Unusual presentation of more common disease

A rare cause of lower GI bleeding

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Summary

The authors present a rare case of lower gastrointestinal (GI) bleed due to GI amyloidosis secondary to multiple myeloma. A 79-year-old lady who presented with bloody diarrhoea for 4 weeks. Flexible sigmoidoscopy showed slight oedematous mucosa extending up to the sigmoid colon. CT abdomen showed lytic lesions in the vertebral bodies. 24 h protein analysis and serum electrophoresis suggestive of multiple myeloma, which was confirmed with bone marrow biopsy, revealed plasma cell myeloma.

BACKGROUND

In 2008, 4516 people in the UK were diagnosed with multiple myeloma and caused 2660 deaths in the UK.¹ This case is interesting as the gastrointestinal (GI) involvement by primary systemic amyloidosis is rare, comprising 8% of all cases and only 1% is symptomatic.² Amyloid rarely presents in GI tract as acute GI haemorrhage especially in the absence of clinical disease.³ We present this rare case of Amyloidosis with bloody diarrhoea as a sole presenting feature. Suspicion of GI amyloidosis in patients without history of amyloidosis is difficult but should be considered in those older than 30 years with unexplained diarrhoea, weight loss, autonomic dysfunction, malabsorption or proteinuria.⁴

CASE PRESENTATION

A 79-year-old lady presented to surgical admission unit with 4-week history of bloody diarrhoea with approximately five to six bowel movements per day. She also reported poor appetite and weight loss of one stone over the same period of time. She did not have any abdominal pain, urgency or tenesmus. There was no history of recent travel or antibiotics use. Her medical history included hypertension and hypercholesterolaemia. Her medications included amlodipine and simvastatin. There was neither family history of inflammatory bowel disease nor colorectal cancer. She is an ex-smoker and does not drink alcohol.

On examination, temperature was 35.8°C, blood pressure 101/50, heart rate 90/min. regular, respiratory rate 12/min, pulse oximetry showed sats of 98% on air. Her cardiovascular, respiratory and abdominal examinations were unremarkable. Digital rectal examination showed dark red blood on gloves, no fissures or piles.

INVESTIGATIONS

Laboratory investigations revealed the following: haemoglobin 10.2 g/dl (11.5–16.5), mean corpuscular volume of 90 fl (78–103), white blood cell count 9.90×10^9 cells/l (4.0–11.0), platelet count 377×10^9 cells/l (150–450). Prothrombin time 13.1 s (10–14.0), activated partial thromboplastin time 30.9 s (26.0–38.0), international normalisation ratio 1.1, fibrinogen 5.4 g/l (1.5–6.0); C reactive protein 13 mg/l (<6), sodium 135 mmol/l (136–145), potassium

4.4 mmol/l (3.5–5.1), urea 15.5 mmol/l (2.8–7.6), creatinine 143 umol/l (44–80); total protein 49 g/l (63–79), albumin 27 g/l (38–47), globulin 22 g/l (18–32), alkaline phosphatase 281 IU/l (32–132), alanine aminotransferase 34 IU/l (10–36), lactate dehydrogenase 507 IU/l (240–480), total bilirubin 5 umol/l (<16); Corrected calcium 2.31 mmol/l (2.19–2.54), phosphate 1.11 mmol/l (0.80–1.45). Stool microscopy was reported negative for bacteria, parasites and cysts. *Clostridium difficile* screening showed one stool sample to be positive for glutamate dehydrogenase but PCR for tox B gene was negative. The flexible sigmoidoscopy showed patchy inflamed and oedematous colonic mucosa extending from rectum to descending colon (figure 1).

Figure 1 Sigmoid colon of flexible sigmoidoscopy.

The histology from rectal biopsies (using Congo red) revealed amyloid deposition within lamina propria and within the walls of blood vessels, confirming the diagnosis of amyloidosis (figure 2).

CT-scan of abdomen revealed multiple lytic lesions in vertebral bodies suspicious of metastasis or myeloma (figure 3).

Further investigations showed low-immunoglobulin (Ig) levels IgG 5.1 g/l (5.3–16.5), IgA 0.48 g/l (0.80–4.0) and IgM 0.19 g/l (0.50–2.0). A 24 h urine collection for Bence-Jones proteins was positive with a high-protein level of 2.22 g/l (0.01–0.15). Urine immunofixation detected λ light chain. Serum immunofixation also revealed a λ free light chain monoclonal in the β -1 region 2g/l. Later on, bone marrow biopsy showed plasma cell myeloma.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of lower GI bleeding in this case includes inflammatory bowel disease, diverticular disease, infective and ischaemic colitis and colonic neoplasia.

TREATMENT

Treatment of GI bleeding caused by amyloidosis is difficult. Localised GI amyloidosis can be treated by surgical resections. Amyloid light-chain (AL) amyloidosis can be treated with chemotherapy and stem cells transplantation. After confirming the diagnosis of multiple myeloma, patient was referred to haematologist and started on chemotherapy.

Figure 2 Lamina propria showing apple green birefringence. Congo red $\times 100$.

OUTCOME AND FOLLOW-UP

The patient is under follow-up with haematologists.

DISCUSSION

Amyloidosis is a group of diseases, all characterised by deposition of protein fibrils with a β -sheet structure. Three types of systemic amyloidosis are important for the clinician: AA (related to underlying chronic inflammation), AL (related to underlying monoclonal light chain production) and ATTR amyloidosis (related to old age or underlying hereditary mutations of transthyretin).⁵

AL amyloidosis is associated monoclonal light chain in serum and urine with 15% of patients having multiple myeloma. It can affect the whole GI tract from mouth to anus. Endoscopically, amyloidosis of colon can appear as polypoid lesions, ulcerations or nodules.⁶

Amyloidosis of the colon presents with bleeding in 25–45% of patients affected. It is frequently located in recto-sigmoid and descending colon. The differential diagnosis of lower GI bleeding includes inflammatory bowel disease, diverticular disease, infective and ischaemic colitis, and colonic neoplasia.⁷

Diagnosis is confirmed with biopsies from the rectum. Histologically, using Congo-Red stain, amyloid appearance is red in normal light and apple green in polarised light.³

Treatment of GI bleeding caused by amyloidosis is difficult. Localised GI amyloidosis can be treated by surgical resections. AL amyloidosis can be treated with chemotherapy and stem cells transplantation.^{3 4 7}

In summary, AL amyloidosis is a rare cause of lower GI bleeding which can also present with common, non-specific complaints.⁸

Learning points

- ▶ GI amyloidosis is the rare cause of lower GI bleed.
- ▶ Multiple myeloma rarely presents with GI amyloidosis. Amyloidosis should be considered in those older than 30 years with unexplained diarrhoea, weight loss, autonomic dysfunction, malabsorption or proteinuria.
- ▶ Amyloidosis of GI tract can present with common, non-specific complaints.

Figure 3 CT scan of abdomen showing multiple lytic vertebral lesions.

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Competing interests None.

Patient consent Obtained.

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