

Hematology



ISSN: (Print) (Online) Journal homepage: www.tandfonline.com/journals/yhem20

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To cite this article: Madunil Anuk Niriella, Hiruni Jayasena, Achini Withanachchi & Anuja Premawardhena (2024) Mistakes in the management of iron deficiency anaemia: a narrative review, Hematology, 29:1, 2387987, DOI: 10.1080/16078454.2024.2387987

To link to this article: https://doi.org/10.1080/16078454.2024.2387987

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Published online: 07 Aug 2024.

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Mistakes in the management of iron deficiency anaemia: a narrative review

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ABSTRACT

Introduction: Anaemia occurs due to an imbalance between erythrocyte production and loss. This imbalance can be due to ineffective erythropoiesis, blood loss or haemolysis. Whilst there are many causes for anaemia, iron deficiency anaemia (IDA) remains the predominant cause worldwide.

Areas covered: There have been many updated guidelines on the management of IDA in the past few years. As the reasons for IDA are many, evaluation requires thorough analysis and focused investigations. As an asymptomatic disease in the early stages, IDA can lead to many mistakes in its management. This review highlights potential mistakes in assessing and managing IDA and recommendations to avoid them.

Conclusion: The effective management of IDA necessitates a comprehensive and multidisciplinary approach. By recognising and addressing the common mistakes highlighted in this narrative review, healthcare professionals can improve patient outcomes, minimise complications, and enhance the overall quality of care.

Plain language summary

Iron deficiency anaemia (IDA) is a major global health issue. Proper diagnosis involves taking a detailed medical history, physical exam, and laboratory tests. A good response to iron replacement therapy, even with inconclusive lab results, strongly suggests IDA. Screening for Helicobacter pylori infection should be done in areas with high prevalence. For adult men and postmenopausal women with IDA, bidirectional endoscopy is recommended to check for gastrointestinal bleeding. Guidelines vary for premenopausal women. If IDA persists despite treatment and negative endoscopies, further small bowel evaluation may be needed. Treatment choice depends on patient factors and how urgently iron levels need correction - options include oral/IV iron or blood transfusions for severe cases. After treating anaemia, follow-ups should monitor compliance, side effects and treatment response.

Article highlights

- Iron deficiency anaemia (IDA) is a significant cause of morbidity worldwide.
- The initial evaluation should contain a thorough history (including dietary habits, medication use, and menstrual cycles), identification of any concurrent disorders, and clinical examination.

- To confirm IDA and rule out other potential causes, a relevant standardised laboratory workup such as iron studies, red cell indices, and haemoglobin electrophoresis (if relevant) should be performed.
- An excellent response (Hb rise ≥10 g/L within a 2week timeframe) to iron replacement therapy (IRT) in anaemic patients is highly suggestive of absolute iron deficiency, even if the results of iron studies are equivocal.
- In cases of inexplicable IDA or IDA from high-prevalence areas, active screening for *Helicobacter pylori* (using non-invasive testing methods) and treatment initiation should be implemented.
- As blood loss from the gastrointestinal tract is the commonest cause of IDA in adult men and postmenopausal females, bidirectional endoscopy must be conducted.
- In pre-menopausal women with IDA, guidance about investigations varies.
- In cases of persistent or recurrent IDA despite adequate IRT and negative bidirectional endoscopy, further evaluation of the small bowel (by capsule endoscopy, enteroscopy or CT/MR enterography) may be warranted.
- Patient factors and the urgency of iron repletion must be considered when determining the appropriateness of the type of IRT.
- IRT to replenish iron stores and improve haemoglobin levels can be done with iron therapy (oral/

Received 2 June 2024 Accepted 30 July 2024

ARTICLE HISTORY

KEYWORDS

Anaemia; iron deficiency; evaluation; haemoglobin; blood loss

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Table 1. Haemoglobin cut-off values to diagnose anaemia [1].

Group	Haemoglobin g/
Men (Age > 15)	130
Non-pregnant women	120
Pregnant women	110

intravenous) or blood transfusions (reserved for severe, symptomatic patients).

