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CASE REPORT PEER REVIEWED | OPEN ACCESS

Dysphagia accompanied with anterior osteophytes of the cervical vertebrate, secondary to the anterior cervical discectomy and fusion

Akira Tempaku

ABSTRACT

Introduction: One cause of osteophyte formation is adjacent segment syndrome, which can result from spinal fixation surgery. Compression of the spinal cord and nerve roots due to spinal canal stenosis causes neurological disorders. However, cases with neurological findings similar to central nervous system abnormalities have been seen without spinal canal stenosis. A rare case of osteophyte formation in the anterior cervical spine causing laryngeal stenosis and dysphagia was encountered.

Case Report: A 72-year-old male with an anterior cervical discectomy and fusion (ACDF) at C5–C6 presented with a complaint of dysphagia. No impairment was observed in pharyngeal and laryngeal function; however, osteophytes at C45 and C67 protruded anteriorly, resulting in laryngeal stenosis. This was identified as the underlying cause of the dysphagia. Osteophyte resection and ACDF at C45 and C67 were performed. Following the surgical procedure, there was an improvement in the patient's dysphagia.

Conclusion: The formation of osteophytes is a consequence of excessive pressure being applied to the intervertebral discs located between neighboring vertebrae. The hypothesis is that the cervical ACDF increased the load on the adjacent vertebrae above and below, leading to the formation of osteophytes. The osteophytes manifested particularly robust formation at the anterior aspect, exerting pressure on the posterior laryngeal wall from a posterior direction and consequently impeding the passage of ingested matter. This case report documents a rare instance of adjacent segment disorder with osteophyte formation. The condition was extremely rare, involving tissue protrusion, narrowing of the airway and difficulty eating and drinking.

Keywords: Anterior cervical discectomy and fusion, Dysphagia, Esophagus obstruction, Osteophyte

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INTRODUCTION

The anterior cervical discectomy and fusion (ACDF) procedure entails the removal of the intervertebral disc and posterior longitudinal ligament with the objective of enlarging the spinal canal and intervertebral foramen [1, 2]. This procedure is undertaken to prevent neurological disorders that result from compression of the cervical spinal cord and nerve roots. Moreover, it is instrumental in ensuring stability between the vertebral bodies, thereby maintaining spinal support. The stability of the vertebral bodies is ensured by the insertion of cages into the intervertebral disc spaces or the placement of plates on the anterior surfaces of the vertebral bodies. Nevertheless, there remain uncertainties regarding the long-term stability between fixed vertebral bodies and the vertebral bodies above and below them, as well as associated complications [3–5].

A case of dysphagia was observed, attributed to the presence of osteophyte formation in conjunction with adjacent segment syndrome following ACDF surgery. Several reports have documented the occurrence of osteophyte formation within the spinal canal subsequent to ACDF surgery [6, 7]. However, the majority of these cases have been observed to extend from the posterior aspect of the vertebral body into the spinal canal itself [8].

Furthermore, although there are scattered reports of dysphagia caused by pharyngeal compression associated with osteophyte formation [7], no reports of ACDF as a cause were found. The present study aims to provide a comprehensive report on the mechanism of osteophyte formation induced by vertebral fixation and osteophytes associated with dysphagia, incorporating a thorough literature review.

CASE REPORT

The patient is a 72-year-old male. Thirteen years prior to the present, the patient underwent C5–C6 ACDF for cervical disc herniation (Figure 1A and B). Over one decade, the presence of osteophyte formation was observed at the C4–C5 and C6–C7 vertebral levels (Figure 1C and D). The protrusion of osteophytes from the cervical vertebral bodies in an anterior direction resulted in posterior compression of the laryngeal wall, leading to dysphagia. A thorough investigation revealed no issues with the function of the pharyngeal and laryngeal muscles. The primary cause of the dysphagia was determined to be esophageal stenosis.

