Dysphagia as a Presenting Complaint in Carcinoma Lung: A Case Report

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Abstract

Carcinoma lung classically present with symptoms of cough, hemoptysis, chest pain and weight loss. Lung cancer presenting with sole manifestation of difficulty swallowing as the initial presenting complaint is very uncommon. Literature reports 1-2 % patients with lung cancer had dysphagia at presentation. We report a case of 45 years male, who is initially evaluated for the complaints of difficulty swallowing and eventually diagnosed to have carcinoma lung with mediastinal lyphadenopathy as the cause of his symptoms.

Keywords: Lymphadenopathy, Extrinsic Compression, Carcinoma.

Introduction

Cough, hemoptysis, chest pain and weight loss, are the classic symptoms of carcinoma (Ca) lung. Dysphagia is rarely mentioned as the presenting symptoms in this symptom complex.[1] Le Roux [2] reported that 1-2% of lung cancer patient had dysphagia at presentation, and when full clinical course of the disease is considered the percentage experiencing dysphagia rise to 6-7%.[3,4] We report this case to highlight that carcinoma lung can present with dysphagia as the sole presenting complaint, in the absence of classical symptoms. We report a case of 45 years old male that presented with progressive dysphagia of one month duration without any classical chest symptoms and eventually diagnosed to have Ca lung with mediastinal lymphadenopathy (LAP) as the cause of his symptoms.

Case Report

A 45 year old male presented to gastroenterology outpatient department with complaints of difficulty in swallowing for 4 weeks. Dysphagia is more for the solid food than liquid. There was no history of heartburn, vomiting and dyspepsia. There was no history of cough, shortness of breath, chest pain and regurgitation. He was a smoker with smoking index of 500 and occasionally consumes alcohol once in a month or two during social occasions. His general physical and systemic examination was within normal limits.

His esophagogastroduodenoscopy (EGD) revealed multiple extrinsic impressions on the esophageal mucosa starting at 28 cm from incisors with luminal compromise with food bolus impaction [Fig 1A and 1B]. Food bolus removed with foreign body forceps. Adult scope (Olympus EXERA II GIF-H180 Gastroscope

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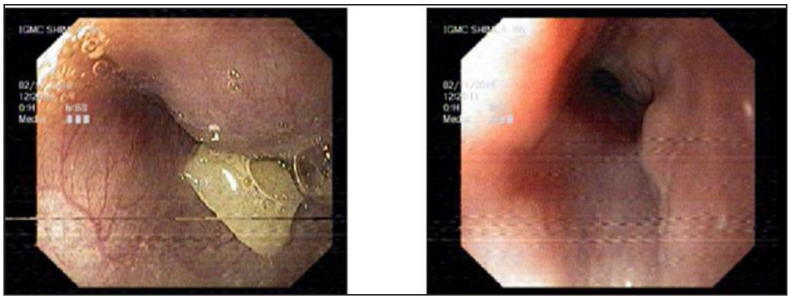

Fig 1A – Showing extrinsic impression over esophagus causing luminal collapse with normal overlying mucosa with food bolus impaction

Fig 1B – Showing extrinsic impression with narrowed esophageal lumen after removal of food bolus

with outer diameter of 9.8 mm) could not be negotiated beyond 30 cm from incisor. Flexible ultrathin scope (Olympus TJF type 180 with outer diameter of 5.8 mm) was then used to negotiate across the narrowing. Final impression of esophageal luminal narrowing (between 28 to 34 cm from incisor) with normal overlying mucosa, due to multiple extrinsic compressions (EC) was given and chest X-ray and CECT – thorax was advised to rule out mediastinal pathology.

Chest X–ray [Fig 2A] revealed non- homogenous ill-defined opacity without air bronchogram in right lower lobe. CECT – thorax [Fig 2B] revealed a lobulated soft tissue mass lesion in right lung lower lobe with mediastinum lymphadenopathy (LAP) compressing esophagus suggestive of Ca lung with mediastinal LAP.

Chest consultation was taken and bronchoscopy revealed fleshy necrotic growth in right intermediate bronchus totally occluding its lumen. Histopathological examination of the biopsy specimen from the growth revealed well differentiated squamous cell carcinoma lung. Patient attached to oncology department for further management.

