Not Your Usual Culprit of Hemorrhagic Shock

Diana Jomaa, MD[1], Hamna Fahad, MD[2], Sanam Husain,MD[3] ,Radhika Aggarwal,MD[2]

1.Department of Internal Medicine, 2. Division of Gastroenterology and Hepatology , 3. Department of Pathology

Henry Ford Health, Detroit, Michigan

Introduction

- Patients with end stage renal disease (ESRD) are at risk of GI bleeding most commonly due AVM or ischemic colitis.
- They are also at risk for developing hyperphosphatemia commonly treated with phosphate binders such as sevelamer.
- Sevelamer can crystallize into concretions that lead to GI mucosal injury and rarely induce colitis.

Case Presentation

- 56-year-old African American female with ESRD on hemodialysis who presents for elective MV replacement
- Post-op placed on anticoagulation (AC) → course c/b shock, 3 g drop in Hgb (10 to 7 g/dL), and large bloody BM requiring vasopressors and pRBC transfusions
- **CTA:** negative for active bleeding
- **Nasogastric lavage:** bilious output
- **Colonoscopy:** localized area of granular, erythematous, and ulcerated mucosa in the descending colon near the splenic flexure that was friable on contact
- **Biopsies:** fragments of resin material consistent with sevelamer crystals admixed with acute and chronic inflammation.
- **Management:** Sevelamer discontinued, AC resumed

Images

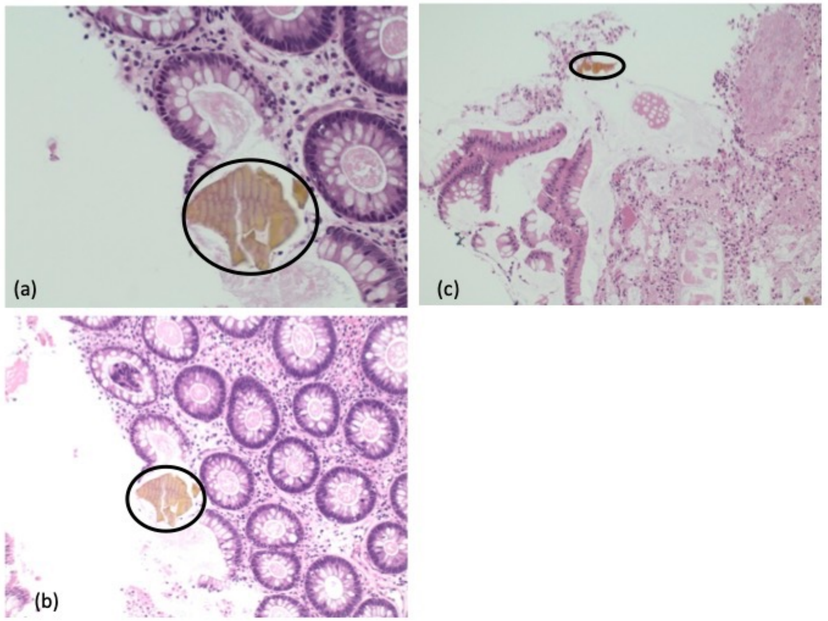

Figure 1: Sevelamer crystals are highlighted in figures (a),(b),and (c) with acute inflammation demonstrated in (c).

Discussion

- GI adverse effects of sevelamer are largely under-recognized and under-diagnosed.
- Sevelamer is dissociated in the stomach, releasing a polymer that binds phosphate within the intestine, producing phosphate crystalline concretions that are excreted in the feces. These concretions can also embed inside eroded or ulcerated GI tract mucosa in patients with ESRD.
- With the increased use of phosphate binders, it is important to recognize GI side effects and discontinue the offending agent.

References

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