Consequently, the anterior aspect of the cervical vertebral body was exposed via a right anterior approach to the cervical spine. The osteophytes that had formed anterior to C45 and C67 were removed. The intervertebral disc was also resected, and ACDF was performed at C45 and C67 to maintain cervical stability (Figure 1E and F). Pharyngeal narrowing and bone lesion size were measured and compared pre- and post-operatively on the mid-sagittal plane of computed tomography (CT) images. Preoperatively, the distance from the anterior vertebral body to the anterior edge of the osteophyte was 9.8 mm and to the anterior pharyngeal wall was 30.3 mm. Postoperatively, this distance was 7.3 mm, while the distance to the anterior pharyngeal wall increased to 33.9 mm.

Immediately following the surgical procedure, there was a rapid improvement in the patient's dysphagia. Functional improvement of dysphagia has been kept more than seven months during the follow-up periods.

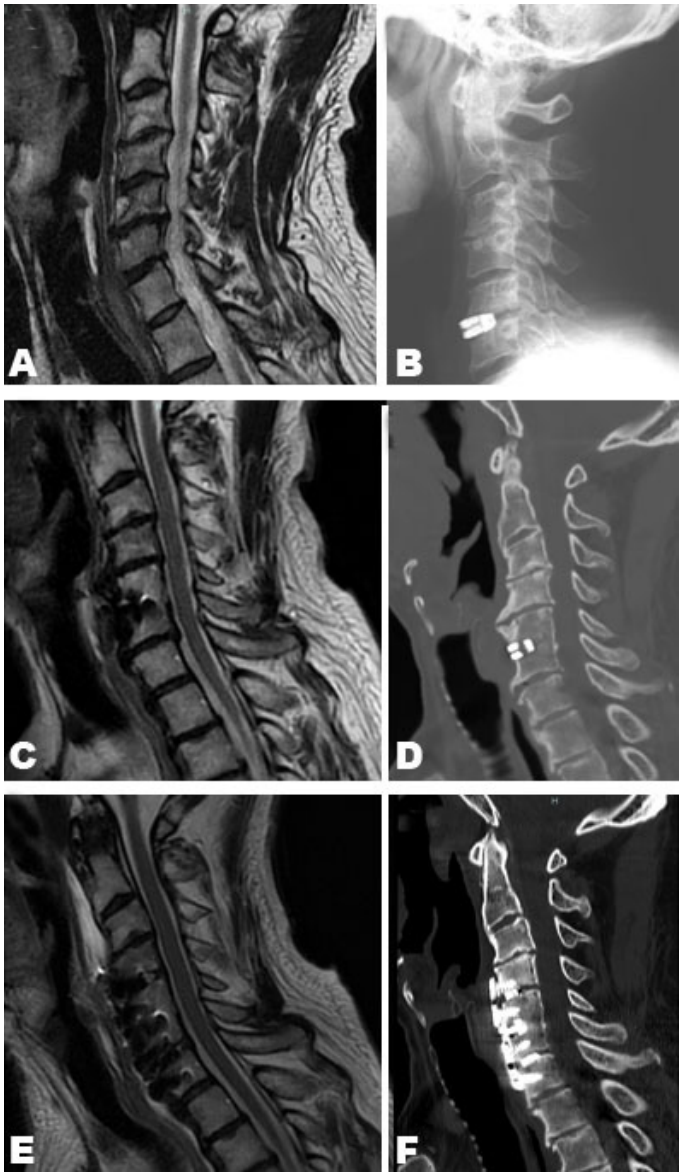

Figure 1: The cervical features are documented. (A) shows a lateral view of the cervical portion obtained via T2-weighted MRI 13 years prior to the present time. Spinal canal stenosis at the C5–C6 level due to lumbar herniation is observed. (B) shows an x-ray taken after the first ACDF procedure. The cervical disc was replaced with a cage at the C5–C6 level. (C) shows the cervical portion by T2-weighted MRI at the present time. Spinal canal stenosis is not observed. (D) shows the cervical region via CT scan at the present time. Anterior protrusion with calcification is observed at the C4–C5 and C6–C7 levels. (E) shows the cervical portion by T2-weighted MRI after the additional operation. (F) shows the cervical portion by CT scan after the additional operation.

DISCUSSION

The formation of osteophyte lesions in the cervical spine is hypothesized to be precipitated by instability of the cervical spine. The condition is triggered by stress on the intervertebral surface due to compression and twisting of the intervertebral disc, applied strongly and over a long period of time. Consequently, the cortical bone of the intervertebral surface undergoes an expansion and formation of osteophytes [9, 10].

