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Achalasia Cardia – A Case Report

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ABSTRACT

Achalasia is a primary oesophageal motility disorder of unclear etiology. It is relatively rare. Affecting approximately 1 in 100,000 individuals a year (1). It is usually diagnosed between 20 and 50 years of age, but can occur at any age, with no predilection for either sex. Achalasia (primary achalasia) is impaired lower oesophageal sphincter relaxation and absence of oesophageal peristalsis, resulting in a functional outflow obstruction at the esophagogastric junction (2,3). Obstruction of the distal oesophagus from other non-functional etiologies may have a similar presentation and has been termed "secondary achalasia" or pseudoachalasia.

Keywords: Oesophagus, achalasia cardia, dysphagia, peristalsis, oesophageal sphincter.

INTRODUCTION

Achalasia is a Greek word that means "failure of relaxation". Primary achalasia is idiopathic. The exact pathogenesis of primary achalasia is still not known. In secondary achalasia, the cause for the degeneration of oesophageal nerve fibres is known. Pathophysiologically, achalasia is caused by loss of inhibitory ganglion cells in the mesenteric plexus. Several studies have attended to explore initiating agents that may cause the disease such as viral infection, other environmental factors, autoimmunity genetic, malignancy etc. Sir Thomas Willis first described the condition as "food blockage in oesophagus" in 1674, and he treated it an event with dilatations using a sponge attached to a whale bone (4). A F Hurst discovered that the motility disorder was due to the lower oesophageal sphincter's inability to relax, and named it "achalasia" (from the Greek khalasis "relaxation") (5).

CASE REPORT:

A 42-year-old female patient presented at our OPD with vomiting with small amount of blood. She has history of vomiting for four days with burning sensation at epigastrium. No loose motion or fever. She could not take food orally. Clinical diagnosis was dysphagia due to achalasia.

Chest x-ray was done and findings were mild cardiomegaly with widening of mediastinum (Fig. 1) -suggests dilated oesophagus (achalasia**).** Later an upper GI tract endoscopy was done and H-pylori was positive. In GI tract endoscopy oesophagus was dilated and the opening at the centre looked like a donut.

CT Thorax done - findings were, the esophagus was grossly dilated till gastro-oesophageal junction with air-fluid level (Fig. 2 – 5). No mass was seen – findings suggests achalasia.

Gastrografin swallow done subsequently which showed grossly dilated oesophagus. There was no flow of contrast into the stomach. A nasogastric tube was given and repeat gastrografin swallow was done which showed flow of contrast in to the stomach from dilated oesophagus (Fig. – 6).

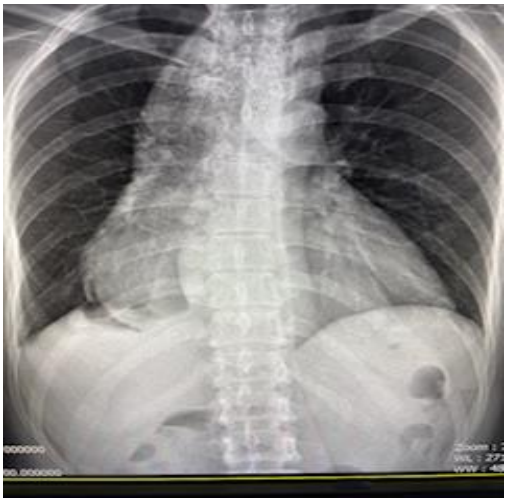

Fig. 1 - Plain X-ray Chest showing mild cardiomegaly with widening of mediastinum.

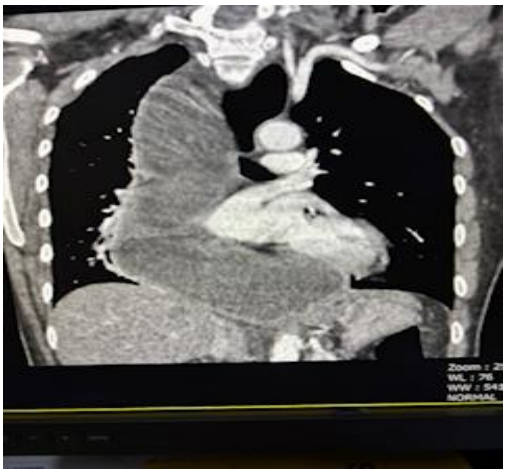

Fig. 2 - CT Thorax Coronal scan showing grossly dilated esophagus till GEJ with air-fluid level.

