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A high chromogranin A: Is it always a tumor?

Yüksek kromogranin A düzeyleri her zaman tümör varlığına mı işaret eder?

To the Editor,

Human chromogranin A (CgA) is a secretory protein that is used as a tissue marker for neuroendocrine tumors (NETs) (1,2). In patients with carcinoids and pheochromocytomas, CgA is a more stable marker than plasma levels of serotonin, catecholamines and their urinary metabolites (3). It may also be elevated due to several other causes; however, the use of proton pump inhibitors (PPIs) is the most important reason if elevated CgA level is not caused by a NET (4).

A 65-year-old woman was referred for endocrine evaluation of an incidentally discovered right-sided adrenal mass measuring 1 cm. She had a medical history of type 2 diabetes mellitus and hypertension for 20 years and long-standing gastroesophageal reflux disease (GERD). She was treated with intensive insulin therapy and metformin for diabetes and had been receiving lansoprazole for six months for GERD. She reported that she had been suffering from abdominal pain, bloating, recurrent diarrhea, and constipation for the last year. Hormonal evaluation showed normal plasma aldosterone/renin ratio and overnight dexamethasone suppression test. Urinary meta and normetanephrines were normal. We concluded that the patient's unilateral adrenal mass was nonfunctional. According to the patient's history, a clear conclusion could not be drawn on the association of her diarrheal episodes and her vague symptoms of carci-

noid syndrome. Therefore, serum CgA levels, urinary 5-hydroxyindoleacetic acid (5-HIAA) and serotonin levels were measured. Serum CgA level was >1000 ng/ml (normal: <100 ng/ml), while urinary 5-HIAA and serotonin levels were normal. A whole body Indium-111 octreotide scintigraphy was negative. Due to the marked elevation of CgA, thorax and abdominopelvic computed tomography (CT) scan, upper endoscopy and colonoscopy were performed to rule out a NET. Colonoscopy revealed a tubular adenoma in the sigmoid colon, and endoscopy showed esophageal varices and pangas-tritis. Biopsy samples of gastric mucosa did not indicate enterochromaffin-like (ECL) cell hyperplasia. The presence of elevated liver enzymes, pancytopenia, imaging consistent with chronic liver disease, and esophageal varices on endoscopy suggested liver failure. As for the elevated CgA levels, the possible causes identified were liver failure, diabetes and PPI therapy. After cessation of PPI therapy for one week, serum CgA level plummeted from >1000 ng/ml to 176 ng/ml (0-100 ng/ml) and remained normal (43 ng/ml) one year after stopping PPI treatment.

Hypochlorhydria-induced hypergastrinemia can have a trophic action on ECL cells; thus, CgA can be released into the blood circulation as a consequence of the activation and proliferation of these ECL cells in the stomach. Treatment with PPIs

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might enhance the release of CgA from non-neoplastic, yet functionally abnormal, neuroendocrine cells, thus giving rise to a false-positive diagnosis. Therefore, during the evaluation of elevated CgA

levels, giving particular attention to the use of PPIs as a possible cause might help to prevent the need for expensive and unnecessary diagnostic procedures ordered for revealing NETs.

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Mesenteric Meckel's diverticulum

Mezenterik Meckel divertikülü

To the Editor,

Meckel's diverticulum is the most common congenital anomaly of the gastrointestinal tract (1). The usual location is on the antimesenteric border of the distal ileum, usually within about 60–100 cm of the ileocecal valve. On rare occasions, it may be located on the mesenteric border of the ileum, the so-called mesenteric Meckel's diverticulum. This Meckel's diverticulum attached to the mesenteric border is a distinct variant of the Meckel's di-

verticulum and has been considered a “forgotten entity” (2,3). Existence of the mesenteric Meckel's diverticulum underlines the need for a revision in the understanding and classification of the Meckel's diverticulum (Table 1) (4).

A nine-year-old boy presented with right lower abdominal pain of 10 hours duration. There was a history of nausea and anorexia. On general physi-

Table 1. Various case reports in the literature of mesenteric Meckel's diverticulum

Chaffin (5)	1941	Mesenteric	Asymptomatic
Segal (3)	2004	Mesenteric	Diverticulitis
Sarioglu-Buke (6)	2008	Mesenteric	Rectal bleeding
Manukyan (4)	2009	Mesenteric	Diverticulitis
Seitun (2)	2011	Mesenteric	Diverticulitis
Present case report	2011	Mesenteric	Asymptomatic

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