WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
SJIF Impact Factor 8.084
Volume 13, Issue 2, 24-32. Review Article ISSN 2277–7105

# A REVIEW ON ACHALASIA

**Thumati Harika[1]*, E. Rajani[2] and Dr. Y. Prapurna Chandra[3]**

[1]B.Pharmacy, Ratnam Institute of Pharmacy.

[2]Associate Professor, Dept. of Pharmacology, Ratnam Institute of Pharmacy.

[3]Principal, Dept. of Pharmacology, Ratnam Institute of Pharmacy.

Article Received on
27 November 2023,

Revised on 17 Dec. 2023,
Accepted on 07 Jan. 2024

DOI: 10.20959/wjpr20242-30306

***Corresponding Author**
**Thumati Harika**
B.Pharmacy, Ratnam Institute of Pharmacy.

## ABSTRACT

Achalasia is a oesophageal motility disorder. It is characterized by the progressive ganglion cell degeneration in the oesophageal myenteric plexus, which results in the impaired lower oesophageal sphincter relaxation upon swallowing in the smooth muscle of the oesophagus. Achalasia is mainly caused by loss of nerve cells in the oesophagus and it is also caused by auto immune responses in the body. Both males and females may be effected by achalasia and senior adults may be effected. The achalasia is mostly to be in the Caucasians, smokers and obese. Achalasia can be overlooked because it has symptoms similar to other digestive disorders. Recent advances in diagnostic techniques including Oesophageal manometry may help in predicting outcome. At present pneumatic dilation and heller myotomy are combined with a procedure are the treatments of choice and have the comparable success rates.

**KEYWORDS:** Achalasia, Oesophageal sphincter, Pneumatic dilation, POEM, Diagnosis, Treatment.

## INTRODUCTION

Achalasia results from damage to nerves in the food tube, preventing the oesophagus from squeezing food into the stomach. It is characterized by the progressive degeneration of the nerve cells in the oesophagus. It is also called as Cardio Spasm. In 1672, Sir Thomas Willis, first described the condition known as achalasia. In 1881, Johann Freiherr von Mikulicz-Radecki described the disease as cardio spasm. In 1929, Hurt and Rake figured out the problem was due to the Lower Oesophageal Sphincter was not relaxing. The main signs and

symptoms of achalasia are Dysphagia (Difficulty swallowing foods or liquids, arising from the throat or oesophagus, ranging from mild difficulty to complete and painful blockage), Regurgitation (Bringing swallowed food up again to the mouth), Chest pain behind the sternum, weight loss, Aspiration (Drawing out of a fluid with a suction), Anorexia (An eating disorder causing people to obsess about weight and what they eat), Heartburn, Belching, Coughing at night, Pneumonia (From aspiration of food into the lungs), vomiting.

**Etiology**

Achalasia occurs from the degeneration of the Vagus nerve fibers and the myenteric plexus of the lower oesophageal sphincter. There is a loss of inhibitory neurons containing peptide and nitric oxide at the oesophageal myenteric plexus, but in some cases the cholinergic neurons are involved. The exact etiology of this degeneration is not clear, although many theories have been proposed. The theories includes an auto immune phenomenon, viral infection and Genetic predisposition. It occurs mostly in the central and South Africa, the oesophageal infection with the protozoan parasite can result in the loss of the ganglion cells and it leads to the aperistalsis and incomplete lower oesophageal sphincter relaxation.it also causes due to the genetics or family history.

**Epidemiology**

Achalasia is a very rare disease, occurs in one per 100000 people and prevalence of 10 per 100000. It does not affect mostly in a particular age, race and gender. However, a recent study showed that increasing in the hospitalizations. For some unknown reasons, the achalasia increases in individuals with spinal cord injury, but these cases related to the damage of the cervical and thoracic vertebrae. The reports of achalasia after the use of Endoscopic Sclerotherapy sessions. Then higher the number of the Sclerotherapy sessions than greater the risk of Achalasia. Most of the patients will have hypotensive peristalsis and defective lower oesophageal sphincter function. Achalasia occurs equally in both males and females. This mostly affects the people between the ages of 30 to 60 years, less than 2%-5% of cases occurs in the children less than 16 years. The incidence of Achalasia is increases with the age. It mostly occurs in the older age group. The data from the United States, United Kingdom, Israel, Singapore and New Zealand have demonstrated the data about 8 cases per 100000 people per year. Most of the cases are diagnosed between the ages of 30 -60 years. Although the condition cannot be cured, the symptoms can be usually be controlled with treatment.

