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# Cervical osteophytes presenting as unilateral vocal fold paralysis and dysphagia

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**Abstract**
Any process involving either the vagus nerve, its recurrent laryngeal branch or the external branch of the superior laryngeal nerve may cause paralysis of the vocal fold. The most common cause is neoplasm. Clinically, the patients often present with a hoarse, breathy voice as well as symptoms of aspiration. The following represents a unique case of unilateral vocal fold paralysis and dysphagia caused by a degenerative disease of the cervical spine, resluting in extrinsic compression of the recurrent laryngeal nerve.

**Key words: Cervical Vertebrae; Arthritis; Degenerative; Vocal Fold Paralysis**

## Case report

The patient is a 67-year-old male, who is a retired fireman. The patient's past medical history is remarkable for mild hypertension, controlled with medication, as well as a left-sided blunt trauma to the shoulder, requiring rotator cuff repair eight years prior to presentation. The patient has never smoked nor drunk alcohol.

The patient presented with a six-week history of dysphonia, that the patient described as hoarse. After 10 days of persistent hoarseness, the patient consulted a family physician, who prescribed antibiotics, with no resolution of symptoms. During the next five weeks, the patient developed a progressive dysphagia and weight loss of approximately 10 kg, without any associated odynophagia. The patient also complained of symptoms consistent with aspiration. The patient denied any other symptoms, including chest discomfort, shortness of breath or haemoptysis.

At this point, an otolaryngology consultation was performed. The head and neck examination was remarkable only for a left-sided vocal fold paralysis, in the paramedian position, with normal function of the opposite fold. There was poor glottic closure, with no evidence of a mass lesion. There was no pooling of secretions. Stroboscopic examination concurred with the flexible laryngoscopic examination, revealing a paralyzed left vocal fold and a normal mucosal wave on the opposite side. Electromyography was not performed. There was no evidence of cervical lymphadenopathy, and palpation of the thyroid gland was normal. Neurological testing, with the exception of the parlayzed fold was within normal limits, without any focal neurological defects. All blood work was within normal limits, including complete cell count and thyroid function testing. A modified barium swallow and chest X-ray were both normal.

It was elected to perform a computed tomography (CT) scan of the neck (Figure 1), which revealed massive osteophytes of cervical spine bodies three through six. The

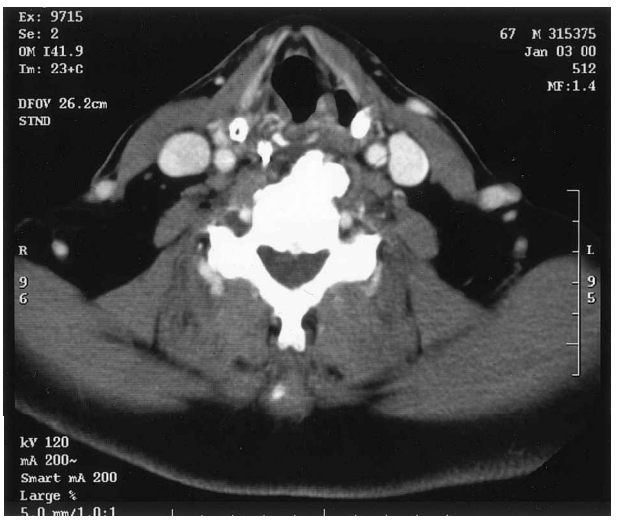


Fig. 1
CT scan showing large left-sided osteophytes of the cervical spine.

osteophytes, that were primarily on the left side, projected anteriorly, causing displacement of the oesophagus, and compression of the left tracheoesophageal groove. There was also mild reactive oedema of the left pyriform sinus, and the patient was taken to the operating theatre for surgical decompression. The patient underwent an anterior approach to the cervical spine, that confirmed the presence of massive osteophytic disease of the bodies of cervical spine three to six. The operation was uneventful and without post-operative complications. At follow-up six weeks later, the fold had near perfect mobility, with complete resolution of the dysphagia and aspiration symptoms.

