r. Pandya began her presentation on anemia in long-term care with the case of LD, an 88-year-old Caucasian man living in a nursing home who is able to socialize, feed himself, and propel himself in a wheelchair. He weighs 132 pounds (60 kg) and has a history of coronary artery disease, congestive heart failure, mitral regurgitation, hypothyroidism, renal insufficiency, previous transient ischemic attack, anemia, and depression. His medications are ferrous sulfate, furosemide, aspirin, lisinopril, levothyroxine, and mirtazapine. He complains of increased shortness of breath and is noted to have edema and anorexia.

Physical examination and laboratory results for LD are in the box on the right. Based on these findings, Dr. Pandya attributed LD’s symptoms to congestive heart failure and anemia.

According to the World Health Organization (WHO), anemia in women is defined as hemoglobin (Hb) < 12 g/dL and in men as Hb < 13 g/dL. The National Kidney Foundation uses a slightly different cutoff point for men (Hb < 13.5 g/dL).

“Many physicians mistakenly believe that it is normal for older adults to be anemic,” said Dr. Pandya. However, older age by itself is unlikely to cause anemia. Therefore, anemia should always be investigated; it may be a sign of a clinically treatable disease.

Aging may predispose individuals to anemia for several reasons, including decreased hematopoietic reserve, reduced absorption of essential nutrients, decline in glomerular filtration rate (GFR), decline in erythropoietin (EPO) secretion, and relative EPO deficiency. In addition, aging may predispose individuals to inflammatory diseases, cancer, or infections that raise levels of cytokines, which may inhibit erythropoiesis and response to growth factors.

In a theoretical model of anemia, an inflammatory disease can lead to cytokine production, which affects bone marrow and leads to anemia. Anemia causes the individual to be more susceptible to disease. As with excess cytokine production, anemia can cause muscle loss, wasting, increased tendency for frailty, and functional depend-
Anemia of aging may be self-maintained and may aggravate the manifestations of aging,” said Dr. Pandya.

A study by Coleman, which looked at causes of anemia in older adults, found that one-third of cases are due to nutritional deficiencies (vitamins or iron deficiency).\(^4\)

One-third of cases are due to chronic inflammation or chronic kidney disease, and in one-third the cause is unexplained.

The Study of Anemia in Long-term Care (SALT) was a retrospective study in U.S. nursing homes, which examined the prevalence of anemia in long-term care and explored the association between anemia and falls in this population.\(^5\) Among the 564 nursing home residents (70% female, mean age 81 years, mean Hb 11 ± 1.8 g/dL), 56% had anemia, approximately four times the rate in community-dwelling elders. Approximately 50% were receiving treatment the rate in community-dwelling elders.

Anemia was associated with African-American race, diabetes, cancer, chronic kidney disease Stage III with a GFR < 60 mL/min/1.73m\(^2\), gastrointestinal bleeding, kidney disease Stage III with a GFR < 60 mL/min/1.73m\(^2\), approximately 70% with ferrous sulfate.

Approximately 50% were receiving treatment the rate in community-dwelling elders. Approximately 70% with ferrous sulfate.

Anemia was associated with African-American race, diabetes, cancer, chronic kidney disease Stage III with a GFR < 60 mL/min/1.73m\(^2\), gastrointestinal bleeding, and inflammatory disease. Anemia was strongly associated with higher risk of falling in this study.

Chronic kidney disease is an important cause of anemia. A study by Garg et al. looked at the prevalence of chronic kidney disease in long-term care residents.\(^6\) Among 9,931 long-term care residents age 65 and older, 40% had GFR < 60 mL/min/1.73m\(^2\) (using the MDRD study equation).

Correcting anemia in older adults is important because anemia has the potential to lead to serious adverse outcomes. The signs and symptoms of anemia are nonspecific (see Table 1). The Long Term Care RAI User’s Manual for the Minimum Data Set (MDS) Version 2.0 contains useful parameters for identifying patients who should be investigated for anemia. These include changes in cognitive status (B6), change in mood (E3), chronic infections (I2) (e.g., osteomyelitis, H. pylori), decline in ADL function (G9), falls (J4), nutritional problems (K4), skin conditions (M1, M4), weight loss, and anorexia (K3).

