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Case report on Achalasia Cardia

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ABSTRACT

Achalasia is one of the often occurring causes of motor dysphagia. Achalasia, a disorder of esophageal smooth muscle motility, is caused by the lower esophageal sphincter's inability to relax. This disorder leads to a functional blockage of the gastroesophageal junction. Despite the fact that the disorder was first described more than 300 years ago, the pathogenesis of this disorder is still unknown. Achalasia cardia may be caused by the loss of the inhibitory ganglion in the myenteric plexus of the oesophagus. Loss of inhibitory neurons that make VIP and nitric oxide synthase in the esophageal myenteric plexus occurs, but in severe cases, it can also impair cholinergic neurons. Although several theories have been advanced, the precise reason for this degradation is still unknown. Dysphagia and regurgitation are the most typical early symptoms of achalasia. Chest pain will be present in more than half of patients when they first arrive. Rarely does esophageal emptying increase lead to pain relief. As the illness progresses, patients may have symptoms such as regurgitation with a risk of aspiration, nocturnal coughing, heartburn, and weight loss owing to feeding difficulties. Because they can't swallow, people often lose weight quickly.

INTRODUCTION

Motor dysphagia has a number of frequent causes, including achalasia. The failure of the lower esophageal sphincter to relax results in achalasia, a condition of esophageal smooth muscle motility[1]. The gastroesophageal junction becomes functionally blocked as a result of this disease. The actual pathophysiology of this disorder is still unknown, despite the fact that the disease was originally recorded more than 300 years ago. Loss of the inhibitory ganglion in the myenteric plexus of the oesophagus can be the pathophysiological aetiology of achalasia cardia. In the esophageal myenteric plexus, there is a loss of inhibitory neurons that produce VIP and nitric oxide synthase, but in extreme situations, it also affects cholinergic neurons. Despite numerous possibilities being put forth, the specific cause of this deterioration remains unknown. The autoimmune phenomenon, viral infection, and genetic predisposition are some of these hypotheses[8].

When inhibitory nerves in the oesophagus degenerate, excitatory neurotransmitters like acetylcholine act unopposed, causing high-amplitude non-peristaltic contractions (vigorous achalasia); as cholinergic neurons gradually die off over time, the esophageal body dilates and simultaneously contracts at a low intensity (classic achalasia). Numerous researchers have sought to investigate potential disease-initiating agents since the initial report, including viral infection, other environmental variables, autoimmunity, and genetic factors[8].

The majority of achalasia patients have dysphagia at presentation, beginning with solids and progressing to liquids in 70 to 97 percent of cases.[1] The most common initial signs of achalasia are dysphagia and regurgitation. More than half of patients will experience chest pain upon presentation. The pain is rarely reduced by an increase in esophageal emptying. Patients may have symptoms including regurgitation with a risk of aspiration, nocturnal coughing, heartburn, and weight loss due to difficulties eating as the condition worsens. Due to the inability to swallow, weight loss is frequently quick[4].

CASE PRESENTATION

The clinical progress of a patient with achalasia cardia is being described. A 71-year-old male patient was admitted with complaints of dysphagia, vomiting, regurgitation, and chest pain.

The patient is a known case of achalasia cardia and has undergone pneumatic dilation on 20/09/2021. The patient also has a history of bipolar disorder and is on treatment with TAB DAYO (DIVALPROEX) 500mg, TAB TAB OLZIC (OLANZAPINE) 10 mg, TAB NITHRA (NITRAZEPAM) 10 mg, TAB TRIDYL (TRIFLUOPERAZINE 5 mg + TRIHEXYPHENIDYL 2 mg).

THE HISTORY OF THE DISEASE

The disease was initially diagnosed one year ago when the patient presented with complaints of difficulty in swallowing for 4 months, with intolerance to liquid and solid diets, worsening regurgitation, and significant loss of body weight. The patient also experienced occasional non-radiating retrosternal chest pain and productive cough. The CT thorax showed a megaesophagus. OGD showed a dilated oesophagus with food stasis and tightness of the gastro-esophageal junction. Upper GI endoscopy reported a roomy oesophagus with minimal food stasis and the case was diagnosed as achalasia cardia. Pneumatic dilatation was done.

