GI Amyloidosis: A Case of Congo Red Staining and Anemia

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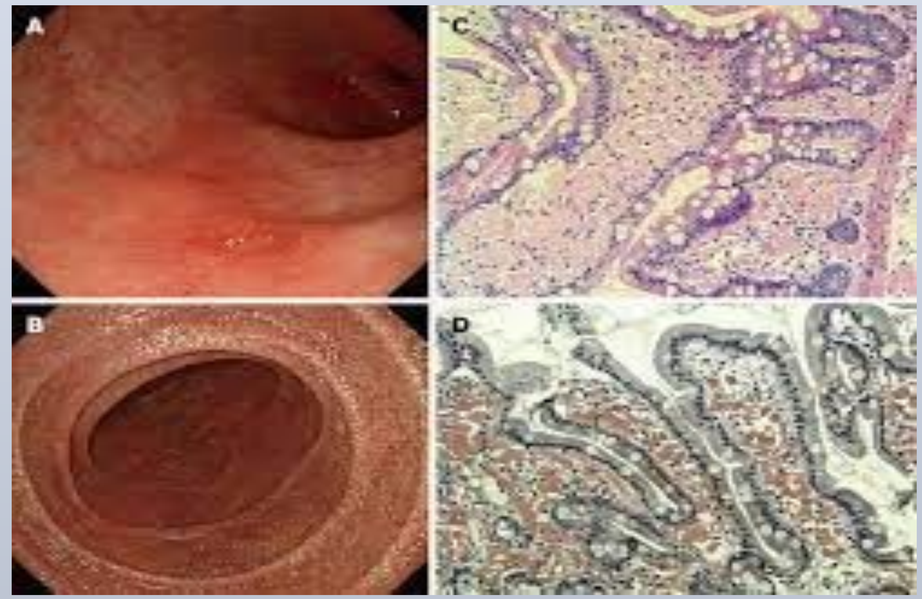
Introduction

Gastrointestinal amyloidosis (GIA), is a protein deposition disorder. It represents a complex common pathway that encompasses multiple etiologies and presentations. Within the GI tract, amyloid deposition occurs in the muscularis mucosae, within close proximity to vasculature, nerves, and nerve plexuses. This deposition increases the frailty of blood vessels, hinders intrinsic peristalsis and decreases the compliance of the gut wall. We are presenting an interesting case where GI amyloidosis was discovered while evaluating for chronic anemia.



Case Description

Patient is a 71 year old female with a clinical history remarkable for hypertension, hyperlipidemia, pre-DM, who was referred to GI in order to evaluate a suspected GI source for her chronic anemia and now presents to the endoscopy unit for an upper endoscopy. The EGD showed a normal appearing esophagus and GEJ, mild non-specific gastritis, scattered erythema in the stomach or antrum, small area of hyperplastic fold appearance in the gastric body, s/p cold biopsy for histopath evaluation, and a normal appearing duodenum. Patient subsequently underwent a colonoscopy in which a suspicious lesion, flat and pale, 7mm in size was identified at 55 cm from the anal verge,. Cold biopsy was taken to evaluate for histopath correlation of a suspected polyp. In addition a moderate sized rectal flat polyp with a depressed center, measuring 18mm (Paris IIc), concerning for a dysplastic lesion vs neoplastic lesion was found and another cold biopsy was taken for further evaluation. The two specimens underwent congo red staining and demonstrated amyloid deposition in the mucosa/submucosa. Patient was recommended for further evaluation for systemic amyloidosis via diagnostic tests including serum and urine protein electrophoresis, serum/urine free kappa and lambda light chain analysis, etc.

Discussion

Gastrointestinal amyloidosis (GIA) results from the deposition of insoluble extracellular protein fragments that have been rendered resistant to digestion. GIA can be acquired or genetic, and most commonly results from chronic inflammatory disorders. The deposition of abnormal proteins interferes with gastrointestinal tract (GI) organ structure and function, most notably in the liver and small bowel. Presentation from GI involvement includes cirrhotic sequelae, abdominal pain, malabsorption, and GI bleeding. In our patient's case, it could explain her diagnosis of chronic anemia

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