 Following the correction of anaemia, a clinical review according to standardised follow-up protocol focusing on symptom management, treatment compliance, and the presence of adverse events should be conducted.

Introduction

Anaemia is a haemoglobin (Hb) concentration below the lower limit of normal for the relevant population (Table 1). The severity of anaemia is classified into mild, moderate and severe. The Hb cut-offs for these severities vary between three groups: 1. men, 2. nonpregnant women and 3. pregnant women (Table 2) [1].

Anaemia occurs due to an imbalance between erythrocyte production and loss. This imbalance is due to either ineffective erythropoiesis, blood loss or haemolysis. Whilst anaemia has various causes, iron deficiency anaemia (IDA) remains the most predominant cause of anaemia worldwide [2]. Globally, there are over 2 billion cases of IDA [2].

Iron deficiency (ID) can be due to increased iron need, reduced availability, or both. Decreased iron availability can result when iron loss exceeds the absorptive capacity of the small intestine. Prolonged, uncorrected ID will result in IDA. Whilst IDA commonly involves the gastrointestinal system, it can be due to blood loss from other sources, such as excessive menstruation in premenopausal women (Table 3) [3]. Furthermore, suboptimal intake of iron and malabsorption of iron within the gastrointestinal tract can also less frequently contribute to IDA.

Iron plays a significant role in many cellular processes within the human body. Individuals may be symptomatic with ID, even without the presence of anemia. ID can lead to various health complications such as fatigue and reduced cognitive function [4]. ID can also increase the risk of heart failure as well as raise the risk of death if untreated [4]. Hence recognition and provision of early treatment with iron replacement therapy (IRT) is paramount to prevent future complications of ID. In the presence of IDA however, blood transfusions may be indicated in those patients

Table 3. Non-gastrointestinal and gastrointestinal causes of iron deficiency anaemia.

Non-gastrointestinal causes	Gastrointestinal causes
Menstruation	Aspirin/ NSAID use
Blood donation	Malignancy:Oesophageal/gastric/small bowel/ ampullary/ colonic
Haematuria	Helminthic infections
Epistaxis	Benign gastric ulceration
	Esophagitis
	Angiodysplasia
	Gastric antral vascular ectasia

with severe symptoms of anaemia rather than needing to correct the Hb.

Although the management of IDA may seem straightforward, numerous pitfalls can lead to suboptimal care and prolonged patient suffering. Hence, the management of ID and IDA can remain a complex challenge for clinicians. This review aims to highlight common mistakes in the management of IDA and provide practical strategies to avoid them, ensuring better patient outcomes.

Mistake 1: incomplete clinical evaluation

A detailed medical history is the cornerstone of any clinical evaluation, and IDA is no exception. Overlooking crucial aspects of the patient's history can result in missed opportunities for timely diagnosis and appropriate management of IDA.

Clinical symptoms of IDA are varied and can be nonspecific. They depend on several factors, such as the patient's age co-morbidities, as well as the severity, speed of onset and chronicity of anaemia [5]. The commonly encountered symptoms are exertional dyspnoea, chest pain, lethargy, fatigue and poor concentration. Furthermore, ID can further exacerbate underlying medical conditions such as ischemic heart disease and heart failure. Pica, the compulsive eating of non-nutritive substances (commonly that of ice), is one of the most important clues to IDA [6]. In the context of anaemia, pica is almost always suggestive of IDA. Plummer-Vinson syndrome and the oesophageal webs, which present with dysphagia, are also associated with IDA [7].

Owing to the many causes and associations of IDA, clinicians must obtain a comprehensive history and recognise any significant associations. It is paramount that any features of upper or lower GI bleeding, menstrual patterns in females, history of gastrointestinal malignancies and conditions that may predispose to chronic blood loss (helminthic infestation, peptic ulcers, angio-ectasia) are checked. Personal and family

Table 2. Haemoglobin cut-off values according to severity of anaemia [1].

