Esophageal Tuberculosis: A Rare Case Report

P. Prasanth¹, Nirmal Chand Kajal¹, Ritu Dadra¹, K. T. Nithin¹, Jasvir Kaur¹
¹Department of Chest and TB, Government Medical College, Amritsar, Punjab, India

Abstract

After a steady decline throughout the 20th century, the incidence of tuberculosis (TB) in industrialized countries has started to rise again. However, in developing countries like India, the menace of TB had never been controlled. Gastrointestinal (GI) TB is rare, and the GI tract is considered only the sixth most frequent site of extrapulmonary TB. Esophageal TB (ET) is still rarer. This is a case report of a rare form of ET in a patient presenting with dysphagia. The patient was subjected to upper GI endoscopy, which revealed an ulcerative growth in the distal esophagus. Histopathology revealed ET. The patient was managed conservatively with anti-TB treatment (ATT). In spite of the rare nature of the disease, it can be managed effectively with ATT to avoid complications (fistula, stricture, and esophageal perforation), which might warrant surgery.

Keywords: Dysphagia, esophageal ulcers, gastrointestinal tuberculosis

Introduction

Gastrointestinal (GI) tuberculosis (TB) can involve any site in the GI tract, with the terminal ileum, cecum, and the peritoneum being the usual sites. Esophageal TB (ET) is a rare condition (accounts for only 2.8% of all cases of GI TB).[1] Dysphagia as a presenting manifestation of TB is rare and occurs either due to primary involvement of the esophagus by TB or secondary to direct extension from adjacent structures.[2] Primary ET is very rare because of various protective mechanisms such as the presence of stratified squamous epithelium covered with mucus. Hence, most of the cases of ET are because of secondary involvement from the surrounding infected structures.[3] We present the case of a 50-year-old male with dysphagia who was diagnosed with ET. This case report emphasizes the importance of suspecting tubercular involvement in esophageal ulcers particularly in endemic regions like India to avoid complications such as bleeding, perforation, fistula formation, aspiration pneumonia, fatal hematemesis, traction diverticula, and esophageal strictures.

Case Report

A 50-year-old male presented to our hospital with complaints of dysphagia for the past 4 months, primarily for solid foods. The patient also had a history of intermittent low-grade fever (unrecorded), loss of appetite, and loss of weight for the past 4 months. The patient had no history of cough and expectorations, breathlessness, and chest discomfort. The patient is an auto-rickshaw driver by occupation for the past 20 years, is a smoker who smokes 5–10 cigarettes in a day for the past 25 years with a smoking index of 12.5 pack-years, and is nonalcoholic. The patient had never taken antitubercular therapy in the past, and he had no contact history with a case of TB. On admission, the patient was febrile (temperature 99.8°F) tachycardic (heart rate 94/min), and tachypneic (respiratory rate 22/min). Blood investigations revealed leukocytosis (total leukocyte count 12,800) with a lymphocytic predominance and erythrocyte sedimentation rate of 110 mm in the 1st hour, tested negative for HIV, hepatitis C virus, and hepatitis B surface antigen. The patient’s sputum examination for acid-fast bacilli (AFB) came out to be negative. Chest X-ray of the patient was normal [Figure 1], and his contrast-enhanced computed tomography (CT) thorax showed distal esophageal thickening.
with luminal narrowing and few enlarged mediastinal lymph nodes. Gastroesophagoduodenoscopy was performed in which ulcerations with nodularity and friability, extending into the gastroesophageal junction, were visualized [Figure 2a and b]. Histopathological examination revealed ill-defined epithelioid granulomas with giant cells and caseating necrosis, with no evidence of malignancy present. On cartridge-based nucleic acid amplification test, *Mycobacterium tuberculosis* was detected. As per the Revised National Tuberculosis Control Programme (RNTCP) guideline, Directly observed treatment short course (DOTS) regimen was started with isoniazid, rifampicin, pyrazinamide, and ethambutol. The patient was advised to continue anti-TB treatment (ATT) for 6 months as prescribed.

