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MEDIASTINAL SARCOIDOSIS MASS REVEALED BY PROGRESSIVE DYSPHAGIA – CASE REPORT

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ABSTRACT: Dysphagia is considered a digestive symptom caused not only by intrinsic gastrointestinal diseases, but also by extradigestive conditions. The mechanism of progressive dysphagia encountered in extraluminal esophageal diseases is related to extrinsic esophageal compression. Besides intraesophageal pathology responsible for dysphagia development, there have been described several causes, less frequently encountered in clinical practice: mediastinal tumors or masses of different etiologies, pericardial effusion, surgical changes, vascular compression. We report a case of sarcoidosis expressed by a giant posterior mediastinal lymphadenopathy that presented in a gastroenterology clinic for progressive dysphagia.
KEYWORDS: dysphagia – mediastinal, mass- sarcoidosis, mediastinal lymphadenopathy

INTRODUCTION:

Generally, the most frequent causes of dysphagia are represented by intrinsic esophageal conditions as motor disorders (achalasia, esophageal dismotility) and obstructive lesions (tumors, strictures, webs, rings) (Yetim et al., 2012; Hemender, 2008). The initial work-up of dysphagia should start by differentiating between oropharyngeal and esophageal causes. Oropharyngeal swallowing difficulties are not considered in our presentation and refer to neurological deficits and neuromuscular disorders (Lemos et al., 2008). Once established the esophagus as the primary site for dysphagia development, the attention should be focused on clarifying the certain cause, either intrinsec or extrinsic lesion (Lind, 2003). Among intrinsec causes of esophageal dysphasia, benign and malignant conditions should be mentioned (Adeyemie et al., 2008). Esophageal webs and rings as well as peptic strictures which state for 60-70% of cases, are the most common benign lesions, while adenocarcinoma and squamous cell cancer represent neoplasic esophageal lesions (Adeyemie et al., 2008; singhal et. Al., 2013). Extrinsic causes of dysphagia are represented by mediastinal masses and vascular compression related to vascular abnormalities (Adeyemie et al., 2008). Mediastinal causes of dysphagia encomprise a variety of conditions divided according to their localization into the mediastinum compartment: anterior, middle and posterior mediastinum (Siew et al.,

2012). The most frequent mass of anterior mediastinum was found to be thymoma which accounts for 50% of anterior mediastinal masses and 20 % of all mediastinal tumors (Riedel et. Al., 2006). Middle mediastinal masses include foregut duplication cysts, tracheal lesions, lymphadenopathies, vascular lesions, pericardial cyst (Siew et al., 2012). The structures localized in the posterior mediastinum consist of lymphadenopathies of several etiologies, neurogenic tumors or vascular abnormalities as descending aortic aneurysm (Siew et al., 2012; Saenz et al., 1993). Mediastinal lymph-node enlargement results from different conditions: metastatic cancer, lymphoma, infectious disease (tuberculosis), systemic processes (sarcoidosis) (Guideline, 2011). The diagnostic tools for identifying a mediastinal adenopathy are represented by imaging techniques (thoracic CT scan), endoscopic ultrasound or even upper digestive endoscopy which reveals esophageal compression by an extrinsic mass (Guideline, 2011). EUS posses a key role in evaluating a posterior mediastinal adenopathy, both for diagnostic purposes and also for guiding transesophageal targeted biopsies through FNA technique (Guideline, 2011). Regarding sarcoidosis as a possible cause of posterior mediastinal lymphadenopahy mass, it should be mentioned that we deal with an inflammatory multisystemic disease without a clear etiology that is characterized histological by noncaseating granulomas (Iannuzzi et al., 2007). Sarcoidosis presents either an active disease with pulmonary symptoms such shortness of breath, coughing, fatigue, fever accompanied by bilateral hilar adenopathy, either an indolent condition discovered incidentally by histopathological examination (Iannuzzi et al., 2007; Giovinale et al., 2009).

