

Dysphagia as an Uncommon Presentation of Disseminated Tuberculosis: A Case Report

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Summary:

This is a case report of a 57-year old farmer who presented with a 4 months history of dysphagia and weight loss. Upper gastrointestinal endoscopy revealed submucosal swellings with overlying ulcers at mid to lower part of esophagus. Repeated histopathological evaluation of endoscopic esophageal tissue biopsies showed nonspecific findings. Endoscopic ultrasound showed a hypoechoic mass at submucosal layer with periesophageal lymphadenopathy. Endosonogram guided FNA materials depicted caseating granulomas. Patient was managed with anti-tuberculosis therapy. Follow up endosonography after therapy revealed resolution of the growth and the mediastinal lymph node enlargement and patient was symptomatically improved.

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Introduction:

Esophageal tuberculosis is a rare entity, constitutes 0.3% of gastrointestinal tuberculosis (GI TB).¹⁻² It may occur in isolation or as a part of disseminated TB. It was first described as postmortem diagnosis by Denonvilliers in 1837.³ Tuberculosis can involve any part of GIT, although the terminal ileum, caecum and the peritoneum are common sites.⁴

Primary esophageal tuberculosis is very rare, as esophagus has various protective mechanisms such as mucosal stratified squamous epithelium, covered with mucus and saliva and rapid transit of food content through the lumen.^{5,6,7} So esophageal tuberculosis usually occurs because of secondary spread from surrounding infected structures like mediastinal nodes, lung, spine or by hematogenous spread from distant site.^{1,2,5-8}

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Tuberculosis usually affects the middle third of the esophagus at the carina level.⁶ The principle presenting symptom is dysphagia but many complain of retrosternal pain, odynophagia and weight loss.^{1,6} Clinical and endoscopic findings are non-specific and can mimic carcinoma esophagus. Endoscopic ultrasound (EUS) may help in diagnosis of esophageal and mediastinal tuberculosis.^{5, 6, 8-10}

We present one case report of esophageal TB in order to uncover rare presentation, diagnosis and outcome in management of this disease at tertiary level hospital.

Case report:

A 57 years old male got admitted in our hospital with the presenting complaints of progressive dysphagia to solid foods for 4 months, anorexia and weight loss of about 18 kg for 04 months, gradually increasing ascites for the last 03 months. Patient also reported occasional low

grade fever for 03 months. He had no history of cough, hemoptysis, abdominal pain and jaundice. He was non-smoker, non-alcoholic, normotensive and non-diabetic. There was no history of contact with smear positive tuberculosis patient. His bowel and bladder habits were normal.

On general examination, patient was ill looking and cachexic. He was mildly anemic. Jaundice, cyanosis, clubbing, koilonychia, leuconychia and edema were absent. No peripheral lymphadenopathy was found. All vitals were within normal limit. On gastrointestinal systemic examination, abdomen was distended. No organomegaly was found, testes were normal. Ascites was present evidenced by shifting dullness. Examination of the other systems revealed no abnormality.

Investigations revealed hemoglobin - 9.8 gm/dl, ESR- 40 mm in 1st hour with normal WBC and platelet count. Random blood glucose, liver and renal functions were normal. Carbohydrate antigen (CA) 19.9, Carcinoembryonic antigen (CEA) and alpha feto protein (AFP) were negative. Thyroid stimulating hormone (TSH) was normal. HBsAg and Anti HCV were Negative. Tuberculin test was negative. Chest X-ray P/A view was normal. Ultrasound of whole abdomen showed moderate ascites. Echocardiography was normal (EF: 62%).

Ascitic fluid study showed exudative fluid with serum ascites albumin gradient (SAAG) - 0.46 gm/dl. Total WBC count in the fluid was 1700/mm³ with lymphocyte predominance. Adenosine deaminase activity (ADA) was 21.93 U/L and AFB was not found in the fluid.

