CASE REPORT

Synchronous MALT lymphoma of the colon and stomach and regression after eradication of *Strongyloides stercoralis* and *Helicobacter pylori*

Kevin Singh,[1] Soren Gandhi,[2] Behzad Doratotaj[3]

[1]Department of Medicine, New York University School of Medicine, Woodhull Medical and Mental Health Center, Brooklyn, New York, USA
[2]Department of Gastroenterology and Hepatology, New York University School of Medicine, Woodhull Medical and Mental Health Center, Brooklyn, New York, USA
[3]Department of Hematology and Oncology, New York University School of Medicine, Woodhull Medical and Mental Center, Brooklyn, New York, USA

Correspondence to
Dr Kevin Singh,
Singh.kevin@gmail.com

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SUMMARY

Mucosa-associated lymphoid tissue (MALT) is vital for host immunological surveillance against pathogens. MALT lymphoma, also known as extranodal marginal zone B cell lymphoma, is a non-Hodgkin's lymphoma subtype that predominantly arises in the gastrointestinal tract. Chronic *Helicobacter pylori (H. pylori)* infection is a common cause of gastric MALT lymphoma, although other infections are reported in association with extragastric MALT lymphomas. To our knowledge, here we report the first case of synchronous MALT lymphomas of the colon and stomach in the presence of *Strongyloides stercoralis* and *H. pylori* infections that resolved after eradication of both organisms.

BACKGROUND

Mucosa-associated lymphoid tissue (MALT) is responsible for immunological surveillance and protection against pathogens in the respiratory, genitourinary and GI tracts (GITs).[1] Microscopically, the MALT consists of lymphoid follicles containing germinal centres enriched with antigen-presenting cells such as dendritic cells scattered among an array of B lymphocytes. When dendritic cells present environmental antigens such as bacterial antigens to B lymphocytes, polyclonal expansion may occur, leading to pathogen neutralisation, antibody production and antigenic memory.

However, repetitive stimulation of polyreactive B cells and Toll-like receptors by environmental antigens during chronic inflammatory states causes free radical damage to B lymphocytes and consequent mutations within these cell populations. As a result, pathways responsible for the regulation and prevention of apoptosis, especially nuclear factor kappa light chain enhancer of activated B cells (NF-κB), can be constitutively activated. This leads to malignant clonal transformation of the B lymphocytes and MALT lymphoma, also known as extranodal marginal zone B cell lymphoma, a subtype of non-Hodgkin's lymphoma (NHL).[2–5] MALT lymphomas can arise anywhere along the GIT and account for 23% of all primary GI NHLs, but most MALT lymphomas arise in the stomach. Colorectal MALT lymphomas are very rare, accounting for less than 1% of large bowel malignancies.[6] Even rarer are synchronous MALT lymphomas of the stomach and colon.[5 7–9]

MALT lymphomas are associated with specific chronic inflammatory conditions including autoimmune diseases such as Sjogren's syndrome and Hashimoto's thyroiditis, but they are also frequently associated with chronic infections.[3 10] *Helicobacter pylor*i (*H. pylori*) is classified by the WHO as a class I carcinogen and is associated with 90% of gastric MALT lymphomas.[11] Other organisms such as hepatitis C virus, *Chlamydia psittaci*, *Borellia burgdorferi* and *Campylobacter jejuni* are also reported in association with non-gastric MALT lymphomas.[12]

Strongyloides stercoralis (*S. stercoralis*), a parasite that infects 30–100 million people worldwide especially in sub-Saharan Africa, South America and South-East Asia, frequently causes chronic infections and has rarely been reported in association with colorectal adenocarcinoma and GIT T cell lymphomas.[13–16] To our knowledge, there are no reports of *S. stercoralis* infection in association with MALT lymphoma. Here we report an unusual case of synchronous colorectal and gastric MALT lymphomas in the presence of *H. pylori* and *S. stercoralis* infections. The lymphomas resolved after eradication of both organisms. As well as being unique, our case is a reminder that chronic inflammatory states such as infection in the GIT can predispose to the development of colorectal malignancies including MALT lymphoma.

CASE PRESENTATION

A previously healthy 60-year-old African-Caribbean man presented to the primary care clinic with a 1 year history of alternating constipation and diarrhoea. The patient reported having at least four bowel movements per day, intermittent haematochezia and fatigue. He did not report any abdominal pain, weight loss, dysphagia, odynophagia or early satiety. Prior to presentation, the patient worked as a fisherman, travelling every few months between the USA and the Caribbean islands.