THE CLINICAL EXAM

The patient was conscious and afebrile with a heart rate of 79 beats per minute and a respiratory rate of 20 breaths per minute. The bowel movements were reduced and the patient had significant weight loss in the past. The airway was patent with normal vascular breath sounds. The cardiac examination showed S1 and S2 to be normal, and the GI examination showed non-tender git.

THE LABORATORY INVESTIGATIONS

On admission, vitals were normal (Blood Pressure of 120/70 mm Hg, Respiratory rate of 20 breath/min and Pulse rate of 70 beats per minute) and blood routine examination showed that neutrophils, ESR, and CRP levels were all elevated with values of 80.9%, 116 mm/hr and 84.6 mg/L respectively, while the lymphocyte count was depleted (12%). Upper GI endoscopy showed a roomy, tortuous esophagus with minimal food stasis. Esophageal secretions were aspirated and pneumatic dilatation of the lower esophageal sphincter was done under endoscopic guidance.

Intravenous Monocef (Ceftriaxone 1 gm) parenteral antibiotic injection and IV fluids DNS and RL were initiated, and the patients' own medicines were continued. On the first day, Inj Celemine 10% (1 bottle) was also administered at a rate of 75ml/hr. The propped up position was maintained. The patient was better after the procedure, and small sips of liquid food were tolerated. He was advised to lie down at 45 degrees and was discharged with Tab Cefuroxime (Cefuroxime) 500 mg 1 - 0 - 1 for 5 days; Tab Nicardia retard (Nifedipine) 10mg 1 - 0 - 0 ; Tab Pantocid (Pantoprazole) 40mg 1 - 0 - 1 and Tab Ganaton (Itopride) 50mg 1 - 1 - 1 for 14 days each.

DISCUSSION

The well-known esophageal motility problem, achalasia cardia, develops as a result of poor lower esophageal sphincter (LES) relaxation during swallowing and peristalsis in the distal smooth muscle segment of the oesophagus. It is characterised by increasing ganglion cell degeneration in the esophageal myenteric plexus. The typical symptoms include dysphagia for both liquids and solids from the time they first appear, as well as regurgitation of unprocessed food, retrosternal discomfort, heartburn, and weight loss.

According to the currently available evidence, the most likely pathogenic event causing achalasia is the autoimmune progressive degeneration of ganglion cells in the esophageal myenteric plexus in genetically susceptible individuals (human leukocyte antigen [HLA]-DQ variants DQA1*0103 and DQB1*0603). Inhibitory nitrergic neurons, which secrete nitric oxide and vasoactive intestinal peptide, and excitatory cholinergic neurons, which secrete acetylcholine, are preferentially lost in the oesophageal smooth muscles, which results in insufficient LES relaxation and peristalsis failure. Latent or ongoing viral infections like herpes simplex virus type 1 are thought to be the initiating mechanism for the loss of ganglion cells. Lewy bodies seen in ganglion cells indicate that achalasia cardia is linked to other neurodegenerative conditions, including Parkinson's disease.

Achalasia cannot be accurately diagnosed by symptoms alone; therefore, diagnostic procedures are necessary to establish the diagnosis when there is clinical suspicion of the condition. Additionally, it's essential to rule out benign and cancerous causes of lower esophageal blockage.

A barium esophagogram is the most effective first test to identify achalasia (barium swallow). The lower oesophagus smoothly tapers to a "bird's beak" look; dilated proximal oesophagus; and absence of peristalsis on fluoroscopy are the classic findings on the barium swallow. To rule out premalignant or malignant esophageal lesions, upper endoscopy