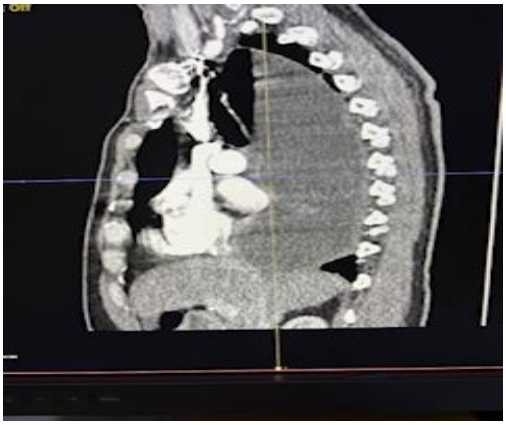

Fig. 3 - CT Thorax Sagittal scan the esophagus is grossly dilated till GEJ with air-fluid level.

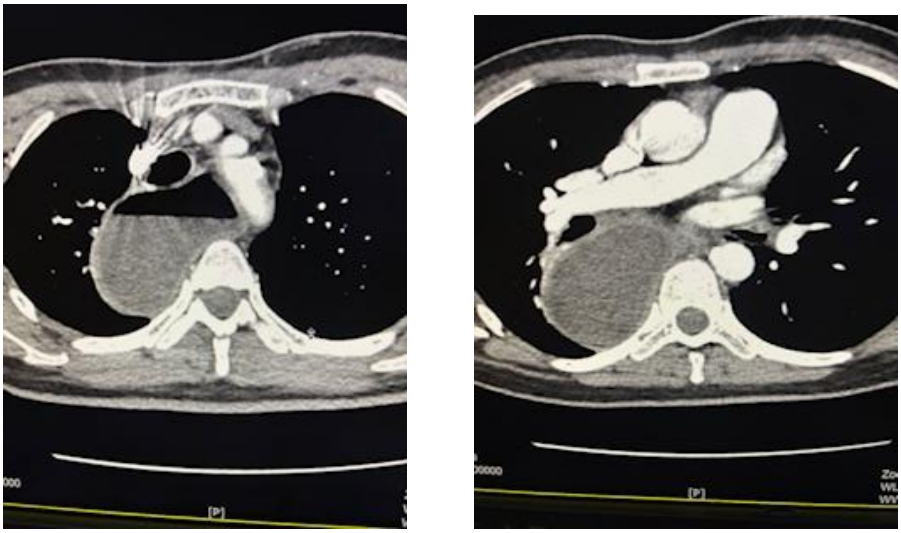

Fig. 4 - CT Thorax Axial scan-findings are, the esophagus is grossly dilated till GEJ with air-fluid level.

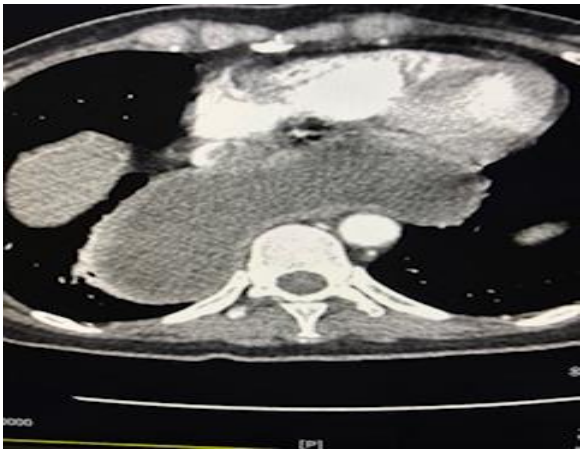

Fig. 5 - CT Thorax Axial scan-findings are, the esophagus is grossly dilated till GEJ with air-fluid level.

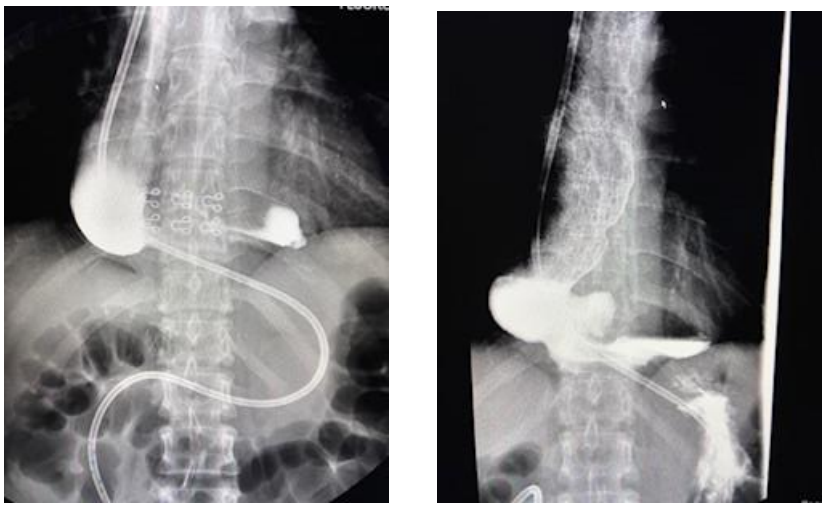

Fig. 6 - NGT given and gastrografin swallow done with gastrografin and flow of contrast is seen in to the stomach from dilated oesophagus.

