Dysphagia due to tuberculosis

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Abstract

Objective: Dysphagia due to tuberculosis is rare in both the developing countries with high prevalence rates and the western population following the recent upsurge linked to the AIDS and immigration. Aim: To study tuberculosis as an aetiological factor in the causation of dysphagia and to evaluate the outcome of anti-tubercular treatment and surgical results in these patients. Methods: Retrospective review of experience with 14 cases of dysphagia due to tuberculosis encountered between 1996 and 2003. Results: The duration of symptoms ranged between 3 and 18 months. All of them underwent oesophagogastroscopy, barium swallow, fiberoptic bronchoscopy and CT scan of the chest. The aetiology was subcarinal node enlargement in seven, tracheo-oesophageal fistula in four, oesophageal ulcer in two and cervical node suppuration in one. Tuberculous involvement was confirmed by pathological examination in all patients. All of them received anti-tuberculous therapy. Seven patients required surgery, transthoracic repair of tracheo-oesophageal fistula in four patients, one patient required subcarinal node excision and two needed abscess drainage. There were no mortalities and there was complete relief of dysphagia in all of them. Conclusions: Tuberculosis as a causative factor for dysphagia should be considered in regions with high incidences of tuberculosis and in immunocompromised patients. Treatment with anti-tuberculous therapy is effective. Surgery is required only for complications of tuberculosis.

Keywords: Tuberculosis; Dysphagia; Oesophagus; Tracheo-oesophageal fistula

1. Introduction

Dysphagia is difficulty or discomfort in moving food from the mouth to the stomach. It refers to difficulty in eating as a result of disruption in the swallowing process at various levels of swallowing. Dysphagia can range from mild forms to those causing serious complications like aspiration pneumonia, malnutrition, dehydration, weight loss and airway obstruction.

Tuberculosis can cause dysphagia by many mechanisms; it can be intrinsic oesophageal tuberculosis or it can be extraneous compression. Even in countries with a high prevalence, tuberculous involvement of the oesophagus is rare [1]. Likewise tuberculous adenitis rarely causes dysphagia.

We share our experience with dysphagia due to tuberculous aetiology in this article.

2. Aim

This study was aimed at reviewing our institutional experience with dysphagia due to tuberculosis over a period of 7 years between 1996 and 2003.

3. Methods

Fourteen consequent patients were identified between 1996 and 2003 who presented with dysphagia and had a histological confirmation of tuberculosis. The case notes, investigations and treatment and outcomes were reviewed.

4. Findings

4.1. Demographics

The age distribution was between 27 and 79, with 9 men (64%). The duration of symptoms ranged between 3 and 18 months. Comorbidities included diabetes in three, chronic...
obstructive airway disease (COAD) in one patient and two patients had previous medical therapy for tuberculosis.

4.2. Presentation

The aetiology of dysphagia was mediastinal and subcarinal lymphadenopathy in seven, tracheo-oesophageal fistula in four, oesophageal ulcer in two and cervical node suppuration in one patient. Two patients had already received medical therapy for pulmonary tuberculosis and one patient who had undergone thoracotomy and drainage for suppurating mediastinal adenopathy now presented with tracheo-oesophageal fistula.

All of them had presented themselves with progressive dysphagia for solids and the patients with tracheo-oesophageal fistula presented with associated aspiration and chest infections. One patient had an oesophageal stent placed in an attempt to close the tracheo oesophageal fistula by the referring unit which was not successful. They also had associated weight loss due to dysphagia and the tuberculous disease process itself.

4.3. Investigations

All the patients underwent routine haematological and biochemical investigations including liver function tests. Investigative workup included oesophagastroduodenoscopy, contrast swallow, fiberoptic bronchoscopy and CT scan of the chest.

Oesophagastroduodenoscopy demonstrated extraneous compression in ten patients and ulcers in two patients. The Chest radiograph demonstrated largely enlarged neck nodes in one patient and loss of the paratracheal stripe in four patients. The CT scan revealed oesophageal and tracheal shift in patients with significantly enlarged cervical and mediastinal nodes (Fig. 1). The Barium swallow demonstrated the extrinsic compression in ten patients (Fig. 2) and the tracheo-oesophageal fistula in four patients (Fig. 3). The bronchoscopy demonstrated tracheal splaying due to enlarged subcarinal nodes. Histological assessment of biopsies produced proof of epithelioid cell granulomas with marginally polygonal Langhans-type giant cells confirming tuberculosis.

4.4. Management

All patients received a drug regimen comprising of Isoniazid, Rifampicin and Ethambutal and Pyrazinamide for 2 months followed by Isoniazid and Rifampicin for 4 months except the two patients who had received the first line drugs earlier were given para amino salicyclic acid as well.

Seven patients required surgical procedures, four patients underwent transthoracic repair of tracheo-oesophageal fistula of which three had intercostal muscle flap and one had pleural flap for reinforcement of the repair. Two required drainage of suppurating abscess and one had excision of subcarinal lymph node (Table 1).

