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A rare cause of colonic stricture: Amebiasis

Kolonik striktürün nadir bir sebebi: Amebiasis

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A 41-year-old man presenting with lower abdominal pain, constipation, abdominal distention, fever (37.5°C) and fatigue was evaluated, and a mass localized to the left lower abdomen was identified. Radiographic and colonoscopic examination revealed a stricture 10 cm in length localized to the sigmoid-descending colon junction. The diagnosis of amebiasis was confirmed by histological examination of a biopsy specimen taken from the stricture and stool examination. One month after the initiation of metronidazole treatment, complete clinical and laboratory improvement was observed. In the differential diagnosis of colonic stricture, amebiasis should also be considered.

Key words: Amebiasis, colonic stricture

Karın alt bölgesinde ağrı, kabızlık, karında şişkinlik, ateş ve yorgunluk şikayetleriyle müracaat eden hastanın karın sol alt bölgesinde kitle tespit edildi. Radyolojik ve kolonoskopik inceleme sonrası sigma-inen kolon birleşkesinde 10 cm uzunluğunda striktür saptandı. Striktürden alınan biyopsi ve gaitanın parazitolojik incelemesi sonrası amebiasis tanısı konuldu. Bir aylık metranidazol tedavisi sonrası tam klinik ve radyolojik düzelme gözlendi. Kolonik striktürlerin ayırıcı tanısında amebiasis de düşünülmelidir.

Anahtar kelimeler: Amebiasis, kolon striktürü

INTRODUCTION

Amebiasis is an infectious disease caused by the protozoan Entamoeba histolytica, which is common in tropical countries and infects 10% of the world's population, resulting in approximately 100, 000 deaths per year. Amebiasis may involve any part of the bowel, but it has a predilection for the cecum and ascending colon (1). Colorectal amebiasis may present in many different clinical forms including the asymptomatic carrier state (2), acute amebic colitis, toxic mega colon (1), and fulminant necrotizing colitis with intestinal bleeding and perforation (3, 4). While tissue necrosis is often encountered in amebic colitis, chronic inflammatory response with formation of a pseudotumor or ameboma (5, 6) and colonic stricture (7) by excessive granulation tissue on the intestinal wall is less common. A quick diagnosis is possible by examining the stool for trophozoites and cysts, but it may be negative (1, 2). The classical colonoscopic appearance is that of discrete areas of ulceration covered by exudate with normal intervening mucosa (1, 8). Rectal biopsy is useful and accurate as it may reveal the presence of the parasites and at the same time exclude the possibility of a carcinoma (3-5). We reported a case with colonic stricture simulating colon cancer.

CASE REPORT

A 41-year-old man was referred to our hospital with a chief complaint of lower abdominal pain, constipation, abdominal distention, fever (37.5°C) and fatigue for four weeks. There was no weight loss. His family history was unremarkable except for the death of his uncle two years ago due to colon cancer. Physical examination revealed slight tenderness and mass localized to the left lower abdomen. Laboratory tests were normal except for leukocytosis (13,000/mm^3) and elevated erythrocyte sedimentation rate (52 mm/hour). All biochemistry parameters including tumor markers were

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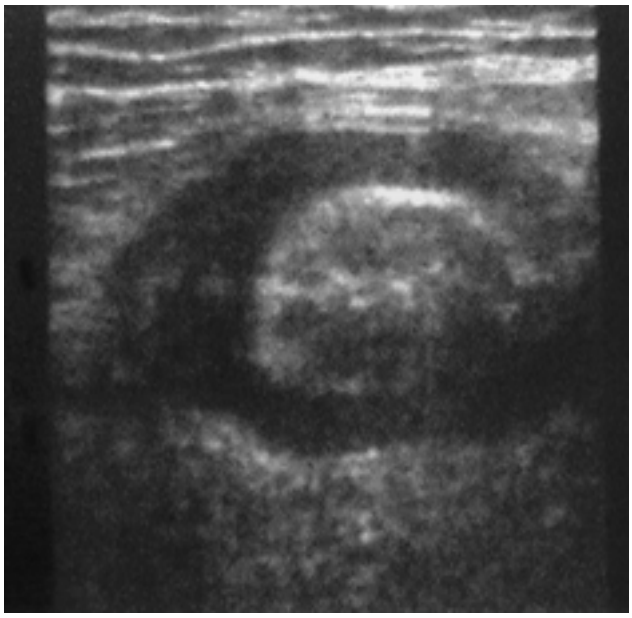

