The Art and Science of Infusion Nursing

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Chronic Anemia and the Role of the Infusion Therapy Nurse

ABSTRACT

Chronic anemia develops over a course of weeks to months and is usually mild to moderate in nature. It is important to understand the etiology of the reduced number of circulating red blood cells to treat the anemia appropriately. Diagnosis is dependent on patient history and laboratory findings, such as complete blood counts, iron studies, a peripheral smear, and occasionally, a bone marrow biopsy. Treatment modalities frequently administered by infusion therapy nurses include treatment of the underlying chronic disease, replacement of deficiencies (iron, vitamin B12, folate, or erythropoietin), or transfusion of red blood cells. Infusion therapy nurses play a vital role in the assessment and delivery of medication therapy to patients with chronic anemia.

Key words: anemia, chronic, ESA, iron, infusion

Anemia is defined as a reduction below normal of the number of erythrocytes, quantity of hemoglobin (Hgb), or volume of red cells in the blood. Anemia occurs by 1 or more of 3 mechanisms: blood loss, decreased production of erythrocytes, and increased destruction of erythrocytes.[1] Chronic anemias result primarily from a decreased production of erythrocytes, with or without accompanying blood loss or increased destruction of erythrocytes. They encompass a broad range of anemias that develop acutely or chronically and may persist for several months if left untreated. Formally, anemia of chronic disease is a specific category of anemia associated with underlying infection, inflammation, or malignancy. For the purpose of this publication, the term chronic anemia will be used to encompass anemia of chronic disease; anemia of chronic kidney disease (CKD); anemia of cancer; and vitamin B12, folate, and iron deficiency anemias. On diagnosis, the infusion therapy nurse may be involved with treatment strategies ranging from treatment of an underlying chronic disease, replacement of deficiencies (iron, vitamin B12, folate, or erythropoietin), or transfusion of red blood cells (RBCs) in symptomatic patients or those with cardiovascular instability. Normal erythropoiesis, clinical and laboratory findings, etiology, medications used for treatment of chronic anemia, and the role of the infusion therapy nurse in the delivery of care to patients will be reviewed.

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ERYTHROPOIESIS

RBCs are unable to divide into new cells and live on average about 120 days. To replenish themselves, aged and ruptured cells must be replaced by new cells. The body replaces approximately 1% of its total RBCs on a daily basis. This process is called erythropoiesis. When the body has an increased need for RBC production secondary to anemia, the kidneys sense tissue hypoxia and increase secretion of the hormone erythropoietin. Erythropoietin travels to the bone marrow by means of the circulatory system and stimulates differentiation of hematopoietic stem cells. As the differentiated cells, known as erythroblasts, mature, they fill with Hgb and release their nucleus. Vitamin B12 and folate are critical to DNA synthesis within the nucleus.[2] The release of the nucleus causes the cell to collapse on itself, creating a disk-like formation. This formation allows for increased cell surface so Hgb can attract more oxygen molecules. Hgb is responsible for the red color of the RBC. The

mature RBC is then released into the bloodstream. Near the end of RBC life, macrophages engulf the RBC, and iron is recycled for a new generation of RBCs. Large amounts of iron, which is stored in ferritin, are required for Hgb production.

CLINICAL AND LABORATORY FINDINGS

A thorough patient history is critical to the evaluation and management of patients with anemia. Classic presentation may include pale appearance, fatigue, weakness, anorexia, insomnia, shortness of breath, dizziness, headache, decreased mental and physical performance, pica, and palpitations. However, it is important to recognize that not all patients with anemia present with these classic symptoms; an underlying chronic illness may frequently mask the symptoms of anemia.

