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Case Report

Collagenous gastritis: A rare entity

Faten Limaiem, Sabeh Mzabi

Department of Pathology, Mongi Slim Hospital, La Marsa, Université de Tunis El Manar, Faculté de Médecine de Tunis, 1007, Tunisa

Address for correspondence:
Faten Limaiem, Department of Pathology, Mongi Slim Hospital, La Marsa, Tunisia.
Tel.: +216 96 55 20 57,
E-mail: fatenlimaiem@yahoo.fr

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ABSTRACT

Collagenous gastritis is a rare entity of unknown etiology characterized histologically by the presence of a thick subepithelial collagen band associated with an inflammatory infiltrate of gastric mucosa. A 40-year-old male presented with a history of chronic intermittent abdominal pain for about 6 months. Physical examination was unremarkable, and biological tests were within normal range. The patient underwent esophagogastroduodenoscopy and colonoscopy which showed a nodular mucosa of the stomach. Biopsies of the duodenum and colon were unremarkable. However, biopsies of the gastric fundus revealed a mild chronic gastritis characterized by lymphocytic and plasma cell infiltration of deep mucosa, without lymphoid follicle formation or active inflammation. No microorganisms were identified on routine hematoxylin and eosin or Giemsa-stained sections. Subepithelial collagen in the gastric biopsies was thickened and showed entrapped capillaries. Subepithelial collagen was highlighted by Masson's trichrome staining and was negative for amyloid by Congo Red. In the areas containing thickened collagen, there were no intraepithelial lymphocytes. The final pathological diagnosis was collagenous gastritis. Collagenous gastritis is an extremely rare disease, but it is important to recognize its characteristic endoscopic and pathologic findings to make a correct diagnosis. Specific therapy for this rare entity has not yet been established.

KEY WORDS: Collagenous gastritis, collagen band, histopathology

INTRODUCTION

Collagenous gastritis is a rare and poorly characterized disease. It is defined histologically by the presence of subepithelial deposition of collagen <10 μm in thickness, entrapping dilated capillaries and inflammatory cells, similar to collagenous colitis [1]. To date, more than 50 patients with collagenous gastritis have been reported in the English-language literature, mainly as reports of one or two cases [2-4]. The three largest previous series of collagenous gastritis comprised 6, 12, and 40 patients respectively [5,6]. In this paper, we report a new case of collagenous gastritis. Our aim was to highlight the clinicopathological features of this rare entity.

CASE REPORT

A 40-year-old male presented with a history of chronic intermittent abdominal pain for about 6 months. Physical examination was unremarkable, and biological tests were within normal range. Imaging studies including abdomen X-ray and ultrasound were normal. The patient underwent esophagogastroduodenoscopy and colonoscopy. The former showed a nodular mucosa of the stomach. Biopsies of the duodenum and colon were unremarkable.However, biopsies of the gastric fundus revealed a mild chronic gastritis characterized by lymphocytic and plasma cell infiltration of deep mucosa, without lymphoid follicle formation or active inflammation. No microorganisms that were morphologically compatible with Helicobacter pylori were identified on routinehematoxylin and eosin or Giemsa-stained sections. Subepithelial collagen in the gastric biopsies was thickened and showed entrapped capillaries [Figure 1A]. Subepithelial collagen deposition was localized to the superficial epithelium and did not involve the gastric foveolae. Subepithelial collagen was highlighted by Masson's trichrome staining [Figure 1B] and was negative for amyloid by Congo red staining. In the areas containing thickened collagen, there were no intraepithelial lymphocytes. The final pathological diagnosis was collagenous gastritis. Omeprazole 20 mg (once a day orally during 1 month) was prescribed to the patient who was lost to follow-up.

