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Dunbar's syndrome: A rare and unclear entity

Dunbar sendromu: Nadir ve anlaşılmamış bir hastalık

To the editor,

The median arcuate ligament (MAL) is located at the T12–L1 level and bridges the crura of the diaphragm, just anterior to the aorta. Dunbar's syndrome, median arcuate ligament syndrome (MALS) or celiac artery compression syndrome (CACS) is caused by external compression of the celiac trunk by the MAL, and is characterized by postprandial abdominal pain, nausea, vomiting, and weight loss (1). MALS is a rare entity, which is diagnosed in only 2 of 100,000 patients with ambiguous upper abdominal pain (2).

A 64-year-old female patient presented with a 2-year history of recurrent postprandial epigastric pain, nausea, and vomiting. Physical examination, laboratory tests, plain radiographs, and upper gastrointestinal endoscopy were unremarkable. The patient underwent abdominal and pelvic computed tomography (CT) (Figure 1). Three-dimensional CT angiography showed compression of the celiac artery trunk by the MAL.

Surgery was performed, and the MAL fibers were transected after identifying the celiac trunk. The postoperative period was unremarkable, and the patient was discharged on postoperative day 3. The patient was symptom-free at 24 months after surgery.

Dunbar et al. reported 15 cases with compression of the celiac artery that were treated with decompression via surgical division of the MAL in 1965 (3). This clinical condition, which was known as MALS and CACS, subsequently became known as Dunbar's syndrome.

The etiology of Dunbar's syndrome is not well-known, but there are several etiological theories, including hereditary, vascular, and neurogenic causes.

Angiography is the gold standard for diagnosing Dunbar's syndrome; duplex Doppler ultrasonography and multidetector-row CT are also commonly used (4). Gastric tonometry and observation of symptoms following papaverin injection during angiography are another diagnostic modalities.

The treatment of Dunbar's syndrome remains contentious. Vasodilators may be used in patients without severe symptoms after establishing the diagnosis. Luminal dilation via percutaneous transluminal angioplasty (PTA) can also be used to treat Dunbar's syndrome. Generally, PTA relieves the symptoms in the short term; however, the mainstay of Dunbar's syndrome treatment remains surgery. Transection and division of MAL fibers and cleaning of periceliac tissue, which may include celiac ganglion, should be performed. Laparoscopic and robot-assisted approaches have become viable options. Another treatment option is revascularization via reimplantation or bypass procedures. Symptom-relief success rates were 53% to 79% in

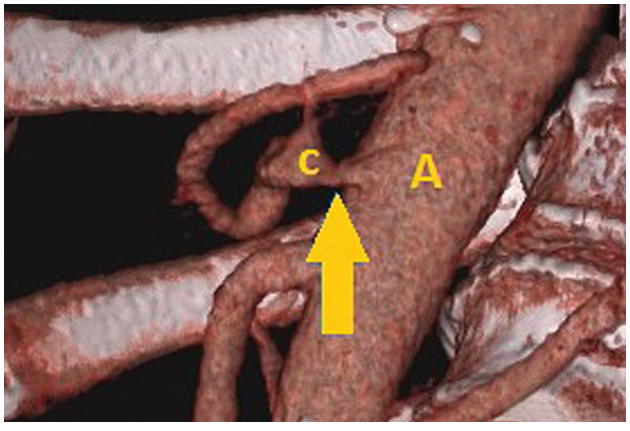

Figure 1. Three-dimensional CT angiography imaging of Dunbar's syndrome. Compressed celiac trunk is shown with arrow. A: aorta. C: celiac trunk.

Address for correspondence: Nezih AKKAPULU
Hacettepe University Faculty of Medicine, Depatment of General Surgery, 06100, Ankara, Turkey
Phone: + 90 312 305 13 18 • Fax: + 90 312 312 27 10
E-mail: akkapulu@gmail.com

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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.

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Nezih AKKAPULU, Yusuf Alper KILIÇ, Onur AYDIN, Ömer ARAN

Department of General Surgery, Hacettepe University School of Medicine, Ankara

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.

Address for correspondence: Mustafa ÜNÜBOL
Adnan Menderes University Medical Faculty, Division of Endocrinology, Aydin, Turkey
E-mail: drmunubol@yahoo.com.tr

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