The provider's physical examination of the patient with suspected anemia should include inspection of general appearance, vital signs, assessment for skin pallor, a cardiac exam, and evaluation for splenomegaly. Laboratory studies remain the mainstay of diagnosis and determining etiology of the anemia. This includes a complete blood count (CBC), iron studies, and a peripheral smear. Important components of the CBC are Hgb, hematocrit (Hct), reticulocytes, mean corpuscular volume (MCV), and RBC distribution. Leukocytes and platelets are also a part of the CBC and are less relevant to the diagnosis of anemia but may have utility in ruling out other hematological causes such as leukemia. Suspicion of iron deficiency anemia warrants iron studies (ie, serum iron, ferritin, total iron binding capacity [TIBC], and transferrin saturation [Tsat]). Vitamin B12 and folate levels are important for diagnosis and differentiation of megaloblastic anemias. Erythropoietin levels should be considered with a diagnosis of anemia of chronic disease, anemia of CKD, and anemia of cancer.

Initial suspicion of anemia is based on a low Hgb or Hct. Anemia is generally classified by cell size. RBC indices will give the MCV, which is the size, thickness, and volume of the RBC. Microcytic anemias are those with low MCV. These cells are also hypochromic, indicating that they have a low Hgb concentration. These anemias are generally associated with iron deficiency and chronic inflammation.[3] Macrocytic anemias are those with elevated MCV and are generally associated with vitamin B12 and folic acid deficiency. Last is normocytic anemia, which presents with normal-sized RBCs. This anemia can be found in renal insufficiency and bone marrow infiltration.[3]

Serum ferritin, Tsat, and TIBC can be used individually or in conjunction to aid in diagnosing anemia. Serum ferritin measures iron provisions in the body. This is important in early diagnoses of iron deficiency anemia. Tsat measures the absorption and transportation of dietary iron. Transferrin is a protein that binds iron. TIBC measures the blood's capacity to bind iron and transferrin. Ferritin and transferrin levels are used to calculate the TIBC. Figure 1 illustrates a classification algorithm for the chronic anemias.

ETIOLOGY

The 3 major mechanisms of anemia are:

1. Blood loss (acute or chronic)
2. Decreased RBC production
   a. Defect in stem cells (aplastic anemia, pure red cell aplasia)
   b. Defect in heme production (iron deficiency, thalassemias)
   c. Defective DNA production (vitamin B12 and folic acid deficiencies)
   d. Destruction of bone marrow (solid tumor metastasis, inherent marrow malignancy, or other marrow invasion)
3. Increased red cell destruction
   a. Caused by external factors
   b. Caused by hereditary internal factors
   c. Caused by an acquired defect[1]

The chronic anemias generally develop as a result of an underlying cause that decreases RBC production and may or may not have an associated blood loss or increase in red cell destruction.

Anemia of Chronic Disease

The pale appearance of patients with chronic infections such as tuberculosis was recognized hundreds of years ago. The pallor was eventually attributed to an anemia of chronic disease, which occurs when an inflammatory process causes erythrocytes to have a decreased life span, as well as a poor bone marrow erythropoiesis response. More recently, the designation anemia of inflammation has been recommended because it is thought to be more reflective of the pathophysiology of this anemia, which also includes anemia of critical illness, a condition that presents similarly to anemia of chronic disease but develops acutely within days of the onset of illness.[5]

For the purpose of this article, anemia of chronic disease will be used. This anemia is thought to be caused by inhibitory effects of inflammatory cytokines released in response to the effects of an underlying chronic disease on erythropoiesis.[6] Erythropoietin production is less than expected, and the inflammatory process also induces a relative resistance to the effects of erythropoietin. Iron availability is also compromised

