REFERENCES

1. Shimer GR, Helweg EB. Inflammatory fibroid polyps of the intestine. Am J Clin Pathol 1984; 1: 708-14.
2. Morandi E, Pisoni L, Castoldi M, et al. Gastric outlet obstruction due to inflammatory fibroid polyp. Ann Ital Chir 2006; 77: 59-61.
3. Johnstone JM, Marson BC. Inflammatory fibroid polyp of the gastrointestinal tract. Histopatholoy 1978; 2: 349-61.
4. Sakamoto T, Kato H, Okabe T, et al. A large inflammatory fibroid polyp of the colon treated by endoclip-assisted endoscopic polypectomy: a case report. Dig Liver Dis 2005; 37: 968-72.
5. Helwig EB, Ranier A. Inflammatory fibroid polyps of the stomach. Surg Gynecol Obstet 1953; 96: 335-67.
6. Suen KC, Burton JD. The spectrum of eosinophilic infiltration of the gastrointestinal tract and its relationship to other disorders of angiitis and granulomatosis. Hum Pathol 1979; 10: 31-43.
7. Eslami-Varzaneh F, Washington K, Robert ME, et al. Benign fibroblastic polyps of the colon: a histologic, immunohistochemical, and ultrastructural study. Am J Surg Pathol 2004; 28: 374-8.
8. Makhlouf HR, Sobin LH. Inflammatory myofibroblastic tumors (inflammatory pseudotumors) of the gastrointestinal tract: how closely are they related to inflammatory fibroid polyps? Hum Pathol 2002; 33: 307-15.
9. Stolte M, Zinkenzeller G. Inflammatory fibroid polyp of the stomach. Endoscopy 1990; 22: 203-7.
10. Tada S, Iida M, Yao T, et al. Granular cell tumor of the esophagus: endoscopic ultrasonographic demonstration and endoscopic removal. Am J Gastroenterol 1990; 85: 1507-11.

Ülkü SARITAŞ[1], Yücel ÜSTÜNDAĞ[2], Gökhan GEDİKOĞLU[3]

Department of [1]Gastroenterology, Medicana International Hospital, Ankara
Department of [2]Gastroenterology, Zonguldak Karaelmas University School of Medicine, Zonguldak
Department of [3]Pathology, Hacettepe University School of Medicine, Ankara

A case of atypical celiac hepatitis presenting with hypogonadism, pancytopenia and lymphadenopathy

Hipogonadizm, pansitopeni ve lenfadenopati ile ortaya çıkan atipik çöliak hepatit vakası

To the Editor,

Celiac disease (CD) is a genetically determined autoimmune disease associated with T cell activation triggered by gliadin followed by damage to the intestinal villi (1). CD has been recognized to be a multisystem disorder that may affect the immune system, hematopoietic system, endocrine system, nervous system, and liver (2,3). Although CD involves many organs, no case has been reported in the literature in which CD manifested itself as liver, hematopoietic system and gonadal dysfunctions all in the same patient.

A 19-year-old male was admitted to the hospital with growth retardation and asthenia. He was pale. Hepatomegaly and splenomegaly were present. Bilateral axillary lymphadenopathy, measuring 0.5x0.5 cm, was detected. The patient's height was 163 cm and weight was 54 kg. Hematological and biochemical values are shown in Table 1.

Serum gonadotropin levels were significantly lower than the normal range. The gonadotropin-releasing hormone stimulation test showed hypogonadotropic hypogonadism. The patient commen-

Address for correspondence: Fatih ALBAYRAK
Department of Gastroenterology, Atatürk University, School of Medicine, Erzurum, Turkey
Phone: + 90 442 231 72 10
E-mail: fatihalbayrakerz@gmail.com

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Table 1. Hematological and biochemical values of the patient during the first and second admittances to the hospital

	Patient values			Patient values	
	Jul 2008	Dec 2008		Jul 2008	Dec 2008
Hemoglobin (12–16 g/dl)	6.6	14.6	Serum calcium (8.8-10.8 mg/dl)	9.6	9.1
Mean corpuscular volume (81–99 fl)	57.7	73.1	Folate (3.1-17.5 ng/ml)	4.14	10.97
White blood cells ($4.1\text{-}9.8\times10^9$/L)	3.95×10^9	6.1×10^9	Ferritin (30-400 ng/ml)	3.63	264.22
Platelets ($156\text{-}373\times10^9$/L)	79×10^9	122×10^9	Vitamin B12 (193-982 µg/ml)	527.7	1159
Alkaline phosphatase (30-120 U/L)	430	504	FSH (0.7-11.1 mIU/ml)	1.26	1.71
Alanine aminotransferase (<45 U/L)	77	80	LH (0.8-7.6 mIU/ml)	3.41	3.25
Aspartate aminotransferase (<35 U/L)	100	68	Thyroid-stimulating hormone (0.27-4.2 mIU/ml)	3.01	2.86
γ-glutamyl transpeptidase (<55 U/L)	47	45	Growth hormone (0-20 mIU/ml)	9.14	12.73
Total bilirubin (0.3-1.2 mg/dl)	0.4	0.6	Total testosterone (3-6 ng/ml)	<0.1	422
Total protein (6.6-8.8 g/dl)	6.1	6.8	Cortisol (5-25 µg/dl, 08:00 a.m.)	14.0	16.2
Albumin (3.5-5.2 g/dl)	3.3	4.3	ACTH (0-100 pg/ml)	47.3	52.6

FSH: Follicle stimulating hormone. LH: Leuteinizing hormone. ACTH: Adrenocorticotropic hormone.

ced testosterone replacement therapy. Growth hormone secretion evaluated by the clonidine stimulation test was normal at that time.