Endoscopy showed moderate size submucosal swellings with overlying superficial ulcers at mid to lower part of esophagus (32- 38 cm) with narrowing of the lumen (Figure 1 and 2). Comment was suggestive of carcinoma esophagus. Histopathology report revealed acute & chronic inflammatory cells infiltration in mucosa without any granuloma or malignancy. Repeat endoscopy and biopsies including PCR were inconclusive. CT scan of chest showed eccentric wall thickening at mid and lower part of esophagus with mild bilateral small pleural effusion and ascites.

EUS showed submucosal swelling with mucosal ulcers at 32-38 cm in endoscopic view. In endosonographic view a hypoechoic soft tissue mass at submucosal layer of esophagus (28 mm X 17mm) invading layer of muscularis mucosa & adventitia was seen. Multiple

enlarged periesophageal lymph nodes were also seen (Figure 3, 4). Features of EUS were consistent with esophageal neoplasm or tubercular lesion.

EUS guided FNA was done from the lesion in fanning pattern. Eight slides were prepared. Cytology of FNA materials showed granuloma (Figure 5) with background caseation necrosis (Figure 6). Cytological finding of FNA was chronic granulomatous inflammation suggestive of tuberculosis.

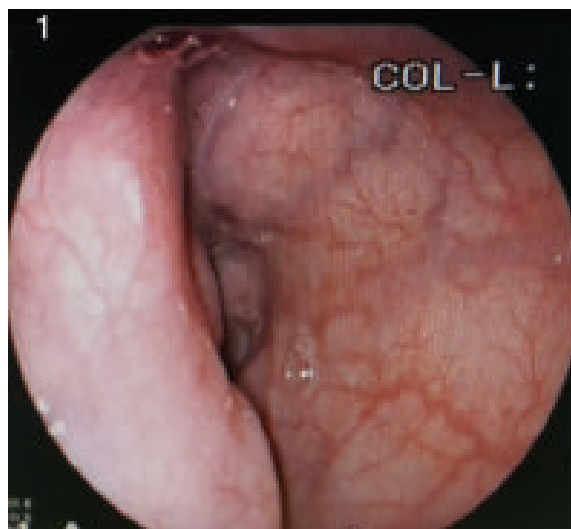


Fig-1: Endoscopic view of esophageal submucosal swelling with partial narrowing of lumen.



Fig-2: Endoscopic image of submucosal swelling in esophageal wall with ulcer.

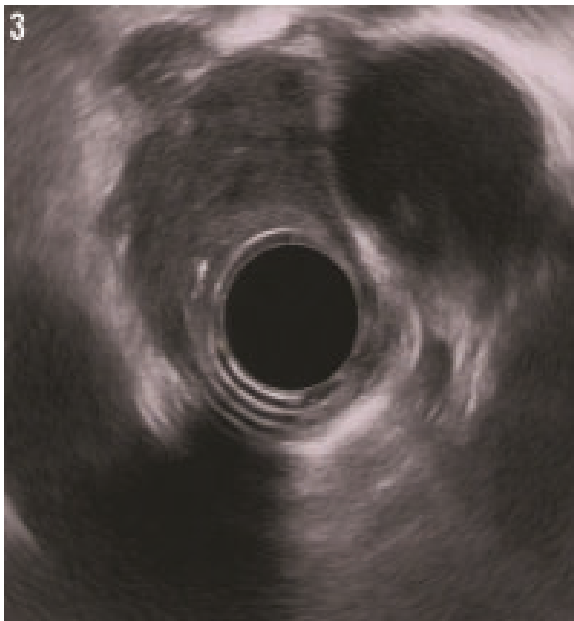


Fig.-3: Endosonographic Image showing esophageal wall thickening with periesophageal enlarged lymph nodes.