Group	Haemoglobin g/l in non-anaemia	Haemoglobin g/l in mild anaemia	Haemoglobin g/l in moderate anaemia	Haemoglobin g/l in severe anaemia
Men (Age > 15)	≥130	110–129	80–109	<80
Non-Pregnant women	≥120	110–119	80–109	<80
Pregnant women	≥110	100–109	70–99	<70

history of bleeding disorders and blood donation must be noted. Conditions causing intestinal mucosal damage resulting in decreased iron absorption from the diet (atrophic gastritis, *Helicobacter pylori* infection and inflammatory bowel disease) and the presence of previous surgeries such as gastrectomy, duodenal bypass, and bariatric procedures must be looked explicitly to in the patient's history. Diligently inquire about the use of medications (over-the-counter medications such as non-steroidal anti-inflammatory drugs) that can cause gastrointestinal bleeding. Notably acid modifying therapies such as proton pump inhibitors and H2 receptor blockers, can impair iron absorption, due to long-term gastric acid reduction in the stomach [8].

It is also important to inquire about dietary habits. Animal food products are rich in haem and are readily absorbable, whilst plants contain non-haem iron, which is non-absorbable [2]. Excessive consumption of cow's milk in children can also contribute to IDA due to the low iron content and/or as a result of occult intestinal blood loss [9]. An individual's cultural and personal beliefs can lead to veganism or vegetarianism, significantly contributing to low haem iron content in the diet. Dual pathology may also coexist for IDA, particularly among individuals with limited dietary iron intake in developing countries, where helminthic infestation or chronic diseases such as end-stage renal failure or rheumatoid arthritis may co-exist [10, 11].

Upon clinical examination, patients may appear pale and lethargic with dry skin and hair. In severe cases, there might be evidence of leukonychia and koilonychia. Loss of tongue papillae and atrophic glossitis have also been noted [5]. Rarely, it may be possible to detect evidence for hereditary haemorrhagic telangiectasia or Peutz-Jeghers syndrome on examination of the oral cavity as a cause of IDA. However, IDA can be an incidental finding, as some individuals remain asymptomatic and undiagnosed for many years.

Strategies to avoid this mistake

When evaluating patients with IDA, using standardised questionnaires or checklists to ensure comprehensive history-taking should be implemented at primary contact. Furthermore, patients should be encouraged to maintain accurate records of their dietary intake, medication use, and menstrual cycles. Establishing a good patient-doctor relationship is essential to foster open communication and create a comfortable environment for patients to disclose sensitive personal and family information.

Mistake 2: inadequate investigation

Given the many aetiologies related to anaemia, relying solely on Hb levels to diagnose IDA is unwise. To

confirm IDA and rule out other potential causes, a relevant laboratory workup should be performed [5, 12, 13].

Iron studies should confirm ID before further investigations [10, 11]. Serum ferritin is the single most helpful marker of IDA, but other blood tests can be beneficial if false-normal ferritin is suspected [10, 11]. In IDA, serum ferritin is less than 45 ng/ml [12, 13]. This level has a sensitivity of 85% with a specificity of 92% for ID and is considered the most appropriate ferritin threshold [14]. However, much controversy remains in determining the most appropriate cut off value for ferritin for IDA. This is reflected in the difference in the ferritin level thresholds used for the diagnosis of ID and IDA between the two guidelines listed in Table 4. Hence, a precise threshold has not been determined, but rather it fluctuate from less than 15 to 100 ng/mL. Given that ferritin is an acute phase reactant, in clinical situations of systemic inflammation or infections, ferritin levels will be reassuringly high. Thus, it is suggested that in patients with suspected infection or inflammation a ferritin cut off level less than 30 ug/L in children and 70 ug/ L in adults be used to determine ID [15].

Several other indices can be used to determine iron status in the body. Serum iron level will be low at less than 50 microgram/dL. Yet serum iron levels can fluctuate throughout the day hence it alone cannot be used for the diagnosis of ID or IDA [4]. The total iron-binding capacity (TIBC), which measures the total amount of iron bound to a carrier protein in serum, will be high in comparison. A transferrin saturation of less than <20% indicates an iron supply deficiency in maintaining normal erythropoiesis [16].