“One of the easiest approaches to diagnosing anemia is to look at the red cell morphology,” said Dr. Pandya. A high mean corpuscular volume (MCV) (macrocytic) indicates the cause may be medication related or due to alcohol use, B\(_12\) or folate deficiency, hypothyroidism, or liver disease. If the MCV is low (microcytic), it is necessary to look at iron status and iron stores as reflected in the ferritin level. Low ferritin suggests iron deficiency or chronic disease.

If the ferritin level is normal, the cause of anemia may be chronic kidney disease, anemia of chronic inflammation, thalassemia, or sideroblastic anemia. Normocytic anemia could be caused by chronic kidney disease or other chronic disease conditions.

Laboratory tests for anemia are listed in Table 2. Table 3 illustrates how to differentiate iron deficiency anemia from anemia of chronic disease. In both disorders, the iron level is low. Total iron-binding capacity (TIBC) is low in chronic disease and elevated in long-term care remains inadequate due to several barriers. For example, a consistent definition of anemia in the elderly is lacking. Best practices in the assessment and management of anemia are still uncertain. Data are lacking on the outcomes of anemia treatment in the frail elderly and very little research has been done on underlying causes of anemia in this population.

### Mechanisms and Diagnosis of Anemia

The basic mechanisms underlying anemia are decreased red blood cell production, loss of red blood cells, or decreased survival of red blood cells. Decreased red blood cell production may be caused by nutritional deficiencies, infiltration of the bone marrow by cancer or myelofibrosis, or medications that suppress the bone marrow. Loss of red blood cells may result from undetected bleeding or medications such as nonsteroidal anti-inflammatory drugs (NSAIDs). Reduced red blood cell survival, or hemolysis, can be caused by diseases of the liver or spleen, sickle cell anemia or thalassemia, transfusions, or autoimmunity.

### Table 1. Signs and Symptoms of Anemia

#### Central Nervous System
- Fatigue
- Headache
- Dizziness
- Syncope
- Depression
- Impaired cognitive function

#### Vascular System
- Cold intolerance
- Edema
- Pallor of the skin, mucous membranes, and conjunctivae

#### Cardiovascular System
- Dyspnea
- Tachycardia
- Hypotension
- Systolic ejection murmur
- Palpitations
- Cardiac hypertrophy
- Wide pulse pressure
- Orthostasis

#### Gastrointestinal System
- Anorexia
- Nausea

### Table 2. Laboratory Tests for Anemia

- Red blood cell indices (CBC)
- Serum ferritin
- Serum iron
- Total iron-binding capacity (TIBC)
- Transferrin saturation
- Vitamin B12
- Folate
- Methylmalonic acid
- Homocysteine
- Reticulocyte count
- Sedimentation rate (ESR)
- Stool for occult blood
- Morphology by peripheral smear
- Hepatic and renal function
- Serum protein electrophoresis
- Thyroid-stimulating hormone (TSH)
in iron deficiency. Transferrin saturation may be low or normal in chronic disease and will be low in iron deficiency. Ferritin level will be elevated or normal in chronic disease but low in iron deficiency. Soluble transferrin receptor levels (a recently available test) will be normal in chronic disease and elevated in iron deficiency.

In anemia of chronic disease, acute or chronic immune activation causes disturbances of iron homeostasis that limits the availability of iron for erythropoiesis. Hepcidin, a protein produced in the liver, causes significant disturbances to iron transport. There is increased uptake of iron by macrophages and impaired proliferation and differentiation of erythroid precursors. This leads to a blunted erythropoietin response.11

Anemia of chronic kidney disease is a form of chronic anemia primarily due to decreased red blood cell production, and it is associated with a decline in GFR. This is measured by most laboratories using the MDRD equation and is utilized to stage chronic kidney disease (Stages I to V). ●

Table 3.
Differentiating Iron Deficiency Anemia (IDA) from Anemia of Chronic Disease (ACD)

<table>
<thead>
<tr>
<th>Blood Test</th>
<th>ACD</th>
<th>IDA</th>
<th>ACD + IDA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iron</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>TIBC</td>
<td>↓</td>
<td>↑</td>
<td>LN or N</td>
</tr>
<tr>
<td>% Transferrin</td>
<td>↓</td>
<td>or N</td>
<td>↓</td>
</tr>
<tr>
<td>saturation</td>
<td>↑</td>
<td>or N</td>
<td>↓</td>
</tr>
<tr>
<td>Ferritin</td>
<td>↑</td>
<td>or N</td>
<td>↑ or N</td>
</tr>
<tr>
<td>Soluble transferrin</td>
<td>N</td>
<td>↑</td>
<td>↑ or N</td>
</tr>
<tr>
<td>receptor</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

N, normal; LN, low normal; TIBC, total iron-binding capacity

In anemia of chronic disease, acute or chronic immune activation causes disturbances of iron homeostasis that limits the availability of iron for erythropoiesis.

