TJG
The Turkish Journal of Gastroenterology

Candida esophagitis mimicking esophageal carcinoma

Özofagus kanserini taklit eden kandida özofajiti

To the Editor,

Candida albicans is the most common cause of infectious esophagitis. Although most patients with candida esophagitis are immunocompromised, nearly 25% have scleroderma, achalasia, or other causes of esophageal stasis that allow the fungal organism to overgrow and colonize the esophagus, with subsequent esophagitis (1).

A 71-year-old man presented to the hospital with anorexia, difficulty in swallowing and epigastric pain. Clinical history included achalasia, esophageal balloon dilatation and Botox therapy, which was performed two years ago. Endoscopy of the patient revealed mid-esophageal diverticula, diffuse edematous mucosa, mucosal granularity, and a 5x10 mm focal mucosal protuberance with non-smooth contours superior to the Z line, with suspicion of malignancy. Computed tomography (CT) of the patient showed diffuse, concentric wall thickening and submucosal edema, which was prominent in the mid- and distal esophagus (Figures 1, 2). Pulmonary nodules and ground glass appearance were present in the lungs, considered to be due to the infectious process. Endoscopic biopsy specimen yielded fungal esophagitis. Culture of the biopsy material yielded Candida albicans as the etiologic microorganism. The patient's complaints and pulmonary nodules regressed after antifungal therapy.

Esophageal wall thickening is a nonspecific response to a variety of conditions involving the esophagus. Reasons for a thickened esophageal wall on CT imaging include benign and malignant tumors, esophagitis, Barrett's esophagus, secondary achalasia, diffuse esophageal spasm, varices, and esophageal intramural pseudodiverticulosis (2). Contrast material–enhanced esophagography and endoscopy remain the reference standards for the evaluation of esophagitis. CT imaging may be performed when the diagnosis is unclear or when a

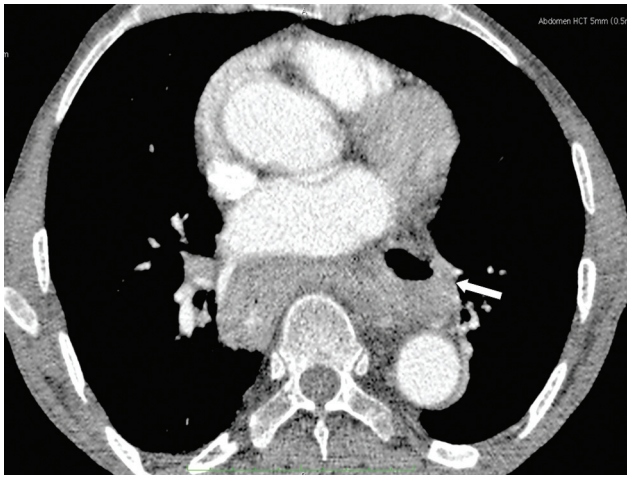

Figure 1. Axial and coronal CTs of the thorax show diffuse symmetric and concentric wall thickening (arrow) with submucosal edema in the distal esophagus suggesting inflammation.

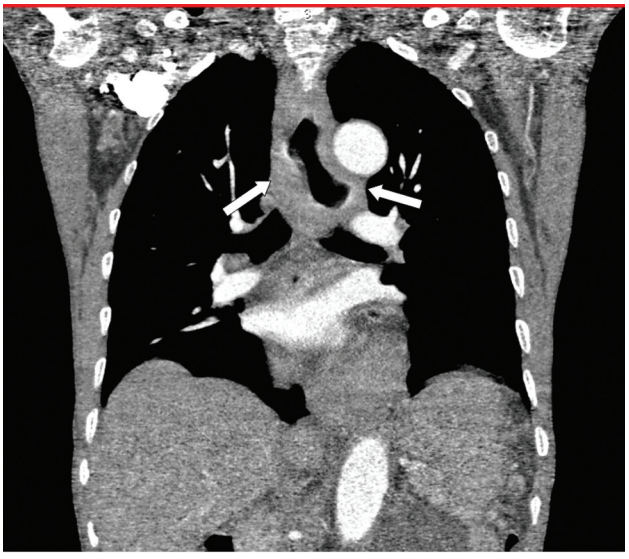

Figure 2. Coronal CT of the thorax shows symmetric wall thickening (arrows) in the mid-esophagus.