4.5. Surgical management

One patient with suppurating neck abscess was drained under general anaesthesia through a cervical incision to result in a nondependent drainage. All the other patients underwent a right thoracotomy for the surgical treatment. They were performed on single lung ventilation with a double...
lumen endotracheal tube. The patients were placed in left lateral position and the posterolateral thoracotomy was performed through the fourth intercostal space. The lungs were released from the chest wall by extrapleural approach if there was presence of extensive adhesions especially in the patient who underwent the reoperation. In the four patients with tracheo-oesophageal fistula, the oesophagus was dissected and a tape was placed around it and the trachea was then dissected to identify the fistula. The fistulous tract was excised and the tracheal site was repaired with 2 '0' vicryl and the oesophageal side was repaired with 3 '0' vicryl in two layers. We interposed a pedicled intercostal muscle flap in three patients and a thick flap of pleura in one patient between the two suture lines. The tracheal repair was checked for air leak.

The patient with the caseaous mediastinal node had the abscess opened and drained. The abscess cavity was excised with a sliver adherent to the trachea. The patient with a large subcarinal lymph node mass had the mass excised completely; fortunately the mass was not infiltrating any of the adjacent organs including the oesophagus and was resected without difficulty. All the patients were closed with an apical and basal chest drains. All the patients were extubated on table and were nursed in the high dependency unit.

4.6. Results

There were no mortalities in this series. One patient had air leak till the sixth postoperative day. One patient had a persistent sinus in the neck, which settled down after 2 weeks. All the patients who underwent surgery had relief of their dysphagia immediately after surgery.

All patients had complete remission of their dysphagia following the course of treatment and were followed up between 6 months and 2 years and discharged back to the care of referring clinician.

5. Discussion

The global burden of tuberculosis remains enormous, mainly because of poor control and coexisting nature of Mycobacterium tuberculosis and HIV. In 1997, new cases of tuberculosis totalled an estimated 7.96 million (range, 6.3—11.1 million), with an estimated 1.87 million (1.4—2.8 million) people dying of tuberculosis [2].

Tuberculosis though a systemic disease rarely causes dysphagia. Tuberculous dysphagia can be due to intrinsic oesophageal ulcers, tracheo-oesophageal fistulae and extrinsic compression due to mediastinal or cervical lymph nodes.

Tuberculosis has been known to involve the oesophagus, either as a primary infection or as a secondary manifestation of reactivated disease. The exposure of the oesophagus to the organism is limited by the rapid clearance of infected sputum by means of coordinated peristalsis, combined with upright posture and an intact lower oesophageal sphincter [3]. Oesophageal tuberculosis is almost always associated with mediastinal lymphadenopathy with or without a tracheo-oesophageal fistula [4]. The two most common differential diagnoses are carcinoma of the oesophagus and Crohn’s disease of the oesophagus [5,6].

The primary complaint is chronic dysphagia in almost 75% of the patients and other symptoms included weight loss, haematemesis and productive cough suggestive of tracheo-oesophageal fistula formation [7].

Most of the reported cases of oesophageal tuberculosis are secondary to tuberculosis elsewhere in the body, most commonly pulmonary tuberculosis [8]. Oesophageal tuberculosis can present as ulcers, strictures or fistulae [9]. Endoscopic features of oesophageal tuberculosis are deep and large oesophageal ulcers, tracheoesophageal fistula, and chronic nonhealing ulcer [10]. Histological assessment of biopsies produce proof of epithelioid cell granulomas with marginally polygonal giant cells of Langhans-type and the presence of acid-fast bacilli confirms the diagnosis. In difficult scenarios, oesophageal tuberculosis has been diagnosed by identification of mycobacteria in paraffin-embedded oesophageal biopsy specimens by polymerase chain reaction [11]. Mediastinal tuberculous lymphadenitis is rare in adults, and it is even rare to present as dysphagia. This may lead to oesophageal ulceration, mucosal or submucosal mass with ulceration, fistula or sinus formation, extrinsic compression, or displacement of the oesophagus [12—16].

Acquired, nonmalignant tracheo-oesophageal fistulae usually result from erosion of the tracheal and oesophageal walls by endotracheal or tracheostomy tube cuffs [17]. However, tuberculosis mediastinal lymphadenitis can also lead to tracheo-oesophageal fistula. These patients usually present with dysphagia and recurrent aspiration and chest infections [18].

Bronchoscopy, possibly with the application of methylene blue dye into the oesophagus, or oesophagography with a water-soluble agent will exactly define even a small fistula. Oesophagoscopy may reveal the fistula, however, the fistula can be overlooked in the mucosal folds of the oesophagus [19].

Tracheo-oesophageal fistulae caused by Mycobacterium tuberculosis usually require a combination of both surgical treatment and medical treatment with anti-tuberculosis drugs.
It can be managed by simple division and closure of the fistula or tracheal resection and reconstruction. Primary reconstruction definitely corrects the fistula and removes concurrent tracheal disease and is preferred to simple division and closure of the fistula [17,20]. The oesophageal defect is closed in two layers and a viable strap muscle interposed between the two suture lines [20]. However, there are case reports with complete healing of the fistula with medical therapy alone [4,21].

Medical therapy is the mainstay of management of these patients. The standard drug regimen incorporating Isoniazid, Rifampicin, Pyrazinamide and Ethambutal usually lead to complete cure. Maintenance of a good nutritional state is imperative especially considering the altered liver metabolism and vitamin deficiencies associated with it.

6. Conclusion

Tuberculosis as a causative factor for dysphagia should be considered in developing countries with high incidences of tuberculosis and in immunocompromised hosts. Treatment with standard anti-tubercular therapy is effective. Surgery is required only for complications of tuberculosis.

References