Red cell indices can be used to identify the presence of IDA in the absence of chronic disease. These include 1. presence of reduced mean cell haemoglobin (MCH), which signifies hypochromia (less than 27pg), 2. reduced mean cell volume (MCV), which indicates microcytosis (less than 75fl), and 3. reduced mean corpuscular haemoglobin concentration (MCHC) (less than 33.4 g/dl) which signify an increased percentage of hypochromic cells. Whilst each of the above-mentioned red cell indices can be low in ID, each parameter can vary due to multitude of reasons (such as sample temperature, storage times, chronic disease states) [4, 17]. Hence red cell indices alone cannot be relied upon to diagnose ID. Furthermore, there still remains the possibility of ID even when red cell indices are normal, as it does not rule out IDA.

The use of soluble transferrin receptor (sTfR) can be used to differentiate between anaemia due to ID and due to chronic disease. sTfR is not influenced by the presence of inflammation, hence it's a more reliable test [17]. sTfR level is inversely related to the iron status. Hence, high levels of sTfR is seen in ID while decreased levels of sTfR may represent adequate levels of iron or anaemia due to chronic disease [18].

Additionally, to eliminate the presence of hemoglobinopathy, haemoglobin electrophoresis must be

Table 4. Comparison of guidelines and limited resource setting practice.

Category	British Society of Gastroenterology (BSG) [10]	American Gastroenterological Association (AGA) [11]		
Diagnosis	Serum ferritin: • <15 ng/mL = IDA • <45 ng/mL = GI tests	Serum ferritin: • <45 ng/mL = iron deficiency		
	If false positive = Transferrin saturation			
Clinical assessment	 Urinalysis or urine microscopy Coeliac serology if appropriate Bidirectional endoscopy in men and postmenopausal women with newly diagnosed IDA. Reserves colonoscopy (LGIE) in addition to UGIE in premenopausal women for patients with risk factors for colon malignancy 	 Bidirectional endoscopy all asymptomatic men and postmenopausal women Bidirectional endoscopy over IRT in asymptomatic pre- menopausal women IDA Helicobacter pylori & celiac tests in patients with IDA without other identifiable aetiology (no routine gastric biopsies) 		
Special assessment	CT colonography if not suitable for colonoscopy	-		
Negative endoscopy	Try IRT If recurrent IDA / no response to IRT = capsule endoscopy Alternatives: CT/MR enterography	Try IRT If failure with a trial of IRT = capsule endoscopy Also, non-invasive testing for <i>Helicobacter pylori</i> and treat		
Management	Don't defer IRT pending testing	Don't defer IRT pending testing		
Treatment	Start: 1 iron tablet/day Adjust if not tolerated to 1 iron tablet/ every other day or IV iron If symptomatic IDA: Consider limited blood transfusion	 Start: 150–200 mg iron/day If no response – assess non-adherence, malabsorption or ongoing blood loss exceeding iron intake Consider IV iron if needed (impaired absorption due to prior gastric surgery, with inflammatory bowel disease or chronic kidney disease, 		
		or in whom blood loss exceeds the ability to replete iron orally)		
Monitoring	First 4 weeks for a Hb response to oral IRT Continue treatment 3 months post – Hb normalisation	First 4 weeks for a Hb response to oral IRT		
Follow up	Monitor blood count every 6 months to detect recurrent IDA	-		

IDA - iron deficiency anaemia; IRT - iron replacement therapy; UGIE - upper gastrointestinal endoscopy; LGIE - lower gastrointestinal endoscopy.

performed in patients from appropriate ethnic and geographical backgrounds. High-performance liquid chromatography (HPLC) will help exclude this entity. Presence of menorrhagia in a person who menstruates, remains an under recognised cause of IDA. Hence it is advisable to screen any person who menstruates with IDA for underlying menorrhagia and seek early input from Gynaecology specialist [16].

