Histopathological investigation confirmed the diagnosis (Figure 2). No history of trichotillomania or evidence of hair loss was found during the physical examination at the time of admission. Psychiatric interview after the surgery revealed no major psychotic disorders.

Among the causes of appendicitis, foreign bodies are the most uncommonly reported pathology. Different types of exotic foreign bodies have been found in the lumen of acutely inflamed appendices; however, there are few reports on trichobezoars as the cause of obstructive appendicitis (1,2). Trichobezoars are classically described as concentrations of hair fibers and are often found in young women with psychotic disorders or in mentally retarded children (3). Trichobezoars are usually noted in patients who have a compulsion to pull out their hair, termed trichotillomania (3, 4). There have been only a few reports thus far on trichobezoar-associated appendicitis, which have all mentioned a history of trichotillomania or other psychosomatic disorders, unlike the present case, in whom no other accompanying disorders were determined. We would like to emphasize not only the rarity of appen-

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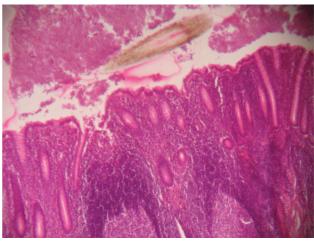


Figure 1. Cross-section of the appendix wall tissue revealing intraluminal hair shaft.

dicitis due to trichobezoar formation, but also the importance of considering a trichobezoar formation during the investigation of a patient with gastrointestinal obstruction symptoms or any possible emergent manifestation of appendicitis, even in the absence of any previous history of trichophagia.

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To the Editor,

Gastrointestinal involvement is common in patients with systemic amyloidosis; however, the ma-

Address for correspondence: Kemal DENIZ Erciyes University, Faculty of Medicine, Department of Pathology 38039 Kayseri, Turkey Fax: + 90 352 651 61 77 E-mail: drkdeniz@yahoo.com jority of the patients with gastrointestinal amyloidosis are asymptomatic (1). Different types of co-

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litis, including ischemic colitis and hemorrhagic bullous colitis, have been described during the course of this disease (2,3). Amyloid colitis mimicking collagenous colitis has been reported rarely.

A 31-year-old male admitted to our hospital with watery diarrhea for 2 months. He had a 15-year history of juvenile rheumatoid arthritis and a 4year history of microalbuminuria. He had been taking prednisolone 5 mg per day regularly for 5 years. Physical examination was unremarkable except for bilateral pretibial 2+ pitting edema. Laboratory tests showed proteinuria (19 g) and increased serum urea nitrogen (67 mg/dl) and creatinine (6.7 mg/dl) levels. Thyroid function test was normal. Abdominal ultrasonography (USG) demonstrated bilateral increased renal dimensions. The rest of the radiological examination, including chest X-ray and abdominal computed tomography, were within normal limits. Diagnostic work-up for diarrhea revealed negative stool cultures for Clostridium difficile and Salmonella, Shigella and Campylobacter species. Rectosigmoidoscopy displayed mild mucosal erythema, and endoscopic examination was performed. Light microscopic examination of the mucosal biopsy showed eosinophilic subepithelial thickening, with a maximum thickness of 20 µm, and detachment of the surface epithelium (Figure 1A). Subepithelial eosinophilic band showed Congo red positivity (Figure 1B), and it exhibited apple green birefringence under polarized light. Immunohistochemically, amyloid deposition further proved to be AA type (Figure 1C). Currently, the patient has chronic renal failure and is under treatment with hemodialysis.

The gastrointestinal tract is one of the most commonly involved organs during the course of systemic amyloidosis. The prevalence of gastrointestinal involvement varies from 35% to 100%. It is preferentially deposited in the interstitium and vessel walls. The interstitial distribution pattern of the deposition depends on the type of the amyloid (4,5). It is deposited in the interstitium of the muscularis mucosae and muscularis propria in primary amyloidosis, while it is mainly deposited in the lamina propria in the secondary (AA) type (5).