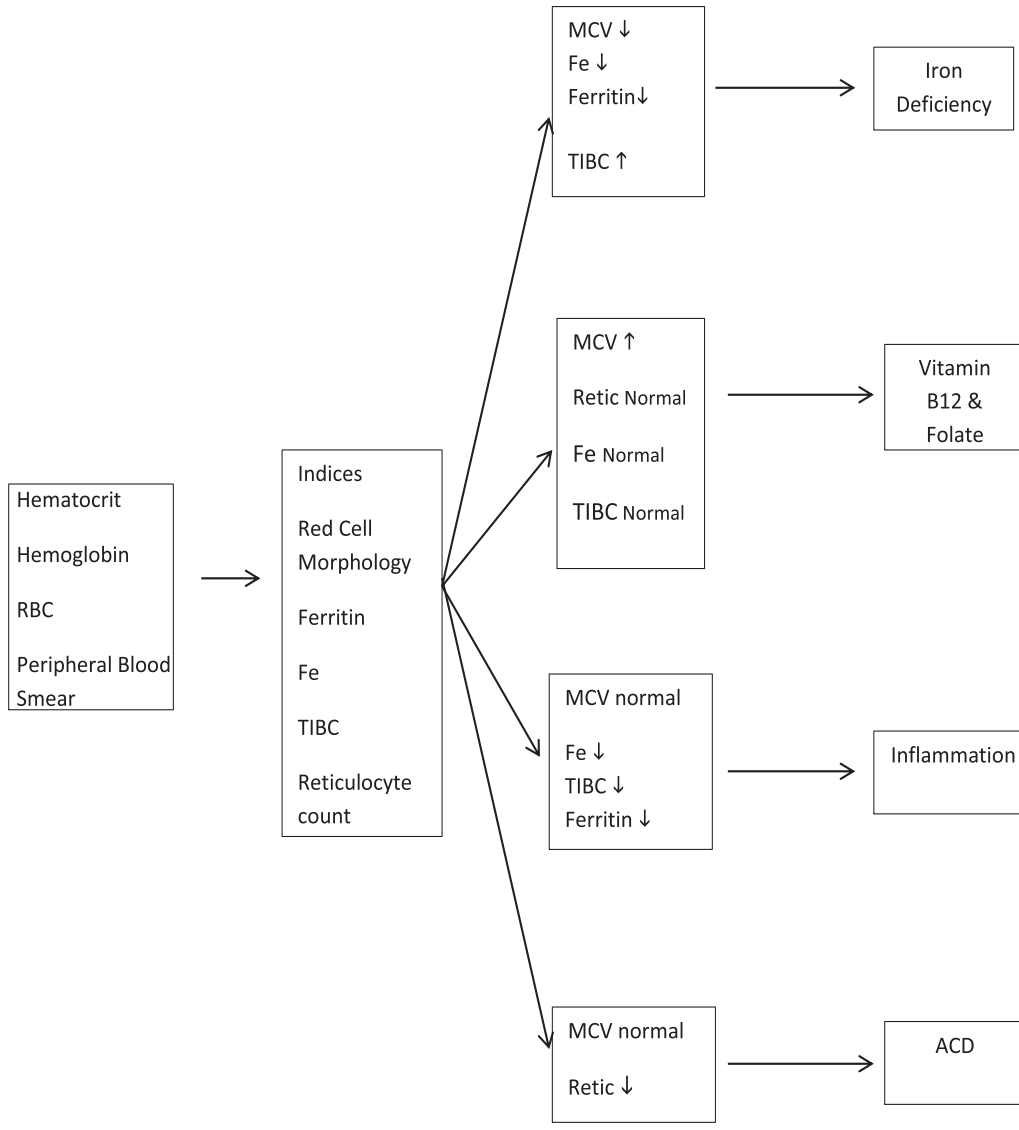

Figure 1 Classification of anemia by laboratory findings. Data from Young and Koda-Kimble (1995).[4] *Abbreviations: RBC, red blood cell; Fe, iron; TIBC, total iron binding capacity; MVC, mean corpuscular volume; Retic, reticulocyte count; ACD, anemia of chronic disease.*

through interleukin-6 inducing the iron regulatory hormone, hepcidin.[7] In turn, hepcidin inhibits the release of iron stores from the macrophages, resulting in hypoferremia.[8] Common conditions associated with anemia of chronic disease may include infections (HIV, tuberculosis, malaria, osteomyelitis, chronic abscess, and sepsis), inflammation (rheumatoid arthritis and inflammatory bowel disease), and malignancy (carcinomas, myeloma, and lymphomas).