The absence of stainable iron on bone marrow biopsy remains the gold standard for IDA [5, 11]. Whilst it is invasive and uncomfortable for the patient, it is not affected by infection or inflammation. Hence, it has high specificity and must be reserved for those difficult-to-diagnose cases.

An excellent response (Hb rise ≥ 10 g/L within a 2week timeframe) to IRT in anaemic patients is highly suggestive of absolute ID, even if the results of iron studies are equivocal. This therapeutic trial can be helpful in a resource-limited setting where iron studies are not freely available [10, 13].

Strategies to avoid this mistake

It is imperative that clinicians do not use Hb alone to screen for ID or IDA. A standardised laboratory panel, including iron studies and red cell indices, must be implemented to complete ID or IDA evaluation. Furthermore, clinicians can consider performing haemoglobin electrophoresis in patients from high-risk ethnic or geographic backgrounds for hemoglobinopathies. It is paramount that healthcare professionals remain updated and educated on interpreting laboratory results in the context of the patient's clinical presentation.

Mistake 3: lack of Helicobacter pylori screening

Helicobacter pylori infection is not an uncommon common cause of IDA, particularly in developing countries. It can lead to chronic atrophic gastritis and impaired iron absorption [19]. Failure to screen for and treat *Helicobacter pylori* can perpetuate IDA despite adequate iron replacement therapy [10, 11].

Using non-invasive testing methods for *Helicobacter pylori* is encouraged as they have high diagnostic accuracy and carry low cost. Urea breath test C13 has been reported to have the best sensitivity at detecting *Helicobacter pylori* [20]. It also benefits from being a safe and cost-effective test [21]. Recent guidelines hence recommend non-invasive testing (urea breath test or stool antigen test) for *Helicobacter pylori* in those patients with unexplained IDA [20, 21].

Timely intervention for patients who test positive for *Helicobacter pylori* is advised to enhance iron absorption and avert potential complications.

Strategies to avoid this mistake

Establish regular screening for *Helicobacter pylori* in individuals with IDA, particularly in areas with high prevalence of *Helicobacter pylori* or those with unexplained IDA. It is also advisable to utilise non-invasive testing techniques such as urea breath test or stool antigen test as they are safe, cost-effective tests with high diagnostic accuracy.

Mistake 4: inconsistent guidelines follow-up

Once IDA is diagnosed, the physician should explore the underlying aetiology effectively. Clinicians must know the different patient populations presenting with IDA and how best to investigate them. Notably, blood loss from the gastrointestinal tract remains the most common cause of IDA in adult men and postmenopausal females [13, 22]. Hence, upper and lower gastrointestinal evaluation should be conducted in adult males and post-menopausal females with newly diagnosed IDA.

The recommendation for bidirectional endoscopy (upper gastrointestinal endoscopy and colonoscopy) in adult males and postmenopausal females with IDA is widely accepted by most guidelines [10, 11]. The same cannot be said for premenopausal women with IDA, as there remains much variation in guidance relevant to this group of patients [10, 11, 23, 24]. The British Society of Gastroenterology (BSG) reserves gastroscopy and colonoscopy in premenopausal women with risk factors for colon malignancy [10]. These risk factors include 1. the presence of a strong family history (two affected first-degree relatives or one first-degree relative before the age of 50 years) or 2. persistent IDA after iron supplementation and correction of potential losses [10].

In those who are suspected of having coeliac disease, duodenal biopsies should be performed during UGIE to confirm the aetiology [10, 11, 25]. The recent American Gastroenterological Association (AGA), on the other hand, advocates for bidirectional endoscopy use for investigating asymptomatic premenopausal women over IRT only [11, 25].

Further evaluation for IDA after bi-directional endoscopy of acceptable quality is not warranted unless there is a poor response to iron replacement therapy or recurrent IDA. Hence, poor adherence to appropriate guidelines may lead to unnecessary procedures or overlooked diagnoses. Ultimately, it results in suboptimal care of the patient.