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## Discussion

Paralysis of a single vocal fold involves the disturbance of the recurrent laryngeal branch of the vagus nerve, which innervates all intrinsic muscles of the larynx, with the exception of the cricothyroid muscle, that is innervated by the superior laryngeal branch. Nearly 90 per cent of the paralyses are related to lesions located peripheral to the central nervous system, while 10 per cent are located in the central nervous system.[1] The recurrent larygneal nerve has a different anatomical course on either side of the neck. On the left side of the neck, the nerve descends towards the great vessels, wraps around the ligamentum arteriosum, and then ascends in the left tracheoesophageal groove, piercing the cricothyroid membrane, and then innervating the intrinsic musculature of the larynx. On the right side of the neck, the recurrent laryngeal nerve wraps around the right subclavian artery, and then ascends in the right tracheoesophageal groove, similarly innervating the musculature of the larynx on the right side.

The differential diagnosis of the paralyzed vocal fold is extensive. In ascertaining the cause, the physician needs to differentiate between central and peripheral lesions, as well as unilateral versus bilateral. The aetiology of bilateral vocal fold paralysis includes Guillain-Barré syndrome, post-thyroidectomy recurrent laryngeal nerve injury, poliomyelitis, Parkinson's disease, cerebrovascular accident, multiple sclerosis, myasthenia gravis, CNS/neck/chest neoplasm, infection or congenital paralysis.[2,3] The aetiology of unilateral vocal fold paralysis is equally diverse and includes subglottic haemangioma, laryngeal papillomas, laryngocele, laryngeal carcinoma, carcinoma of the lung, central nervous system malignancy, schwannoma of the vagus nerve, post-cardiac surgery, post-intubation paralysis, post-thyroidectomy paralysis, blunt trauma, cystic hygroma, thyroid neoplasm, or oesophageal neoplasm as reported in the literature.[4–6]

Degenerative disease in the spine includes two interrelated conditions; one involves the intervertebral disc joints (degenerative disc disease), and the other, the posterior articulating facet joints (degenerative joint disease). Both processes represent an exaggeration of the normal ageing process and may be aggravated by injury, deformity and pre-existing disease of the spine.[7] The resultant pain is the most common of all musculoskeletal symptoms.

The initial degeneration in the human spinal column occurs in the nucleus pulposus of the intervertebral disc, which loses its water content, resilience, height, and turgor progressively with advancing age. Similarly, the annulus fibrosus gradually loses some of its elasticity. Its fibres become torn, allowing for the potential disc herniation. As a result of these degenerative changes in the intervertebral disc joints, smooth motion in each involved segment of the spine is lost and is replaced by motion that is not only uneven, but also excessive. In this stage of segmental instability, the joint margins react by forming small traction spurs, or osteophytes. As the degeneration erodes the articular surface, the ability of this surface to distribute stress begins to fail, and stress on the underlying bone increases. The bone responds to increased stress by laying down increased bone. Thus more surface area is produced to cover the increased stress.[8,9] These osteophytes are detectable radiographically in up to 90 per cent of individuals older than 60 years of age.[10] As this degenerative process wears down the disc space, the intervertebral joint space narrows and loses much of its motion. As the joint stiffens the instability diminishes, as does the pain.

In the cervical spine, the most common segments to be affected by such degenerative changes are C5-6 and C6-7, which are particularly mobile and in an area of maximal lordosis, making the loads to which they are subjected significant. This is analogous to the situation in the lumbar spine to the L4-5 and L5-S1 levels.[11]

Management of cervical spine degenerative arthritis is predominantly conservative in nature. Symptomatic treatment with anti-inflammatory combined with analgesic medications are the first line agents. Physiotherapy to achieve paravertebral muscle strengthening thereby enhancing spinal segmental stability is also routinely used. Surgical intervention is usually reserved for those patients who have failed conservative options. In addition, invasive treatment may be indicated for those who have demonstrable neurological impairment or protracted, incapacitating pain. Cheilectomy (or excision of osteophytes) may be useful in cases where the osteophytes cause extrinsic compression of other anatomical structures resulting in radiculopathies or myelopathies. In the case of this patient who exhibited both significant clinical and radiological disease, an anterior approach to the cervical spine just medial to the carotid sheath, exposing C3 to C6[12] allows a safe and direct approach to this area. The quick recovery of the patient was attributed to only mild neuropraxia of the involved nerves secondary to compression. Once the compressive factor was removed i.e. the osteophytic disease, function returned to normal.

In summary, vocal fold paralysis may be attributed to numerous aetiologies, spanning a wide spectrum of clinical entities. Cervical osteophytic disease, although rare as a cause of vocal fold paralysis, should be included in this differential diagnosis.

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