Treatment for Anemia

Manju T. Beier, PharmD
Senior Partner, Geriatric Consultant Resources LLC
Clinical Associate Professor of Pharmacy
University of Michigan, Ann Arbor, Michigan

Dr. Beier reviewed treatment for anemia, beginning with a chart of treatment options from the AMDA Anemia Clinical Practice Guideline (Table 4). She noted that iron deficiency anemia is often treated with iron after the cause of the condition is treated. Supplemental iron generally is given in the form of iron sulfate (325 mg 1-3 times daily). Possible side effects of iron sulfate include constipation, diarrhea, nausea, and bloating, and therefore many frail older adults can not tolerate the recommended dose. Dr. Beier cited a study of patients over age 80 with anemia who improved on a lower dose of elemental iron.12

Vitamin B12 deficiency is usually seen only in strict vegetarians. Older adults may have a malabsorption syndrome (most common etiology) in which B12 cannot be absorbed or they lack the intrinsic factor. Because some B12 is absorbed passively, supplemental B12 can be delivered parenterally or orally. B12 is also available in an intranasal formulation, which may be advantageous in cognitively impaired patients or patients with difficulty swallowing.

To diagnose vitamin B12 deficiency and differentiate it from folate deficiency, Dr. Beier recommended measuring serum methylmalonic acid. Methylmalonic acid is also useful to establish B12 deficiency in those with borderline or low normal B12 levels. Treatment for folate deficiency is folate-rich foods, such as leafy green vegetables.

To treat anemia of chronic disease or anemia of chronic inflammation, first identify and then stabilize the underlying cause. For hemolytic anemia, identify the underlying cause and discontinue medications that may be contributing to the condition. For anemia associated with chronic kidney disease, treatment is epoetin alfa or darbepoetin alfa SC.

Correcting anemia has important clinical consequences. It has been demonstrated that successful treatment of anemia with erythropoietin may improve left ventricular hypertrophy. In a study by Portolés, correction in hematocrit levels was associated with a significant reduction in left ventricular mass (P < .01).13 In another study, correction of anemia resulted in marked improvements in left ventricular ejection fraction, New York Heart Association status, and fatigue/shortness of breath symptoms.14 The study also showed preservation of, or improvement in, cardiac and renal function. A separate study demonstrated improved quality of life with correction of anemia.15

Evaluation and Treatment
For anemia patients with chronic kidney disease, the National Kidney Foundation (NKF) recommends measuring Hb at least annually, regardless of stage or cause of kidney disease. If Hb is < 12 g/dL in an adult woman or < 13.5 g/dL in an adult man, the NKF recommends obtaining a complete blood count, absolute reticulocyte count, serum ferritin to assess iron stores, and serum transferrin saturation to assess adequacy of iron for erythropoiesis.16
iron therapy," said Dr. Beier. Once ESA therapy has begun, it is important to monitor Hb and adjust dosages as needed to reach the target of 10 to 12 g/dL.

Iron status should be monitored regularly using transferrin saturation (TSAT) and serum ferritin levels. Iron should be administered to maintain a TSAT level ≥ 20% and serum ferritin ≥ 100 ng/mL.21

Several iron preparations are available, including ferrous sulfate, ferrous gluconate, ferrous fumarate, iron polysaccharide, and heme iron polypeptide. The recommended dose of oral elemental iron is 200 mg/day, which may be problematic for older adults who may require a lower dose to prevent side effects. Iron replacement therapy may be individualized based on the urgency for correction of anemia and tolerability factors.

Iron is available in enteric-coated and sustained-release formulations, as combination products with ascorbic acid, and in pediatric elixirs. Dr. Beier noted that enteric-coated and sustained-release formulations may reduce iron absorption due to iron being transported past the duodenum where it is optimally absorbed. Iron combined with ascorbic acid may have improved absorption, although this option may be more costly. Pediatric elixirs may be useful for residents who have difficulty swallowing.

Iron doses should ideally be administered 1 hour or more before meals if tolerated to minimize any alterations in absorption due to food. Medications that may decrease iron absorption include fluoroquinolones, antacids, tetracycline, cimetidine, levodopa, methyldopa, and penicillinamide.

If oral iron is insufficient for correcting iron deficiency after 2 to 3 months of therapy, intravenous (IV) ferric gluconate, iron sucrose, or iron dextran should be given. The dosage for ferric gluconate or iron sucrose is 200 mg to 250 mg over 1 to 2 hours. Iron dextran may be utilized, but because of a potential for anaphylactic reaction, it requires the administration of test dose. The NKF guidelines prefer parenteral iron sucrose because of the superior safety and ease of administration.20 Potential side effects include metallic taste, headache, nausea, vomiting, muscular pain, and pruritus.

The importance of correcting iron deficiency prior to initiation of ESA therapy was demonstrated in a study by Kim et al. The investigators found that patients with high ferritin stores and with greater transferrin saturation were more likely to respond to epoetin alfa therapy than those with depleted iron stores.22

A comparison of the two available ESAs, epoetin alfa and darbepoetin alfa is shown in Table 5. Dr. Beier noted that while there are pharmacokinetic differences, the two agents are pharmacodynamically fairly equivalent. The goal of therapy is to achieve Hb of 10 to 12 g/dL. If Hb rises above 12 g/dL, the ESA should be discontinued until the Hb falls to 12 g/dL. If Hb does not increase 1 g/dL after 4 weeks, the ESA dose should be reinitiated at a lower dose. If Hb is between 10 and 12 g/dL, the ESA should be maintained. If Hb is less than 10 g/dL and Hb does not increase 1 g/dL after 4 weeks, the dose should be increased.

Dr. Beier recommended adjusting the dose of erythropoietin (SC) based on the patient’s weight.23 For patients weighing 60 to 100 kg, she suggested giving erythropoietin alfa 10,000 units every week or darbepoetin alfa 40 mcg every week. For patients weighing less than 60 kg, give erythropoietin alfa 6,000 units every week or darbepoetin alfa 25 mcg every week.

For anemia patients with chronic kidney disease, the National Kidney Foundation (NKF) recommends measuring Hb at least annually, regardless of stage or cause of kidney disease.
The PROMPT study evaluated the efficacy of dosing once every 2 weeks, darbepoetin alfa and who were previously receiving darbepoetin once every 2 weeks, darbepoetin alfa was given every 4 weeks. Most patients (79%) achieved an Hb concentration between 10 and 12 g/dL, which was maintained for the duration of the 29-week study. The PROMPT study evaluated the efficacy and safety of extended maintenance dosing of epoetin alfa in patients with chronic kidney disease being treated for anemia. Patients received dosing every week, every 2 to 4 weeks initially, and aim for Hb 10 to 12 g/dL and TSAT ≥20%.

Double the dose for every-other-week dosing, monitor hematocrit/hemoglobin every 2 to 4 weeks initially, and aim for Hb 10 to 12 g/dL. Patients in all of the groups maintained Hb ≥ 11 g/dL. Extended dosing currently is not FDA-approved for these agents.

Potential Risks Associated with ESA

In 2006, a few studies were published showing potentially serious adverse events associated with use of ESAs. One study was the Correction of Hemoglobin and Outcomes in Renal Insufficiency (CHOIR) study, which was a prospective study of patients with GFR 15 to 50 mL/min and Hb < 11 g/dL. Patients were randomized to a target Hb of 13.5 g/dL (n = 715) or 11.3 g/dL (n = 717). The investigators found that the group with the higher Hb target had higher rates of congestive heart failure, death, and hospitalization. There was no difference in stroke, myocardial infarction, renal replacement therapy, or quality of life between the two groups.

In March 2008, the black box warning label was changed again to include more detailed instructions for use of ESAs in cancer patients and it reiterated the need to individualize dosing in patients with renal failure to achieve Hb levels in the 10 to 12 g/dL range. In July 2008, still more changes were made to the black box warning label, which referred to use of ESAs in cancer patients.

For patients with chronic kidney disease, it has been well established that the optimal Hb target is < 12 g/dL. For patients with chronic kidney disease, it has been well established that the optimal Hb target is < 12 g/dL. Hb > 13 g/dL is associated with increased risk of hypertension and mortality, especially in patients with cardiac disease. Blood pressure should be monitored in all patients with chronic kidney disease, particularly during initiation of ESA therapy. If there is a rapid rise in Hb/Hct accompanied by hypertension, it may be necessary to initiate or increase antihypertensive medications and reduce the ESA dose.

### Case Study

Returning to the earlier case of LD (see box on page 1), Dr. Beier discussed evaluation and treatment of the patient. LD was transferred to the hospital for treatment of congestive heart failure and anemia. He received a blood transfusion and diuresis. Upon returning to the long-term care facility, his heart failure stabilized, he lost 4 pounds, and dyspnea and edema were reduced. He was still fatigued and anorexic. Laboratory results were as follows: Hb 9 g/dL, creatinine 1.9 mg/dL, estimated GFR 24 mL/min/1.73 m², thyroid-stimulating hormone (TSH) 1.37 mIU/mL. Results of an anemia workup were normal. Based on this GFR he had Stage IV kidney disease.

Several weeks after returning from the hospital LD became more fatigued. The nursing assistant reported that he had trouble transferring between the bed and a chair, had decreased appetite (eating only 50%), and he seemed more confused. Laboratory results were the following: Hb 8.5 g/dL, creatinine 2 mg/dL, estimated GFR 24 mL/min/1.73 m², TSAT 16% (20%-50%), serum ferritin 75 ng/mL (100 ng/mL-800 ng/mL). His weight was 128 pounds (58 kg). At this point, the clinician may opt to check his iron status, initiate therapy with an ESA, or call a family meeting to discuss palliative care. Dr. Beier recommended that all three are viable options and need to be discussed. Moreover, decompensation of congestive heart failure in an 88-year-old patient may indicate that palliative care is a prudent choice.

In summary, Dr. Beier emphasized the need to make sure iron stores are adequate, use algorithms to guide treatment, maintain an Hb between 10 and 12 g/dL, wait at least 4 weeks before increasing the dosage, and take advantage of alternative dosing schedules based on the patient’s weight.

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<table>
<thead>
<tr>
<th>Epoetin alfa</th>
<th>Darbepoetin alfa</th>
</tr>
</thead>
<tbody>
<tr>
<td>▪ IV half-life = ~8.5 hours</td>
<td>▪ IV half-life = ~26.3 hours</td>
</tr>
<tr>
<td>▪ Biologic activity is less than that of darbepoetin alfa</td>
<td>▪ Biologic activity is greater than that of epoetin alfa</td>
</tr>
<tr>
<td>▪ Epoetin alfa should be given subcutaneously (or intravenously) 1 to 3 times weekly</td>
<td>▪ Darbepoetin alfa should be given once every 2 weeks if a patient was receiving epoetin alfa once weekly</td>
</tr>
<tr>
<td>▪ Starting dosage: 0.45 µg/kg once weekly</td>
<td></td>
</tr>
<tr>
<td>▪ Dose response: 2-6 weeks</td>
<td></td>
</tr>
</tbody>
</table>

Once goal is achieved, monitor hemoglobin at each injection. Recommendations provided for a typical 70-kg (154-lb) patient.
Role of the Interdisciplinary Team in Recognizing and Monitoring Anemia

Naushira Pandya, MD, CMD

Dr. Pandya discussed a team approach to anemia management in long-term care settings. In terms of monitoring treatment, she explained the need to take into account both the individual patient's goals as well as the goals of the facility. Examples of patient goals include improved stamina, minimal or no adverse effects, and improved nutritional intake. Facility goals may include improvement in physical, social and cognitive function, increase in Hb concentrations, and reduced hospitalizations for anemia-related complications.

Achieving these goals requires improving the process of care. Some changes in processes recommended in the AMDA Anemia Clinical Practice Guideline (March 2007) include systematic follow-up of abnormal CBCs, systematic evaluation for the causes of anemia when appropriate, appropriate treatment when anemia is diagnosed, and systematic laboratory monitoring of patients with anemia.

Some questions that all members of the healthcare team should consider relate to efficacy and safety of ESAs. Questions include the following: Is the starting dose of the ESA appropriate based on the indication and package insert? Is Hb monitored at appropriate intervals? Are iron stores monitored? Are they within therapeutic range? Are dose adjustments at least 4 weeks apart? Is a dose adjustment needed based on laboratory values and package insert guidelines? Has Hb reached target levels? Are there any adverse events noted from pharmacotherapy? If response is inadequate, are factors responsible for hyporesponsiveness investigated?

There are circumstances in which patients do not respond to ESAs. Some of these are as follows: missed doses, inadequate iron stores, drug/disease interactions (iron needs an acidic environment to be maximally absorbed), B12/folate deficiencies, protein deficiencies, occult blood loss, infection/inflammation processes, coexisting medical conditions (malignancies, hematological disorders), and hemolysis.

Each member of the healthcare team has a role in recognizing and monitoring anemia.

References


Dietitians monitor the diet for lack of foods rich in iron, B12, or folate, and monitor residents for anorexia, nausea, and weight loss. Nurses monitor residents for signs of fatigue and bleeding (such as hematochezia, melena, and pale mucous membranes, skin, or nail beds), evidence of heart failure despite medical treatments, complaints of lightheadedness or dizziness upon standing, hypotension, and tachycardia. Certified nursing assistants monitor residents for decreased ability to perform activities of daily living. Pharmacists monitor the use of medications that can cause or contribute to anemia, and monitor for laboratory test results that indicate abnormal hematological status or renal function. Physical therapists monitor residents’ progression and ability to follow a physical therapy plan and assess endurance during therapy.

In conclusion, the etiology of anemia in long-term care is multifactorial and unexplained anemia is common. The estimated GFR is a useful test, which should be followed by a check of hemoglobin. Iron and erythropoietin deficiencies should be treated. Dosing strategies for epoetin alfa and darbepoetin alfa can range from every week to every month. Treating anemia can have a positive impact on many comorbid conditions in long-term care residents. It can improve level of function and may reduce falls.
Continuing Education Examination

To receive continuing education (CE) credit, follow the instructions on the following page.

1. Which laboratory profile corresponds with anemia of chronic disease?
   a. Low ferritin and high total iron-binding capacity (TIBC)
   b. High ferritin and high TIBC
   c. Low ferritin and low TIBC
   d. Normal ferritin and low TIBC

2. Which of the following conditions may cause macrocytic anemia (i.e. elevated MCV)
   a. Iron efficiency
   b. Thalassemia
   c. Vitamin B₁₂ deficiency
   d. Chronic kidney disease

3. Elderly patients with anemia may have which of the following manifestations?
   a. Fatigue
   b. Functional decline
   c. Tachycardia
   d. All of the above

4. A reasonable recommendation for nursing homes when managing patients with anemia is:
   a. Implementation of a set protocol for diagnosis and treatment for all residents
   b. Individualized assessment and treatment based on treatment goals agreed upon by the resident or responsible party
   c. Laboratory assessment for B₁₂, folate, iron deficiency, and thyroid disease initiated by the treatment nurse
   d. Obtaining a hematology consultation for all residents with anemia

5. An 84-year-old resident with a history of peptic ulcer disease, hypertension, and osteoarthritis is found to have a microcytic anemia on a laboratory evaluation for dizziness, and decline in self-care. The most appropriate next step in her evaluation would be:
   a. Review all her medications and check stool for occult blood
   b. Increase the dose of her omeprazole from 20 mg to 40 mg daily
   c. Prompt neurological consultation for her dizziness
   d. Trial of therapy with an ESA

6. Which of the following is NOT a common side effect of iron supplementation therapy in patients with iron-deficiency anemia?
   a. Dark discoloration of stools
   b. Glossitis
   c. Diarrhea
   d. Nausea

7. Enteric-coated or sustained-release formulations of iron may reduce the optimal amount of iron absorbed because:
   a. They transport iron past the duodenum where it is maximally absorbed.
   b. The preparations do not contain the necessary amount of elemental iron.
   c. They contain ascorbic acid which reduces the amount of absorbable iron.
   d. They must always be given with meals, which reduces absorption.

8. Which laboratory test is the most useful to differentiate iron-deficiency anemia from anemia of chronic disease?
   a. Serum iron
   b. Transferrin saturation
   c. Total iron-binding capacity
   d. Serum ferritin

9. Which laboratory test is the most useful to differentiate folate deficiency anemia from B₁₂ deficiency anemia?
   a. Serum B₁₂ level
   b. Transferrin saturation
   c. Homocysteine level
   d. Serum methylmalonic acid

10. According to the NKF KDOQI guidelines (2007), the target Hb in chronic kidney disease patients receiving ESA therapy should be:
    a. 9-10 g/dL
    b. 10-11 g/dL
    c. 11-12 g/dL
    d. 12-13 g/dL
Continuing Education Examination & Evaluation

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6. You must complete the online evaluation form and pass the online examination with a score of 70% or better. If you do not receive a minimum score of 70% or better, you are permitted one (1) retake.
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