Acute Onset Dysphagia Secondary To Cervical Injury In A Young Male – A Case Report
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Citation

Abstract
Dysphagia may have oropharyngeal or esophageal causes. Extrinsic compression of oesophagus or intraluminal abnormalities in the esophagus can cause dysphagia. Anatomic causes of dysphagia are tumors, abscesses, and cervical bony outgrowths (osteophytes). Anterior cervical osteophytes can rarely cause dysphagia due to mechanical compression and inflammatory reactions in the tissues around the esophagus. For this reason Non steroidal anti-inflammatory drugs might have a role in improving patient symptomatology. We present a case of a young man with cervical osteophytes presenting with an acute onset of dysphagia following mechanical injury to the neck that improved rapidly with non steroidal anti-inflammatory medications. The report is discussed with a review of relevant literature.

INTRODUCTION
Mechanical oesophageal dysphagia is caused by either an extrinsic compression of oesophageal lumen or intrinsic pathologies of oesophageal wall. Various local structural lesions such as Oropharyngeal tumors, thyroid gland enlargement, vascular pathologies, mediastinal mass, or a retropharyngeal abscess, and anterior cervical osteophytes may lead to mechanical oesophageal dysphagia. Although rare, patients with anterior cervical osteophytes may complain of dysphagia particularly when the osteophytes are extraordinarily large. Anterior cervical osteophytes occur in 20-30% of the elderly population and generally remain asymptomatic.

Large anterior osteophytes of the cervical spine may also be seen in diffuse idiopathic skeletal hyperostosis (DISH), also known as Forestier's disease. Large osteophytes, however, do cause swallowing disorders through a variety of mechanisms, including: (1) direct mechanical compression of the pharynx and esophagus, (2) disturbances of normal epiglottis tilt over the laryngeal inlet by the osteophytes at C3–C4 level, (3) inflammatory reactions in the tissues around the esophagus and (4) cricopharyngeal spasm.

We present a case of a young man with cervical osteophytes presenting with an acute onset of dysphagia following mechanical injury to the neck with a review of relevant literature.

CASE
A 38 year old male gentleman presented at the Out Patient Department of the Ear Nose and Throat clinic at a tertiary referral hospital in Bangalore, South India with a history of progressive painless dysphagia for solids since 1 year following a fall which lead to injuries to his upper neck and teeth. He had no other known co-morbidities apart from chronic neck pain since three years, prior to the event that caused the trauma. A 90 degree rigid videolaryngoscopy was performed which showed a normal endolarynx except for a prominent bulge at the posterior pharyngeal wall at the level of the epiglottis.(figure 1) Clinical examination of the neck revealed significant tenderness over the third to fifth cervical vertebrae (C3-C5) and there was no evidence of any other palpable neck mass or growth. He was afebrile and the remaining ear, nose and throat examination was unremarkable.
Figure 1
Figure 1- A 90 degree rigid videolaryngoscopy showing a prominent bulge at the posterior pharyngeal wall at the level of the epiglottis

A soft tissue x-ray of the lateral view of the neck was taken which revealed severe anterior spondylosis from the second cervical vertebrae to the fourth cervical vertebrae and fifth and sixth cervical vertebrae along with anterior spondylophytes and severe osteophytic changes. (Figure 2)

Figure 2
Figure 2 - * severe anterior spondylosis from the second cervical vertebrae to the fourth cervical vertebrae and fifth and sixth cervical vertebrae along with anterior spondylophytes and severe osteophytic changes

These findings confirmed the cause for dysphagia to be secondary to cervical osteophytes. A conservative line of management was selected and he was administered oral non-steroidal anti-inflammatory drugs along with antacids for 2 weeks. A cervical collar was also given to ensure adequate immobilization. There was a remarkable improvement in symptomatology after a follow-up of two weeks.

DISCUSSION

Mechanical oesophageal dysphagia is caused by either an extrinsic compression of oesophageal lumen or intrinsic pathologies of oesophageal wall. Dysphagia may have oropharyngeal or esophageal causes. Anatomic causes of dysphagia are tumors, abscesses, and cervical bony outgrowths (osteophytes). The differential diagnosis of spinal bony outgrowths is extensive, but the most common etiologies are diffuse idiopathic skeletal hyperostosis (DISH) and ankylosing spondylitis (AS). Cervical bony outgrowths are common, and most are asymptomatic. However, dysphagia secondary to compression of the esophagus by these osteophytes is unusual. Other causes of dysphagia include diffuse idiopathic skeletal hyperostosis, also known as Forestier’s disease (typified by bony bridges between vertebrae and absence of disc degeneration, congenital bone bars, anterior herniation of a calcified nucleus pulposus, atlantoaxial dislocation, trauma, “ekchondromata”, osteochondroma and calcification of spinal ligaments or muscles from other causes, including severe cervical lordosis.