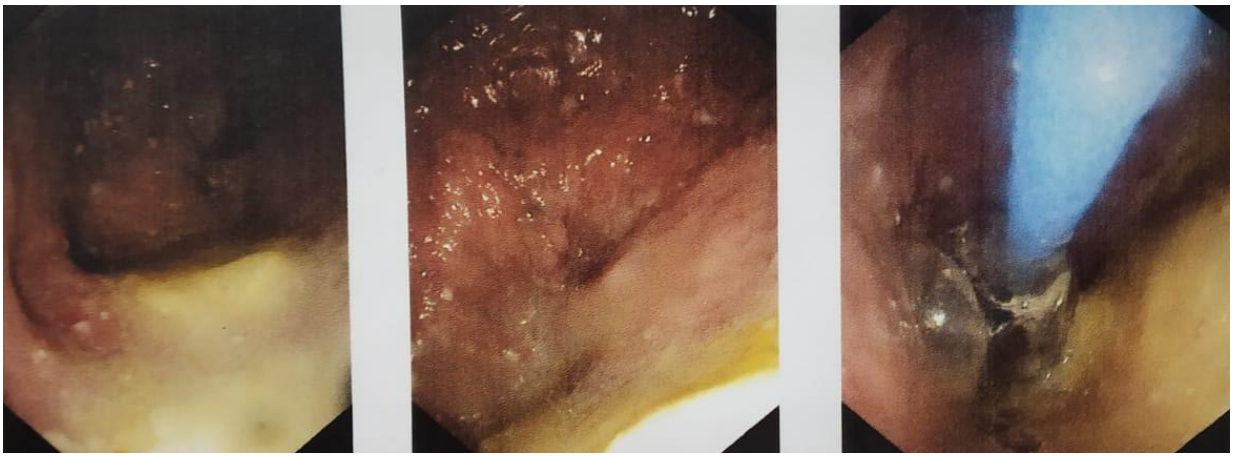

Fig. 1 : Upper GI Endoscopy showing a roomy, tortuous oesophagus

(esophagogastroduodenoscopy, or EGD) is advised in all patients with suspected achalasia or dysphagia. In the early stages of the disease, EGD may be normal but has poor diagnostic accuracy for achalasia. In severe instances, a rosette-shaped esophagogastric junction or an oesophagus that has dilated and convoluted with trapped food and saliva are among the findings. Additionally, CT scans, endoscopic ultrasounds (to rule out submucosal lesions), and transabdominal ultrasounds are additional helpful tests in instances of pseudoachalasia. The most accurate test for achalasia diagnosis is still esophageal manometry. Manometry will show partial lower esophageal sphincter relaxation in response to swallowing, occasionally a lack of peristalsis in the lower oesophagus, and an increase in lower esophageal sphincter pressure.

The goal of treatment for achalasia is to reduce the outflow resistance brought on by a rigid and hypertensive lower esophageal sphincter. Currently, surgical or nonsurgical treatments are available for primary idiopathic achalasia. Pharmacotherapy, endoscopic botulinum toxin injection, and pneumatic dilatation are nonsurgical alternatives. Two surgical options are laparoscopic Heller myotomy (LHM) and peroral endoscopic myotomy (POEM).

To lower the lower esophageal sphincter (LES) pressure, pharmacologic therapies may involve the administration of nitrates, calcium channel blockers, and phosphodiesterase-5 inhibitors. Calcium entry into cells that impedes smooth muscle contraction is inhibited by calcium channel blockers, which lowers LES pressure. Nitrates raise nitric oxide levels in smooth muscles, which raises levels of cyclic adenosine monophosphate and causes smooth muscle relaxation.

The surgical myotomy procedure, which can be carried out laparoscopically, is the suggested method for lowering pressure across the lower esophageal sphincter. Through this treatment, the lower esophageal sphincter's circular muscle fibers will be severed, causing relaxation. LHM is frequently combined with an anti-reflux operation, such as Nissen, the posterior (Toupet), or the anterior (Dor) partial fundoplication, because it has the potential to produce uncontrolled gastroesophageal reflux. In high-risk individuals or those who relapse following myotomy, endoscopic injection of botulinum toxin may be employed. A powerful biological neurotoxin that is generated from the bacterium Clostridium botulinum, botulinum toxin, blocks the release of acetylcholine at the level of the lower esophageal sphincter.

CONCLUSION

The most economical non-surgical treatment for achalasia is pneumatic dilatation of the oesophagus via endoscopy. When using a graded dilator technique, air pressure is used to break the LES's circular fibres in order to dilate the oesophagus. 50% to 93% of patients report an improvement in their symptoms, although 30% of patients report symptom recurrence at five years. In a few centres, peroral endoscopic myotomy (POEM) is a successful, minimally invasive alternative to laparoscopic Heller myotomy for the treatment of achalasia. Endoscopic dissection of the LES's circular fibers results in LES relaxation, but because there is no antireflux technique involved, there is a substantial chance of gastroesophageal reflux. Esophageal surgery is the last option.

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