Anemia of Cancer

Anemia of cancer may be caused by the underlying disease in cancers that cause damage to the bone marrow, as with myeloma and some lymphomas. It also can result from the hematologic toxicity of the treatment modalities of radiation and/or chemotherapy. The same mechanism of cytokine release found in anemia of chronic disease is thought to be responsible for the suppression and resistance to erythropoietin.

Anemia of CKD

This anemia is usually moderate to severe in nature and is primarily due to a reduction of erythropoietin production from the kidneys. Patients on hemodialysis may develop an iron deficiency, which is important to consider when treating this anemia.

Iron Deficiency

Iron deficiency is the most common cause of anemia. The primary cause of iron deficiency in adults is blood loss. Other causes include increased demand for iron or

decreased iron intake or absorption. The reticuloendothelial system is responsible for recycling iron from destroyed RBCs to be used for the next generation of RBCs. In cases of blood loss, the iron is lost, and body iron stores may be unable to meet the demand of increased erythropoiesis.

Megaloblastic Anemias

These anemias result from impairment of DNA synthesis, which results in ineffective erythropoiesis. Clinically, they are difficult to distinguish without testing the levels of both vitamin B12 and folic acid.

Vitamin B12 deficiency

A megaloblastic anemia from vitamin B12 deficiency results from inadequate dietary intake or a defective absorption and transport chain for B12. The latter is known as pernicious anemia when caused by autoantibodies against parietal cells that decrease production of intrinsic factor, resulting in inadequate uptake of vitamin B12. Neurologic deficits may present with this anemia.

Folic acid (folate) deficiency

Folic acid deficiency can result in megaloblastic anemia, which occurs from inadequate dietary intake, increased folic acid requirements (pregnancy, hemodialysis, hemolytic anemia, and exfoliative skin conditions), or alcoholism. Folic acid deficiency is not associated with pernicious anemia or neurologic deficits.

TREATMENT

Treatment options for the chronic anemias discussed in this article include increased dietary intake of vitamin-rich foods, supplementation of vitamins or iron, erythrocyte-stimulating agents (ESAs), transfusions, or treatment of an underlying disease that caused the anemia.

Treatment of the Underlying Disease

Anemia of chronic disease and some anemias of cancer, especially those that have an impact on the bone marrow, tend to resolve on treatment of the underlying chronic disease or cancer. Therefore, diagnosis and treatment of the underlying condition is the first step in treatment of these anemias. In some cases, as with cancer, the anemia may worsen initially, but it improves when the underlying bone marrow disease and marrow regeneration are resolved. When treatment of the underlying condition is not effective, symptomatic patients may require therapy with ESAs, iron therapy, or transfusions.

Replacement of Vitamin Deficiencies (Vitamin B12 and Folic Acid)

Vitamin B12 anemia requires treatment with monthly intramuscular or deep subcutaneous injections of 1 mg (1000 mcg) of vitamin B12 daily for 7 days, followed by weekly injections for 4 weeks, then each month.[9] Conversion to oral vitamin B12 therapy of 1 mg by mouth daily may be appropriate once vitamin B12 levels return to normal.

In patients with an underlying malabsorption (eg, pernicious anemia or gastric bypass), oral therapy is not indicated, and continuation of monthly parenteral B12 injections is required. Treatment is lifelong unless the underlying cause of the deficiency is eliminated. Sublingual and nasal routes of treatment are available; however, they have not been studied adequately, and the available formulations are expensive.[9] B12 parenteral and oral therapy is relatively devoid of side effects.

Folic acid deficiency anemia treatment starts by increasing consumption of foods rich in dietary folic acid (eg, green leafy vegetables, beans, fruits, nuts, wheat germ, liver). Replacement with oral folic acid in doses of 1 to 5 mg daily is used to treat the underlying deficiency, with 1 mg being the most common dose. Return to normal folate levels occurs in approximately 6 to 8 weeks, at which time replacement can be stopped. Folic acid is well tolerated.