Physical examination at presentation was unremarkable. His laboratory results revealed microcytic anaemia (haemoglobin (Hb) 11.3 g/dL (12.0–16.0 g/dL), mean corpuscular volume (MCV) 77.5 fL (80–100 fL)) and peripheral eosinophilia with 20% eosinophils. Iron studies revealed iron saturation 11% (20%–55%), ferritin 10 ng/mL (22–322 ng/mL), serum iron 31 μg/dL (60–180 μg/dL) and total iron-binding capacity 278 ug/dL (250–450 μg/dL),

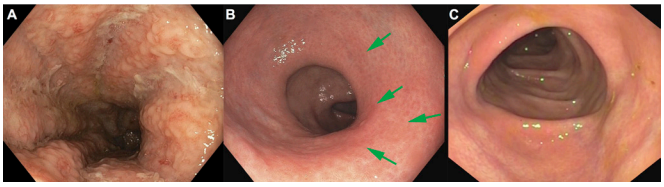

Figure 1 (A) Diagnostic colonoscopy revealing diffusely friable, nodular and erythematous mucosa. (B) Repeat colonoscopy 3 months after treatment with ivermectin revealing a less friable colonic mucosa with patchy areas of erythema (green arrows). (C) Colonoscopy repeated 1 year after diagnosis revealing a normal colon.

indicative of iron deficiency anaemia. The HIV test was negative. A colonoscopy performed 10 years earlier was normal.

DIFFERENTIAL DIAGNOSIS

Due to the patient's age and longstanding history of diarrhoea, constipation, haematochezia and iron deficiency anaemia, a colorectal malignancy was suspected. However, the differential diagnosis for chronic diarrhoea is vast and includes less likely causes such as Crohn's disease, ulcerative colitis, irritable bowel syndrome, microscopic colitis, coeliac disease and chronic infections including *Clostridium difficile*, *Aeromonas*, *Campylobacter* and Whipple's disease.[17]

INVESTIGATIONS

Our patient underwent diagnostic colonoscopy that revealed a diffusely friable, nodular and erythematous mucosa throughout the colon (figure 1A). Because these findings were suggestive of colitis and there was peripheral eosinophilia, our evaluation of colitis included three stool collections for ova and parasite assays that revealed infection with *S. stercoralis* and, later, serum immunoglobulin (Ig)G antibodies were detected, further confirming *S. stercoralis* infection. The *S. stercoralis* infection was treated with two doses of ivermectin.

Subsequently, histopathological examination of colonic mucosal biopsies showed sheets of small, angulated B lymphocytes over-running scattered lymphoid follicles (figure 2A,B). The atypical lymphocytes expressed cluster of differentiation (CD)20, CD79a and B-cell lymphoma (Bcl)-2 antigens without expression of CD3, CD10, Bcl-6 or cyclin D1 (figure 3), and there were gene rearrangements of mainly kappa Ig light chains (IgL). Ig heavy chain (IgH) rearrangements were detected for predominantly IgG restriction. These findings were immunohistopathologically compatible with low-grade MALT lymphoma. For staging purposes, a positron-emission tomography (PET) scan did not detect involvement of other organs. Interdisciplinary Tumour Board discussions that emphasised the National

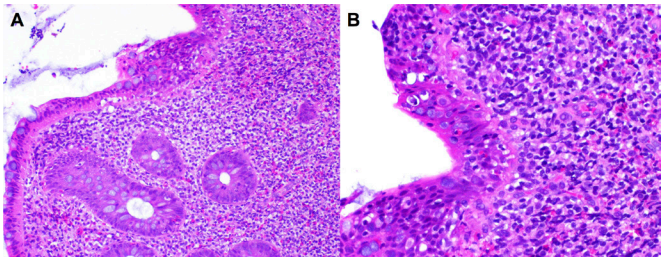

Figure 2 Histopathological examination of colonic mucosal biopsies showed sheets of small, angulated B lymphocytes over-running scattered lymphoid follicles. (A) 4× and (B) 40×.

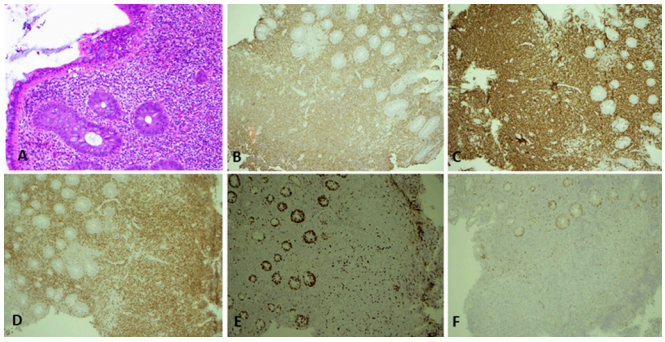

Figure 3 Representative images of H&E and immunohistochemical staining. (A) H&E staining of the colonic epithelium (20×). (B) CD20 positivity in tumour cells (10×). (C) CD79a positivity in tumour cells (10×). (D) Bcl-2 positive tumour cells (10×). (E) Low Ki-67 proliferation index (<5%). (F) Cyclin D1 negative tumour cells (10×). Bcl, B cell lymphoma; CD, cluster of differentiation.

