DOI: 10.7860/JCDR/2024/73812.20325

Case Report

Internal Medicine Section

# Non Tubercular Mycobacterial Infection as a Cause of Non Resolving Aspiration Pneumonia in a Case of Achalasia Cardia: A Case Report

SHAILESH BHANUDAS MESHRAM[1], HIRAL GULAB RAMNANI[2]



## ABSTRACT

Achalasia is an oesophageal motor disorder characterised by the absence of peristalsis and swallowing difficulties, which results in poor clearance of the oesophagus. The Lower oesophageal Sphincter (LES) fails to relax, either partially or completely, with elevated pressures demonstrated monometrically. Hereby, the authors present an interesting case of a 38-year-old male who presented to a tertiary care hospital with high-grade fever, difficulty in breathing, chronic cough with whitish-yellowish sputum, and generalised weakness for one month. He had made multiple hospital visits for similar complaints and was treated as a case of aspiration pneumonia. Upon admission, he was febrile but otherwise had stable vital signs. Initial investigations revealed bilateral inhomogeneous opacities on chest radiography, a total leukocyte count of 25,000/mm$^3$, and haemoglobin of 7 gm%. He was started on empirical antibiotics and antipyretics, and due to difficulty swallowing, a nasogastric tube was inserted under fluoroscopic guidance. High-Resolution Computed Tomography (HRCT) showed a dilated thoracic oesophagus with mild tracheal and Superior Vena Cava (SVC) compression, bilateral mass-like consolidation, and an air-fluid level, raising suspicions of malignancy or lung abscess. Upper gastrointestinal endoscopy revealed a dilated, tortuous oesophagus with white plaques. A 2D echocardiography showed an ejection fraction of 60% with moderate pulmonary arterial hypertension. Despite initial antibiotic therapy reducing the leukocyte count, his cough and fever persisted. Fiber optic bronchoscopy indicated mild tracheal compression and bilateral mucosal congestion, and a transbronchial lung biopsy was performed. Bronchoalveolar Lavage (BAL) samples tested negative for the Cartridge-based Nucleic Acid Amplification Test (CBNAAT) but showed acid-fast bacilli on Gram and Ziehl-Neelsen (ZN) staining. Repeated sputum samples and CBNAAT tests revealed similar results, confirming the presence of Non Tubercular Mycobacteria (NTM), and appropriate treatment was started. The patient gradually improved, with a reduction in fever spikes over a month. He later underwent laparoscopic Heller's cardiomyotomy with Dor's fundoplication, the treatment of choice for achalasia cardia. Currently, patient has completed nine months of treatment for NTM with visible clinical and radiographic improvement.

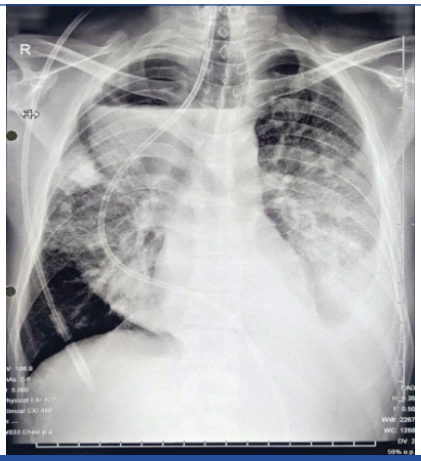
**Keywords:** Bronchoalveolar lavage, Consolidation, Dysphagia, Mediastinal disease, Oesophagitis

## CASE REPORT

A 38-year-old male presented to the Emergency Department with complaints of high-grade fever (103.1° Fahrenheit), difficulty in breathing with modified Medical Research Council (mMRC) grade 2, chronic cough with whitish-yellow expectorant, and generalised weakness for the past month. The patient was vitally stable, maintaining 98% saturation on room air. Additionally, he had visited multiple hospitals for similar complaints and had received several antibiotics, including tablet Augmentin 625 mg three times a day for seven days, followed by tablet Levofloxacin 500 mg once daily for ten days. As the symptoms did not subside, he was referred to us for further management.