Strategies to avoid this mistake

Consistent revision and distribution of guidelines for investigating IDA should be done at a locoregional

level, thus allowing for a tailored approach to specific populations. Using decision-support tools or algorithms to guide appropriate endoscopic evaluation based on patient characteristics and risk factors will allow for individualised treatment of IDA. Fostering multidisciplinary collaboration between gastroenterologists, gynaecologists, and primary care providers will also ensure consistent management approaches.

Mistake 5: delay in small bowel evaluation when indicated

In cases of persistent or recurrent IDA despite adequate iron replacement therapy and negative bidirectional endoscopy, further evaluation of the small bowel may be warranted [10, 11, 26, 27].

Video capsule endoscopy (VCE) is the preferred test for examining the small bowel in IDA because it can detect mucosal lesions and has a lower complication rate [10]. However, it is noteworthy that most of the lesions detected by VCE are reachable by UGIE, thus making the case once more for a repeat UGIE in the first instance in those who have failed adequate iron replacement therapy for IDA before opting for VCE [10]. Push or balloon enteroscopy must be considered once the lesions are identified on VCE for tissue biopsy retrieval [28]. However, enteroscopy is costly, requiring expertise, and often warrants both antegrade and retrograde routes to be employed [29]. The diagnostic yield of push enteroscopy broadly varies between 3-78% [30, 31]. The diagnostic yield of balloon enteroscopy is around 60–90% [32–34]. Delaying specialised investigations, such as VCE or enteroscopy, can prolong the diagnostic odyssey and delay appropriate treatment [28–34]. After a negative VCE of acceptable quality, it is recommended that further gastrointestinal investigations be considered only if there is refractory IDA after IRT [10, 11].

It is also recommended that long-term IRT be an appropriate strategy when the cause of recurrent IDA is unknown or irreversible. Other investigations, such as mesenteric angiography, are of limited value in evaluating IDA without overt bleeding. However, it can rarely detect vascular malformations. There is no place for labelled red blood cell scans or Meckel scans to evaluate IDA. If endoscopic procedures are contraindicated or unavailable, imaging techniques, such as CT or MR enterography, should be exploited [35, 36].

Strategies to avoid this mistake

Establishing clear criteria and protocols for referring patients to specialised centres for assessment of small bowel evaluation will facilitate early diagnosis and treatment. Clinicians must ensure timely access to advanced diagnostic modalities, such as VCE and enteroscopy. Above all, in managing such cases, foster collaboration between gastroenterologists and radiologists to leverage complementary imaging techniques, such as CT or MR enterography, when endoscopic procedures are contraindicated or unavailable.

Mistake 6: inadequate patient education

Effective management of IDA requires active patient participation and adherence to treatment [37, 38]. Inadequate patient education can lead to misunderstandings, poor compliance with treatment, and, ultimately, suboptimal treatment outcomes. Patients should receive comprehensive information about IDA, including the importance of iron supplementation, proper timing and administration, potential interactions with other medications or dietary components, and the need for long-term monitoring.

Hepcidin, the primary regulator of iron homeostasis in the human body, follows a circadian rhythm. Research has demonstrated that the circadian increase in plasma hepcidin can be further augmented by a morning dose of iron [39]. Hence, morning dose of oral iron leads to poor absorption of an afternoon or evening dose of oral iron. Therefore, a single dose of oral iron in the morning is preferred. Additionally, oral iron should not be taken with food, mainly calcium-containing foods, calcium tablets, high-fibre foods, tea, coffee, or eggs, as phytates, phosphates, and tannates in food may bind and thus impair iron absorption [37, 38]. When taken with meals, there is up to a 40% reduction in iron absorption [12]. Therefore, iron replacement should be taken between meals. However, when taken with ascorbic acid, the bioavailability of dietary iron is increased. Iron replacement therapy should also be taken well apart from antacid medication (two hours before or four hours after, if possible) [40].