Comprehensive Cancer Network (NCCN) recommendations on the treatment of extragastric MALT lymphomas resulted in an expectant management plan of follow-up with close observation with serial history and physical examinations, repeat colonoscopy and imaging.

After treatment with ivermectin, the patient's diarrhoea resolved, and eradication of *S. stercoralis* was confirmed with a repeat stool assay 1 month later. Repeat colonoscopy 3 months after presentation showed that the colonic mucosal abnormalities had significantly improved with only areas of patchy erythema present (figure 1B). However, the biopsies still revealed MALT lymphoma.

Due to the known high incidence of gastric MALT lymphoma and to investigate whether the patient had synchronous gastric MALT lymphoma, an oesophagogastroduodenoscopy (EGD) was performed to screen for upper GIT MALT lymphoma. The mucosa appeared mildly erythematous (figure 4A), and biopsies revealed MALT lymphoma in the presence of *H. pylori* infection. The patient was treated with 14 days of amoxicillin, clarithromycin and omeprazole to eradicate *H. pylori*, which was undetectable on a stool *H. pylori* antigen test 1 month after completion of therapy.

TREATMENT

S. stercoralis infection was treated with two doses of ivermectin, and *H. pylori* infection was treated with 14 days of amoxicillin, clarithromycin and omeprazole.

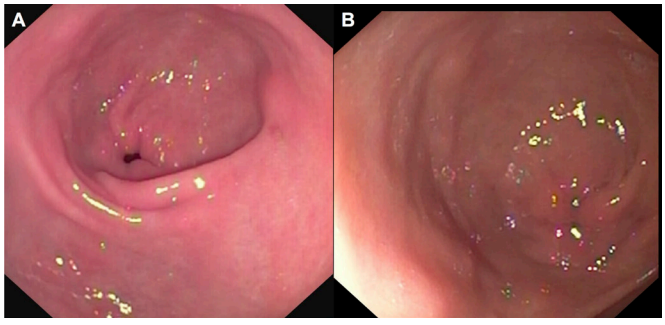

Figure 4 (A) Gastric mucosa revealed erythematous areas later diagnosed as mucosa-associated lymphoid tissue (MALT) lymphoma. (B) Endoscopic resolution of the MALT lymphoma in the stomach 1 year after *Helicobacter pylori* eradication therapy.

OUTCOME AND FOLLOW-UP

One year after initial presentation, a repeat EGD and colonoscopy showed no evidence of MALT lymphoma at either site (figures 1C and 4B). The patient has now been asymptomatic for 3 years without evidence of recurrence with imaging or endoscopy with random biopsies.

DISCUSSION

MALT lymphomas account for 7% of all newly diagnosed lymphomas and can, on rare occasions, transform into aggressive diffuse large Bcl.[2] Most patients are clinically asymptomatic at presentation, so the diagnosis is easily missed. However, a few patients present with a combination of vague and generalised signs and symptoms including abdominal pain, chronic fatigue, anaemia, night sweats, weight loss and a positive occult blood stool assay that can suggest the suspected diagnosis. Some patients may also have site-specific symptoms similar to chronic gastritis or peptic ulcer disease such as epigastric pain, nausea, vomiting, early satiety, haematemesis or melaena with gastric MALT lymphomas, while colonic MALT lymphomas often cause constipation, diarrhoea and haematochezia.[18 19] Hence, the symptoms of other colorectal cancers and MALT lymphomas overlap, and these patients with 'red flag' symptoms such as anorexia, weight loss, unexplained iron deficiency anaemia, non-specific abdominal pain and changes in the frequency or calibre of the stool should be referred for endoscopic evaluation.

The gold-standard test for the diagnosis of gastrointestinal MALT lymphoma is the histopathological evaluation of biopsies obtained from endoscopy or surgery.[20] Common gross manifestations are ulcers, polyps or a protruding mass, although the mucosa can appear nodular, as observed here.[21–23] On histopathological examination, atypical B lymphocytes with irregular nuclei and hyperchromasia infiltrating the muscularis propria are suggestive of MALT lymphoma.[24] Non-neoplastic marginal zone B lymphocytes and mantle cell lymphomas are in the histological differential diagnosis. Although non-neoplastic marginal zone B lymphocytes and neoplastic cells from MALT lymphoma express CD20 and IgM, IgA and IgG (in order of decreasing antibody prevalence), a dense CD20+ lymphocyte infiltrate between glands and reactive follicles is highly suggestive of MALT lymphoma.[25] Immunohistochemistry is useful for distinguishing MALT lymphoma from mantle cell lymphoma because MALT lymphoma cells do not express IgD, CD5, CD10, Bcl-6 or cyclin D1, unlike mantle cell lymphoma.

