Large osteophytes causing dysphagia: an interesting case

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Abstract

As otolaryngologists, we are the first to be consulted for dysphagia. One must consider both extrinsic and intrinsic etiologies in the differential diagnosis. We report a patient with dysphagia due to large osteophyte at level of cricoid and pressing on the esophagus. Patient was diagnosed to be suffering from Diffuse Idiopathic skeletal Hyperostosis and was managed conservatively.

INTRODUCTION

Diffuse Idiopathic skeletal Hyperostosis (DISH) is the skeletal disease of unknown etiology, characterized by flowing ossification along the anterior and lateral aspects of spinal column. We present an interesting case presenting with dysphagia and on investigation found to be suffering from DISH. An astute otolaryngologist needs to be aware of the spine pathology in cases of dysphagia. Here we briefly describe the presenting features, radiographic findings and management options.

CASE REPORT

A 60 year old male presented to our out patient department with complaint of difficulty in swallowing for one month. The difficulty was mainly limited to solids, non-progressive and got relieved with fluids. Patient was non-smoker and teetotaler. There was no history of similar complaints in the past and history of regurgitation was absent. On examination oral cavity and oropharynx were normal but the indirect laryngoscopy revealed midline bulge on the posterior wall of pharynx just above the level of arytenoids. X-ray soft tissue neck lateral view was done which revealed osteophytes at three different levels in

Cervical spine namely C2-3, C3-4 and C5-6 as depicted below (Figure 1).

Figure 1

Figure 1 X-ray cervical spine lateral view showing straightening of spine and large anterior osteophytes at multiple levels



X-ray lumbar-sacral spine revealed calcification of anterior spinal ligament at places as seen in the picture (Figure 2).

Figure 2

Figure 2 X-ray lumbosacral spine lateral view showing calcification of anterior spinal ligaments.



Patient was diagnosed to be suffering from diffuse idiopathic skeletal hyperostosis and managed conservatively with dietary modification.

DISCUSSION

Degenerative changes in the spine can produce osteophytic spurs on the anterior surface of the cervical vertebrae. Dysphagia can be caused by hypertrophic anterior cervical osteophytes¹, and about 100 such cases have been reported in the literature². Large bridging anterior osteophytes of the cervical spine are usually the result of diffuse idiopathic skeletal hyperostosis (DISH) and they cause compression of the oropharyngeal swallowing structure. Also known as Forestier disease, senile ankylosing hyperostosis and ankylosing vertebral hyperostosis, it is a common skeletal disease of unknown etiology. It is characterized by flowing ossification along the anterior and lateral aspects of the spinal column.

DISH is more frequent in men than in women. It affects up to 10% of patients older than 65 years of age³, and back stiffness in the thoracic region is the usual presenting symptom. These patients can develop osteophytes, which can lead to extrinsic compression on local tissues. Depending on their location, this can lead to otolaryngologic manifestations such as dysphagia, hoarseness and stridor. The dysphagia may be due to mechanical blockage of the esophagus due to large size or even due to small osteophyte located opposite to the fixed points of the esophagus. The

fixed points are the cricoid cartilage (C-6 level) and the point where the esophagus passes through the diaphragm. Most symptomatic osteophytes are located at the level of the cricoid cartilage, as in our case. Although stridor is rare, this can result when osteophyte formation occurs in the C2-3 region. Additional head and neck symptoms include aspiration, Horner's syndrome, globus, odynophagia and otalgia. The latter two manifestations may result from hypopharyngeal ulceration at a point of pressure between the posterior cricoid cartilage and a protruding osteophyte. Typically, patients have a long history of dysphagia, and the C3-6 levels are most commonly involved.

Even with radiographic evidence of osteophytes, one must not overlook the possibility of a malignant etiology for dysphagia given the age of these patients. The differential diagnosis of dysphagia should include tumors of the larynx, esophagus, lung and mediastinum, esophageal motility disorders, esophagitis, esophageal stricture, spinal tumors, vascular abnormalities, Zenker diverticulum, Plummer-Vinson syndrome, gastroesophageal reflux and globus hystericus⁴.

The diagnosis of DISH is based on the following findings: 1) flowing calcification and ossification within the anterior longitudinal ligament involving four or more contiguous vertebral bodies, 2) minimal to no degenerative disc changes, and 3) absence of apophyseal joint ankylosis and sacroiliac erosion⁵. These radiographic criteria allow DISH to be differentiated from ankylosing spondylitis and intervertebral osteochondrosis, which are two other diagnoses that can be responsible for vertebral osteophytosis.

Management of patients with DISH depends on the degree of symptomatology. Most patients respond to diet modification, swallowing therapy, nonsteroidal antiinflammatory drugs, muscle relaxants, antibiotics and steroids⁶. When dysphagia impairs nutritional status, symptoms are distressing or the airway becomes compromised, surgical intervention must ensue. Excision of the osteophyte via anterolateral, posterolateral or transoral approach may be done. The anterolateral approach provides better exposure of the great vessels and vagus, but it does place the recurrent laryngeal nerve at greater risk⁷. The posterolateral approach offers wide exposure of the prevertebral space but requires more retraction of the carotid sheath. The transoral approach has the advantage of cosmetic appeal as well as limited risk to the major vessels and nerve. However disadvantages include limited exposure

and potential risk for fascial infection or osteomyelitis.

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