Hypertensive Urgency Secondary to Tablet Retention in a Patient with Achalasia

by Stanley Yakubov, Kadirawel Iswara, Ira Mayer, Rabin Rahmani

Achalasia is a rare disease (incidence of 1.6 cases per 100,000) that is due to degeneration of inhibitory neurons in the myenteric plexus that leads to failure of lower esophageal sphincter relaxation. It can occur at any age from childhood to old age, with both sexes equally affected. Patients complain of dysphagia of solids and liquids, non cardiac chest pain, regurgitation, weight loss and cough. Surgery is the standard of care for achalasia, but endoscopic intervention (e.g. botulinum toxin injection) can server as a temporizing measure. We report a rare case of an elderly female who presented with dysphagia, weight loss, headaches and diaphoresis and found to be in hypertensive urgency secondary to esophageal tablet retention.

INTRODUCTION

Achalasia, meaning "failure to relax", is a rare disease that may occur from childhood to old age, but most commonly happens between the ages of 25 and 60 with both sexes equally affected.[5,6] Achalasia was initially known as cardiospasm and was treated with a sponge attached to the distal end of a carved whalebone. In 1937 Lendrum noted that the syndrome was caused by incomplete relaxation of the lower esophageal sphincter (LES) and renamed it as achalasia.[1] We report a rare case of an elderly female who presented with dysphagia, weight loss, headaches and diaphoresis and found to be in hypertensive urgency secondary to esophageal tablet retention.

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Case Description

An 82 year-old female with past medical history of hypertension and achalasia presented to the emergency department with severe headaches and diaphoresis. On further questioning, she reported difficulty swallowing solids and liquids and unintentional weight loss of 13 pounds over a 5-month period. Home medications included amlodipine and valsartan. On admission, her temperature was 96.7 °F, blood pressure 207/110 mmHg, pulse 90 beats/min, respiratory rate of 20 breaths/min, and oxygen saturation of 95% on room air. Physical examination and laboratory analysis were unremarkable. Telemetry monitoring and intravenous (IV) administration of anti-hypertensive medication for presumed hypertensive urgency was instituted with subsequent improvement of her blood pressure (BP). Computed tomography of her chest and abdomen showed markedly dilated distal esophagus with fluid and debris (Figure 1). Esophagogastroduodenoscopy (EGD)

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revealed a fluid filled esophagus with 41 undigested tablets (Figure 2). The tablets were removed (Figure 3) using a Roth net and subsequent achalasia pneumatic balloon dilation was performed with good results. The patient was subsequently discharged from the hospital tolerating a soft diet and with significant improvement in her BP.

Discussion

Achalasia is failure of relaxation of the LES due to degeneration of inhibitory neurons in the myenteric plexus. Inhibitory neurons release nitrous oxide and vasoactive intestinal peptide neurotransmitters and cause muscle relaxation.[6] It has been hypothesized that the degeneration of these inhibitory neurons in achalasia is associated with class II HLA antigen DQw1 and related to herpes and measles infection.[1] Achalasia is a rare disorder with incidence of 1.6 cases per 100,000 and is about 200 fold more frequent in patients with Down syndrome.[5,6] Symptoms often include dysphagia of solids and liquids, non cardiac chest pain, regurgitation, weight loss and cough.[2,3] Complications include retention esophagitis, aspiration pneumonia in up to 8% of patients and esophageal squamous cell carcinoma with estimated risk ranging 30 times higher than in the general population.[6] Diagnostic studies include barium swallow, endoscopy and esophageal manometry.[2] Achalasia typically appears on radiographs as a dilated esophagus that terminates with a "bird beak" appearance. The therapeutic goal in achalasia is to reduce the LES pressure and allow gravity to facilitate esophageal emptying. For patients that are surgical candidates, Heller myotomy is the treatment of choice. Alternative treatment options include medications, botulinum toxin injection and endoscopic intervention. The efficacy of botulinum toxin injection at 1 month is up to 90%, but is only up to 46% at 12 months.[3] After 5 years, endoscopic pneumatic dilation has a response in up to 85% of patients in retrospective study, but only in 26% in prospective study. Complications include hemorrhage in roughly 0.4% and perforation in up to 10%.[6] Endoscopic balloon dilation was found to be superior to endoscopic botulinum toxin injection with better symptom relief, 68.2% vs. 40.6% respectively, and required less additional therapy, 25% vs. 46.6% respectively. Laparoscopic myotomy with fundoplication provided better symptom relief than all other surgical and endoscopic techniques, however, the

Figure 1.

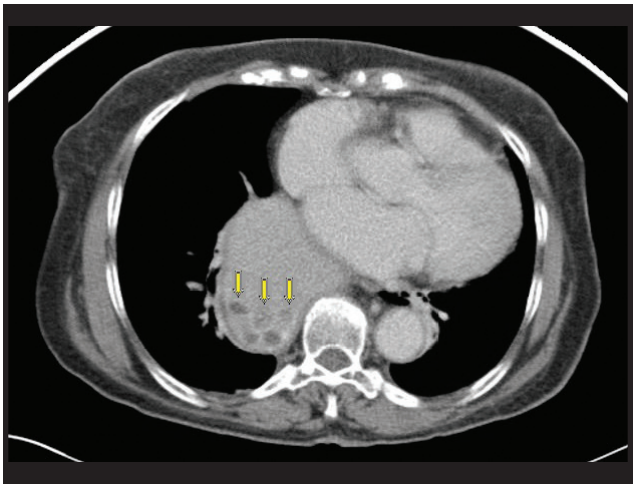

Figure 2.

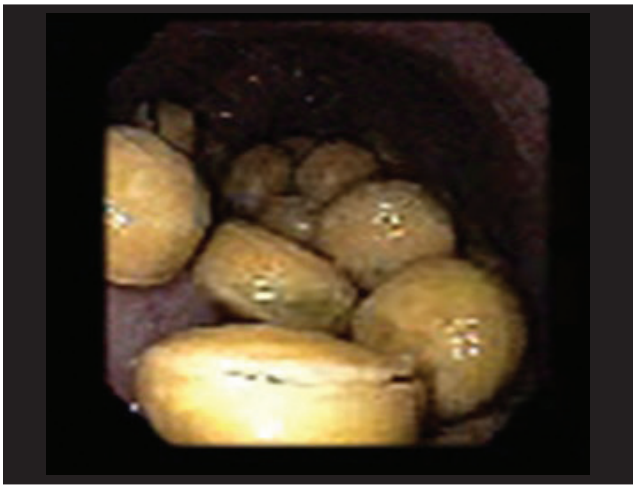

Figure 3.

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overall complication rate after laparoscopic myotomy was higher than for endoscopic balloon dilatation, 6.3% vs. 1.6% respectively.[4] Patients who are unable or unwilling to undergo any procedures, or in whom botulinum toxin injections fail can choose to undergo medical treatment for achalasia. Medical treatment often revolves around nitrates and calcium channel blockers as they relax the smooth muscle of LES. Studies have shown that calcium channel blockers efficacy varies greatly and is 50-90% at 6-18 month follow-up and side effects such as peripheral edema, hypotension, and headache were seen in up to 30%.[3] In patients with concomitant hypertension, calcium channel blockers have the additional benefit of acting as an anti-hypertension medication.

CONCLUSION

Although EGD and balloon dilation were successful in relieving dysphagia in our patient, careful BP monitoring and periodic dysphagia questioning should be considered in all patients with achalasia and hypertension who are treated with calcium channel blockers.

Furthermore, achalasia should be considered in all individuals with dysphagia. Early recognition and treatment of achalasia will prevent complications and allow for a better quality of life. ■

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