

Editor's quiz: GI snapshot

An unexpected cause of iron deficiency

CLINICAL PRESENTATION

A 70-year-old man presented with progressive weight loss and intermittent lower abdominal pain. Bowel habit was unchanged, there was no overt blood loss and there were no constitutional symptoms. His medical history included vascular Parkinson's, hypertension, osteoarthritis, type 2 diabetes mellitus and colonic polyps. His medications included pantoprazole and clopidogrel.

On physical examination, his abdominal and digital rectal examinations were unremarkable. Laboratory tests revealed normocytic (mean cell volume 84.1 fL) hypoferritinaemia ($15\mu\text{g/L}$) with low haemoglobin (123g/L). Coeliac serology, other haematinics and immunoglobulin profile were normal. Urinalysis excluded haematuria.

Gastroscopy and duodenal biopsies were normal and urease test was negative for *Helicobacter pylori*. CT colonography identified a 22mm proximal sigmoid lesion with a depressed centre concerning for extension beyond the bowel wall and in keeping with a T2/T3a non-metastatic cancer. The only other relevant findings were significant sigmoid diverticulosis and a 7mm sessile polyp at the hepatic flexure. On subsequent endoscopy the proximal sigmoid lesion lay within a prominent diverticular segment; the overlying mucosa was very erythematous but smooth with a normal pit pattern (figure 1A,B). The central depression was more likely representative of a shallow diverticulum. On biopsy this felt relatively firm

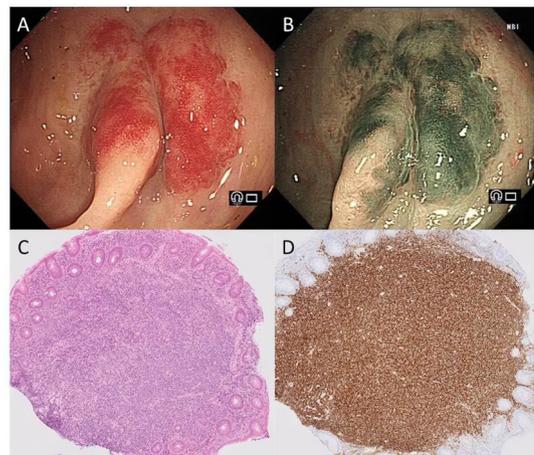


Figure 1 (A, B) Endoscopic view of the lesion in white light and narrow band imaging. (C) Low-power H&E stain showing nodular lymphoid infiltrate. (D) Low-power CD20 immunohistochemistry showing a markedly dominant B cell population.

and exposed mucosal/submucosal tissue that had a homogeneous white/grey appearance.

QUESTION

What is the aetiology of the colonic lesion?

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See page 1434 for question

ANSWER

Histopathological examination revealed morphological and immunophenotypic features of an extranodal marginal zone lymphoma of mucosa-associated lymphoid tissue (MALT lymphoma) (figure 1C,D).

First described in 1983 by Isaacson and Wright,¹ MALT lymphoma can affect the GI tract, respiratory tract, breast, conjunctiva and salivary glands. Although the prevalence of GI MALT lymphoma is on the rise, involvement of the colorectum is rare in comparison with other sites (<1%).²⁻⁴ Typically colonic MALT lymphoma affects patients in the fifth to seventh decade of life, with approximately 50% diagnosed on screening endoscopy.⁵ As in our case, there are reports of colonic MALT lymphoma presenting with weight loss and mild anaemia in the absence of B symptoms.²

The endoscopic appearance of colonic MALT lymphoma is variable, ranging from flat/epithelial and subepithelial lesions to polypoid and semipedunculated lesions.^{2,5} The surface characteristics can vary greatly; however, ulceration/erosions are rare in colonic compared with upper GI MALT lymphoma.²

Histologically it can be challenging to differentiate MALT lymphoma from florid reactive lymphoid tissue, especially in small biopsy specimens, owing to its lack of pathognomonic features. Clinicopathological correlation is therefore crucial. Colonic MALT lymphoma has an excellent prognosis and treatment depends on disease stage. Our patient was treated with rituximab monotherapy and responded well. The iron deficiency anaemia normalised within 6 months (haemoglobin 153 g/L, mean cell volume 94 fL, ferritin 80 µg/L) and repeat colonoscopy confirmed complete resolution of the lesion.

Thomas Edward Conley ¹, Timothy Andrews,² Arvind Arumainathan,³ Paul O'Toole,¹ Philip J Smith,¹ Sreedhar Subramanian¹

¹Gastroenterology, Royal Liverpool and Broadgreen Hospitals NHS Trust, Liverpool, UK

²Histopathology, Royal Liverpool and Broadgreen Hospitals NHS Trust, Liverpool, UK

³Haematology, Clatterbridge Cancer Centre NHS Foundation Trust, Wirral, UK

Correspondence to Dr Thomas Edward Conley, Gastroenterology, Royal Liverpool and Broadgreen Hospitals NHS Trust, Liverpool L7 8XP, UK; thomasconley@doctors.org.uk

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ORCID iD

Thomas Edward Conley <http://orcid.org/0000-0002-0604-0770>

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