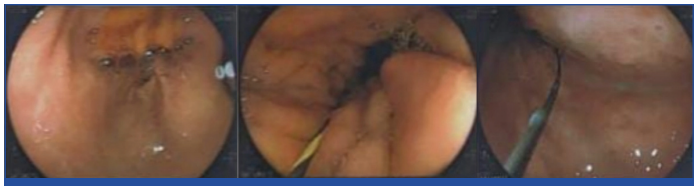
Upon admission, the patient was febrile, and auscultation revealed bilateral coarse crepitations. Chest radiography showed bilateral inhomogeneous opacities, raising suspicion of aspiration pneumonia [Table/Fig-1]. Routine blood investigations reported a total leukocyte count of 25,000/mm$^3$ and haemoglobin of 7 gm%. Empirical antibiotics, such as injection Monocef 1 gram intravenously twice a day, were started for the initial two days. The patient was then shifted to injection Meropenem 500 mg intravenously three times a day, and injection paracetamol 1 gram was given on an as-needed basis for fever.

During the course of treatment, the patient was unable to swallow, so a nasogastric tube was inserted through the mouth into the stomach, and a 14 French nasojejunal tube was passed over a guidewire into the stomach. The guidewire and nasojejunal tube were then pulled out. The final position of the nasogastric tube was confirmed using fluoroscopy, and a repeat chest radiograph suggested dilated oesophagus, indicating achalasia cardia that mimicked aspiration pneumonia [Table/Fig-2].



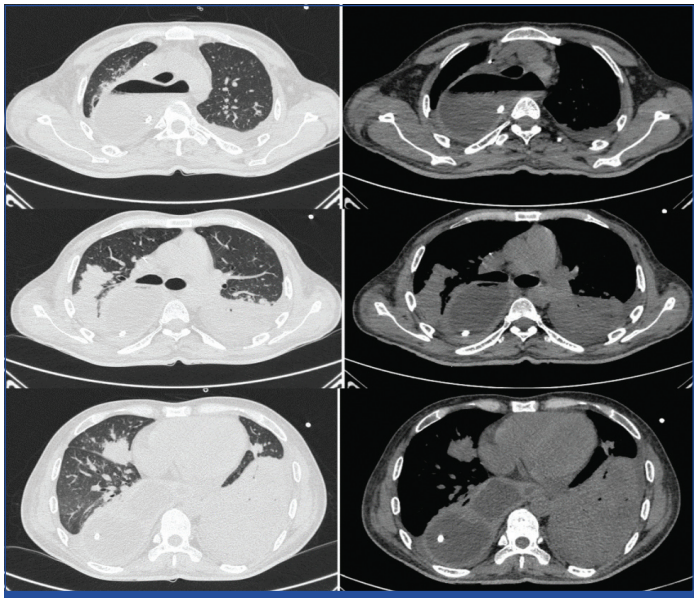
**[Table/Fig-1]:** Chest radiograph Posteroanterior (PA) view showing aspiration pneumonia.

High Resolution Computed Tomography (HRCT) of the thorax suggested a dilated thoracic oesophagus up to the Gastro-Oesophageal Junction (GEJ) with mild tracheal and superior vena cava compression. The dilated oesophagus returned to midline just proximal to the esophago-gastric junction, with a maximum



**[Table/Fig-2]:** Nasogastric tube insertion under fluoroscopic guidance.

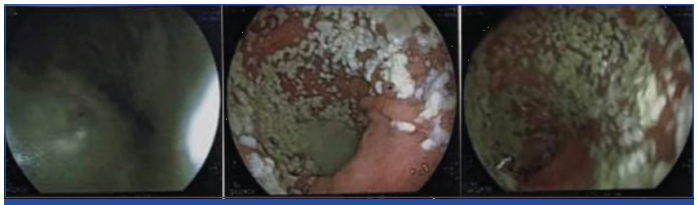
transverse diameter of 8 cm. There was a mass-like consolidation in the right upper and middle lobes and dense consolidation in the left lower lobe. An air-fluid level was seen with the Ryle's tube in-situ, mild wall thickening in the lower third of the oesophagus along the lateral aspect was noted, with a maximum thickness of 8 mm and a length of 7 cm. The differentials considered included malignancy, lung abscess, and some rare variant of tuberculosis [Table/Fig-3].