Figure 1. Ultrasonographic appearance of circular wall thickness at the sigmoid-descending colon junction

within the normal limits. The stool was positive for cysts of Entamoeba histolytica. Ultrasonography showed marked bowel wall thickness in the sigmoid-descending colon junction (Figure 1). Maximal wall thickness was 20 mm. Computerized abdominal tomography showed bowel wall thickness and luminal narrowing in the sigmoid-descending colon junction. Barium enema examination revealed stricture 10 cm in length localized to the defined bowel segment (Figure 2). Colonoscopy showed stricture and markedly diffuse edematous mucosa localized to the 50th cm of the sigmoid colon. We could not traverse the stricture because of

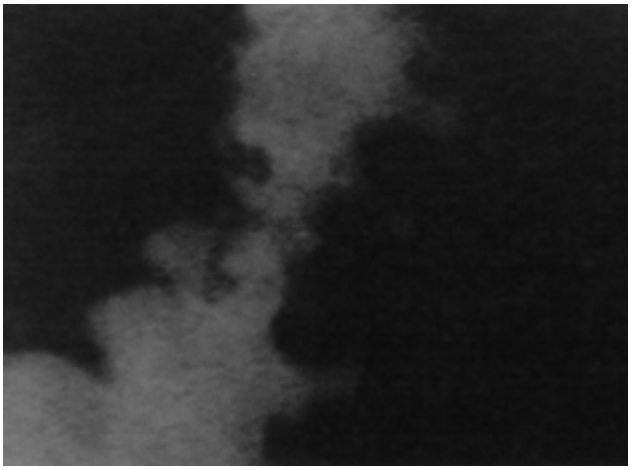

Figure 2. Radiographic appearance of stricture at the sigmoid-descending colon junction

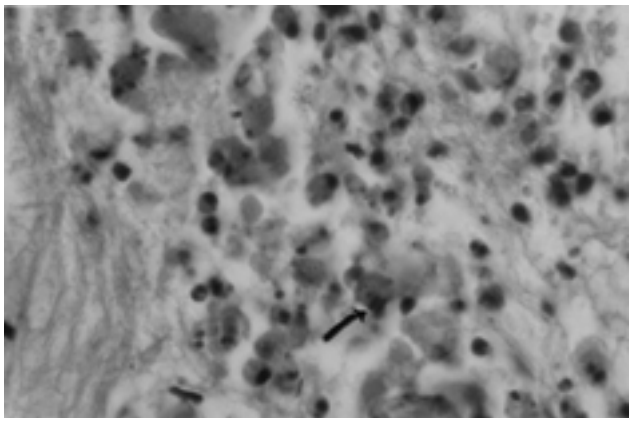

Figure 3. Numerous Entamoeba histolytica trophozoites, some containing ingested erythrocytes (arrow) with sparse inflammatory cells in the biopsy specimen taken from colonic stricture (H&EX400)

severe luminal narrowing. However, no notable findings were observed below the defined lesion. Histological examination of a biopsy specimen taken from the edematous mucosa by colonoscopy revealed inflammatory cell infiltration without ulceration and numerous trophozoites of Entamoeba histolytica, some of which contained ingested erythrocytes (Figure 3). The patient was treated with metronidazole (1500 mg/day) and ciprofloxacin (1000 mg/day) for 15 days. His symptoms completely resolved and abnormal laboratory findings improved after completion of therapy. One month after the initiation of therapy, abdominal