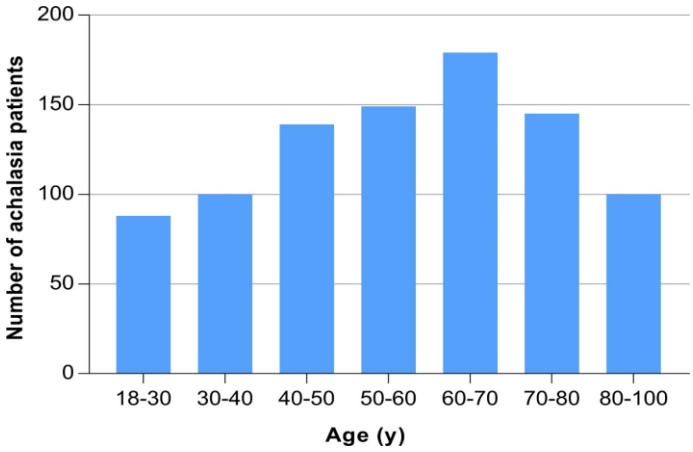


**Figure 1: The epidemiological study of achalasia.**

**Pathophysiology**

The pathophysiology of Achalasia is not well understood but, in some theories it is due to an inflammatory neurodegenarative insult with the possible viral involvement. Achalasia results from the degenaration of neurons in the esophageal wall. Histologic examination reveals decreased numbers of neurons (Ganglion cells) in the myenteric plexuses, and the ganglion cells that remain often are surrounded by lympocytes and, less prominently, by eosinophils. This inflammatory degenaration preferentially involves the nitric oxide-producing, inhibitory neurons that effect the relaxation of esophageal smoth muscle.However the cholinergic neurons that contribute to lower oesophageal sphincter tone by causing smooth muscle contraction are relatively spared.

Genetic and Autoimmune components may cause the neuronal damage. Inflammatory changes with in the oesophagus causes the loss of inhibitory neurons in the myenteric plexus and reduction in the inhibitory transmitters, nitric oxide and vasoactive intestinal peptide. The excitatory neurons are not affected, which results in the imbalance between excitatory and inhibitory neurons preventing lower oesophageal sphincter relaxation. Due to aperistalsis and a non-relaxing lower oesophageal sphincter cause dysphagia.

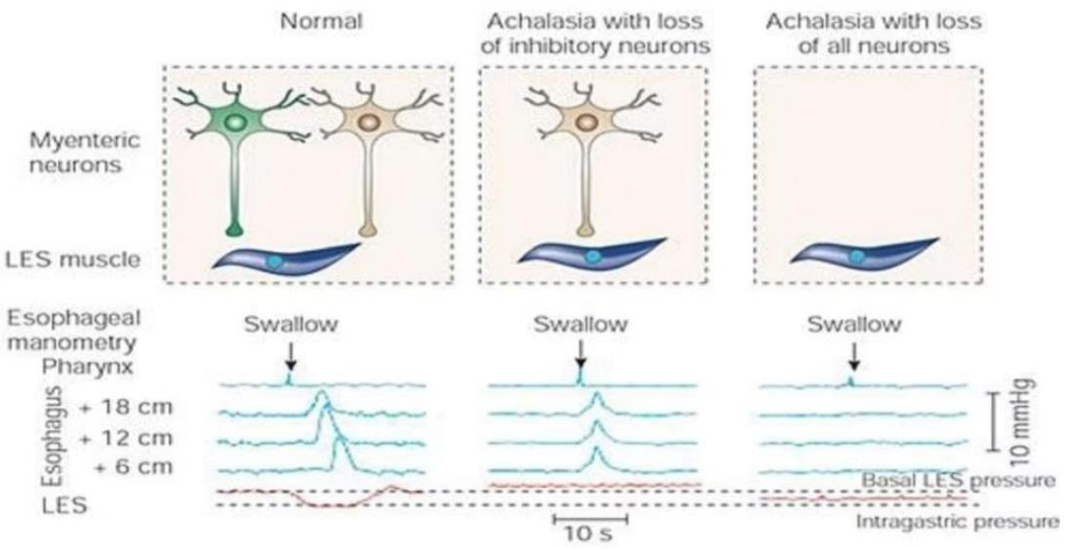


**Figure 2: Pathophysiology of achalasia.**

**Diagnosis**

Achalasia can be overlooked because it has the symptoms similar to the other digestive disorders. There are 3 types of diagnostic techniques. there are 1. Esophageal Manometry 2. X-rays of your upper digestive system (Esophagram). 3.Upper Endoscopy.