**[Table/Fig-3]:** CT Chest showing dilated thoracic oesophagus upto Gastro-Esophageal Junction (GEJ) with mild tracheal and Superior Vena Cava (SVC) compression with a mass-like consolidation in the right upper and middle lobes and dense consolidation in the left lower lobe with air-fluid level in right lung and with Ryle's tube in situ.

Ultrasound of the thorax revealed bilateral pleural effusion, with 300-350 cc on the right-side and 20-30 cc on the left-side. Right-sided pleural tapping yielded inconclusive results.

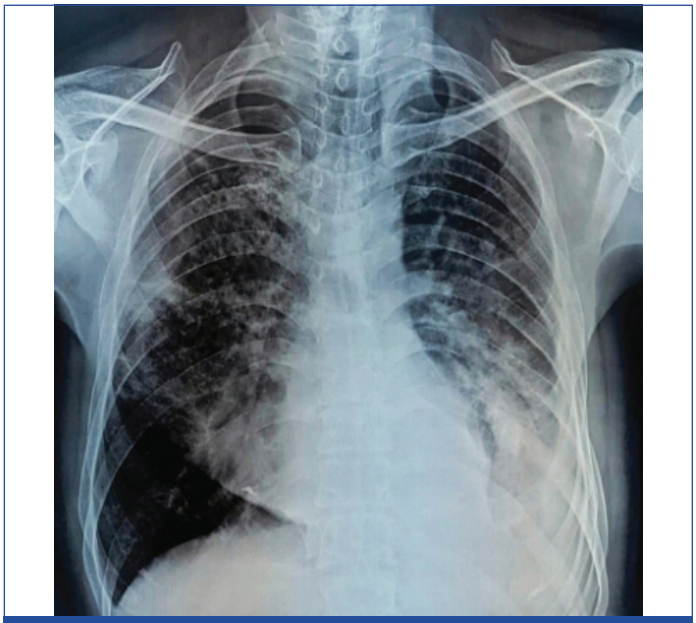
An upper gastrointestinal endoscopy was performed using a Fujinon ultrathin scope, which revealed a dilated, tortuous oesophagus with solid and liquid food residue. Liquid residues were suctioned. Following suctioning, whitish plaques were noted, suggesting candidiasis. The pinpoint GEJ was visible through the ultrathin scope, but the scope could not be passed further. A Ryle's tube was placed for decompression, but feeds were not given through it [Table/Fig-4].



**[Table/Fig-4]:** Upper gastrointestinal scopy was done using Fujinon ultrathin scope showing dilated, tortuous oesophagus.

The 2D echocardiography showed an ejection fraction of 60% with moderate pulmonary arterial hypertension. Antibiotic therapy initially reduced leucocyte counts to 14,000; however, cough and fever persisted at 102 degrees Fahrenheit. A fiber optic bronchoscopy was performed, revealing mild compression of the trachea, bilateral mucosal congestion, and transbronchial lung biopsy taken from the superior segment of the lower lobe. A BAL sample was sent for CBNAAT, which turned out to be negative, while Gram and ZN staining showed acid-fast bacilli. To confirm, a sputum sample was sent for CBNAAT, and Gram, ZN staining, and culture. The staining revealed acid-fast bacilli, while the ZN stain and CBNAAT were negative. A repeat smear and CBNAAT were performed with similar results, pointing towards NTM, and treatment for the same was initiated. The patient gradually improved with a reduction in fever spikes over the course of one month.

Procedural fitness was delayed due to weakness and non-resolving pneumonia. Later, he underwent laparoscopic Heller's cardiomyotomy with Dor's fundoplication for achalasia cardia. Currently, he has completed nine months of treatment for NTM with tablet Rifampicin 450 mg once a day, tablet Ethambutol 800 mg once a day, and tablet Clarithromycin 500 mg once a day, after being crushed and dissolved in one glass of water through a Ryle's tube. He has no symptoms, and there is resolution in the chest radiographs [Table/Fig-5].