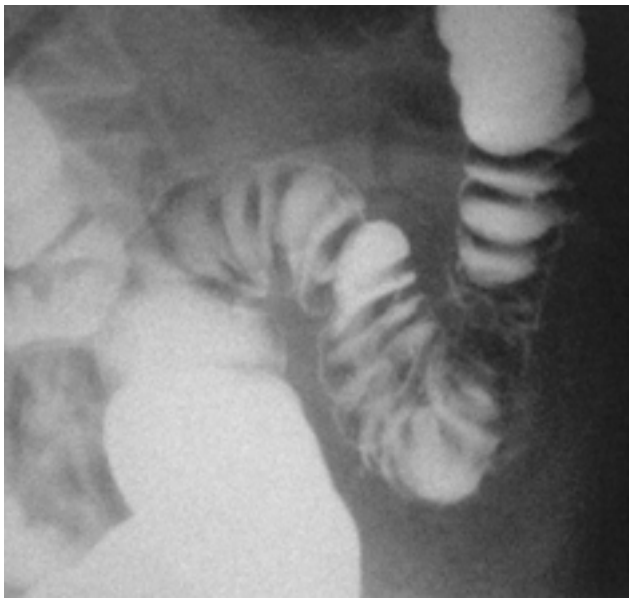

Figure 4. Stricture localized to the sigmoid-descending colon disappeared one month after the initiation of therapy

ultrasonography and tomography showed significant regression in the bowel wall thickness (maximal bowel wall thickness was 8 mm), barium enema study did not show any stricture (Figure 4) and colonoscopy did not reveal any remarkable findings from the rectum to the cecum except for minimal mucosal edematous appearance localized to the sigmoid-descending colon junction. During 10 months' follow-up, the patient was asymptomatic.

DISCUSSION

Entamoeba histolytica is endemic in most tropical and subtropical countries (9). Infection usually begins with the ingestion of the cysts in food or water that is contaminated by human feces. Many individual with Entamoeba histolytica infection have no symptoms and can clear their infection without any signs of disease. Patients with amebic colitis present with bloody diarrhea, abdominal pain and tenderness. Multiple small volume mucoid stools are common, but profuse, watery diarrhea might be noted. Fever, weight loss and anorexia can be present. Occasionally, individuals develop fulminant amebic colitis, with profuse bloody diarrhea, fever, and widespread abdominal pain often with peritoneal signs (1). In addition, colorectal amebiasis may occasionally manifest as an ameboma which can resemble a carcinoma (5, 10, 11), and manifest as colonic strictures (7, 12, 13). In our patient, the presence of familial history of colon cancer, localized mass lesion, significantly thickened colonic wall and severe narrowing in colonic lumen and obstruction symptoms supported the possibility of malignant stricture. But, the presence of leukocytosis, elevated erythrocyte sedimentation rate, fever and tenderness over left lower abdomen were consistent with co-existence of infection. Occurrence of amebiasis is relatively high in the patient's city of residence, but amebomas and amebic strictures are rare. The patient had no previous history of amebic colitis. Although we identified Entamoeba histolytica trophozoite in the patient's stool, we did not consider amebic stricture before exclusion of other possible diagnoses.

Colonoscopic features of ameboma may be erosions, ulcers (6, 8, 13), edema, and nodularity that narrow the lumen (6), ring-like stenosis (14), or polypoidal stricture (5) within the colorectum. Accurate clinical diagnosis and early histological confirmation are mandatory in order to institute rational management. The co-existence of an underlying malignant lesion in association with an inflammatory lesion in an endemic area should always be considered, particularly if it fails to respond to empirical therapy within a short period of time (7). In our patient, colonoscopic features were more compatible with inflammatory mass lesion rather than neoplasm. Histological examination of a biopsy specimen taken from the lesion revealed severe inflammatory infiltration with numerous trophozoites of Entamoeba histolytica, and there were no histological findings to support the colon cancer. In addition, complete clinical and radiological improvement after metronidazole treatment excluded the possibility of the co-existence of an underlying malignant lesion in association with amebic stricture in our case.