1. **Esophageal manometry:** Oesophageal Manometry is used to confrim the diagnosis of Achalasia. The test typically reveals three abnormalities in people with Achalasia:high pressure in the Lower Oesphageal Sphincter at rest, failure of the lower oesophageal sphincter to relax after swallowing, and an absence of useful contractions in the lower oesophagus. This test measures the rhythmic muscle contractions and relaxations in the oesophagus during the swallowing of the food. During oesophageal manometry, a thin, flexible tube that contains pressure sensors is passed though nose, down your oesophagus and into your stomach. Esophageal Manometry can be helpful in diagnosing certain disorders that can affect your oesophagus.

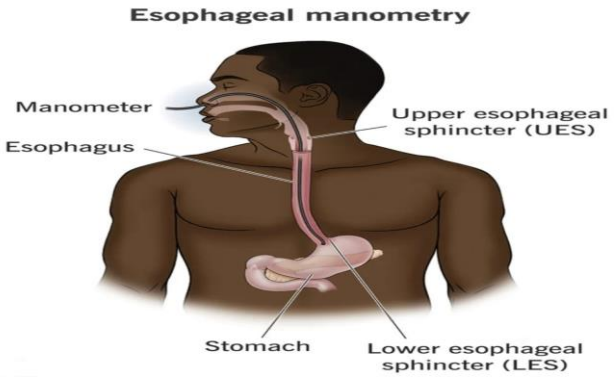


**Figure 3: Esophageal manometry of achalasia.**

2. **X-Rays of your digestive system or esophgram:** Before taking the X-rays of the oesophagus we have to drink a chalky liquid that coats and that fills inside the linning of your digestive tract. That coating allows the doctor to see a silhouette of your oesophagus and stomach.sometimes the doctor may advice to swallow barium pill that can help to show the blockage of the oesophagus.

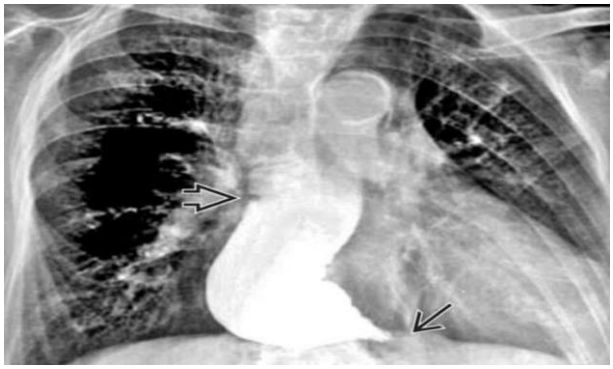

**Figure 4:X-Rays of the oesophagus with achalasia.**

3. **Upper endoscopy:** The Upper Endoscopy is used to know about the partial blockage of the oesophagus. The Gastroenterologist inserts the thin, flexible tube fitted with the light and camera (endoscope) down your throat, to examine inside of your stomach and oesophagus. It is also used to collect a sample of tissue to be tested for complications of reflux such as Barrett's oesophagus.

