An Unusual Case of Upper GI Bleeding in the Setting of Injection Drug Use

Mohammed Rifat Shaik MBBS[1], Nishat Anjum Shaik MBBS[2], Elvina Yunasan MD [1], Erika Wheeler MD [3], Yuting Huang MD PhD [4], Robert Chow MD [1,3]
[1]University of Maryland Medical Center Midtown Campus, [2]Guntur Medical College [3]University of Maryland Medical Center [4]Mayo Clinic, Jacksonville

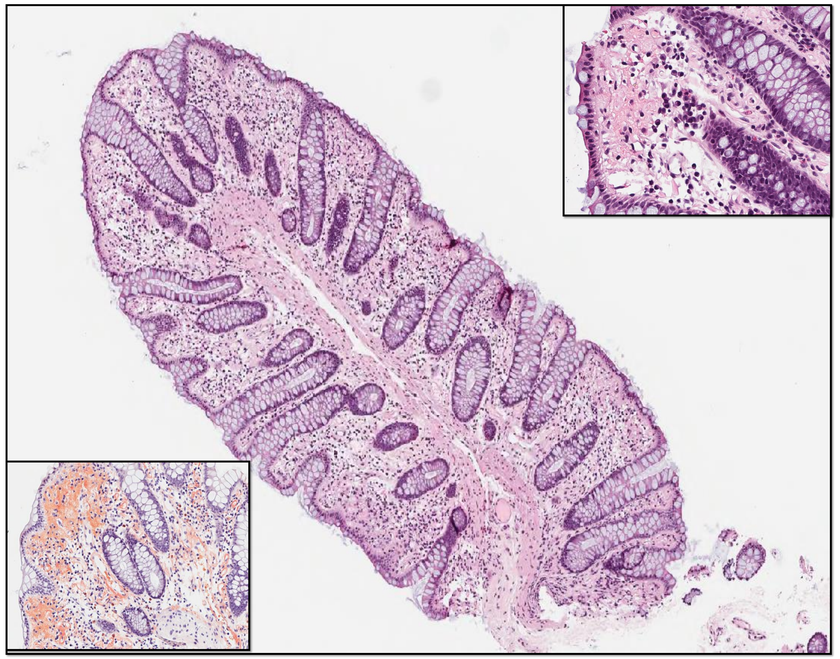
INTRODUCTION

- Autoimmune and chronic inflammatory or infectious disorders increase the risk of developing systemic AA amyloidosis.
- Renal and cardiac involvement is most common, followed by the nervous system, soft tissues, lungs, and liver.
- Gastrointestinal (GI) involvement is uncommon and often asymptomatic, leading to a delayed diagnosis.
- Here we present a patient with history of injection drug use who presented with hemoglobin (Hgb) decline from ongoing occult GI bleed. This prompted endoscopy, leading to a diagnosis of GI amyloidosis.

CASE PRESENTATION

- A 37-year-old woman with a longstanding history of injection drug use was admitted with sepsis secondary to infection of chronic, non-healing forearm wounds.
- Physical examination showed large wounds measuring 12 x 7 x 0.5 cm and 12 x 6 x 0.5 cm on the right and left forearm respectively.
- Her hospital course was complicated by a Hgb decline to 6.1 from 9 g/dL mandating transfusion support with 6 PRBCs to maintain her Hgb > 7 g/dL.
- She denied hematemesis, melena, or hematochezia.
- CT angiography indicated no obvious source of bleeding in the abdomen or pelvis.
- Upper GI endoscopy revealed erythematous, friable gastric mucosa, and duodenal mucosal atrophy.
- Colonoscopy revealed grossly normal but friable mucosa.
- Gastric, duodenal, and colonic biopsies showed extensive amyloid deposition in the lamina propria confirmed with Congo red stain (see figure).
- Mass spectrometry was positive for amyloid A.
- She was discharged on Pantoprazole, and responded well.

HISTOPATHOLOGY – COLONIC BIOPSY



This low power image of a colon biopsy shows increased deposition of an amorphous pink material within the lamina propria, which can be more easily seen at higher magnification (see inset, upper right,20x). A Congo red special stain confirms the amorphous material is comprised of amyloid protein (see inset, orange coloring, bottom left).

DISCUSSION

- GI symptoms while not uncommon in the setting of amyloidosis, biopsy proven GI amyloidosis is rare with an incidence of only 3% according to a large retrospective case series[1].
- The most common type of amyloid type in the GI tract was ALλ (53%) followed by ATTR (16%), AA was the less common and occurred 10% of the time[1].
- Having GI symptoms as the first sign of systemic amyloidosis is exceedingly unusual, especially in the absence of clinical signs of the disease elsewhere in the body[2].
- Clinical manifestations are the result of tissue infiltration or GI dysmotility from autonomic nerve dysfunction[3].
- These can commonly be categorized as impaired motility (leading to esophageal reflux, delayed gastric emptying, chronic intestinal pseudo obstruction) malabsorption (leading to steatorrhea, diarhoea), vascular insufficiency (presenting as bleeding, infarction or perforation).
- AL preferentially depositing in the muscularis propria and muscularis mucosa and AA depositing in the lamina propria[4](as in our patient).
- In accordance with the pattern of deposition described above, AA amyloidosis is usually presents with diarrhea, malabsorption, and gastrointestinal bleeding, whereas AL amyloidosis is characterized by mechanical obstruction and chronic intestinal pseudo obstruction[4].
- Gastrointestinal bleeding results from vascular occlusion and fragility induced by amyloid infiltration in the tunica media/intima[1].
- Treatment is aimed to reduce SAA levels by managing the underlying cause i.e, wound infection and drug rehabilitation in our case.
- Because the fragility of small blood vessels leads to the impairment of hemostasis, endoscopic treatment is generally ineffective[5].

REFERENCES

CONTACT INFORMATION

Mohammed Rifat Shaik MD shaikrifat13@gmail.com