**[Table/Fig-5]:** Chest radiograph Posteroanterior (PA) view showing resolving aspiration pneumonia post-treatment.

## DISCUSSION

Achalasia is derived from the Greek term meaning "failure to relax." The Lower Oesophageal Sphincter (LES) remains contracted due to defective innervation of the smooth muscle, resulting in achalasia. This motor disorder appears to be due to the loss or destruction of neurons in the Auerbach or myenteric plexus. In the early stages of achalasia, the LES tone may be normal, or subtle changes may be observed. Primary achalasia most frequently occurs in individuals during the 3rd to 7th decades of life, with no gender predilection [1,2]. Most cases are idiopathic; however, a similar presentation may occur in Chagas disease [3]. The primary aetiology of achalasia is usually idiopathic, but secondary causes may include gastric carcinoma extending to the oesophagus, lymphoma, Chagas disease, irradiation, and certain medications and toxins [4].

Achalasia is a rare disease that mimics respiratory symptoms. The oesophagus filled with debris and undigested food increases the risk of aspiration [5]. Chest pain is the most common symptom, occurring in 50% of cases, while aspiration pneumonia may occur rarely. In the absence of radiological suspicion of achalasia, a patient may initially be managed for respiratory conditions, which can lead to complications like aspiration pneumonia and a subsequent diagnostic delay [6].

The usual clinical presentation includes progressive dysphagia for both liquids and solids, regurgitation, and chest pain. Symptoms are frequently misdiagnosed as gastroesophageal reflux disease, which can delay diagnosis by two to three years. In a recumbent

position, the symptoms worsen. It is important to rule out pseudoachalasia due to a tumour in individuals who have recently experienced dysphagia (less than six months), have lost weight, and are older than 50 years. In this case, a 76-year-old patient with hypoxic respiratory failure and bronchorrhea was diagnosed with aspiration pneumonia attributed to achalasia cardia, similar to present case, with the difference that our patient tested positive for Non-Tuberculous Mycobacteria (NTM) [7].

In patients presenting with dysphagia, chest pain, and regurgitation, pulmonary aspiration may occur due to the overflow of saliva and ingested food lodged in the oesophagus [8]. Regurgitation gradually increases as the disease progresses. Aspiration of oesophageal contents into the respiratory tract leads to chronic and/or acute pulmonary infection. The majority of reports describe cases of patchy bilateral alveolar opacities that resemble aspiration pneumonia [6]. In the present case, the patient was initially misdiagnosed, resulting in the prescription of multiple courses of antibiotics. This delayed the correct treatment and ultimately led to the development of aspiration pneumonia. A diagnosis of pneumonia should be considered, particularly in younger patients with pneumonia who are suspected to have aspiration [9]. Additionally, aspiration of gastric content may cause respiratory failure in these patients with achalasia, and etiological factors other than pneumonia should also be evaluated in patients presenting with high fever and cough [6,10].

Aspiration pneumonitis implies the presence of an inflammatory response to aspirated material not associated with infection, while aspiration pneumonia implies the presence of infection along with pneumonitis [8]. In another case involving a 33-year-old male with chronic cough and fever, chest tomography showed pneumonia revealing oesophageal obstruction, which led to the diagnosis of achalasia cardia. In present case, the findings were positive for acid-fast bacilli [11].

The incidence of NTM has increased, possibly due to better detection methods, increased clinical awareness, and a growing number of immunosuppressed individuals. The present case highlights the association between NTM and patients with oesophageal motility problems [12]. The association between achalasia and NTM has been recognised since 1953 [13]. *M. fortuitum* has been identified as the most common pathogen in this situation. The pathogenesis remains unclear, but there is growing evidence that lipoid pneumonia is a predisposing factor for NTM infection. It is postulated that lipids may act as a trigger to promote mycobacterial growth. In achalasia, the stagnant food may serve as this trigger when aspirated into the bronchopulmonary tree. NTM is generally susceptible to macrolides, tetracyclines, fluoroquinolones, and sulfonamides, with treatment lasting 9 to 12 months. *M. fortuitum* is typically considered curable with antibiotic therapy, although cure rates for other members of the rapidly growing mycobacteria are lower [12].

## CONCLUSION(S)

The present rare and interesting case describes a patient with achalasia cardia complicated by aspiration pneumonia, which was unresponsive to multiple antibiotic regimens. The lack of improvement raised suspicion of an atypical infection or tuberculosis. BAL identified acid-fast bacilli; however, multiple CBNAAT tests for tuberculosis were negative. This led to a diagnosis of NTM, which were unresponsive to previous antibiotic regimens. The patient was treated with a combination of rifampicin, ethambutol, and clarithromycin, resulting in significant clinical improvement, including reduced fever and resolving radiological findings. In such cases where treatment does not yield the expected results, it is crucial to consider other differentials. Rare causes of bacterial colonisation, such as NTM, must be identified and treated to reduce mortality and improve healthcare outcomes.