Furthermore, certain common medications, such as proton pump inhibitors (PPI), can result in gastric acid hyposecretion, reducing the absorption of dietary iron or iron tablets [41]. To counteract this reduction in iron absorption, an adequate time gap must be maintained between PPI and iron tablet ingestion.

Strategies to avoid this mistake

Health services should develop standardised educational materials regarding dietary intake of iron and strategies to improve absorption (e.g. brochures, videos) in local languages and at appropriate literacy levels. There should be transparent communication and discussion between the clinician and the patient regarding worries, ideas and misconceptions at clinical encounters. It is also essential to engage relatives of caregivers in the education process when appropriate.

Mistake 7: using high-dose oral replacement therapy

The aim of treatment in IDA consists of two parts: 1. correction of haemoglobin, improving the red cell indices and 2. replenishment of the iron stores. Once the underlying cause is identified, treatment for iron loss and anaemia should be instituted. Correcting anaemia and ID will help to improve symptoms, quality of life and the patient's long-term prognosis. Oral IRT is the first-line treatment for IDA. A daily dose of 150–200 mg of elemental iron is recommended [25].

There are many formulations of oral IRT. These include ferrous sulphate, ferrous sulphate exsiccated, ferrous gluconate, and ferrous fumarate. The commonly used formulation is ferrous sulphate, available in tablet form. Traditionally, oral IRT has been prescribed in high doses, often twice daily. However, current guidelines recommend lower daily or every-other-day dosing of oral iron preparations, as this approach is better tolerated and promotes better adherence to treatment [36, 37]. High-dose regimens result in gastrointestinal side effects, such as constipation, abdominal discomfort, and nausea, which ultimately reduces treatment efficacy and adherence.

Strategies to avoid this mistake

Clinicians should implement guidelines for appropriate dosing and administration of oral iron preparations, with preference for once-daily or every-other-day regimens. Patients should also be educated on adhering to the prescribed dosing schedule. Patients must be encouraged to report any side effects promptly. In instances of intolerance or non-adherence to treatment, alternative formulations or routes of admission of iron should be explored.

Mistake 8: over-reliance on blood transfusions

In severe, symptomatic IDA, early blood transfusions may be necessary. Furthermore, blood transfusions should be used judiciously and only in selected cases [10]. Overreliance on transfusions can result in potential complications, such as transfusion reactions, transfusion-transmitted infections, and increased healthcare costs. Moreover, transfusions may mask the underlying cause of IDA, delaying appropriate diagnostic workup and management.

Therefore, blood transfusions to correct haemoglobin levels should be used carefully only in selected situations. Limited packed red cell transfusion is required to treat symptomatic IDA [10, 11]. It is reasonable to achieve a target Hb of 7–8 g/dL if the patient is symptomatic and the initial Hb is less than this level. In such cases, IRT remains essential post-transfusion.

Strategies to avoid this mistake

It is essential to establish clear guidelines, protocols and criteria for the use of blood transfusions in the management of IDA. Oral IRT should be implemented as the primary treatment modality, thus reserving blood transfusions for those severe and symptomatic cases. Clinicians must ensure that timely investigations are done to identify the underlying cause of IDA, even after transfusion.

Mistake 9: failure to consider parenteral iron therapy

In certain situations, such as malabsorption syndromes, intolerance to oral IRT, or the need for rapid iron repletion, parenteral IRT may be indicated [11, 13, 22]. Failure to consider this option promptly can prolong the patient's anaemic state and associated symptoms, impacting their quality of life.

For those unable to tolerate or absorb oral preparations, parenteral IRT is the next appropriate choice. However, this consideration should be early if oral IRT is judged unlikely to be effective and the correction of IDA is particularly urgent [10]. Parenteral IRT can also be used in those who have undergone gastric and small bowel surgeries such as gastrectomy, gastrojejunostomy or bariatric surgery [11, 13]. Commonly, parenteral IRT is also given in patients with unresolved bleeding, inflammatory bowel disease, malabsorptive patients with coeliac disease and chronic kidney failure with anaemia [13, 22, 42].