As the patient had pancytopenia, a bone marrow biopsy was performed, which revealed mild granulocytic hypoplasia, markedly erythroid hyperplasia and normal megakaryocytes. Parenteral iron therapy was initiated for presumed iron deficiency.

Viral serologies were negative. Abdominal ultrasonography revealed a coarse and homogeneous liver echotexture, minimal dilatation of the portal vein, hepatomegaly, and splenomegaly. The liver biopsy findings were consistent with chronic hepatitis.

Duodenal biopsy showed partial villous atrophy with increased number of intra-epithelial lymphocytes. Antigliadin IgA and IgG and antiendomysial antibodies were positive. Other autoimmune antibodies were negative.

Computed tomography (CT) showed mesenteric lymphadenopathy. Furthermore, an axillary lymph node biopsy revealed reactive lymph nodes.

The patient was started on a gluten-free diet for six months, and by the end of this period he weighed 62 kg. Anemia and leukopenia, as well as some other laboratory findings, had normalized. In addition, the mesenteric lymphadenopathy was smaller on CT.

The clinical presentation of CD can vary from a classical malabsorption syndrome to more subtle atypical gastrointestinal manifestations or extraintestinal presentations (4). CD can have multiple hematologic, liver and endocrinologic manifestations (4,9,14). However, lymphadenopathy is a rare presenting sign of CD (12). As with our case, the explanation of many seemingly unrelated conditions can only be CD. Therefore, CD must definitely be considered when investigating either single organ/system or multi-organ/system disorders.

REFERENCES

1. Farrell RJ, Kelly CP. Celiac sprue. N Engl J Med 2002; 346: 180-8.
2. Guandalini S, Setty M. Celiac disease. Curr Opin Gastroenterol 2008; 24: 707-12.
3. Rostom A, Murray JA, Kagnoff MF. American Gastroenterological Association (AGA) Institute technical review on the diagnosis and management of celiac disease. Gastroenterology 2006; 131: 1981-2002.

4. Rubio-Tapia A, Murray JA. The liver in celiac disease. Hepatology 2007; 46: 1650-8.
5. Halfdanarson TR, Litzow MR, Murray JA. Hematologic manifestations of celiac disease. Blood 2007; 109: 412-21.
6. Bona G, Marinello D, Oderda G. Mechanisms of abnormal puberty in coeliac disease. Horm Res 2002; 57: 63-5.
7. Simmonds JP, Rosenthal FD. Lymphadenopathy in coeliac disease. Gut 1981; 22: 756-8.

Fatih ALBAYRAK[1], Hakan DURSUN[1], Rahşan YILDIRIM[1], Ayşe ALBAYRAK[2], Sare ALTAŞ[3], Serkan CERRAH[1], Nihat OKÇU[1], Murat AKBAŞ[1]

Department of [1]Gastroenterology and [3]Pathology, Atatürk University, Faculty of Medicine, Erzurum, Department of [2]Clinical Bacteriology and Infectious Disease, Erzurum Region Education and Research Hospital, Erzurum

Endoscopic removal of an iatrogenically induced rectal foreign body

İyatrojenik nedene bağlı rektumda yabancı cisim ve endoskopik olarak çıkarılması

To the Editor,

Discovery of foreign bodies in the lower gastrointestinal system is rare in clinical practice. Their removal requires experience and attention. Rectal foreign bodies occur either by the oro-anal route or by insertion via the anal canal. Additionally, they can reach the rectum by migration from adjacent organs (1-3). Insertion of foreign bodies into the rectum can be due to autoerotism, sexual or criminal assaults, accidents, or for concealment (2,4). Thermometers and the tip of enemas in the rectum may occur iatrogenically (5).

A female patient was hospitalized in the internal medicine clinic to investigate the etiology of chronic constipation. An enema was applied by a nurse to relieve her constipation. The tip of the enema broke during the application, leaving the tip inside the rectum. She was then consulted to our department. The rectum was empty and no foreign body was palpated during digital rectal examination. Rectosigmoidoscopy was performed in the left lateral decubitus position, and the foreign body was found 12 cm proximal to the anus (Figure 1A). The foreign body was captured with foreign body forceps and then turned parallel to the long axis of the colon and extracted from the anal canal (Figure 1B). Follow-up rectosigmoidoscopic examination after the procedure did not show any complications or even mucosal erosion.

Foreign bodies ingested by mouth generally reach the rectum spontaneously without any problems, leaving the body from the anal canal, having already passed several physiologically narrow sites (5). They reach the outlet with a fibrous diet and by sedation. Foreign bodies that originate from the anal canal can actually be more problematic than those that originate from the oral route. Patients can be asymptomatic or can present with abdominal pain, rectal bleeding, perianal pain, and constipation. Complications such as peritonitis, perforation and obstruction are reported rarely (3,4). They also cannot be detected on plain abdominal radiographs. Rectosigmoidoscopy can be performed both for diagnosis and treatment (6,7). Enema application before the extraction procedure can cause movement of the foreign body to the proximal colon. Thus, extraction procedures should not be carried out blindly, and the clinician should not hesitate in consulting the patient to the surgery department whenever indicated (4).

Address for correspondence: Hilmi ATASEVEN
Department of Gastroenterology Cumhuriyet University School of Medicine, Sivas, Turkey
E-mail: hilmiataseven@yahoo.com

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