## REFERENCES


[1] Sinan H, Tatum RP, Soares RV, Martin AV, Pellegrini CA, Oelschlager BK. Prevalence of respiratory symptoms in patients with achalasia. Dis Esophagus. 2011;24(4):224-28.
[2] Bailey BJ, Johnson JT, Newlands SD. Head & neck surgery--otolaryngology. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2006.
[3] Gaillard F, Niknejad M, Knipe H, et al. Achalasia. Radiopaedia. 2024. Available from: https://doi.org/10.53347/rID-835.
[4] Arslan M, Ercen Diken O, Adali G. A case of achalasia causing aspiration pneumonia. Turk Klin Arch Lung. 2016;17:01-04. Doi: 10.5336/archlung.2015-45272.
[5] Hunter JG, Trus TL, Branum GD, Waring JP. Laparoscopic Heller myotomy and fundoplication for achalasia. Ann Surg. 1997;225(6):655-64.
[6] Akritidis N, Gousis C, Dimos G, Paparoas K. Fever, cough, and bilateral lung infiltrates. Achalasia associated with aspiration pneumonia. Chest. 2003;123(2):608-12.
[7] Ali HA, Murali G, Mukhtar B. Respiratory failure due to achalasia cardia. Respir Med CME. 2009;2(1):40-43.
[8] Robinson KM, Zorowitz RD. Mechanism of aspiration disorders. In: Elias JA, Fishman JA, Grippi MA, Senior RM, editors. Fishman's pulmonary diseases and disorders. 3rd ed. New York: McGraw-Hill; 1997. p. 1211-24.
[9] Hirata M, Kohno K, Murakami S, Yoneda S, Kajita Y, Fujita Y, et al. A case of recurrent aspiration pneumonia by achalasia. Nihon Kokyuki Gakkai Zasshi. 2002;40(2):149-53. Japanese.
[10] Kendall AP, Lin E. Respiratory failure as presentation of achalasia of the oesophagus. Anaesthesia. 1991;46(12):1039-40.
[11] Ono Y, Kobayashi H, Nagasaki K, Ariga H, Kashimura J. Aspiration pneumonia associated with achalasia. J Gen Fam Med. 2022;23(3):195-96. Doi: 10.1002/jgf2.524. PMID: 35509346; PMCID: PMC9062550.
[12] Church AC, Watkin S. Non-tuberculous mycobacteria masquerading as aspiration pneumonia in patients with gastrointestinal problems. Respir Med. 2006;100(9):1663-65.
[13] Gibson JB. Infection of the lungs by saprophytic mycobacteria in achalasia of the cardia, with report of a fatal case showing lipoid pneumonia due to milk. J Pathol Bacteriol. 1953;65(1):239-51.



**PARTICULARS OF CONTRIBUTORS:**
1. Associate Professor, Department of Respiratory Medicine, Dr. D. Y. Patil Medical College, Hospital and Research Centre, Pune, Maharashtra, India.
2. Resident Doctor, Department of Respiratory Medicine, Dr. D. Y. Patil Medical College, Hospital and Research Centre, Pune, Maharashtra, India.

**NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:**
Dr. Hiral Gulab Ramnani,
A-9, Carnation Girl's Hostel, Dr. D. Y. Patil Medical College and Hospital,
Sant Tukaram Nagar, Pimpri, Pune-411018, Maharashtra, India.
E-mail: hiralramnani23@gmail.com



**PLAGIARISM CHECKING METHODS:** [Jain H et al.]
- Plagiarism X-checker: Jul 03, 2024
- Manual Googling: Sep 17, 2024
- iThenticate Software: Oct 04, 2024 (7%)

**ETYMOLOGY:** Author Origin

**EMENDATIONS:** 5

**AUTHOR DECLARATION:**
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Jul 02, 2024**
Date of Peer Review: **Sep 09, 2024**
Date of Acceptance: **Oct 05, 2024**
Date of Publishing: **Nov 01, 2024**