Address for correspondence: Mehmet Ruhi ONUR
University of Fırat, Faculty of Medicine,
Department of Radiology, Elazığ, Turkey
Phone: + 90 424 233 35 55
E-mail: ruhionur@yahoo.com

Manuscript received: 08.01.2011 **Accepted:** 07.02.2011

Turk J Gastroenterol 2011; 22 (6): 648-657
doi: 10.4318/tjg.2011.0307

complication is suspected. In our patient, the infectious process may have been due to possible mucosal damage by previous balloon dilatation and Botox therapies. Whatever the cause of severe esophagitis, CT imaging appearance is predominantly characterized by diffuse esophageal thickening, submucosal edema and mucosal enhancement (3). Benign or malignant tumors of the esophagus are usually manifested on CT imaging by focal, asymmetric thickening of the esophageal wall (2), whereas the wall thickening in esophagitis is concentric and circumferential. Since severe edema and mucosal changes in candida esophagitis may simulate malignancy on endoscopy, as in our patient, CT imaging may be a useful imaging modality by showing diffuse, concentric wall thickening of the esophageal wall against the asymmetric, focal wall thickening in malignancies. Furthermore, possible accompanying pulmonary infestation, which adds diagnostic value for the differential diagnosis of esophageal lesions, may be seen on CT imaging.

REFERENCES

1. Levine MS, Macones AJ, Laufer I. Candida esophagitis: accuracy of radiographic diagnosis. Radiology 1995; 154: 581-7.
2. Desai RK, Tagliabue JR, Wegryn SA, Einstein DM. CT evaluation of wall thickening in the alimentary tract. Radio Graphics 1991; 11: 771-83.
3. Young CA, Menias CO, Bhalla S, Prasad SR. CT features of esophageal emergencies. Radiographics 2008; 28: 1541-53.

Mehmet Ruhi ONUR[1], Cem AYGÜN[2], Muammer AKYOL[1], Halil İbrahim BAHÇECİOĞLU[2]

Departments of [1]Radiology and [2]Gastroenterology, Fırat University, School of Medicine, Elazığ

Proton pump inhibitor and clopidogrel interaction: Is it clinically significant?

Proton pompa inhibitörü ve klopidogrel etkileşimi: Klinik olarak önemli mi?

To the Editor,

Clopidogrel is an antiplatelet therapy that has been approved for the treatment of cardiovascular events with aspirin as a dual antiplatelet therapy. Proton pump inhibitor (PPI) therapy was recommended as a gastrointestinal protection for those patients with dual therapy (1). Clopidogrel is a prodrug that must be converted to its active metabolite by cytochrome P450, mainly 3A4 and 2C19. The active metabolite will inhibit platelet aggregation by binding irreversibly to the platelet $P2Y_{12}$ receptor. However, variation in response to clopidogrel has been observed due to genetic mutations in CYP2C19 (*2, *3, *4, and *5), which will lead to clopidogrel resistance. Such variation may lead to increased risk of cardiovascular events in patients undergoing percutaneous coronary angioplasty (PCA) intervention (2). PPI therapy is a competitive inhibitor of CYP2C19, which will diminish the activity of clopidogrel. It has been observed in several randomized trials through measuring the platelet function assay, which led to the Food and Drug administration (FDA) warning that omeprazole reduced the antiplatelet effect by 50% (3). The first double-blind randomized trial was conducted to assess the effect of the interaction between clopidogrel and omeprazole on cardiovascular events.

Address for correspondence: Bandar AL-JUDAİBİ
Department of Gastroenterology, London Health Science Centre, Ontario, Canada
E-mail: bandaraljudaibi@gmail.com

Manuscript received: 16.06.2010 **Accepted:** 21.09.2010

doi: 10.4318/tjg.2011.0315