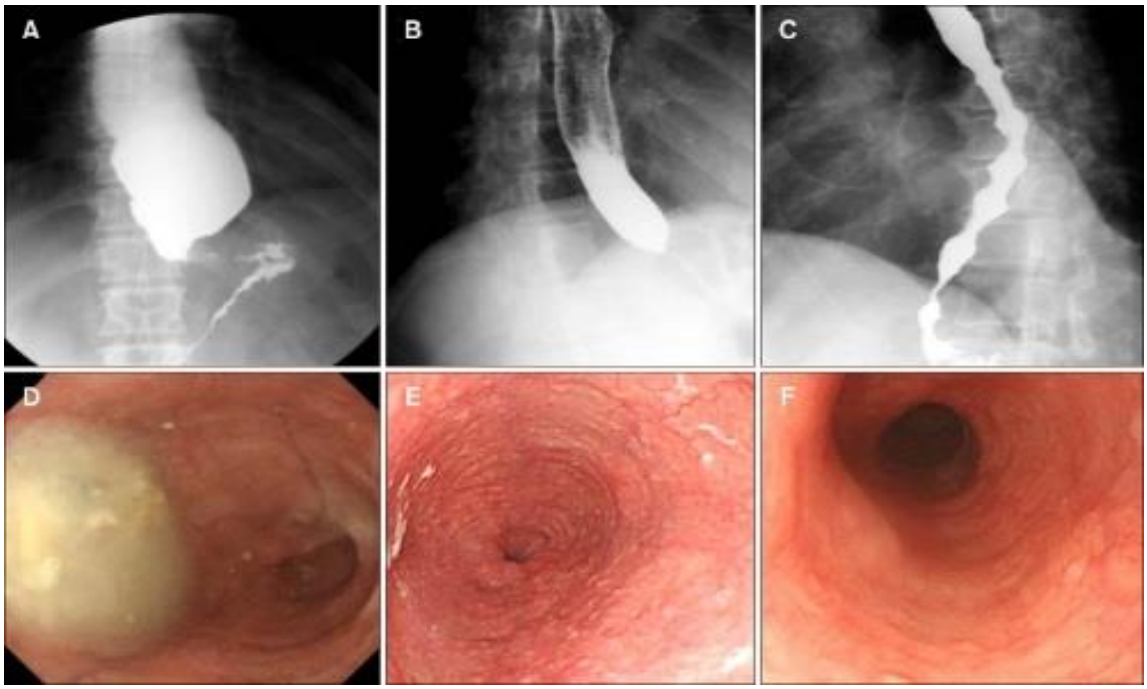


**Figure 5: Upper endosacopy of achalasia.**

**Teatment:** The treatment of Achalasia mainly focuses on stretching open the lower oesophagus sphincter. The treatment depends on your age, health condition and the severity Achalasia Patient. The treatment includes-1.Non-Surgical Treatment 2.Surgical Treatment.

1. **Non-Surgical treatment:** This treatment is done without surgery. It includes –i). Pneumatic Dilation ii). Botox (Botulinum Toxin Type A) iii). Medication. i). Pneumatic Dilation: The pneumatic dilation is a highly effective endoscopic procedure with minimal complications and mortality. With the help of the Endoscopy a balloon is inserted into the oesophageal sphincter, it enlarges the opening of the Oesophagus. The patient may repeat this procedure if the oesophageal sphincter doesn't stay open. This treatment should repeat the patients within five years. This treatment requires sedation. A single ballon dilation session continues to relieve symptoms of achalasia in about 60 percent of people. The pain occurred directly after dilation and reduced within 48 hours.

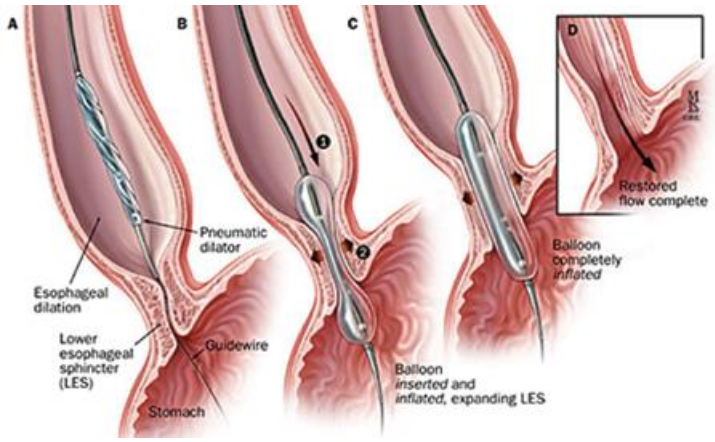


**Figure 6: Pneumatic dilation in achalasia.**

ii). Botox (Botulinum Toxin Type A): Injection of Botulinum toxin into muscle causes temporary paralysis of specific muscle, which lasts for months to over one year. Botulinum Toxins is injected into the lower oesophageal Sphincter. Botulinum toxin is used primarily to treat achalasia. Botox injection is generally recommended for the people not willing to do the Pneumatic dilation and surgery. This injections do not lost for six months.

