Interesting case of spontaneously resolved dysphagia in a young female due to complicated esophageal tuberculosis

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Abstract

Mycobacterium tuberculosis can affect any organ of the body. Gastrointestinal tubercular involvement is fairly common. Esophageal tuberculosis though is uncommon. Esophageal tuberculosis accounts for only 0.3% of gastrointestinal tuberculosis. It presents with dysphagia, retrosternal pain, cough or constitutional symptoms. Complications like hemorrhage from the ulcer and development of fistulas like esophagomediastinal fistula is extremely uncommon. We report a case of a 27 years old female who presented with retrosternal pain, dysphagia and hematemesis. The patient had esophageal ulcer secondary to erosion of the esophagus by the subcarinal lymph nodes. Imaging was suggestive of esophagomediastinal fistula. Esophageal ulcer biopsy showed chronic tubercular infection. Culture from the esophageal biopsy confirmed the presence of tubercular bacilli. Patient responded to anti-tubercular therapy. Spontaneous dysphagia resolution prior to starting therapy was likely due to the rupture of the lymph node into the esophagus, which was compressing it initially. Esophageal tuberculosis presenting with hematemesis and fistula is extremely uncommon.

Introduction

Mycobacterium tuberculosis can affect any organ of the body. Esophageal tuberculosis accounts for only about 0.3% of all gastrointestinal tuberculosis.[1] It usually presents with dysphagia, odynophagia, retrosternal pain, cough or constitutional symptoms like fever, anorexia and weight loss. Complications like hemorrhage from the ulcer, development of arterioesophageal fistula, esophagomediastinal fistula, esophagocutaneous fistula or tracheoesophageal fistula are extremely uncommon.[2] We report a patient who presented to us with retrosternal pain, dysphagia and hematemesis.

Case Report

A 27 years old female came with complaints of retrosternal pain since one month and difficulty in swallowing solids for 15 days. She gave history of spontaneous decrease in her symptom of dysphagia since seven days. She also had one episode of streaky hematemesis seven days back. There was no history of fever, cough, anorexia, weight loss, any lump, ingestion of any pill or corrosive substance. There was no family history of tuberculosis. Her general and systemic physical examination did not reveal any abnormality. Her hemogram showed hemoglobin of 10.9 g/dL. Erythrocyte sedimentation rate was elevated to 37 mm/hr. Rest of the blood investigations were normal. Serology for Human Immunodeficiency Virus infection was negative. Her chest X ray was normal. Her barium swallow showed a filling defect in the left lateral wall of the esophagus with mild luminal narrowing. Her esophagogastroduodenoscopy revealed a deep eccentric ulcer in the esophagus at 25 cm from the incisors (Figure 1). There was no fistulous tract seen. Biopsy from the ulcer edge showed ulcerated esophageal mucosa covered with extensive granulation tissue and dense mixed inflammation. There was presence of non-caseating epitheloid cell granuloma suggestive of tuberculosis (Figure 2). Nucleic acid amplification test (GENE EXPERT MTB RIF) of the esophageal biopsy specimen showed presence of Mycobacterium tuberculosis without rifampicin resistance. Culture from the esophageal biopsy sample grew Mycobacterium tuberculosis susceptible to all the first line medications. Patient underwent contrast enhanced CT scan of chest which showed 5×2.3×4 cm lobulated conglomerated subcarinal lymph nodal mass with areas of necrosis and few areas of air foci, extending from superior endplate of D5 to inferior plate of D7 with effaced fat planes with left atrium anteriorly and mid 1/3rd of esophagus posteriorly (Figure 3). The pulmonary parenchyma appeared normal. The patient was started on anti-tubercular treatment following which she became completely asymptomatic. Repeat endoscopy after 1 month of starting therapy revealed normal esophageal mucosa without any ulceration.

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Discussion

Gastrointestinal tubercular affection is commonly seen. However, involvement of the esophagus by tuberculosis is uncommon. It is of two types – Primary and Secondary. Primary esophageal tuberculosis is defined as isolated affection of esophagus without evidence of tuberculosis anywhere else in the body. It is exceptionally rare. Secondary esophageal tuberculosis is involvement of the esophagus by spread from the adjacent organs. The infection spreads to esophagus from the swallowed tuberculous sputum, contiguous extension from the laryngeal lesion, pharyngeal lesions or other adjacent infected structures like mediastinal or hilar lymph nodes or from the vertebrae, through the lymphatic's and hematogenous infection in case of disseminated miliary tuberculosis.[2] Mid-esophagus is the most common site of affection (as seen in our patient) due to the proximity with the mediastinal lymph nodes.[3] In an analysis of 19 cases of esophageal tuberculosis, Damtew *et al*. found that the majority of patients had direct extension from an adjacent caseous mediastinal or hilar lymph node. Most of these cases had involvement

of the upper or middle third of the esophagus.[4] Symptoms of esophageal tuberculosis include dysphagia, chest pain, cough and constitutional symptoms like fever, anorexia and weight loss. Complications include hematemesis either from ulcer or arterio-esophageal fistula. Direct presentation with