In 1926, Moshe was the first to report dysphagia secondary to cervical osteophytes. Dysphagia due to extrinsic factors in the cervical region is most commonly associated with degenerative disc disease accompanied by osteophyte formation, although the cause of the hyperostosis is not known. Factors that have been considered include an endocrine factor, hypervitaminosis A, fluorosis and secretion of a bone morphogenetic factor with stimulation of growth of bony tissue. Dysphagia caused by cervical osteophyte formation is rare. Three theories have been postulated to explain the mechanism of the dysphagia caused by osteophytes. First, large osteophytes cause direct mechanical blockage of the esophagus or hypopharynx. Second, dysphagia may even be caused by small osteophytes, if they are located at the fixed points of the esophagus (cricoid cartilage at the C6 level). Third, osteophytes may cause an inflammatory reaction around the oesophagus.

Although cervical osteophytes are seen in 20%–30% of the geriatric population, they are an unusual (and treatable) cause of dysphagia. Dysphagia occurs because of mechanical blockage as well as inflammation of the peripharyngeal and peri-oesophageal tissue. As enlarged cervical osteophytes may be an incidental finding, it is important to exclude other potential causes, such as neoplasm.
The evaluation of cervical osteophytes consists of cervical spine radiograph and barium swallow to confirm their presence. Extending the neck and swallowing a barium-coated marshmallow to reproduce dysphagia during the barium swallow may confirm the obstructive nature of the osteophytes. Video fluoroscopy shows the sequence or muscular changes needed to transfer ingested material from mouth to upper esophagus and rules out neuromuscular causes of dysphagia. In patients with cervical osteophytes, upper gastrointestinal endoscopy may be performed, but cautiously because of the risk of esophageal perforation. Manometry and pH stimulation studies may help to exclude motility disorders and gastro esophageal reflux disease as a cause of dysphagia in the neck.

Conservative treatment has been indicated for the initial management of these cases. Most authors advise conservative treatment with anti-inflammatory drugs, muscle relaxants, antibiotics, and dietary nutrition for patients with anterior cervical osteophytes and dysphagia or dyspnea. Such patients have some difficulty in swallowing the tablet forms of nonsteroidal anti-inflammatory drugs (NSAIDs) or steroids. Hence liquid forms are recommended to prevent local irritation and ulcerative effects of these drugs on the esophageal mucosa. Patients should be fed soft food and should swallow solid food after it is completely chewed. If these conservative precautions are not adequate in serious cases, surgical resection may be needed.

Phonophoresis has been used successfully to deliver anti-inflammatory medication to inflamed subcutaneous tissues. In addition, when using pulsed-wave ultrasound there is anti-inflammatory and analgesic non-thermal effects. It has been stated that Phonophoresis of ketoprofen allows the attainment of higher local concentration; whereas systemic exposure was lower. Phonophoresis with anti-inflammatory medication to inflamed subcutaneous tissues. Phonophoresis has been used successfully to deliver anti-inflammatory and local irritation and ulcerative effects of these drugs on the esophageal mucosa. Patients should be fed soft food and should swallow solid food after it is completely chewed. If these conservative precautions are not adequate in serious cases, surgical resection may be needed.

Surgical resection of the osteophyte has been reported to be an effective treatment for severe cases and/ or cases with airway obstruction. It is appropriate in patients with severe and progressive symptoms. Speech and language therapies and nutritional counseling are of great importance in the assessment and management of dysphagia.

CONCLUSION

It is very important to remember the unlikely possibility of an esophageal compression in patients with cervical spondylosis to avoid misdiagnosis when considering the several causes of dysphagia, and to avoid inappropriate treatment. In this case a prompt diagnosis and initiation of NSAIDS brought an immediate relief in the patient’s symptomatology. This highlights the importance of considering a diagnosis of dysphagia secondary to cervical osteophytes in spite of the younger age group and shorter duration of history.

References


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