Non-malignant lesions accounted for strictures in two-thirds of patients and included amebiasis, tuberculosis, ischemic colitis, diverticulosis, radiation colitis, nonspecific colitis, ulcerative colitis/Crohn's colitis and other lesions (7). The typical mucosal alterations that are specific for ulcerative colitis are continuous involvement from the anus and proximal extension, diffuse cryptitis, erosions, and loss of vascular pattern, and for Crohn's disease are skip lesions, cobble stoning, aphthous ulcers, longitudinal ulceration, ileocecal involvement and anal lesions. Benign or malignant strictures in the colon can be seen in patients with ulcerative colitis and Crohn's disease. To distinguish between malignant and benign stricture, multiple biopsy specimens should be taken from the edges and the lumen of the strictures (15). In our patient, we considered Crohn's disease and not ulcerative colitis in the differential diagnosis because of clinical history and the presence of localized bowel wall thickness with stricture formation. However, the colonoscopic features, including the presence of diffuse mucosal inflammation without ulceration, skip lesions, cobble stoning or perianal lesions, were not compatible with Crohn's disease, and the biopsy specimen taken from the stricture contained no histological findings such as granuloma formation that can be considered indicative of Crohn's disease. Complete clinical, radiological and endoscopic improvement without specific treatment for inflammatory bowel disease also excluded the possibility of Crohn's disease.

Diverticular disease is rare in individuals younger than 50 years, and most affected patients have multiple diverticula. Pa rtial colonic obstruction can occur during an attack of acute diverticulitis

because of the relative luminal narrowing resulting from pericolic inflammation or compression by an abscess. Recurrent attacks of acute diverticulitis, which may be sub-clinical, can initiate progressive fibrosis and stricturing of the colonic wall (16). We considered stricture formation caused by diverticulitis with co-existence of amebiasis in the differential diagnosis. However, absence of any other diverticula outside the stricture, the relatively younger age of the patient (when considering development of diverticular disease) and lack of peri-structural abscess formation did not support stricture formation secondary to diverticulitis.

Ischemic colitis is generally more common among the elderly with considerable cardiovascular morbidity. The typical clinical presentation is acute sudden abdominal pain and distention with bloody diarrhea. Common early radiographic signs include thickening with thumb-printing of the bowel wall and late signs are ulceration and stricture. Findings vary greatly depending on the stage at which colonoscopy is performed. At the outset, purplish blebs representing mucosal and submucosal hemorrhage may be seen. As the hemorrhage is resorbed, varying degrees of necrosis, inflammation, ulceration and mucosal sloughing occur, resembling ulcerative colitis or Crohn's disease (17). Our patient had no cardiovascular disease that can cause ischemic bowel disease. We did not consider ischemic colitis in our patient because clinical presentation, radiographic findings, colonoscopic appearance of colonic mucosa and response to antimicrobial treatment were not compatible with ischemic colitis.

It has been reported that gray-scale ultrasonography can demonstrate wall thickening of the cecum, sigmoid colon and rectum of a patient with amebic colitis. Maximum wall thickness was reported as 29.9 mm at the cecum, 26.5 mm at the sigmoid colon and 18.1 mm at the rectum. The layered structure of the wall was conserved, and the submucosal layer showed remarkable thickening (18). In the present case, ultrasonography revealed marked and discrete thickening of the bowel wall (maximal wall thickness was 20 mm) which corresponded well to the findings of barium enema and colonoscopic examination. One month after the initiation of therapy, there was significant regression in the maximal bowel wall thickness (from 20 mm to 8 mm). Ultrasonography may be a useful and noninvasive method for detection and follow-up of bowel wall abnormality of amebic stricture.

In conclusion, in the differential diagnosis of colonic stricture, amebiasis should also be considered.

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