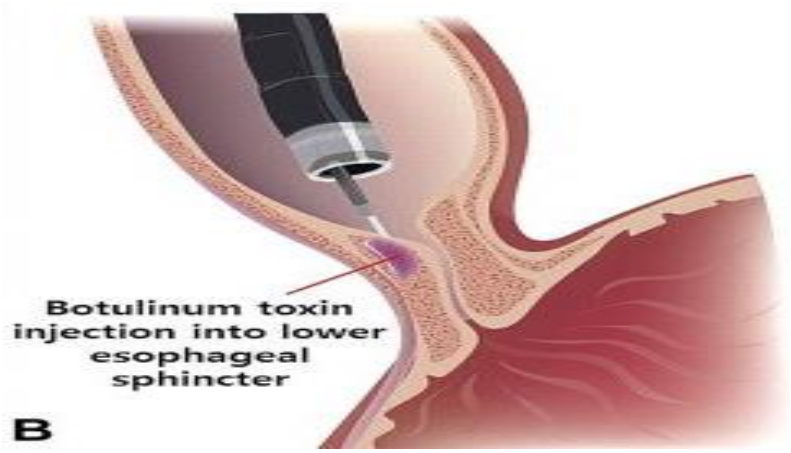


**Figure 7: Botox is injected in Achalasia patient.**

iii). Medication: The muscle relaxants Such as Nitroglycerin and Nifedipine is used before eating. This treatment has less effect and more side effects. So, this is used when people are not ready for the Pneumatic dilation, Botox and Surgery. This is very rare therapy.

2. **Surgical treatment:** The done is taken through Surgery. It includes – i).Heller Myotomy ii).Peroral Endoscopic Myotomy (POEM).

i) **Heller myotomy:** It is a minimally invasive procedure that opens the tight lower oesophageal sphincter (the wall between the oesophagus and stomach) by cutting the thick muscle of the lower part of the oesophagus and the upper part of the stomach to relieve the contraction of muscles.

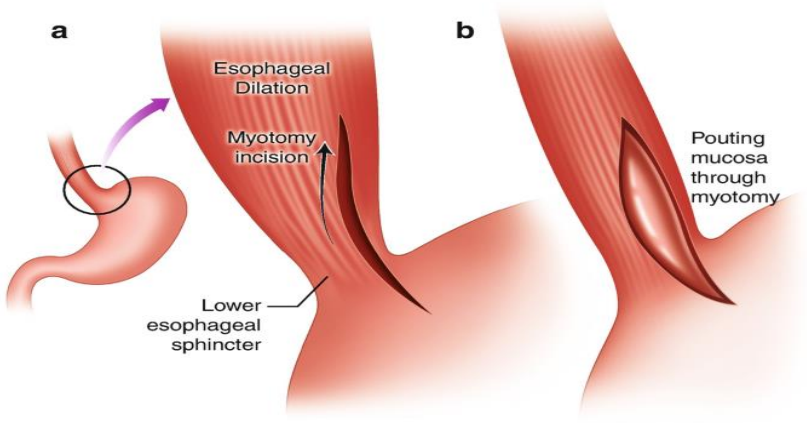


**Figure 8: Heller myotomy in achalasia patient.**

ii) **Peroral Endoscopic Myotomy (POEM):** POEM is mainly used to treat Achalasia, an excessive tightness of the ring of muscle (sphincter) between the oesophagus and stomach. POEM is a form of natural orifice transluminal endoscopic surgery that is

completed by creating a sub mucosal tunnel in the lower part of oesophagus to reach the inner circular muscle bundles of the lower oesophageal sphincter to perform myotomy, while preserving the outer longitudinal muscle bundles. With the help of the Endoscope inserted into the throat to create an incision inside the linning of oesophagus. Then the surgeon cuts the muscle at the lower oesophageal sphincter.