**DISCUSSION**

ET is a rare condition, which accounts for 2.8% of all cases of GI TB.[9] Primary TB of the esophagus is extremely rare, perhaps owing to intrinsic protective mechanisms, such as stratified epithelial lining, presence of saliva, mucous-coated tubular structure, and peristalsis. Several mechanisms have been proposed to explain the spread of infection to the esophagus, resulting in secondary ET, as follows: (1) infection of the esophageal mucosa from swallowed tuberculous sputum; (2) contiguous extension from laryngeal and pharyngeal lesions; (3) contiguous extension from other adjacent infected structures, such as the mediastinum, hilar lymph nodes, or vertebrae; (4) retrograde lymphatic spread; and (5) hematogenous infection in the course of generalized disseminated miliary TB.[9] The symptoms of ET include dysphagia, odynophagia, chest pain, low-grade fever, and weight loss. Esophageal involvement by TB usually occurs at the middle third of the esophagus at the level of the carina, but in our patient, the lesion was in the distal esophagus. Three histomorphologically distinct types exist, namely (1) Ulcerous type (most common): mycobacteria initially involve the submucosa of the esophagus followed by the formation of tuberculosis. As the disease progresses, caseous necrosis occurs within the nodule, followed by ulceration; (2) hyperplastic type: it is due to excessive amount of tuberculous granulation tissue and fibrous tissue hyperplasia. Sometimes, due to massive hyperplasia, there can be false tumor-like mass (pseudotumor) formation into the esophageal lumen, resulting in luminal narrowing; and (3) granular ET (least common): it occurs in the severe systemic disease where the mucosa and submucosa show many gray-white nodules.[9] The clinical, radiological, and endoscopic features of ET are not well defined because of its rarity and also its close resemblance with other symptomatic esophageal disorders.[1] Approximately 65% of the patients with ET have nonspecific findings on chest radiograph; however, CT of the chest shows characteristic tuberculous lymphadenitis.[6] Histopathology and TB-polymerase chain reaction (PCR) are the mainstay investigations for confirming the diagnosis of ET. Histology shows epithelioid granuloma with Langhans cells and central caseous necrosis. Classical granulomas are seen only in 50% of cases, whereas AFB are demonstrated in <25% of cases. Endoscopic mucosal biopsy has a sensitivity of 22% as reported by Mokoena et al.[4] Recently, cytology and PCR have also proven useful in cases where the initial biopsies showed nonspecific changes.[7] The differential diagnosis of ET includes esophageal carcinoma, Crohn’s disease, moniliasis, actinomycosis, syphilis, and esophageal injury secondary to the ingestion of caustic material. Most of the patients respond well with ATT.[8] Even patients with ET

![Figure 1: Chest X-ray of the patient was normal](image1)

![Figure 2: (a) Gastroesophagoduodenoscopy showing mucosal ulcerations with nodularity in the lower 1/3rd of the esophagus. (b) Gastroesophagoduodenoscopy showing mucosal ulcerations with nodularity in the lower 1/3rd of the esophagus](image2)
complicated with esophagotracheal and esophagomediastinal fistulas were safely treated with Antituberculous therapy (ATT) synonymous alone. As per the RNTCP guidelines, ET is treated with isoniazid, rifampicin, pyrazinamide, and ethambutol for 2 months initially and then continued with isoniazid, rifampicin, and ethambutol for 4 months.[9] The reported complications of ET are aspiration pneumonia, fatal hematemesis, esophagotracheal fistula, esophagomediastinal fistula, traction diverticula, and esophageal strictures which may require surgery and amyloidosis. Amyloidosis can be diagnosed after a variable interval of diagnosis of TB as also with concomitant active TB. In a series of two cases, the diagnosis of secondary amyloidosis was reported as early as 2–4 weeks of the diagnosis of TB. The presence of pedal edema and proteinuria suggests the possibility of amyloidosis in patients with TB. The diagnosis of secondary amyloidosis portends a grave prognosis and treatment is usually supportive.[10]

**Conclusion**

In patients with a risk factor for TB, ET should be included in the differential diagnosis of dysphagia and odynophagia even despite its rarity. Dysphagia is the most common presenting feature of ET, and this condition should be considered as a differential diagnosis whenever a lesion is negative for malignancy. Histopathology and TB-PCR are the key to confirm the diagnosis. Delay in diagnosis can lead to complications, which might require surgical intervention; otherwise, this condition is effectively treated with ATT.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

**Financial support and sponsorship**

Nil.

**Conflicts of interest**

There are no conflicts of interest.

**REFERENCES**

1. Patel N, Amarapurkar D, Agal S, Baijal R, Kalshrestha P, Pramanik S. Gastrointestinal luminal tuberculosis: Establishing the diagnosis. J Gastroenterol Hepatol 2004;19:1240-6.
2. Rana SS, Bhasin DK, Sharma V, Chaudhary V, Singh K. Dysphagia as the first manifestation of tuberculosis. Endoscopy 2011;43 Suppl 2 UCTN: E300-1.
3. Welzel TM, Kawan T, Bohle W, Richter GM, Bosse A, Zoller WG. An unusual cause of dysphagia: Esophageal tuberculosis. J Gastrointestin Liver Dis 2010;19:321-4.
4. Mokoena T, Shama DM, Ngakane H, Bryer JV. Oesophageal tuberculosis: A review of eleven cases. Postgrad Med J 1992;68:110-5.
5. Rosario MT, Raso CL, Comer GM. Esophageal tuberculosis. Dig Dis Sci 1989;34:1281-4.
6. Nagi B, Lal A, Kochhar R, Bhasin DK, Gulati M, Suri S, et al. Imaging of esophageal tuberculosis: A review of 23 cases. Acta Radiol 2003;44:329-33.
7. Fujiwara T, Yoshida Y, Yamada S, Kawamata H, Fujimori T, Imawari M. A case of primary esophageal tuberculosis diagnosed by identification of mycobacteria in paraffin-embedded esophageal biopsy specimens by polymerase chain reaction. J Gastroenterol 2003;38:74-8.
8. Baijal R, Agal S, Amarapurkar DN, Kumar P, Kotli N, Jain M. Esophageal tuberculosis: an analysis of fourteen cases. J Dig Endosc 2010;1:14-8.
9. Available from: https://tbcindia.gov.in/showfile.php?lid=3245. [Last accessed on 2019 Sep 24].
10. Sharma V, Prasad KK, Mandavdhare HS. Double trouble: Mediastinal lymph nodal tuberculosis complicated by amyloidosis and esophago-nodal fistula after endoscopic ultrasound fine-needle aspiration. Int J Mycobacteriol 2018;7:296-7.