Despite the high frequency of gastrointestinal involvement in amyloidosis, the patients are clinically asymptomatic. The disease may present with hemorrhage, motility disorders, ischemic colitis, malabsorption, obstruction, protein-losing enteropathy, and perforation. These manifestations mainly depend on the location of the amyloid deposits within the intestinal wall. Submucosal vascular amyloid deposition usually results in hemorrhages because of the fragility of the affected vessel wall. Gastrointestinal bleeding may also be caused by erosions and ulcerations, which can also lead to subsequent perforation. The underlying mechanism of the dysmotility includes myopathic and neuropathic effect. Smooth muscle and nerve infiltration may cause pseudo-obstruction, which presents as paralytic ileus. Obstruction may also be due to submucosal amyloid tumors. The possible causes of malabsorption may be dysmotility, pancreatic insufficiency or ischemia (1,6).

In the natural course of the disease, the occurrence of colitis is described in patients with primary amyloidosis. Ischemic colitis is a rare but wellknown entity in cases with amyloidosis. Hemorrhagic bullous colitis, which was endoscopically described with hemorrhagic bullous lesions, is a

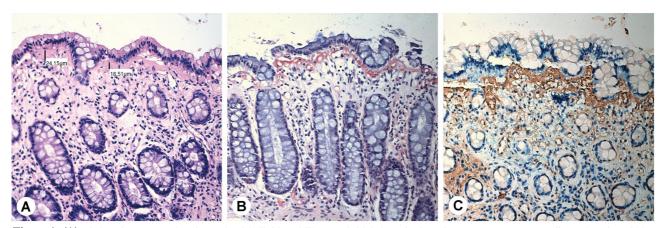


Figure 1. (A) Light microscopy showing subepithelial band-like amyloidal deposits in colon mucosa (Hematoxylin and eosin, x200). **(B)**. Congo red positivity in the subepithelial band (x200). **(C)**. Immunohistochemically positive reaction with amyloid AA (x200).

Author	Age/ Gender	Underlying disease	Symptoms	Endoscopic findings	Histologic findings	Type of amyloidosis
Groisman	65/M	Urinary bladder tuberculosis	Anemia, intermittent diarrhea	Friable orange red mucosae in the left colon and rectum	Thickened subepithelial band of amyloid	AA
García- González	69/M	NA	Intermittent watery diarrhea, anemia	Left colonic and rectal friable orange red mucosae	Thickened subepithelial band of eosinophilic- hyaline substance	AA
	29/M	Crohn's disease	Routine investigation	NA	Thickened subepithelial band of eosinophilic- hyaline substance	AA
Our case	31/M	Juvenile rheumatoid arthritis	Watery diarrhea for 2 months	Mild mucosal erythema	Congophilic subepithelia eosinophilic band	al AA

Table 1. Clinicopathologic features of the previously reported cases of amyloid colitis (7,8)

form of ischemic colitis reported in patients with amyloidosis. Amyloid angiopathy is the main underlying cause of these types of colitis. Possible vascular narrowing by amyloid infiltration leads to ischemia and necrosis (2,3). Amyloid colitis is a rare distinct entity, and is characterized by subepithelial linear amyloid deposition strongly mimicking collagenous colitis. Three cases were presented in the English literature (Table 1). Two of these three cases were associated with chronic inflammatory disorders including Crohn's disease and urinary tuberculosis. The authors did not identify the underlying cause in the remaining third patient. The initial presenting symptom of watery diarrhea, as seen in our case, was shown in two of the three cases. In contrast to ischemic and hemorrhagic bullous colitis, which are usually related with primary amyloidosis, amyloid colitis is associated with secondary amyloidosis. In all three reported cases, the type of amyloidosis proved to be AA (7,8).