Although *H. pylori* eradication is a clear-cut first-line treatment for gastric MALT lymphoma due to their strong association, the management of colorectal MALT lymphoma is complicated and unclear. Most commonly, symptomatic patients with low-grade colorectal MALT lymphomas have been treated by endoscopic resection, surgery, immunotherapy (such as with rituximab) and even *H. pylori* eradication therapy in the absence of *H. pylori* infection, suggesting other aetiologies such as other chronic infections or autoimmune disease.[8 12 26–30] Given that: (1) MALT lymphomas are indolent tumours; (2) some of these therapeutic interventions may place patients at risk of serious post-therapeutic complications and (3) an absence of clear guidelines, especially for rare MALT lymphomas involving the colon, the NCCN and individual case reports have recommended that asymptomatic patients or those with low-grade MALT lymphomas can be monitored with serial history and physical examinations, repeat imaging and haematological testing. However, these options should be weighed against the patient's age, comorbidities and preferences.[31–33]

Our case of MALT lymphoma is rare since the association between MALT lymphoma and *S. stercoralis* infection has not been reported. This infection starts after the patient comes into contact with soil containing filariform larvae that subsequently penetrate the skin, but the disease also can be transmitted via the faecal–oral route or after anal intercourse.[34] Clinically, patients with *S. stercoralis* infection often lack symptoms and may only have peripheral eosinophilia. However, when patients are symptomatic, the clinical features frequently reflect the parasite's life cycle.

In our patient's case, the exact pathogenesis of the colonic MALT lymphoma is unclear, because the symptoms and endoscopic appearances resolved after treatment with ivermectin but microscopic evidence of colonic MALT lymphoma persisted at the time the biopsies were taken. It is therefore possible that *H. pylori*, *S. stercoralis* or another unidentified mechanism was responsible for the colonic MALT lymphoma.[35] *H. pylori*, a known cause of gastric MALT lymphoma and gastric and colorectal adenocarcinomas, could have potentiated the development of colonic MALT lymphoma.[36] However, this possibility is relatively unlikely because it is generally thought that extragastric MALT lymphomas are unrelated to *H. pylori* infection, but are rather being associated with other organisms that commonly colonise or infect the colon like *S. stercoralis*.[6 29 37–40] Therefore, it remains possible that *S. stercoralis* contributed to the colonic MALT lymphoma via an antigen-driven mechanism in the colon. Due to the short interval between treatment and colonic biopsies at 3 months, our case should not be construed as treatment failure since there may have been insufficient time for the MALT lymphoma to completely resolve at the microscopic level because such changes may take as long as 2 years to completely regress.[41 42] We believe that *H. pylori* contributed to MALT lymphoma development in the stomach but that *S. stercoralis* was unlikely to be an incidental finding and opportunistic infection secondary to the MALT lymphoma, since patients with MALT lymphoma do not tend to develop clinically significant immunosuppression and opportunistic infections like those with human T cell lymphotropic virus type 1 and related lymphomas.[3 43 44]

In summary, here we report a unique case of *S. stercoralis* infection that possibly led to the development of colonic MALT lymphoma. The patient's symptoms resolved and the lymphoma regressed endoscopically after treatment of the *S. stercoralis* infection. This patient also had synchronous gastric MALT lymphoma caused by *H. pylori* infection that also regressed with standard treatment. Although there are currently no consensus guidelines on the staging and monitoring of recurrent disease

Learning points

- This is the first reported case of colonic mucosa-associated lymphoid tissue (MALT) lymphoma presenting synchronously with gastric MALT lymphoma in the presence of *Strongyloides stercoralis* and *Helicobacter pylori* infections, respectively.
- *H. pylori* and possibly *S. stercoralis* may contribute to MALT lymphomagenesis through repeated antigenic stimulation or chronic inflammation.
- Antimicrobials are safe, inexpensive and non-invasive therapeutic options in low-grade MALT lymphomas associated with infection.

in this type of setting, our patient remained asymptomatic and later had a normal EGD and colonoscopy. This is the first case describing the possibility of *S. stercoralis* contributing to the development of MALT lymphomas, and additional studies are warranted to confirm this possibility. In the future, additional research might also be helpful to establish guidelines on the treatment and post-therapeutic monitoring of MALT lymphomas so that the complexity of managing rare presentations as seen in our patient can be simplified for clinicians.

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