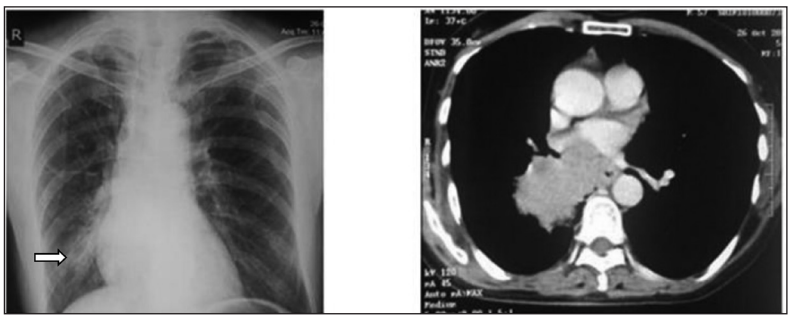

Fig 2a – Chest X-ray – showing inhomogeneous ill-defined opacity right lower zone

Fig 2b – CECT Chest – Mediastinal Window image – showing soft tissue lobular mass in right lower lobe with mediastinal lyphadenopathy with effected fat planes with esophagus

From oncology department he was advised for esophageal stent placement for relief of dysphagia. Repeat EGD was performed [Fig 3A] and 11 cm Fully Covered – Self Expandable Metallic Stent (FC-SEMS) placed over guide wire under endoscopic guidance, across area of luminal narrowing with proximal end at 26 cm from incisor, for palliation of dysphagia.

After stent placement patient was advised to follow in oncology department.

Discussion

Dysphagia is a known complication of carcinoma lung, but lung cancer presenting with dysphagia as a sole initial manifestation is uncommon. There are several different possible mechanisms that can lead to dysphagia in carcinoma lung. According to Stankey *et al* [5] dysphagia associated with lung cancer could be accounted for in all cases by three possible mechanisms—first and most commonly, extrinsic compression of the oesophagus within the mediastinum; second, compression of the pharynx and upper oesophagus by lymph-node deposits within the neck; and third and most infrequently, oesophageal stenosis secondary to antecedent mediastinal radiotherapy. To these three causes Makker *et al*, [6] added secondary achalasia produced by interference with oesophageal motility.

The most common cause of lung cancer associated dysphagia is mediastinal disease. In lung cancer, direct oesphageal invasion may occur with lesions in the left main bronchus but perioesophageal or subcarinal lymph node deposits are more often responsible.[5] Anatomically, the subcarinal lymph nodes, limited in their potential for lateral growth and therefore tending to expand posteriorly, are the group most likely to intrude on the adjacent oesophagus, as is seen in our case.[7]

Kaasa *et al.* reported that 64% of non-small cell lung cancer patients treated with radiotherapy experienced dysphagia compared to 8% of the chemotherapy patients 6 weeks after the start of treatment.[8] Maguire *et al*.[4] similarly found that 75% of their patients receiving high-dose conformal radiotherapy for lung cancer (64.2–85.6 Grey) experienced dysphagia. However, the degree of dysphagia was very variable and only 11% had grade 3 acute oesophageal toxicity (requiring parenteral fluid support). Factors that seemed to

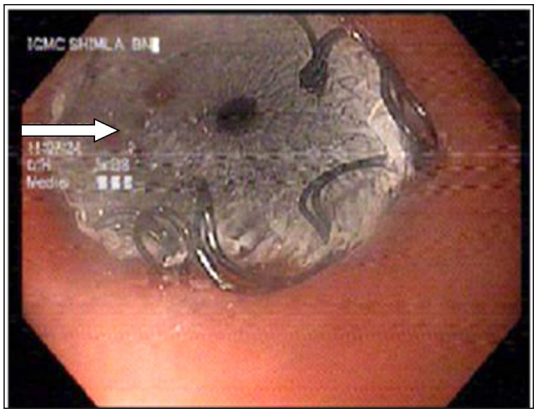

Fig 3a – EGD image showing proximal end of the FC-SEMS

predispose to severe acute toxicity were the existence of dysphagia before treatment and the use of hyperfractionated treatment regimens. Chronic oesophageal toxicity was much rarer than acute toxicity with grade 3 toxicity (requiring dilatation) developing in only 3% of patients. The incidence of severe late toxicity seems to be increased when chemotherapy and radiotherapy [9] are used together.

Patients with secondary achalasia tend to be older (>50 years), to have shorter duration of dysphagia (<1 year) and to have greater weight loss when compared to those with idiopathic achalasia.[10] In most cases secondary achalasia has been reported due to oesophageal obstruction caused by tumour infiltration or encasement of the oesophagogastric junction. Other suggested explanations include physical disruption of the myenteric plexus by the tumour and deposition of eosinophilic cationic protein associated with the tumour causing damage to the myenteric plexus.[10] Intestinal motility disorders in small cell lung cancer have been reported due to paraneoplastic enteric neuropathy linked to the presence of antineuronal antibody (also known as anti-hu antibody).[10]

Other less common causes of dysphagia in carcinoma lung may include, brainstem and gastrointestinal tract metastasis, associated systemic disorder like dermatomyositis, second primary malignancy like coincident carcinoma esophagus and oropharyngeal and esophageal infections like candidiasis and viral and bacterial stomatitis and esophagitis.[11]

Conclusion

Carcinoma lung can present with sole manifestation of dysphagia as the presenting complaint, in the absence of classical symptoms like cough, hemoptysis, chest pain and weight loss. The most common cause of dyphagia in Ca lung is extrinsic compression of the esophagus due to mediastinal disease.

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