Jejunal Hemosiderosis Detected with Small Bowel Capsule Endoscopy

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Case Report

A 64 year old man with chronic hepatitis C and end stage renal disease was referred for evaluation of obscure, occult gastrointestinal blood loss. He experienced recurrent symptomatic anemia for years without melena or hematochezia. His hemoglobin consistently remained between 7 and 9 g/dL with a normal MCV. Iron studies demonstrated iron deficiency with an iron level of 25 mcg/dL, iron binding capacity of 466 mcg/dL, and iron saturation of 25%. Bidirectional endoscopy did not reveal a source of blood loss. His anemia evaluation also included a bone marrow biopsy which was unremarkable and negative hemolysis studies. Oral double balloon enteroscopy performed two years ago demonstrated small arteriovenous malformations in the jejunum which were treated with bipolar coagulation. Despite the absence of overt bleeding, he continued to require intermittent blood transfusions. One year later, he remained iron deficient with an iron saturation of 10% and ferritin 57 ng/mL on intermittent oral iron supplementation. An erythropoietin level was within normal limits at 8 mU/mL with a hemoglobin of 10 g/dL. The patient’s renal function continued to decline (cryoglobulins were undetectable and C3, C4 were within normal limits) and he was initiated on hemodialysis. Erythropoietin and intravenous iron infusions were subsequently administered with sessions of hemodialysis.

We repeated an upper endoscopy and colonoscopy which revealed small esophageal varices, mild portal hypertensive gastropathy, and rectal varices without signs of recent hemorrhage. Multiple speckled areas of brown pigmentation in the stomach and duodenum were also observed (Figure 1). Biopsies of these areas demonstrated hemosiderin deposits with positive iron staining (Figure 2 and Figure 3). A liver biopsy which was recently performed for staging and grading of hepatitis C prior to starting parenteral iron revealed scant Kupffer cell iron and was not consistent with hemochromatosis. In the following months, he required 8 units of packed red cell transfusions for Hgb readings less than 7mg/dL on multiple occasions. Small bowel capsule endoscopy (Pillcam SB, Given Imaging) was performed with multiple, small, nonbleeding arteriovenous malformations visualized in the ileum (Video 1). The hyperpigmented, speckled

Figure 1. Hyperpigmentation of the gastric antrum secondary to hemosiderin deposition.
pattern previously visualized in the stomach and duodenum on upper endoscopy was observed to continue into the distal jejunum (Figure 4, Video 2).

Discussion

Hemosiderosis is increasingly recognized among patients receiving parenteral iron and erythropoiesis stimulating agents with hemodialysis. The liver is the main site of iron storage in humans and reports of hemosiderosis most often involve hepatic iron overload. There are rare reports of endoscopically recognized hemosiderosis of the gastrointestinal tract, most commonly seen in the duodenum with only three prior cases of hemosiderosis distal to the duodenum. Our patient demonstrated hemosiderosis in multiple areas of the GI tract including the stomach, duodenum, and jejunum. In
the majority of cases, there is a history of oral iron supplementation, cirrhosis, or hemodialysis. Interestingly, the patient in this case was not exposed to oral iron supplementation for over a year prior to this capsule study and received parenteral iron for only a month prior to manifestation of hemosiderosis. It is unknown, the length or quantity of parenteral iron infusions required prior to the development of endoscopically visible hemosiderin deposits but appears to be as little as a month based on this case. We hypothesize that our patient may have experienced accelerated gastrointestinal deposition of iron due to portal hypertension with portocaval shunting which allows increased exposure of iron to the gastrointestinal mucosa.

References:


