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Achalasia as a cause of congestive heart failure

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Achalasia is an idiopathic disorder of motility that affects the smooth-muscle portion (distal two thirds) of the esophagus. It is characterized by a loss of normal peristalsis, impaired relaxation of the lower-esophageal sphincter (LES), and resultant dilatation of the esophagus proximal to the LES. The classic presentation is dysphagia to solids and liquids; regurgitation, chest pain, and weight loss are also common. The diagnosis can be difficult to make, especially in the elderly, and the diagnosis is often delayed because of the gradual onset of the symptoms. We present a case of achalasia that was diagnosed because of the rarely associated cardiac complications.

CASE REPORT

An 81-year-old woman was transferred to our institution for evaluation of new-onset congestive heart failure. Of note, before transfer, she complained of dysphagia. A subsequent barium swallow at the referring institution revealed a dilated esophagus and bird's beak consistent with achalasia. The patient was confused and unable to give a history. Physical examination revealed dry mucous membranes, jugular venous distension, and bilateral basilar crackles. A chest radiograph was consistent with pulmonary edema.

The patient was admitted to telemetry and underwent echocardiography with color-flow mapping, which showed extrinsic compression of the left atrium (Fig. 1). The left atrium was crescentic in shape and measured only 1.1 cm in diameter. No flow was detected in the compressing mass. Left ventricular systolic function was normal. A CT showed that the compression of the left atrium (Fig. 2) was, in fact, secondary to a massive amount of food in the esophagus. A nasogastric (NG) tube was placed into the esophagus, and approximately 200 mL of liquid food material was suctioned. A repeat echocardiogram was done to assess the left atrium after NG-tube suctioning. It revealed a more patent left atrium. The tubular mass (dilated esophagus) was much smaller (Fig. 3).

For management of the achalasia, a botulinum toxin injection was used to relieve the LES pressure. Although botulinum toxin therapy has a higher symptom relapse rate than pneumatic dilation, it was felt that this woman who was elderly and debilitated would not be able to survive the potential complications of endoscopic dilation (ie, perforation). An EGD showed liquid food in the esophagus and distal esophagitis (Fig. 4). No stricture or mass was noted. One hundred units of Botox (Allergen Pharmaceuticals, Irvine, Calif) was gently dissolved in 4 mL preservative-free normal saline solution; 0.8-mL aliquots were injected in 4 quadrants into the LES in the distal esophagus. The scope was then retroflexed in the stomach, and 1 injection was given in the LES region of the cardia. The patient tolerated the procedure well and received a clear liquid diet the next

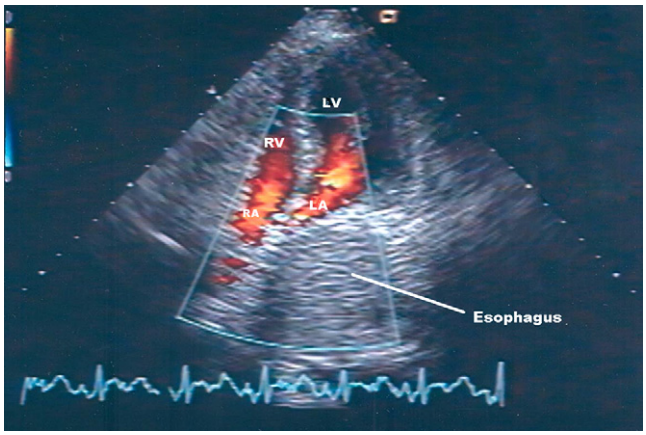

Figure 1. Transthoracic echocardiogram, showing left atrial compression from an extrinsic lesion, as well as a thrombus in the left ventricle. The diameter of the left atrium is 1.1 cm.

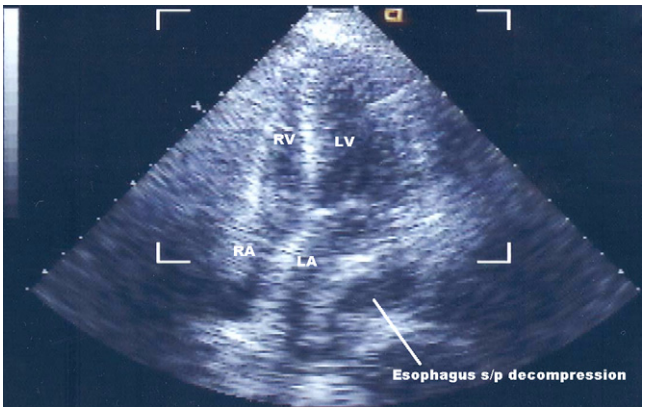

Figure 3. Follow-up transthoracic echocardiogram after decompression with NG-tube suction. Note that the left atrial diameter increased.

