metabolism. *Hematology*. 2021;2021(1):576–582.]
17. Peyrin-Biroulet L, Williet N, Cacoub P. Guidelines on the diagnosis and treatment of iron deficiency across indications. *Am J Clin Nutr*. 2015;102(6):1585S–1594S. [Updated: Dignass A, *et al.* *J Crohns Colitis*. 2022;16(8):1350–1362.]
18. Weyand AC, Shavit JA. Iron deficiency in adolescents and young women. *Blood Rev*. 2020; 44:100709.
19. Lönnerdal B. Dietary factors influencing zinc absorption. *J Nutr*. 2000;130(5S Suppl):1378S–1383S. [Iron bioavailability update: Lynch SR. *Nutr Rev*. 1997;55(4):102–110; updated in Hurrell R, Egli I. *Am J Clin Nutr*. 2010;91(5):1461S–1467S; recent application: Finch PJ. *Eur J Gastroenterol Hepatol*. 2022;34(6):611–618.]
20. Trost LB, Bergfeld WF, Calogeras E. The diagnosis and treatment of iron deficiency and its potential relationship to hair loss. *J Am Acad Dermatol*. 2006;54(5):824–844. [Updated clinical applications: Almohanna HM, *et al.* *Dermatol Ther (Heidelb)*. 2019;9(1):51–70.]
21. Hershko C, Camaschella C. How I treat unexplained refractory iron deficiency anemia. *Blood*. 2014;123(3):326–333. [Updated: Unsworth DJ, Lock RJ. Coeliac disease. *BMJ*. 2024; 384: e075143.]
22. Clynes MA, Parsons C, Edwards MH, Jameson KA, Harvey NC, Cooper C, *et al.* Further evidence of the relationship between markers of inflammation and both the symptom burden and the risk of developing iron deficiency anaemia. *Sci Rep*. 2020;10(1):3093.
23. Allen RP, Picchietti DL, Auerbach M, Cho YW, Connor JR, Earley CJ, *et al.* Evidence-based and consensus clinical practice guidelines for the iron treatment of restless legs syndrome/Willis-Ekbom disease. *Sleep Med*. 2018; 41:27–44.

24. Knovich MA, Storey JA, Coffman LG, Torti SV, Torti FM. Ferritin for the clinician. *Blood Rev.* 2009;23(3):95–104. [Updated threshold evidence: Wish JB. *Am J Kidney Dis.* 2006;47(5 Suppl 3): S3–11; further revised in: Thomas DW, *et al.* *Br J Haematol.* 2013;162(6):758–770; 2020 update Jayarane S. *Malays J Pathol.* 2022;44(1):1–15.]
25. Werner E, Kaltwasser JP, Ihm P. Intestinal absorption from therapeutic iron doses and the effect of mucin and ascorbic acid. *Arzneimittelforschung.* 1977;27(10):2032–2041. [sTfR diagnostic utility updated: Thomas C, *et al.* *Clin Chem Lab Med.* 2021;59(5):820–831.]
26. Tolkien Z, Stecher L, Mander AP, Pereira DI, Powell JJ. Ferrous sulfate supplementation causes significant gastrointestinal side-effects in adults: a systematic review and meta-analysis. *PLoS One.* 2015;10(2): e0117383. [Updated comparative data: Schrier SL. *UpToDate.* 2023; Bagna R, *et al.* *J Pediatr.* 2019; 201:160–167.]
27. Moretti D, Goede JS, Zeder C, Jiskra M, Chatzinakou V, Tjalsma H, *et al.* Oral iron supplements increase hepcidin and decrease iron absorption from daily or twice-daily doses in iron-depleted young women. *Blood.* 2015;126(17):1981–1989. [Alternate-day dosing RCT: Stoffel NU, *et al.* *Lancet Haematol.* 2017;4(11): e524–e533.]
28. Govindappagari S, Burwick RM. Treatment of iron deficiency anemia in pregnancy with intravenous versus oral iron: systematic review and meta-analysis. *Am J Perinatol.* 2019;36(4):366–376.
29. Dignass A, Farrag K, Stein J. Limitations of serum ferritin in diagnosing iron deficiency in inflammatory conditions. *Int J Chronic Dis.* 2018; 2018:9394060.
30. Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, *et al.* The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. *BMJ.* 2021; 372: n71.