ESAs

ESAs are the primary therapy for anemia of CKD and anemia of cancer. ESAs bind to erythropoietin receptors on bone marrow cells to stimulate RBC production through division and differentiation of erythroid progenitors. Baseline iron studies are important, especially in patients undergoing hemodialysis, to determine the need for adjuvant iron therapy. The use of ESAs in the setting of low iron limits their effectiveness in producing RBCs. Epoetin alpha and darbepoetin alfa are the 2 ESAs available in the United States.

ESA use for anemia of cancer

The ESAs are approved for use in patients with metastatic (incurable), nonmyeloid malignancies when Hgb is less than 10 g/dL and if a minimum of 2 months of chemotherapy is planned. Table 1 illustrates the treatment guidelines for ESAs in patients with anemia of chemotherapy. When used for anemia of cancer, health care providers must be aware that both products have a US Food and Drug Administration (FDA)-mandated Risk Evaluation and Mitigation Strategy (REMS) program to educate patients and health care providers about the known risks of death and cardiovascular events. Prescribers and hospitals must enroll in and

TABLE 1

Dosing Guidelines for Erythropoietis-Stimulating Agents Anemia of Chemotherapy[10,11]

	Epoetin Alfa		Darbepoetin Alfa	
When to start	Hgb < 10 g/dL if at least 2 additional months of planned chemotherapy			
Initial dose	150 units/kg 3×/wk	40 000 units weekly	2.25 mcg/kg weekly	500 mcg every 3 weeks
Dose increase	300 units/kg if no reduction in transfusions or rise in Hgb after 4 weeks	60 000 units if increase in Hgb < 1 g/dL after 4 weeks	4.5 mcg/kg every week if increase in Hgb < 1 g/dL after 6 weeks	NA
Dose reduction	Decrease by 25% when Hgb > 1 g/dL in 2-week period or when transfusions avoided		Decrease by 40% when Hgb > 1 g/dL in 2-week period or when transfusions avoided	
Dose holding	If Hgb exceeds a level required to avoid transfusion, restart at 25% dose reduction when Hgb reaches level where transfusions may be required		If Hgb exceeds a level required to avoid transfusion, restart at 40% dose reduction when Hgb reaches level where transfusions may be required	
Discontinuation	After completion of chemotherapy course If no response after 8 weeks			

Abbreviations: Hgb, hemoglobin; NA, not applicable.

comply with the ESA APPRISE Oncology Program to prescribe and/or dispense the ESAs to patients with cancer.[10,11] There are concerns of increased mortality and/or increased risk of tumor progression or recurrence. For these reasons ESAs are not recommended in cancer patients who are not receiving chemotherapy or in patients treated with curative intent.[12]

ESA use in anemia of CKD

The guidelines for use of ESAs in anemia of CKD differ for patients on dialysis compared with those not receiving dialysis.

ESA use for all patients with anemia of CKD:

- When initiating or adjusting therapy, monitor Hgb levels at least weekly until they are stable, then monitor at least monthly. When adjusting therapy, consider Hgb rate of rise, rate of decline, ESA responsiveness, and Hgb variability. A single Hgb excursion may not require a dosing change.
- Do not increase the dose more frequently than once every 4 weeks. Decreases in dose can occur more frequently. Avoid frequent dose adjustments.
- If the Hgb rises rapidly (eg, more than 1 g/dL in any 2-week period), reduce the dose by 25% or more as needed to reduce rapid responses.
- For patients who do not respond adequately, if the Hgb has not increased by more than 1 g/dL after 4 weeks of therapy, increase the dose by 25%.
- For patients who do not respond adequately over a 12-week escalation period, increasing the dose further is unlikely to improve response and may increase risks. Use the lowest dose that will maintain a Hgb level sufficient to reduce the need for RBC transfusions. Evaluate other causes of anemia. Discontinue if responsiveness does not improve.

For patients with CKD on dialysis:

- Initiate treatment when the Hgb level is less than 10 g/dL.
- If the Hgb level approaches or exceeds 11 g/dL, reduce or interrupt the dose.
- Recommended starting doses:
  - Darbepoetin alfa 0.45 mcg/kg intravenously or subcutaneously as a weekly injection or 0.75 mcg/kg once every 2 weeks, as appropriate
  - Epoetin alfa 50 units/kg to 100 units/kg 3 times a week intravenously or subcutaneously. For pediatric patients, a starting dose of 50 units/kg 3 times a week intravenously or subcutaneously.
  - The intravenous (IV) route is recommended for patients on hemodialysis.

