these various treatment modalities (5-7).

In conclusion, Dunbar's syndrome is uncommon, but it should be kept in mind in presence of unexplained gastrointestinal symptoms. Diagnostic tools vary, but CT angiography is favorable method for diagnosis. Medical and surgical treatment modalities are available, although there is lack of consensus about which treatment is the best.

REFERENCES

1. Loukas M, Pinyard J, Vaid S, et al. Clinical anatomy of celiac artery compression syndrome: a review. Clin Anat 2007; 20:612-7.
2. Foertsch T, Koch A, Singer H, et al. Celiac trunk compression syndrome requiring surgery in 3 adolescent patients. J Pediatr Surg 2007; 42:709-13.
3. Dunbar JD, Molnar W, Beman FF, et al. Compression of the celiac trunk and abdominal angina. Am J Roentgenol Radium Ther Nucl Med 1965; 95:731-44.
4. Ozbulbul NI. CT angiography of the celiac trunk: anatomy, variants and pathologic findings. Diagn Interv Radiol 2011; 17:150-7.
5. Jaik NP, Stawicki SP, Weger NS, et al. Celiac artery compression syndrome: successful utilization of robotic-assisted laparoscopic approach. J Gastrointestin Liver Dis 2007; 16:93-6.
6. Reilly LM, Ammar AD, Stoney RJ, et al. Late results following operative repair for celiac artery compression syndrome. J Vasc Surg 1985; 2:79-91.
7. Grotemeyer D, Duran M, Iskandar F, et al. Median arcuate ligament syndrome: vascular surgical therapy and follow-up of 18 patients. Langenbecks Arch Surg 2009; 394:1085-92.

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Coexistence of type 1 diabetes mellitus and Crohn's disesae

Tip 1 diabetes mellitus ve Crohn hastalığı birlikteliği

To the Editor

A 38-year-old male patient was hospitalized with diabetic ketoacidosis and diarrhea ongoing about 2 months. He had type 1 diabetes mellitus (DM) for 19 years. The supporting images in favor of the inflammatory bowel disease that are mucosal fragility, millimetric aphthous ulcers and loss of vascularity in some areas from caecum to rectum, and large aphthous ulcers in rectum have been demonstrated by colonoscopy. Anal canal was normal. Crohn's disease (CD) was confirmed by colon biopsy that revealed loss of mucin in some glands; intense mixed inflammation consisting of neutrophils, eosinophils, histiocytes, lymphocytes, and plasma cells; microgranuloma structures in a few areas (Figure 1), and by the pattern of staining for CD 68. The patient's antiendomysium IgA, antigliadin IgA, antithyroglobulin antibody, antithyroid peroxidase, glutamic acid decarboxylase antibodies were negative, anti-insulin antibodies 26.2% (reference range <8.2%). Anti-Saccharomyces cerevisiae antibodies (ASCAs) were negative. The patient's HLA DNA typing was HLA-A11 A33, HLA-B14 B35, HLA-DRB1*01, DRB1*04, and DRB4. The patient was discharged with therapeutics composed of mesalazine, methylprednisolone, and multiple-dose insulin regimen.

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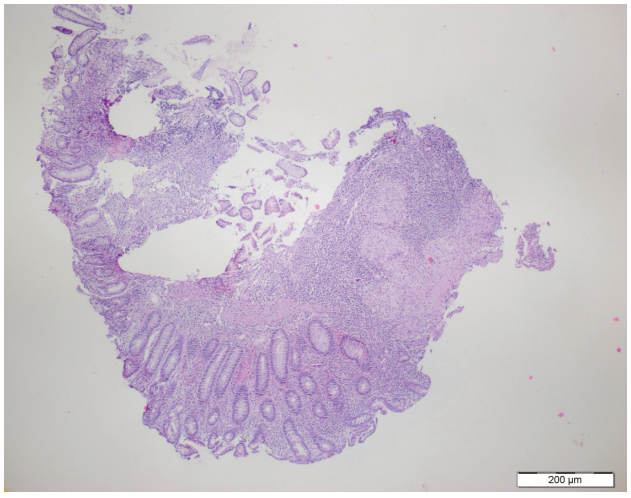

Figure 1. Colon biopsy specimen shows the inflammatory cells and granulomas.

Type 1 DM is strongly associated with autoimmunity (>90%). Most individuals with type 1 DM have the HLA DR3 and/or DR4 haplotype. Refinements in genotyping of HLA loci have shown that the haplotypes DQA1*0301, DQB1*0302 and DQB1*0201 are most strongly associated with type 1 DM (1). CD is a chronic, inflammatory, recurrent, multifactorial disease. The exact pathogenesis is still largely unknown. The most prominent theory describes the CD as a dysregulated, inappropriate immune response to otherwise innocuous bowel flora in a genetically susceptible host (2). Inappropriate down-regulation of an activated immune system is considered as the main pathogenetic mechanism in inflammatory bowel disease (3). ASCA is known to be associated with CD (4). The most consistently replicated association of CD with a common HLA allele is with HLA-DRB1*07. HLA loci have shown that the haplotypes DRB1*0103, DRB3*0301 are the ones associated with CD (5). Data from the meta-analysis of seven small studies also demonstrated a non-significant increase in the common allele HLA-DRB1*04 in CD (6). Autoimmune polyglandular syndromes (APS) are characterized by the coexistence of at least two autoimmune endocrinopathies. APS type 2, type 1 DM, autoimmmune thyroid disease, Addison's disease, primary hypogonadism, hypophysitis, celiac disease, atrophic gastritis, pernicious anemia, vitiligo, alopecia, and myasthenia gravis. In the APS patients, the alleles DRB1*03, DRB1*04, DQA1*03, DQB1*02 were significantly more often present than in healthy controls (7). The coexistence of these two diseases which have autoimmune origin was not reported. This association should be supported by more studies.

REFERENCES

1. Eisenbarth GS, Polonsky KS, Buse JB. Type 1 Diabetes Mellitus. In: Kronenberg HM, Melmed S, Polonsky KS, Larsen PR (ed) Williams Textbook of Endocrinology 11th Edition. Saunders Elsevier Philadelphia: 2008; 1391-416.
2. Chamberlin WM, Naser SA. Integrating theories of the etiology of Crohn's disease. On the etiology of Crohn's disease: questioning the hypotheses. Med Sci Monit 2006; 12: RA27-33.
3. Alkim C, Balci M, Alkim H, et al. The importance of peripheral immune cells in inflammatory bowel disease. Turk J Gastroenterol 2007;18:82-8.
4. Kiliç ZM, Tunç B, Ayaz S, et al. Antineutrophil cytoplasmic autoantibodies and anti-Saccharomyces cerevisiae antibodies in inflammatory bowel diseases. Turk J Gastroenterol 2004; 15:238-42.
5. Ahmad T, Marshall SE, Jewell D. Genetics of inflammatory bowel disease: the role of the HLA complex. World J Gastroenterol 2006; 12:3628-35.
6. Stokkers PC, Reitsma PH, Tytgat GN, et al. HLADR and -DQ phenotypes in inflammatory bowel disease: a meta-analysis. Gut 1999; 45:395-401.
7. Weinstock C, Matheis N, Barkia S, Haager MC, et al. Autoimmune polyglandular syndrome type 2 shows the same HLA class II pattern as type 1 diabetes. Tissue Antigens 2011; 77:317-24.

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