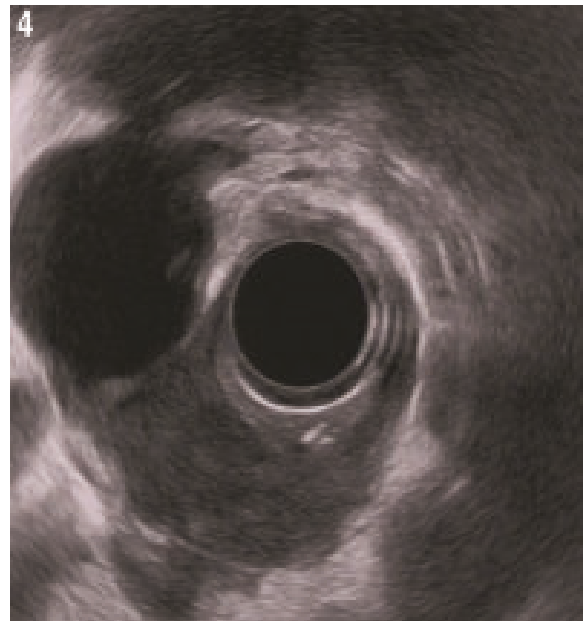


Fig.-4: Endosonographic Image showing esophageal wall thickening with periesophageal enlarged lymph nodes.

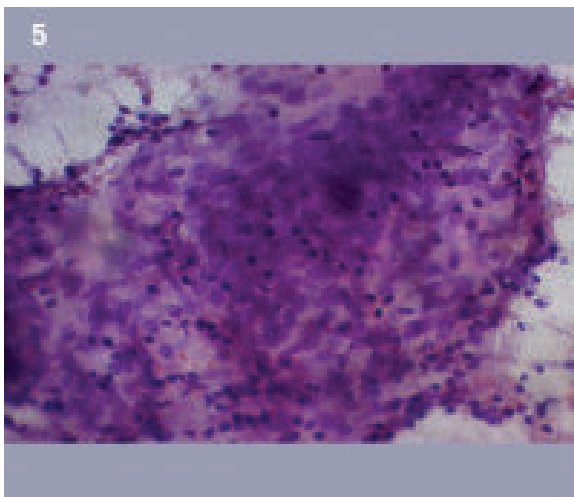


Fig.-5: Cytology of FNA materials showing granuloma, groups of epithelioid histiocytes.

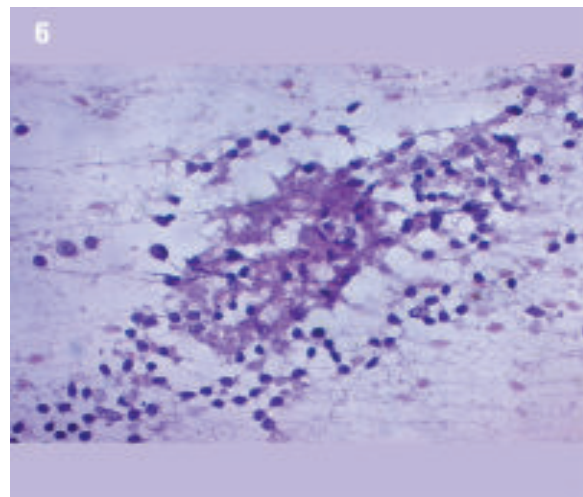


Fig.-6: Cytology of FNA materials showing lymphocytes with background caseation necrosis.

Antitubercular therapy was started accordingly. Patient was followed up; he was improved symptomatically with 12 kg weight gain in 02 months. Dysphagia was totally improved. In subsequent follow up after completion of anti-tubercular therapy, patient was clinically well. Follow up ultrasonogram showed no ascites.