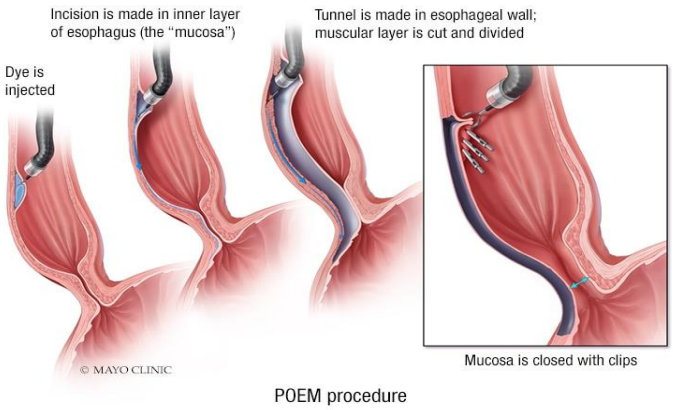


**Figure 9: POEM procedure in Achalasia patient.**

## CONCLUSION

Achalasia is an oesophageal motility disorder. It is caused by the Auto immune responses in the oesophagus. The main signs and symptoms are dysphagia, regurgitation, chest pain and weight loss, these may reduce the patient's life. Mostly Endoscopy is used to diagnose the Achalasia. The treatment of Achalasia is incurable, the treatment is to relieve the symptoms of the Achalasia. The POEM procedure shows more output than comparatively other treatments. POEM proven that it is effective to treat all the disorders of Oesophagus. More studies are required to properly determine the long-term efficacy and safety of treatment of oesophagus.

## REFERENCES

1. Edoardo Savarino, shobna Bhatia, Sabine Roman, Prakash Gyawali. Achalasia. Nature Reviews Disease Primers, 2022; 8(28).
2. Mohan Ramchandani, Partha Pal. Achalasia Cardia: A comprehensive Review. E M J Gastroenterol, 2023; 9(1): 106-117.
3. Ming-Yueli, Dong -Yang Wang, Qing-Hua Wang, Run-Peng. Pathogenesis, clinical manifestations, diagnosis and treatment progress of Achalasia Cardia. World J Cases, 2023; 11(8): 1741-1752.

4. Orla MO' Neil, brain T Johnston, Hellen G Coleman, Achalasia: A review of clinical diagnosis, Epidermology, treatment and outcomes. World J of Gastroenterol, 2013; 19(35): 5806-5812.
5. Amir Mari, Fadi Abu Baker, Rinaldo Pellicano, Jawfik Khoury. Diagnosis and management of Achalasia. J Clin Med, 2021; 10(16): 3607.
6. Joshua Tuason, Haruhiro Inoue. A review on diagnosis and treatment of Achalasia. J Gastroenterology, 2017.
7. Froukje B.Van Hoeji, Lean I. Prims, Andre J.P.M. Smout, Arjan J Bredenoord. A comprehensive review on Achalasia. Neurogastroenterology & Motility, 2019; 31(7): 13548.
8. Dhyanesh A. Patel, Hannah P.kim, Michael F.vaezi. Achalasia: A review. Orphanet J Rare Diseases, 2015; 10(89).
9. Sadowski DC, Ackah F, B.Jiang, L.W.Svenson. Achalasia: Incidence, prevalence & survival. Neurogastroenterol & Motility, 2010; 22(9): 256-261.
10. Sinan H, Tatum RP, Soares RV, Martin AV. Prevalence of respiratory Symptoms in patients with Achalasia. Dis Esophagus, 2011; 24(6): 224-228.
11. Von Renteln D, Inoue H, Minami H. Peroral Endoscopic Myotomy for the treatment of Achalasia: a prospective single center study. Am J Gastroenterol, 2012; 107(3): 411-417.
12. Fangxiao Gong, Yuanyan Li, Sen Ye. Effectiveness and Complications of Achalasia treatment: A systemic review. Asian Journal of Surgery, 2023; 46(1): 24-34.
13. Castagliuolo I, Brun P, Costantini M, Rizzetto C, Palu G, Costantino M, Baldan Zaninotto G. Esophageal achalasia: is the herpes simplex virus really innocent. J Gastrointest Surg, 2004; 8: 24-30.
14. Lau KW, McCaughey C, Coyle PV, Murray LJ, Johnston BT. Enhanced reactivity of peripheral blood immune cells to HSV-1 in primary Achalasia. Scand J Gastroenterol, 2010; 45: 806-813.
15. Ghoshal UC, Daschakraborty SB, Singh R. Pathogenesis of Achalasia cardia. World J Gastroenterol, 2012; 18: 3050-3057.