The rotation and extension-flexion movements of the cervical spine are formed by the sum of misalignments between each vertebra.

Axial rotation (*y*-axis rotation) is primarily formed by deflection at the atlantoaxial joint and the sum of rotational movements at each intervertebral joint from C1–C2 to C6–C7 segments [11]. When C5–C6 is fixed via ACDF, the rotational movement of six segments is compensated for by the rotational movement of five segments. As demonstrated in White's paper [11], the mean change in each intervertebral space is expressed in degrees. Accordingly, the intermediate levels of the cervical vertebrae (C3–C4, representative angle: 11 degrees), C4–C5 (12 degrees), and C5–C6 (10 degrees) are primarily affected, and the required angle of misalignment increases in each intervertebral space, mainly including with C4–C5.

Flexion-extension (*x*-axis rotation) is formed by the sum of the angular changes (tilt) in the seven intervertebral spaces from the occipital bone to C1 through C6–C7 [11]. Following the implementation of ACDF in C56, the distribution of flexion-extension angle, which was previously dispersed across seven locations, is now distributed across six locations. As the intermediate segments C3–C4 (13 degrees), C4–C5 (12 degrees), C5–C6 (17 degrees), and C6–C7 (16 degrees) divide the change in angle, the required angle of tilt between adjacent vertebrae, such as C4–C5 or C6–C7, becomes larger.

Lateral bending (*z*-axis rotation) is formed by the sum of the angular changes (tilt) to the left and right at six intervertebral joints from the occipital bone to C1 through C6–C7, excluding the atlantoaxial joint [11]. Following the implementation of an ACDF procedure on the C5–C6 vertebra, the distribution of tilt, which was previously allocated across six joints, is now distributed across five joints. The distribution of changes in tilt is generally uniform across the upper and middle vertebrae, with the following measurements: C2–C3 (10 degrees), C3–C4 (11 degrees), C4–C5 (11 degrees), and C5–C6 (8 degrees). However, the required angle of tilt becomes larger at vertebral joints such as C4–C5.

In summary, axial rotation reduces the primary range of motion to two-thirds, increasing the range of motion load per joint pair by 17%. Flexion-extension reduces the primary range of motion to three-quarters, increasing the range of motion load per joint pair by 8%. Lateral bending reduces the primary range of motion to three-quarters, increasing the range of motion load per joint pair by 8%.

Consequently, ACDF imposes limitations on intervertebral mobility, thereby augmenting the stress on the vertebrae situated above and below the fixed vertebrae. It is hypothesized that, if this movement load and weight load on adjacent intervertebral spaces continues over a long period of time, osteophyte formation will be promoted in the vertebrae above and below the vertebrae on which ACDF was performed [8, 12–14].

Adjacent segment disorder refers to disc degeneration or spondylolisthesis that occurs in the intervertebral space next to the space that underwent fusion surgery (the responsible space). The long-term follow-up after spinal fusion surgery has been shown to be a period during which cases of adjacent segment syndrome have been reported [15, 16]. It is thought that the cause of adjacent segment syndrome is excessive fixation due to the use of rigid materials in the pedicle screw and rod system (PS fusion), which reduces the range of motion of the responsible segment and thereby increases the range of motion and torque of the adjacent segments [17]. Consequently, there is an increase in instability between the adjacent vertebral bodies and osteophyte formation is promoted in the anteroposterior direction. Osteoblast bone formation is promoted by pressure stress [18, 19]. Mechanical stress on the vertebral bodies due to movement of the cervical spine increases after ACDF surgery in non-fixed vertebral bodies. This increase stimulates osteoblasts. Consequently, signal pathways relating molecules such as Wnt, Notch, ERK, RhoA, and NF-κB are activated [19], which is believed to promote bone formation by osteoblasts.

Strongly protruding osteophytes at the front of the cervical vertebrae can cause dysphagia [20–24]. Even when no organic abnormalities are identified in the cranial nerves, pharyngeal narrowing may be the cause if pharyngeal swallowing movements are impaired. Endoscopic examination of the pharynx or assessment of swallowing function may reveal physiological narrowing; however, evaluation using computed tomography (CT) or magnetic resonance imaging (MRI) of the neck is also effective for identifying the cause.