DISCUSSION

Achalasia patients typically present with- 1. Dysphagia (for both solid and liquids) 2. Chest pain/discomfort 3. Eventual regurgitation 4. Aspiration pneumonia 5.Candida esophagitis 6.Esophgeal carcinoma.7. Acute airway obstruction.

Pathology of achalasia is the lower oesophageal sphincter fails to relax, partially or completely due to loss /destruction of neurons in the Auerbach/myenteric plexus. Peristalsis in the distal smooth muscle segment of the oesophagus is eventually lost due to a combination of damage to the Auerbach/myenteric plexus and vagus nerve.

Achalasia can be divided in to three subtypes. Type-I: Minimal contractility in the oesophageal body. Type II: Intermittent periods of pan-oesophageal pressurisation. Type III: Premature or spastic distal oesophageal contractions.

Differential diagnosis; Achalasia – distal segment of narrowing <3.5 cm. Central and peripheral neuropathy. Scleroderma- gastroesophageal junction will be open, less severe dilatation. Oesophageal stricture. Chagas disease- achalasia with neurenteric plexus damage due to trypanosoma cruzi infection, paraneoplastic syndrome. Diffuse oesophageal spasm etc.

Radiological stages of achalasia

Radiological stage	oesophageal diameter	oesophageal shape
I	< 4 cm	-
II	4-6 cm	-
III	> 6 cm	-
IV	>6 cm	Sigmoid

Diagnosis usually done by radiological investigation and upper endoscopy. Chest radiograph findings include- convex opacity overlapping the right mediastinum. Occasionally may present as a left convex opacity. Air-fluid level due to stasis in thoracic oesophagus filled with retained secretions and food. Small or absent of fundic gas shadow. Anterior displacement and bowing of the trachea on the lateral view. Bilateral alveolar opacities due to pneumonitis may be seen.

Barium swallow study may be used to confirm oesophageal dilatation and assess for mucosal abnormalities. Findings include bird beak sign or rat tail sign. Oesophageal dilatation. Tram track appearance central longitudinal lucency bounded by barium on both sides. Incomplete lower oesophageal sphincter relaxation. Pooling or stasis of barium in the oesophagus. Uncoordinated, non-propulsive, tertiary contractions etc.

In CT Scan patients with uncomplicated achalasia demonstrate a dilated, thin walled oesophagus filled with fluid/food. CT also helps to identify other findings and complications like malignancy.

Upper endoscopy shows dilated oesophagus with food and water retention and a tight esophagogastric junction (EGJ). All patients referred for dysphagia should first undergo esophagogastroduodenoscopy with mucosal biopsies to exclude other causes of dysphagia (6-8). Endoscopy is important test but not very sensitive because more than 40% patients with achalasia have normal endoscopic findings (9). Passage of scope raise the clinical suspicion of achalasia.

Endoscopic ultrasonography is another useful test for distinguishing between primary achalasia and pseudoachalasia. In the early stages, cancers of the EGJ may grow within the oesophageal wall, leaving the mucosa intact. As this condition can be confused with oesophageal achalasia on standard endoscopy, endoscopic ultrasonography could be very useful in shedding light on such cases (10).

Treatment includes lifestyle changes -eating slowly, increasing water intake with meals, avoiding eating near bedtime. Avoiding foods that aggravate gastroesophageal reflux. Calcium channel blockers. Pneumatic dilatation. Surgical myotomy etc.

CONCLUSION

Achalasia is caused by loss of inhibitory ganglion in the myenteric plexus in the oesophagus. Gradual progression of neuronal degeneration is associated with progression of the disease from vigorous to classic achalasia. Though several studies have attempted

to explore initiating agents that may cause the disease, the exact factors responsible for the degeneration of ganglion cells in the myenteric plexus are poorly understood. The disease is likely to be multi-factorial involving host genetic factors, autoimmunity, and environmental factors such as infections. More studies are needed to explore the exact cause of this enigmatic disease.

Conflict of interest: None.

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