CASE REPORT

A 49-years old female patient was admitted to our clinic for progressive dysphagia that started a few months ago, accompanied by vomiting and significant weight loss (10 kilos in three months). Initially, only solid food produced difficulty to swallowing, but subsequently dysphagia became total, including liquids. Her past medical history as well as her family history were unremarkable. She was not a smoker, not a drinker and denied illicit drugs use. The physical examination showed a good-looking patient, with a low BMI (17,5 kg/m^2), without other abdominal, respiratory or cardiac pathological signs. Routine laboratory studies were within normal range. Considering that dysphagia was the main symptom, the diagnostic work-up started with upper digestive endoscopy. Esophagus assessment revealed a stenosis through extrinsic compression, localized at 35 cm from dental arcade; the endoscope passed easily the stenotic area and permitted to complete the investigation. Barium swallow procedure evidenced incomplete medium esophageal stenosis with no malignant signs (Fig 1).

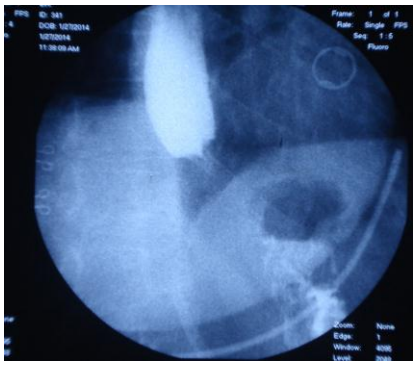

Fig 1. Barium X –ray – esophageal stenosis

Abdominal ultrasound did not show abnormalities, nor did the X-ray of the chest. Under such circumstances, the next step for establishing the diagnosis was thoracic CT scan. The scan evidenced a tumoral mass, with well-defined rims, located in the posterior mediastinum, which extends above the level of carina; the tumor causes extrinsic compression of the medium esophagus on 30 mm distance. In addition, no pulmonary tumors or hilar adenopathies were seen (Fig 2a,2b).

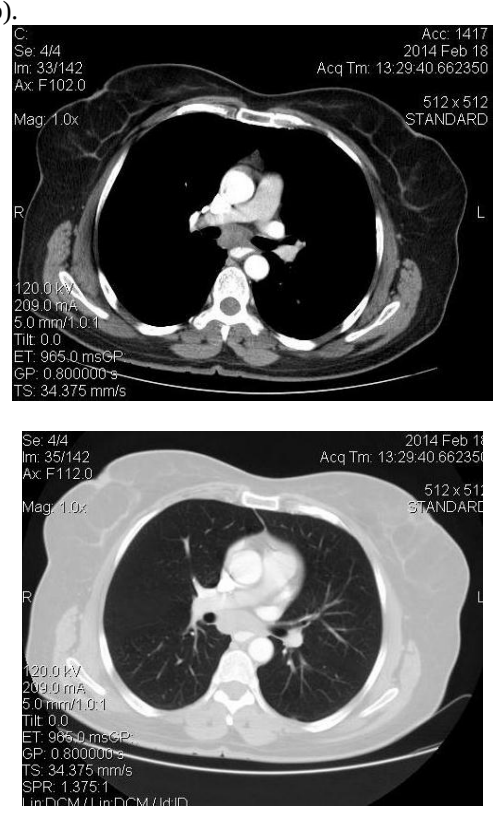

Fig 2a,2b. CT scan – posterior mediastinal tumor

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Endoscopic ultrasound was then performed and identified a solid mass with hypoechoic foci, about 35 mm diameter, hypovascular at Doppler examination; elastography was doubtful in differentiating a benign aspect from a malignant one (Fig 3).

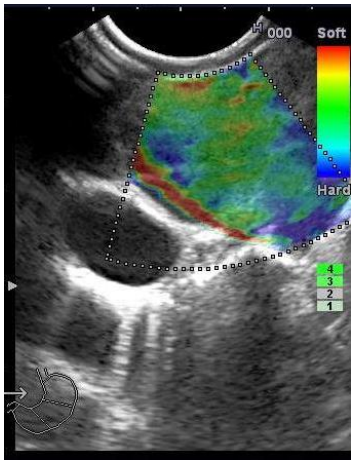

Fig 3. EUS – solid mass with hypoecoic foci and elastographic appearance

A FNA was performed using the linear EUS endoscope with 22 Gauch needle, but the cytology smear did not show malignant features. The patient was refered to thoracic surgery department for surgical resection of the mediastinal tumor (Fig 4).

