Acute Calcific Tendonitis of the Longus Colli

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ABSTRACT

Objective: Report a rare case of acute calcific tendonitis of the longus colli (ACTLC).

Method: Describe the clinical presentation, radiographic findings, and management of a patient with ACTLC and review the literature.

Results: A 45-year-old female presented with a 3-day history of progressive neck pain and tenderness over the right posterolateral neck with limited range of motion. Flexible fiber-optic laryngoscopy revealed moderate posterior pharyngeal wall edema. Computed tomography (CT) scan of the neck revealed a calcification in the longus colli muscle and a prevertebral fluid collection without rim enhancement. A diagnosis of ACTLC was presumed. The patient was successfully managed with non-steroidal anti-inflammatory drugs (NSAIDs), steroids, analgesia, and antibiotics and displayed marked improvement on hospital day 1. She was discharged with a short course of NSAIDs and antibiotics (precautionary measure) with complete resolution of symptoms on follow-up.

Conclusion: ACTLC is an often misdiagnosed disease of the head and neck with limited reports in the literature. CT scan revealing a calcific deposition in the retropharyngeal space confirms the diagnosis. Spontaneous resolution over several weeks warrants conservative treatment with analgesia and anti-inflammatory medications. Awareness of ACTLC is necessary to avoid diagnostic errors and prevent the usage of unnecessary medical intervention.

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INTRODUCTION

Acute calcific tendinitis of the longus colli muscle (ACTLC) is an aseptic inflammatory response to deposition of calcium hydroxyapatite crystals in the superior fibers of the longus colli muscle tendons. Though reports in the literature are scarce, ACTLC likely represents an underreported disease process that routinely goes misdiagnosed. Computed tomography (CT) of the neck with contrast showing calcifications in the prevertebral space clenches the diagnosis. Successful management includes the use of non-steroidal anti-inflammatory drugs (NSAIDs) and corticosteroids.[1] We report a case of ACTLC herein and a provide a brief review of the literature.

CASE REPORT

A 45-year-old otherwise healthy female presented with a 3-day history of progressive neck pain, decreased neck mobility, and odynophagia. The patient was afebrile with normal vital signs. She exhibited tenderness over the right posterolateral neck with limited range of motion on flexion and rotation of the neck. Flexible fiber-optic laryngoscopy revealed moderate posterior pharyngeal wall edema extending from the palatal-pharyngeal sphincter down to the level of the epiglottis. White blood cell count was in the upper limit of normal. Computed tomography (CT) scan of the neck with contrast revealed an amorphous calcification in the longus colli muscle and a prevertebral fluid collection without rim enhancement (Figures 1 & 2). A diagnosis of ACTLC was presumed, though retropharyngeal abscess was still in consideration. The patient was successfully managed with non-steroidal anti-inflammatory drugs (NSAIDs), corticosteroids, analgesia, and antibiotics and displayed marked improvement on hospital day 1. She was discharged with a short course of NSAIDs and antibiotics as a precautionary measure and reported complete resolution of symptoms on follow-up.

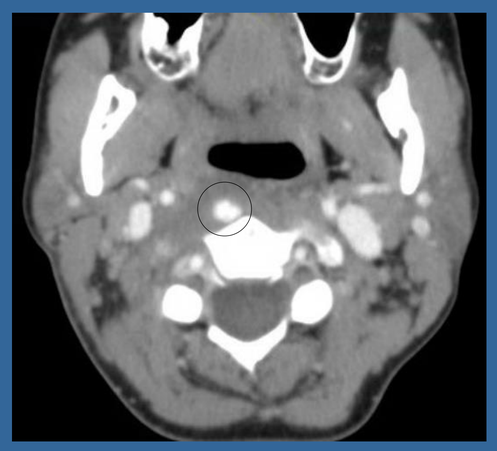

Figure 1. Axial CT at C2 level showing calcific deposit in the mass of the longus colli muscle (circle).

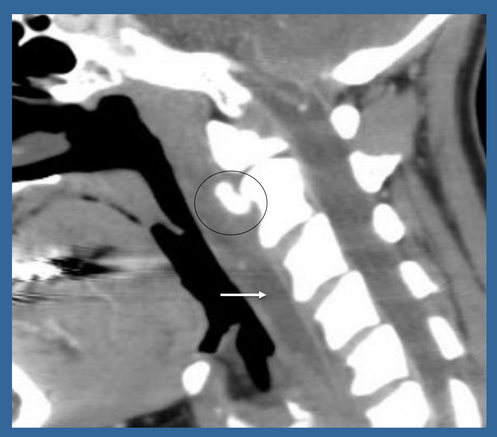

Figure 1. Sagittal CT demonstrates a smooth, lenticular-shaped fluid collection within the prevertebral soft tissues (arrow), with amorphous calcification embedded within the proximal fibers of the longus colli tendon, anterior to the C2 vertebral body (circle).

DISCUSSION

Anatomy
The longus colli muscle is one of the four deep cervical flexor muscles and consists of superior oblique, vertical, and inferior oblique fibers. This muscle acts to provide cervical flexion, ipsilateral flexion, and some rotational movement. The superior oblique fibers, which originate from the transverse processes of C3 to C5 and fuse into a tendon that inserts onto the anterior tubercle of the atlas, are the most vulnerable to calcific deposits.[2]

Epidemiology
Acute calcific tendonitis of the longus colli (ACTLC) mostly affects adults between 30 and 60 years old and has no sex predilection. While only a small number of cases have been presented in the ENT literature, some authors suggest ACTLC may be more prevalent than previously thought due to missed diagnoses and underreporting of the disease.[1,3-10] According to an epidemiological study done in 2013, the age matched incidence of ACTLC may be as high as 1.31 per 100,000 individuals.[1]

Pathophysiology
The etiology behind ACTLC is unclear, though repetitive trauma, ischemia, necrosis, and degeneration of the longus colli tendon are hypothesized to precede calcium and hydroxyapatite crystal deposition. The calcium deposition acts as a localized attempt to compensate for decreased tendon strength, while rupture and release of hydroxyapatite crystals into the surrounding soft tissue lead to an acute inflammatory response.[8,9]

Clinical Presentation
The symptoms of ACTLC include acute onset neck pain, neck stiffness, dysphagia, odynophagia, mild fever, and decreased neck mobility. These symptoms, however, are non-specific as this condition presents similarly to various head and neck pathology including retropharyngeal abscess, meningitis, cervical disc herniation, and vertebral fractures to name a few.[5,9,10]

Diagnosis and Imaging
While the diagnosis of ACTLC ultimately relies on radiologic studies, laboratory findings such as mild leukocytosis and raised inflammatory markers support the diagnosis.[10] Lateral plain films are helpful but are unable to rule out similarly presenting pathologies including retropharyngeal infection, cervical disk herniation/infection, cervical vertebral body subluxation/fracture, meningitis, and extradural hemorrhage. Magnetic resonance imaging, useful in detecting prevertebral fluid collection, may not recognize the calcium depositions. CT is the gold standard for diagnosing ACTLC as it can detect calcific deposition at the longus colli and prevertebral edema (Figure 1 and 2). