

### QUESTION

A 65-year-old male patient with a long history of hypertension and a recent diagnosis of type 2 diabetes mellitus is being treated with lisinopril and metformin. He has been experiencing increasing fatigue and weakness over the past few weeks. His physical examination is unremarkable, and his laboratory tests show a hemoglobin of 10 g/dL, hematocrit of 30%, and a mean corpuscular volume (MCV) of 85 fL. His serum ferritin is 100 ng/mL, and his serum iron is 150 µg/dL. His total iron-binding capacity (TIBC) is 300 µg/dL, and his transferrin saturation is 20%. His renal function is normal, and his liver function tests are within normal limits. Which of the following is the most likely cause of his anemia?

- A) Iron deficiency anemia
- B) Vitamin B12 deficiency
- C) Folate deficiency
- D) Anemia of chronic disease
- E) Hemolytic anemia

ANSWER: D

EXPLANATION: The patient's anemia is most likely due to anemia of chronic disease (ACD). The key features supporting this diagnosis are the normal MCV (85 fL), normal serum ferritin (100 ng/mL), and low transferrin saturation (20%). ACD is a common cause of anemia in patients with chronic medical conditions, such as hypertension and diabetes mellitus. The anemia is typically normochromic and normocytic, and the serum ferritin is often normal or elevated. The low transferrin saturation is a characteristic finding in ACD. Iron deficiency anemia (IDA) would typically present with a low MCV (microcytic anemia) and a low serum ferritin. Vitamin B12 deficiency and folate deficiency would typically present with a high MCV (macrocytic anemia). Hemolytic anemia would typically present with a high reticulocyte count and a low haptoglobin level.

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The diagram illustrates the pathophysiology of anemia of chronic disease. It shows the relationship between inflammation, hepcidin, and iron metabolism. Inflammation leads to the production of interleukin-6 (IL-6), which stimulates the liver to produce hepcidin. Hepcidin binds to and downregulates the ferroportin (FcR) receptors on the surface of macrophages and other iron-storing cells. This results in decreased iron export from these cells into the circulation. Simultaneously, inflammation also increases the activity of the iron regulatory protein (IRP), which binds to and increases the expression of transferrin receptor (TFR) on the surface of erythroid precursors. This leads to increased iron uptake by the bone marrow. However, the overall effect is a decrease in the amount of iron available for erythropoiesis, leading to anemia. The diagram also shows that inflammation leads to a decrease in total iron-binding capacity (TIBC) and a decrease in transferrin saturation (TSAT), which are characteristic findings in anemia of chronic disease.

ANSWER: D

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