Several preparations are available: iron sucrose, dextran, ferumoxytol, sodium ferric gluconate, and ferric carboxymaltose. Of these preparations, the highest bioavailability is with iron sucrose at 20 mg/ml, followed by ferumoxytol at 30 mg/ml [42]. Additionally, patients report mild side effects such as headache and nausea with iron sucrose, whilst anaphylaxis was reported with iron dextran [43]. Severe allergic reactions may defer intravenous iron use by providers with some of the formulations. There is reduced association of severe allergic reaction with ferric carboxymaltose and hence may defer the need for a test dose and allow faster infusions [10, 44].

The dose of parenteral iron may be calculated using the original Ganzoni formula [45]. Modifications using a lower target Hb level (130 g/L) have also been used [10]. The total dose replacement (TDR) provides more rapid replenishment of body iron. Iron sucrose requires multiple infusions because the maximum dose per administration is 200 mg. For iron dextran and ferric carboxymaltose the maximum single dose for TDR in 20 mg/kg [10].

Strategies to avoid this mistake

It is necessary to educate healthcare professionals on the appropriate use and indications for parenteral IRT. Patient factors and the urgency of iron repletion must be considered when determining the appropriateness of the type of IRT. Hence, clinicians should adhere to guidelines and protocols, especially when commencing parental IRT. Clinicians should also ensure access to different parenteral iron formulations and adequately trained personnel to administer parenteral IRT safely.

Mistake 10: lack of long-term monitoring

Following the correction of haemoglobin levels and replenishment of iron stores, patients should undergo long-term monitoring to detect recurrence or persistent IDA [10, 46]. The patient must receive adequate knowledge and education regarding IDA, treatment, monitoring, and follow-up. Neglecting this crucial step can lead to missed opportunities for intervention and perpetuation of the patient's anaemic state.

Usually, response to both oral and parental IRT is evident within the first month [10, 13]. Reticulocytosis is noted to occur within 7-10 days, followed by normalisation of haemoglobin within 1 month and resolution of ID in 3 months with oral iron therapy [47]. Initially, complete blood count (CBC) can be checked monthly to monitor the progress of haemoglobin and red cell indices with treatment. Following the correction of anaemia, the patient can be reviewed every three months in the first year in the outpatient setting. Treatment should be continued for around three months after normalising the Hb level to ensure adequate repletion of the marrow iron stores [10]. After restoring Hb and iron stores with IRT, the CBC is recommended to be monitored periodically (every six months initially) to detect recurrent IDA [10].

Further treatment with oral iron tablets can be commenced if haemoglobin or red cell indices are low [10, 11]. However, small bowel imaging and Helicobacter pylori testing should be done if there is no improvement in haemoglobin or red cell indices despite oral or parenteral iron. Additionally, if not already performed, repeat UGIE can be done with duodenal biopsies taken to rule out coeliac disease.

Strategies to avoid this mistake

It is essential to implement a standardised follow-up protocol, including regular blood testing and clinical evaluations, that can be adhered to in the outpatient setting. Patients must be informed of the significance of treatment adherence by attending regular followup appointments. Furthermore, healthcare providers must be notified of recurring symptoms or adverse events. It is pivotal that cooperation between primary care providers and specialists is encouraged to ensure continuity of care.

Conclusion

The effective management of IDA necessitates a comprehensive and multidisciplinary approach. By recognising and addressing the common mistakes highlighted in this narrative review, healthcare professionals can improve patient outcomes, minimise complications, and enhance the overall quality of care. Continuous education, adherence to evidencebased guidelines, and effective communication with patients and their caregivers are crucial elements for successful IDA management. Prompt action in addressing these errors can improve patient outcomes in the management of IDA.

Disclosure statement

No potential conflict of interest was reported by the author(s).

Author's contributions

MAN conceptualised the paper. HJ drafted the manuscript. MAN, AW and AP were substantially involved in revising the manuscript. All authors checked the final manuscript before submission.

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