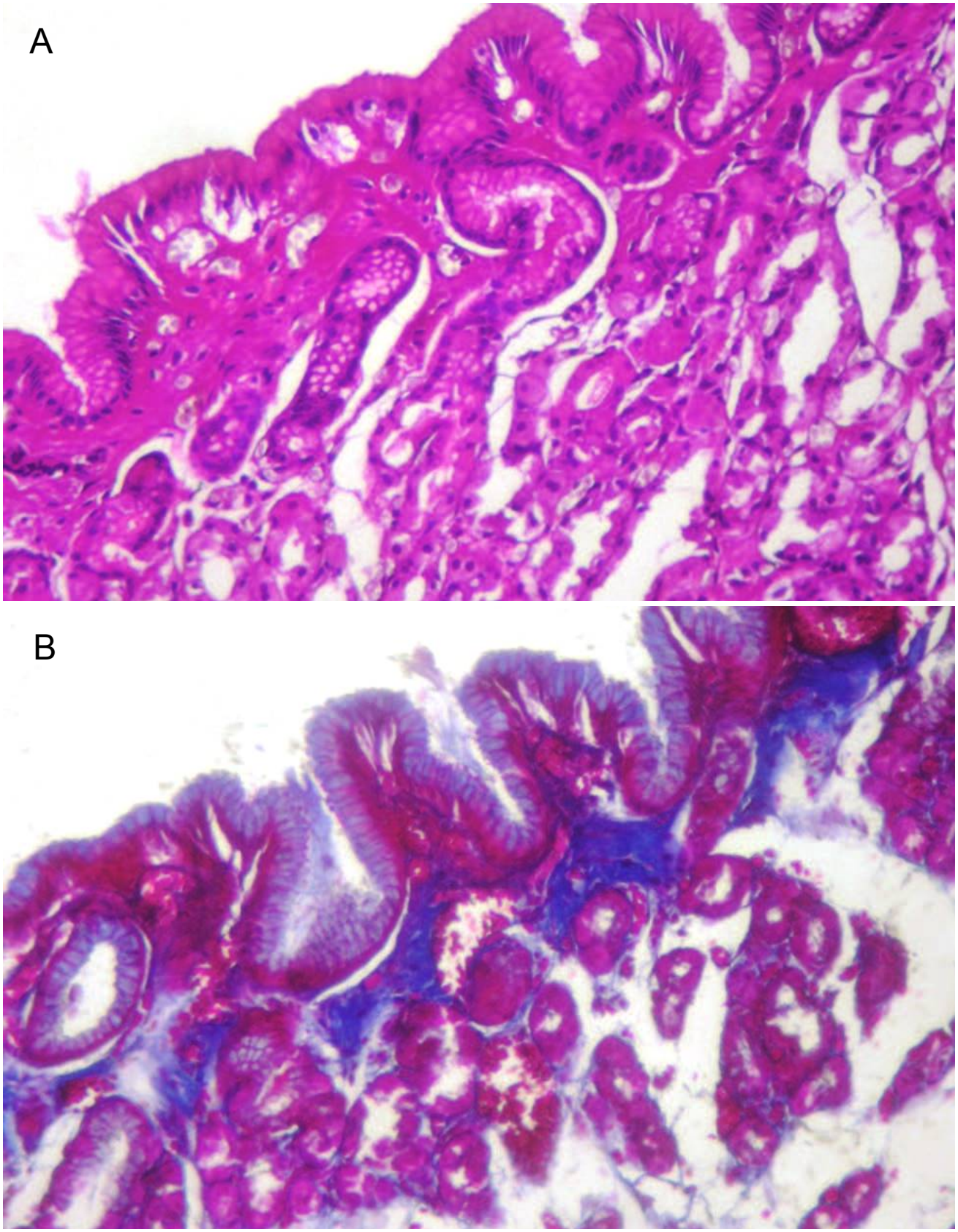

Figure 1: [A] Histological examination of the biopsy specimen of the gastric fundus revealed subepithelial collagenous band >10 µ (Hematoxylin and eosin, x400). **[B]** Subepithelial collagen band with entrapped capillaries highlighted by Masson's trichrome staining (Masson's trichrome, x400).

DISCUSSION

On the basis of patient age and clinical presentation, Lagorce-Pages *et al*. recognized two general clinical subgroups in collagenous gastritis [5]. The first group included children with isolated collagenous gastritis, a nodular stomach on gastroscopy and iron deficiency anemia, hypothesized to be due to bleeding from superficial capillaries entrapped in collagen [5]. The second group consisted of adults presenting with chronic watery diarrhea and displaying associated collagenous colitis [4-6]. While a significant proportion of patients with collagenous gastritis will fall into one of these two categories, a number of reported patients do not easily fit in one of these categories as it was the case in our patient [5]. Collagenous gastritis has a characteristic of female predominance both in pediatric and adult cases [4]. Although an association with autoimmune diseases, particularly celiac sprue, has been noted, the pathogenesis of collagenous gastritis remains uncertain [4,5]. The different age groups and presentations raise the possibility that more than one etiologic mechanism may be responsible. Increased stromal collagen deposition occurs in other disorders of the digestive system, including eosinophilic esophagitis and IgG4-related disease, in particular, autoimmune pancreatitis [2-6]. In addition to the diagnostic irregular subepithelial collagen deposition, three distinct inflammatory patterns can be seen including a lymphocytic gastritis-like pattern, an eosinophil rich pattern and an atrophic pattern [4]. Awareness of the association of these three inflammatory patterns with collagenous gastritis may prompt pathologists to look carefully for collagen in subtle cases. Collagen often persists for years but may resolve histologically in a minority of patients [2-4]. The etiology of collagenous gastritis remains uncertain, but investigations seem to exclude a mechanism related to increased eotaxin or IgG4 [2-6]. Tenascin immunohistochemistry appears to be a sensitive marker for the disease and is an area for future investigation in this disorder. The differential diagnosis of collagenous gastritis includes fibrosis associated with autoimmune gastritis or radiation therapy in which the collagen deposition is usually more diffuse and not specifically subepithelial. Similarly, patients with scleroderma show fibrosis at all levels of the mucosa, and it may involve deeper levels of the bowel wall as well. Artifact of sectioning (tangential sectioning) is another important differential diagnosis of collagenous gastritis [4]. There have been no reports of carcinoma, lymphoma, or definitive dysplasia developing in association with collagenous gastritis [6]. There are no established treatment protocols for collagenous gastritis, and resolution of the abnormalities either endoscopic or histologic has not been documented. Various therapies have been tried for collagenous gastritis including corticosteroids, ranitidine, omeprazole, misoprostol, sucralfate, aminosalicylates, sulfasalazine, cholestyramine and a hypoallergenic diet with marginal results [7,8]. The long-term outcome for collagenous gastritis remains uncertain. A large series of collagenous gastritis indicates that the process persists for years histologically in the majority of patients [4].

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