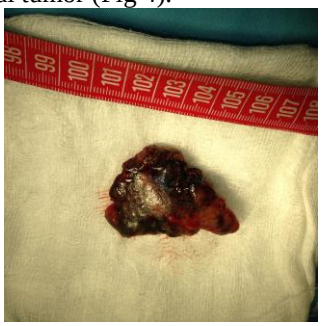

Fig 4. Postoperatory aspect of the tumor

The histopathological examination of the resected specimen revealed lymph-nodes structures with epithelioid granulomas suggestive for the diagnosis of sarcoidosis (Fig 5, 6).

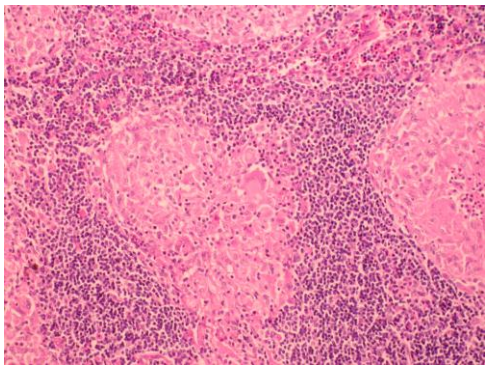

Fig 5. Granulomatous node without central necrosis, including epithelioid and Langhans cells; HE X10

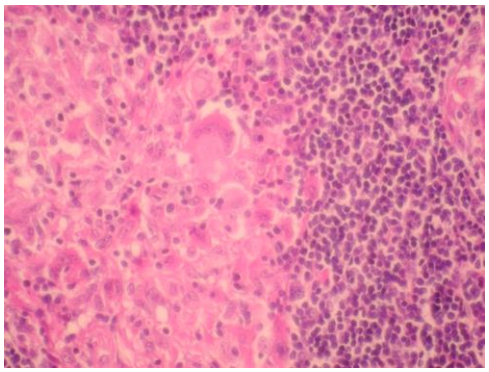

Fig 6. Multinucleated Langhans cell, HEX40

CONCLUSIONS

Dysphagia is often a challenging and confusing symptom because initially it directs the medical attention towards a digestive pathology. Dysphagia can be produced either by esophageal diseases or by extrinsec conditions that cause luminal compression (Yetim et al., 2012; Hemender, 2008; Lind, 2003). The etiology of "extraesophageal dysphagia" is complex and includes both benign and malignant disorders, sometimes indolent at the time of presention, as happened in our patient case. The patient's good clinical condition along with the absence of biological abnormalities stands for a benign disease. As dysphagia was the predominant symptom, upper digestive endoscopy was imposed as first diagnostic procedure and clarified at this point the etiology as being extraesophageal, thus limiting the spectrum of the diseases. The CT scan played in this case a key role in the diagnosis work-up, but insufficient for establishing the etiology of the mediastinal mass. Only the histological examination of the resected mass evidenced clearly its nature, the diagnosis being consistent with sarcoidosis. Sarcoidosis is a multisystemic disease of unknown etiology, that associates in more than 90% of cases pulmonary involvement either symptomatic or silent expressed by

X-ray changes, bilateral hilar adenopathies being a characteristic of the disease (Iannuzzi et al., 2007). In addition, there have been described extrapulmonary manifestations, such as skin and ocular lesions, that both lack in our patient (Iannuzzi et al., 2007; Giovinale et al., 2009). As the diagnosis was certain through histologic evidence, supplementary tests specific for sarcoidosis as angiotensin converting enzyme were unnecessary. In conclusion, we dealed with an asymptomatic patient as respects the clinical manifestations of sarcoidosis, condition that was finally revealed by a common digestive symptom, dysphagia.

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