The exact mechanism of the diarrhea in patients with gastrointestinal amyloidosis is unclear. Possible pathogenetic mechanisms of diarrhea in these patients as proposed in previous reports include autonomic dysfunction-induced rapid transition of the gastrointestinal content, gastrointestinal dysmotility that results in bacterial overgrowth, pancreatic insufficiency, and bile acid malabsorption (6). Pathogenesis of the diarrhea in amyloid colitis seems to be different from that of gastrointestinal amyloidosis and the other types of amyloid-associated colitis. Subepithelial amyloid deposition appears to be a factor acting as a diffusion barrier, which results in diarrhea similar to that in collagenous colitis (9). This lesion resembles a special form of microscopic colitis strongly mimicking the collagenous colitis. Therefore, the term amyloid colitis instead of gastrointestinal amyloidosis may be appropriate, as Groisman et al. (7) suggested.

From the pathologist's perspective, amyloid colitis closely resembles collagenous colitis. The histologic features of these two lesions are essentially the same on routine hematoxylin-eosin-stained slides. They are characterized by varying degrees of subepithelial thickening. Detachment of the surface epithelial lining has no diagnostic importance. The most important diagnostic clue for the differential diagnosis of amyloid colitis is the special staining. Amyloid stains such as Congo red and crystal violet can highlight the nature of the subepithelial deposition.

In conclusion, amyloid colitis is a unique presentation of secondary amyloidosis in patients with chronic inflammatory disease. In the case of suspected collagenous colitis, histologic examination of the endoscopic biopsies should be evaluated with special stains for amyloid to prevent misdiagnosis.

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Anaplastic pancreas carcinoma diagnosed by fine needle aspiration biopsy technique

İnce iğne aspirasyon biyopsi tekniği ile teşhis edilen anaplastik pankreas karsinomu

To the Editor,

Anaplastic carcinoma (undifferentiated) of the pancreas (ACP) is a rare variant of ductal adenocarcinoma, which commonly displays sarcomatoid spindle-cell and pleomorphic growth patterns. Other rare growth patterns have been reported, including rhabdoid and squamous patterns. Diagnosis of this type of tumor may be challenging due to the lack of glandular structures or other features of differentiation. However, it is very important to recognize this distinct entity because of the highly aggressive nature of this type of tumor (1).

A 60-year-old female presented with the complaints of band-like abdominal pain, fatigue, weight loss, and itching. She had been receiving oral antidiabetic treatment with the diagnosis of type 2 diabetes mellitus for the last five months. Her physical examination showed scratches all over her body. A firm, fixed and painful 10-cm mass with indiscernible borders was palpable in the left epigastrium. Laboratory investigations revealed hemoglobin (Hb): 7.3 g/dl, hematocrit (Hct): 22.5%, mean corpuscular volume (MCV): 79.8 fl, white blood cells (WBC): 11,800/mm³, erythrocyte sedimentation rate (ESR): 130 mm/hour, fasting glucose 132 mg/dl, CEA: 187.3 (0-3.4) ng/ml, and CA19-9: 811.7 (<39) U/ml (high). Esophagogastroduodenoscopy detected external compression that extended to the antrum. Computed tomography showed a mass lesion measuring 13 x 12 x 5 cm in the pancreas that extended to the stomach and the small intestine, hypodense mass lesions in the right lobe of the liver with irregular and indiscernible borders (the largest one with a diameter of 5 cm) and paraaortic/caval millimetric lymph nodes (Figure 1). Percutaneous fine needle aspiration biopsy performed ultrasonographically and pathologic investigation revealed anaplastic carcinoma with pleomorphic and sarcomatoid features (Figure 2). The patient did not receive oncologic treatment due to her poor condition and she died 40 days after the morphologic diagnosis.

Anaplastic carcinoma of the pancreas (ACP) is a rare aggressive pancreatic tumor and accounts for 2-7% of all pancreatic cancers, with a male predo-

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