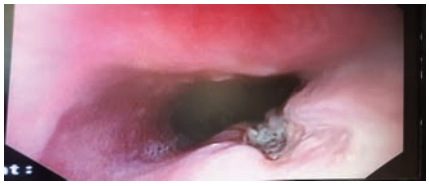

Figure 1. Esophagogastroduodenoscopy showing a deep eccentric ulcer in the esophagus at 25 cm from the incisors.

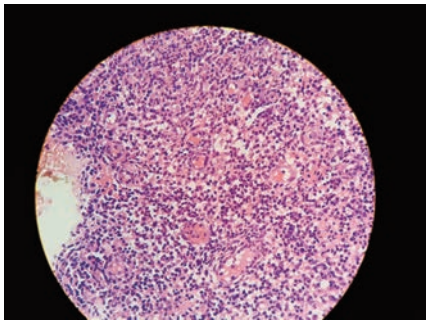

Figure 2. Histopathology of the esophageal ulcer showing presence of ulcerated mucosa with dense mixed inflammation and granulation tissue.

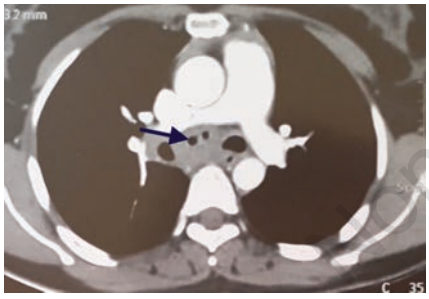

Figure 3. Computed tomography scan of chest showing subcarinal lymph nodal mass with necrosis and air foci suggestive of esophagomediastinal fistula.

complications is even rare.[2] Dysphagia may sometimes decrease spontaneously due to the development of caseous necrosis within the lymph nodal mass or by erosion of the mass into the esophagus.[5] Our patient had caseation and erosion into the esophagus. Hematemesis resulted from the esophageal ulcer, occurring as a result of erosion of tuberculous subcarinal lymph nodes. Diagnosis usually requires a high index of suspicion due to uncommon nature of the illness. Pulmonary or mediastinal lymph node involvement should be ruled out by plain radiograph of the chest and a contrast enhanced CT scan of the chest. CT scan may also reveal changes in esophagus like esophageal wall thickening and local complications. Our patient had air foci in the subcarinal lymph nodal mass, suggestive of esophagomediastinal fistula. The presence of periesophageal gas in patients with tuberculous mediastinal lymphadenitis suggests esophagomediastinal fistula.[6]

Endoscopy can directly visualize the lesion and helps in taking biopsies for histopathological examination and isolation of the organism.[3] Histology shows epithelioid granuloma with Langhans cells and central caseous necrosis. Classical granulomas are seen only in 50% of the cases, whereas acid-fast bacilli are demonstrated in less than 25%.[7] Ulcer edge biopsy can really help in cases with high index of suspicion. Mokoena et al reported a sensitivity of 22% in endoscopic biopsy. Polymerase chain reaction, Nucleic acid amplification tests like gene expert and culture further help in making the diagnosis. Treatment includes anti-tubercular chemotherapy (Rifampicin, Isoniazid, Pyrazinamide, Ethambutol) for six to nine months. She was treated with above four drugs for 6 months. Surgical intervention is warranted only in complicated cases like persistent bleeding, perforation or fistula formation. Our patient respondent to medical therapy and did not need surgical therapy. Our patient was interesting as she had a uncommon disease with three unique features in a single patient namely, spontaneous resolution of dysphagia due to development of necrosis within the nodal mass and its rupture into the esophagus, two complications in the form of esophageal ulcer presenting as hematemesis and esophagomediastinal fistula, along with a positive nucleic acid amplification test (GENE EXPERT).

Conclusions

We suggest that young patients with dysphagia and a mid esophageal lesion should be evaluated for tuberculosis. One should have a high index of suspicion if the presentation is like our patient especially in high endemic countries. Awareness of such manifestation will lead to timely and appropriate investigation, early treatment and good clinical outcome.

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