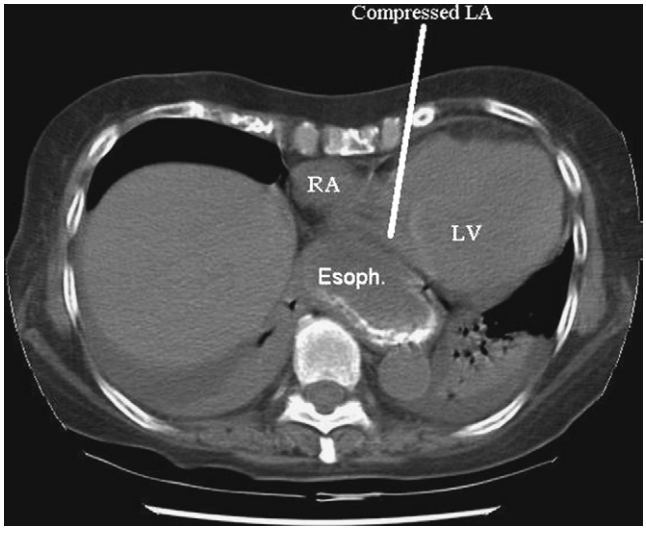

Figure 2. CT of the chest, showing extrinsic compression of the left atrium by massively dilated esophagus.

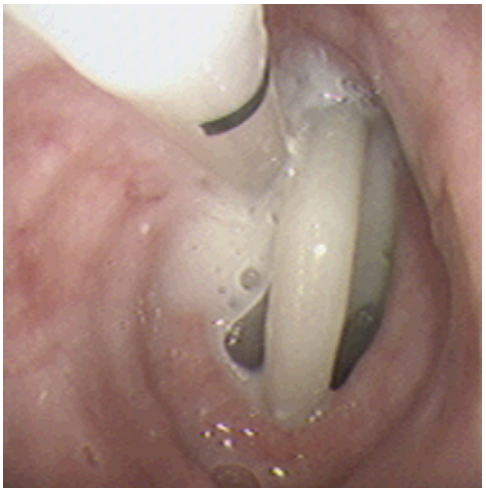

Figure 4. Endoscopic image, showing dilated distal esophagus with coiled NG tube and fluid.

day. The patient was subsequently discharged to a rehabilitation facility and was doing well at 18 months after admission.

DISCUSSION

There have been few documented cases of left atrial compression by an extrinsic source that leads to hemodynamic compromise. These include bronchogenic cysts,[1] carcinoma,[2] lymphoma,[3] thymoma,[4] aortic aneurysm,[5] diaphragmatic hernia,[6] hiatal hernia,[7] schwannoma,[8] and aortic dissection.[9] There have been 2 previous cases in the literature of decompensated achalasia that resulted in left-heart compression as seen on 2-dimensional echocardiography. In 1 case, the patient had no symptoms of cardiac compromise,[10] but the second was a case similar to ours in which the patient had significant symptoms of congestive heart failure but no overt signs of tamponade.[11] Possible mechanisms for the decreased cardiac output and pulmonary congestion include obstruction of the left atrial inflow and direct impairment of ventricular filling, thereby causing functional tamponade.[7]

This case highlights achalasia as a rare but reversible cause of congestive heart failure because of extrinsic left-heart compression. A previous report has shown that echocardiography is a useful method for monitoring the treatment of decompensated esophageal achalasia.[11] We also recommend echocardiography as a monitoring tool, in conjunction with a CT. In addition, we emphasize the

need for acute NG-tube evacuation of food contents, followed by botulinum toxin injection therapy as a less invasive but effective treatment in these generally ill patients.

When extrinsic causes of heart failure are suspected, the clinical scenario and the CT should prompt the next appropriate evaluation. In our patient, the symptoms of dysphagia and the dilated esophagus on CT called for endoscopic evaluation. Endoscopy and EUS play a potentially important role to rule out secondary causes of achalasia. Mucosal or submucosal neoplasms can present as dysphagia, which is often difficult to distinguish in elderly patients from achalasia by history alone. In patients in whom secondary achalasia is suspected but CT and upper endoscopy are unremarkable, EUS may be of value in helping to rule out a submucosal lesion at the level of the gastroesophageal junction.[12]

DISCLOSURE

The authors of this case report have no conflicts of interest to disclose.

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Watermelon stomach treated with endoscopic band ligation

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Watermelon stomach or gastric antral vascular ectasia is an important cause of chronic blood loss and anemia. It is diagnosed on endoscopy by prominent flat or raised erythematous stripes radiating in a spoke-like fashion from the pylorus to the antrum and resembles a watermelon.[1] It may also present as diffuse red spots that are located in the antrum, in comparison to portal hypertensive gastropathy where changes are prominent in the fundus or the corpus.[2] It can present with iron-deficiency anemia secondary to occult blood loss,[3,4] with intermittent melena, or, rarely, with hematemesis.[5] Watermelon stomach has been treated with medical, endoscopic, and surgical approaches.[6] Endoscopic therapies include neodymium-yttrium aluminum garnet laser,[3,7] argon plasma coagulation (APC),[8] heat probe,[9] bipolar electrocautery,[9] argon laser,[10] and endoscopic sclerotherapy injections by using absolute alcohol