Adjacent segment disorders following spinal fusion surgery have been reported in cases involving surgery on the lumbar spine, which bears a greater load [12–17]. The cervical spine, which facilitates movements such as head rotation and flexion, is also a common site for such conditions. However, osteophytes extending from the vertebral body towards the spinal canal are frequently reported in both regions [6]. Due to spinal stenosis, these conditions are more likely to present with symptoms such as spinal cord or radicular symptoms, leading to more frequent clinical evaluations. In the long-term follow-up after spinal fusion surgery, osteophyte formation towards the anterior aspect of the vertebral body may be observed. However, since this is less likely to cause symptoms, it is often overlooked. In the cervical spine, however, the pharynx, esophagus and airway are located in close proximity to the anterior aspect, so stenosis caused by osteophytes may bring a clinical problem. Careful postoperative monitoring based on imaging tests and clinical symptoms is necessary to detect osteophyte formation in the anterior cervical spine.

On the other hand, with regard to the direct invasion of head and neck nerves, the vagus nerve is located outside the carotid sheath; therefore, compression damage is unlikely to spread over a distance. Additionally, the recurrent nerve runs along the lateral side, between the esophagus and the trachea. However, since the esophagus is positioned between the cervical vertebrae, direct compression against the recurrent nerve by osteophytes is anatomically unlikely. It is unlikely that neurological dysfunction of the larynx would arise from the early stages of osteophyte expansion.

Osteophyte formation accompanied by dysphagia is primarily recognized as a complication of diffuse idiopathic skeletal hyperostosis (DISH). According to reports from 134 cases of DISH, dysphagia is present in 6% of cases [25]. There are numerous case reports and case series describing surgical treatment for osteophyte formation with dysphagia in DISH. Comprehensive criteria are increasingly used to determine indications, such as sleep disorders, weight loss and 1 cm+ osteophytes protrusion, as recommended by Sebaaly [26]. Bunmaprasert reported 86 cases in a literature review covering 2000 to 2020 [27], while Harlianto reported 276 cases in a literature review covering 2010 to 2021 [28].

Conversely, Chen reported that approximately half of the cases of anterior osteophytes associated with dysphagia did not meet the criteria for DISH [29]. In the discussion, he raised the possibility of conditions such as ankylosing spondylitis [30, 31]. Furthermore, Fujita reported anterior osteophytes causing dysphagia due to acromegalic arthropathy, which is distinct from DISH [32].

These are thought to represent osteophyte formation due to degenerative disorders of the spine against a background of endogenous hyperostosis, resulting in dysphagia. In contrast, hyperostosis due to adjacent segment disorder following ACDF is thought to result from changes in exogenous stress.

There are reports of osteophyte regrowth following surgical treatment for DISH, over several years [33, 34]. Since the triggers for hyperostosis differ, recurrence after spinal fusion surgery in conditions caused by exogenous adjacent segment disorders requires careful evaluation. Long-term follow-up will likely remain necessary.

This case report still has a clinical limitation for the critical analysis. The severity of dysphagia was assessed using a simplified questionnaire on swallowing function. Subjects were asked to rate the difficulty they experienced when swallowing solids, noodles and liquids. Objective evaluations, such as a video fluoroscopic swallowing study (VFSS), a barium swallow or esophageal manometry, were not performed. This constitutes a limitation when evaluating the results of this case report.

CONCLUSION

Anterior protrusion of osteophytes was observed in the aftermath of ACDF surgery. The presence of excess osteophytes resulted in posterior pharyngeal wall elevation, manifesting as dysphagia. Surgical removal of the protruded osteophytes led to the restoration of pharyngeal function.

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Author Contributions
Akira Tempaku – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

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Conflict of Interest
Author declares no conflict of interest.

Data Availability
All relevant data are within the paper and its Supporting Information files.

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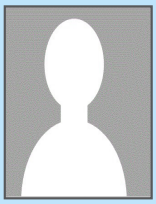

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