For patients with CKD not on dialysis:

- Consider initiating treatment only when the Hgb level is less than 10 g/dL and the following 2 considerations apply:
  - The rate of Hgb decline indicates the likelihood of requiring an RBC transfusion.
  - Reducing the risk of alloimmunization and/or other RBC transfusion-related risks is a goal.
- If the Hgb level exceeds 10 g/dL, reduce or interrupt the dose and use the lowest dose sufficient to reduce the need for RBC transfusions.
- Recommended starting doses:

- Epoetin alfa 50 units/kg to 100 units/kg 3 times a week intravenously or subcutaneously
- Darbepoetin alfa 0.45 mcg/kg body weight intravenously or subcutaneously given once at 4-week intervals as appropriate[10,11]

Side effects are similar for the 2 ESA products and may include hypertension, headache, tachycardia, nausea, vomiting, hyperkalemia, and diarrhea.

Iron Replacement Therapy

Most cases of iron deficiency can be treated with oral iron therapy 100 to 300 mg/d of elemental iron in divided doses. Oral iron preparations are commercially available over the counter in various salt forms (eg, sulfate, fumarate, gluconate). Care must be taken with dosing because ferrous fumarate 325-mg tablets contain 106 mg of elemental iron, while ferrous sulfate 325-mg tablets contain only 65 mg. Oral iron is best absorbed on an empty stomach because food inhibits absorption. However, some patients may need to take it with food to minimize gastrointestinal side effects. Some preparations are paired with ascorbic acid to enhance absorption or a stool softener to reduce the constipating effects of the iron. These products are often much more expensive, and it is unclear whether the benefits outweigh the cost. Oral iron may cause gastrointestinal discomfort, nausea, constipation, or diarrhea. Patients who do not tolerate oral iron therapy, have malabsorption, or have acute blood loss require IV iron. Additionally, patients receiving ESAs should receive IV iron supplementation because oral iron absorption is not able to meet the acute demand necessary for effective erythropoiesis.

IV iron was originally available only as iron dextran. The high-molecular-weight (MW) iron dextrans have a higher incidence of serious adverse reactions, including anaphylaxis and hypotension, than the low-MW counterpart. For this reason, a clinic in Arizona removed the high-MW iron dextran from its formulary in 2008. More recently, several new products have become available with a lower incidence of serious adverse events and do not require a test dose as specified in the product labeling. These include sodium ferric gluconate complex, iron sucrose, ferumoxytol, and ferric carboxymaltose. CKD patients on dialysis are usually treated with smaller doses over several weeks of dialysis sessions, while non–dialysis-dependent CKD patients and non-CKD patients typically receive larger doses over fewer infusion center visits.

Iron dextran and ferric carboxymaltose have the broadest FDA indications for use in iron deficiency in patients for whom oral therapy is unsatisfactory or impossible.[13,14] The FDA-labeled indications for all other available IV iron products are restricted to iron deficiency anemia in CKD. Furthermore, sodium ferric gluconate complex is indicated only in combination with ESA therapy in CKD.

The iron dextran dose is calculated using the equation dose (mL) = 0.0442 (desired Hgb-observed Hgb)· LBW + (0.26 · LBW), where LBW is lean body weight.[13] Iron dextran requires a test dose of 25 mg by slow IV push or in 100 mL of normal saline administered by IV over 15 minutes, followed by 60 minutes of observation. If the patient experiences no chest pain, wheezing, hypotension, or other signs of anaphylaxis, premedication (which is not required) with acetaminophen and diphenhydramine may be given and followed after 30 minutes with the remaining dose of iron dextran. The calculated dose is then administered by IV in at least 250 mL of normal saline infused over 1 to 6 hours.

