A Case of Iron Deficiency Anemia

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Mr. WH is a 72-year-old-man who presented in April 2004 with a hematocrit of 21%. His previous hematocrit was 34% in February 2004. He complained of bright red blood per rectum and rectal pain secondary to external hemorrhoids. Mr. H was admitted with a presumed gastrointestinal bleed.

Mr. H’s past medical history is significant for bovine aortic valve prosthesis in 1997 secondary to aortic stenosis, Addison’s disease, hypocalcemia, seizure disorder, B12 deficiency, colon cancer with partial colon resection, and small bowel obstruction due to adhesions. Mr. H had chronic diarrhea secondary to bowel surgeries. His last colonoscopy was in 1997 and it revealed hemorrhoids. An esophagastroduodenoscopy done at the time showed duodenitis.

The patient’s medications on hospital admission were as follows: calcium carbonate 500mg three times a day, vitamin B12 1000mcg I.M. monthly, divalproex sodium 750mg twice a day, hydrocortisone 25mg every 12 hours, vitamin D 100,000 IU every three months, phenobarbitol 30mg every 12 hours, lasix 20mg by mouth daily, and potassium chloride 20m.e.q. daily. He has no known drug allergies.

Mr. H lives in an assisted living facility. He is independent with his activities of daily living, though his brother has Power of Attorney for finances and personal care.

On admission to the hospital, Mr. H was in no acute distress. His vital signs were stable with a blood pressure of 100/60. He had normal heart and lung sounds. His abdomen showed an old scar and was scaphoid in appearance. He had external hemorrhoids and guaiac positive stool. He had 2+ lower extremity edema extending to the mid thigh. Varicose veins were present in the lower extremities. A musculoskeletal exam revealed contractures of bilateral fingers. He had normal upper and lower extremity strength and normal gait. Mr. H was oriented to person, place, and time.

Laboratory results revealed the following: sodium 134mmol/L, potassium 3.5mmol/L, chloride 102mmol/L, bicarbonate 20mmol/L, BUN 6.4mmol/L, creatinine 79.5µmol/L, and glucose 4.6mmol/L. Phenobarbitol levels and valproic acid levels were in the therapeutic range. White blood count and platelet count were normal, though the neutrophil count was 83%. Hematocrit was 23.8%. Other results showed that calcium was 1.7mmol/L; parathyroid studies were normal; thyroid stimulating hormone level was normal; albumin was 26g/L; urinalysis was negative for protein; and transferrin was 8%.

Esophagastroduodenoscopy and colonoscopy showed mild gastritis and glandular hyperplasia but did not determine a source of bleeding. His hemoglobin was stable after two units of packed red blood cells. His hypocalcemia was treated with intravenous calcium and 100,000 IU of vitamin D. He was discharged back to his assisted living facility on a proton pump inhibitor. He continued to complain of rectal bleeding, which was attributed to hemorrhoids. He required another two units of blood in May 2004. He was hospitalized again in June 2004. Sigmoidoscopy and OGD were repeated. Neither test revealed the source of bleeding. A bone marrow biopsy was done and was consistent with iron deficiency and myelodysplasia. His gastroenterologist did not believe that the hemorrhoids were the cause of his iron deficiency anemia. It was thought that Mr. H was malabsorbing oral iron, and I.V. iron treatments were started. He was also started on erythropoietin.

Mr. H did have outpatient follow-up with his gastroenterologist. A small bowel series was done, which showed rapid transit time in the colon. Subcapsular endoscopy was considered, but this test was deferred because it was felt that iron deficiency was related to malabsorption from short gut syndrome. He was started on tincture of opium to control diarrhea. Cholestyramine and pancreatic enzymes were also added. He continued his iron treatments and erythropoietin. His oral calcium was increased and he was started on calcitriol due to suspected vitamin D malabsorption.

His hematocrit is still only 26% despite weekly iron transfusions and 60,000 units of erythropoietin weekly.
The case of Mr. WH presents some common yet challenging issues in the treatment of anemic patients. The case shows that the causes of anemia are often multifactorial. Mr. WH has vitamin B12 deficiency. We are not told if this presented as a macrocytic anemia, neurologic symptoms, or as an asymptomatic blood test. However, he is now presenting with a recurrent anemia.

There are some practical points that can be taken from the case. It is usually helpful to discuss the hemoglobin rather than the hematocrit when describing an anemia. The hemocrit is helpful in patients who may have been bleeding. The mean corpuscular volume (mcv) helps differentiate the etiology of the anemia and guide investigations as to the approach to a microcytic anemia is slightly different from that of a normocytic or macrocytic anemia. The mean cell hemoglobin concentration (mchc) also helps in that regard.

Prior to doing the bone marrow in this case, some other lab values would have been helpful. The ferritin is the best test for the determination of iron deficiency in the normal population. We were not given a ferritin in this case but are told the transferrin was eight percent. However, Rimon et al. suggest that neither test is that sensitive nor specific in the older adult population. Instead, the transferrin to the log of ferritin is the most sensitive (88% vs. 16% for ferritin) in the over-80 age group.

Investigation of the GI tract is the primary and most important first step in the investigation of IDA. Estimates of prevalence of GI disorders account for 27–95% of the causes of IDA. In this case, the first step was testing the stool for guiac. However, I would not recommend this step because it should not make one feel that the urgency for GI investigation is lessened by a negative test. In our case, this patient complained of bright red blood per rectum and we would be surprised if the test were negative, as guiac testing is designed to discover obscure bleeding. Therefore, the role for guiac testing should be more directed to looking for asymptomatic bleeding and colorectal cancer screening.

Our patient was appropriately investigated with both upper and lower endoscopy. The American and British Gastrointestinal associations have clinical practice guidelines for evaluation of occult bleeding and IDA (www.gastro.org). Patients who can tolerate endoscopic investigation should have full evaluations, including a duodenal biopsy. This is important to patients with IDA and their investigation because iron is absorbed via a divalent metal transporter in the proximal duodenum. Duodenal biopsy is often overlooked in the investigation of patients with IDA as it apparently was in our case (or we were not given that information). A study from a university hospital in Britain found that only 47% of patients, average age 71.5, undergoing upper endoscopy for IDA had duodenal biopsies. Of those, seven percent had celiac disease. Celiac disease is often subclinical and presents as IDA but can also be screened for with an anti-tissue transglutamase (ttg) which is highly sensitive and specific and not affected by immunoglobulin deficiencies.

Double contrast barium enema (DCBE) may sometimes be recommended in this type of investigation but it lacks both the sensitivity and specificity to be considered a useful test. Winawer et al. found that DCBE is only accurate in 39% of cases where polyps have been found at colonoscopy in high-risk patients (i.e., those who had previous polyps removed).

Finally, follow-up of patients with previous colorectal cancer is necessary. There is only one appropriate test for these patients and that is colonoscopy. Mr. WH had gone seven years since his last follow-up colonoscopy. However, the best available data suggest that these patients should undergo screening at least every three years.

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**References**