Discussion

Tuberculosis (TB) results in death of 3 million people globally in each year. An estimated 1 billion people will be infected by the year 2020 and 35 million will die from tuberculosis, if control is not strengthened.¹¹

Esophageal involvement of disseminated TB is rare, hence high degree of clinical suspicion is needed. Esophageal TB is considered primary when there is no other detectable tubercular site and secondary esophageal TB due to spread from adjacent structure.^{2,7}

Symptoms are related to the extent of infection but patient usually present with dysphagia (>90% of cases).² It is usually because of esophageal ulcers but may also be due to stricture, extrinsic compression and diverticula. Occasionally patients present with esophagobronchial fistula producing symptoms like cough on swallowing.^{2,3,7} Other symptoms include odynophagia, retrosternal pain and constitutional symptoms like evening rise of temperature, weight loss and anorexia etc. A hypertrophic growth may mimic esophageal cancer.¹²⁻¹³ In our case, dysphagia and weight loss were the presenting symptoms.

A plain chest X-ray should be done for patients with dysphagia. If it shows any abnormality particularly apical lesion suggestive of present or past TB or mediastinal nodes, the possibility of esophageal TB should be strongly suspected. A contrast esophagogram does not demonstrate extra luminal lesion, for which CT scan of thorax and/or EUS is required to document secondary nature of diagnosis.⁷ CT image is also helpful to detect complication of esophageal TB such as perforation, pneumomediastinum and Pott's disease etc. CT scan of chest of this patient showed eccentric wall thickening at mid and lower part of esophagus with mild bilateral small pleural effusion and ascites.

An endoscopy and biopsy from the lesion is the key investigation in evaluation of dysphagia. It can involve any segment of esophagus but middle third just proximal to carina is commonly involved due to increased number of lymph node around the carina.⁷ In this case, esophageal involvement was at the mid to lower part of esophagus. The most common macroscopic finding on endoscopy is an esophageal ulcer. However, hypertrophic growth may also be present.^{14,15} A differential diagnosis of esophageal TB includes esophageal carcinoma, Crohn's disease, moniliasis, actinomycosis and esophageal injury secondary to ingestion of caustic materials.⁸ In our case, the endoscopic finding was moderate size submucosal swellings with overlying superficial ulcers at mid to lower part of esophagus (at 32- 38 cm) causing luminal narrowing.

Endoscopic biopsies are useful for the diagnosis of esophageal TB which reveals classic caseation granulomas in endoscopy samples in 25% to 60.8% of cases.¹⁶⁻¹⁸ In the present case, repeated endoscopy and biopsy were inconclusive and the diagnosis of the esophageal tuberculosis was delayed. The diagnostic difficulties as reported herein are in line with previous case reports or case series of patients with gastrointestinal tuberculosis.¹⁶⁻¹⁸

In our patient, EUS guided FNAC (eight slides) from the lesion and surrounding lymph nodes were needed to finally detect tuberculous granulomas. This is related to the fact that the density of tuberculous granulomas in the infected organ tissue may be low. Furthermore, tuberculous granulomas are located in the submucosal layer that frequently is not adequately represented in endoscopic tissue biopsies, highlighting the need of multiple and deep tissue samples in patients with suspected esophageal or intestinal tuberculosis. The present study demonstrated that esophageal TB was caused by secondary spread from surrounding lymph nodes.

The treatment of esophageal TB is anti-tubercular drugs. A 6 to 9 months course of anti TB chemotherapy is sufficient to treat the patient with four first line drugs regimen (Rifampicin, INH, Ethambutol and Pyrazinamide) for initial 2 months and then continuation phase with Rifampicin, INH for further 4 to 7 months. We initiated a standard treatment in our patient, and advised to continue the treatment over a period of nine months. In follow-up endoscopy and endoscopic ultrasound two months after therapy initiation, there was complete remission of the tuberculous esophageal lesion and the enlarged mediastinal lymph node, and the patient was symptomatically improved. Ascites disappeared after completion of therapy.

Conclusion

Dysphagia is one of the presenting features of esophageal tuberculosis. This condition should be considered as a differential diagnosis in populations of high prevalence countries and who are at risk such as immunocompromised. The diagnostic confirmation of the disease may be hampered and delayed by the low sensitivity of the available diagnostic methods. Early diagnosis and therapy are critical for the disease outcome. The undue delay in the diagnosis can lead to complications, which might require surgical intervention.

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