The selection of IV iron therapy is dependent on diagnosis, iron requirement (total dose versus smaller weekly doses), convenience, and patient accessibility to infusion therapy services. Ferric carboxymaltose has the most patient-friendly dosing as an IV push, which is administered in 2 brief visits, while iron dextran has the benefit of a single total dose infusion (TDI). The use of TDI iron dextran is off-label but widely accepted as the preferred method of administration.[15] The most common side effects of parenteral iron products are phlebitis, dyspnea, hypotension, dizziness, and headache. Serious adverse events include anaphylaxis, pulmonary embolism, circulatory collapse, and death. Iron sucrose and ferric gluconate have been shown to have lower rates of adverse events than iron dextran and ferumoxytol.[16] Table 2 provides a summary of dosing guidelines for the IV iron products available in the United States.

ROLE OF THE INFUSION THERAPY NURSE

When caring for a patient with anemia, the assessment findings provide information necessary to deliver comprehensive care. The assessment begins by reading through the medical history, which can provide information about the underlying disease that has led to the anemia. Once the patient has arrived in the infusion center, a physical assessment is performed. Assess for symptoms of anemia. It is important to remember that these may be due to either the underlying disease or the anemia itself. Some chronic diseases, such as chronic renal failure, may mask the symptoms of anemia.[5] The patient may present with a number of the following symptoms: weakness, fatigue, dizziness, headaches, anorexia, shortness of breath, insomnia, pale skin, exercise intolerance, or palpitations. A common symptom of iron deficiency anemia is pica—craving for ice, clay, or other unusual substances. Providing a safe environment for patients, as well as educating them on how to manage these symptoms, is just as important as administering the iron infusion itself.

TABLE 2

Intravenous Iron Products[13,14,17,18,19]

Drug	Iron Content	Dose in IDA With CKD	Dose in IDA Without CKD
Low-MW iron dextran (INFeD)	50 mg/mL	100 mg IV over 2 minutes weekly or 3 times weekly until TD reached	TD IV in 250 to 1000 mL NS over 1 to 6 hours × 1 dose
Ferumoxytol (Feraheme)	30 mg/mL	510 mg IV in 100 mL NS over at least 15 minutes × 2 doses, 3 to 8 days apart	Not indicated
Iron sucrose (Venofer)	20 mg/mL	100 mg IV push over 2 to 5 minutes or in 100 mL NS over 15 minutes 3 times per week × 10 doses	• 200 mg IV push over 2 to 5 minutes or in 100 mL NS over 15 minutes × 5 doses in a 14-day period • 300 mg IV in 250 mL NS over 1.5 hours every 14 days × 2, then 400 mg IV in 250 mL NS over 2.5 hours × 1, 14 days later • 500 mg IV in 250 mL NS over 3.5 to 4 hours every 14 days × 2
Sodium ferric gluconate complex (Ferrlecit)	12.5 mg/mL	125 mg IV push over 10 minutes or in 100 mL NS over 1 hour, 3 times weekly × 8 doses	Not indicated
Ferric carboxymaltose (Injectafer)	50 mg/mL	15 mg/kg (max 750 mg) IV push (100 mg/min) or in 250 mL NS over 15 minutes weekly × 2 doses	15 mg/kg (max 750 mg) IV push (100 mg/min) or in 250 mL NS over 15 minutes weekly × 2 doses

Abbreviations: IV, intravenous; TD, total dose; NS, normal saline; IDA, iron deficiency anemia; CKD, chronic kidney disease; MW, molecular weight.

Assessing the patient's venous access is crucial before beginning IV therapy. The pH and osmolality of the prescribed iron will guide the nurse in deciding the appropriate vascular access for treatment. Undiluted iron sucrose has a pH of 10.5 to 11 and an osmolarity of 1250 mOsm/L.[18] INS' *Infusion Nursing Standards of Practice* indicates that this should be administered via a central venous access device. The standard states that it is safe to give medications peripherally if the pH is 5 to 9 and the osmolarity is less than 600 mOsm/L.[20] In addition, the nurse needs to access the tolerance of previous treatments. Before starting the treatment, the nurse must evaluate the lab results. With this information collected, the nurse can identify a nursing diagnosis. The nursing diagnosis will depend on the underlying disease and severity of symptoms. Examples of nursing diagnoses include intolerance related to weakness, fatigue, and general malaise.[21] When vitamin B12, folic acid, or iron deficiency is the medical diagnosis, a nursing diagnosis of altered nutrition less than body requirements related to inadequate intake of essential nutrients can be applied. The nursing diagnosis will guide the individual care of the patient.

The nurse must organize the plan of care according to the priority needs of the patient, including safety. It is important for the nurse to involve the patient to ensure that the outcomes are attainable. For additional support, it is recommended that the nurse collaborate with the provider to determine whether ancillary support may be required (eg, dietary consult, physical therapy, home health). Short- and long-term goals should be set with the patient. An example of a short-term goal is that the patient doesn't fall. Long-term goals include the patient making adjustments to the activities of daily living to preserve his or her energy level.

When administering the prescribed treatment, the nurse must be knowledgeable about the potential side effects and assess the patient often. Iron infusions can lead to anaphylactic reactions and a number of other symptoms, including, but not limited to, itching, difficulty breathing, and redness or pain at the infusion site. A thorough understanding of the IV iron product prescribed is important. IV iron supplementation dosing can vary according to indication, as can the preferred route of administration. The use of a test dose, if indicated, as well as monitoring during and for 30 minutes after each dose, is important to ensure safe care.

To promote safe use of the IV iron products, a metropolitan clinic developed preprinted physicians' orders for available IV iron products, including dosing options, routes, rates of administration, test dose (if indicated), and monitoring guidelines. An emergency medication box—containing diphenhydramine, methylprednisolone, hydrocortisone, albuterol inhaler, and epinephrine—is kept at the bedside for the prompt treatment of infusion reactions. In addition, the iron dextran TDI has been associated with delayed reactions of 24 to 48 hours after administration; these may include arthralgia; backache; chills; dizziness; moderate to high fever; headache; malaise; myalgia; nausea; and vomiting. Infusion nurses should educate patients receiving iron dextran TDI to recognize these symptoms and provide a plan for the patient to report any serious side effects.

Continual assessment for fluid overload and signs and symptoms of transfusion reactions are essential when administering blood transfusions. Most organizations have established policies and protocols to follow when a patient experiences a transfusion reaction.

When administering ESA therapy, the Hgb level should be assessed to ensure that it is within range as identified by FDA guidelines. Collaboration with a pharmacist is helpful to ensure the appropriate use of the ESAs. Oncology providers who prescribe ESAs are required to be a part of the REMS program. This program is mandated by the FDA and regulated by the product manufacturer. To prescribe ESAs, providers must complete a 15-minute education program and register.[22,23] ESAs can be effective, but when their use results in an Hgb level greater than 11 g/dL, serious cardiovascular complications can occur.[22] Controlled clinical trials have not demonstrated an improvement in the symptoms of anemia, quality of life, fatigue, or patient well-being with the use of ESAs.

One role of the infusion therapy nurse is to ensure that the patient has been educated about all treatments that are administered, as well as the potential side effects. Educating patients about what they can do to help themselves is as vital as administering the medication. Ensuring patient understanding can be achieved through the teach-back method. Teach-back requires the patient to explain back to the teacher the information that was just taught. This allows for better evaluation of the patient's understanding of education.[24] Instruction on how to organize activities to preserve energy, education on fall precautions, dietary tips, or underlying disease control will create an improved outcome for the patient.

When evaluating treatment outcomes, nurses should provide adequate documentation that includes tolerance to the procedure as well as symptoms the patient may have experienced. This may be important to subsequent treatment visits to reduce patient discomfort or anxiety. Accurate documentation of tolerance can be an effective tool to assist in making plan-of-care adjustments.

Understanding that anemia can be a life-changing diagnosis for patients and collaborating with patients, providers, and allied health professionals to develop an individual, comprehensive care plan is essential to provide safe outcomes for patients.

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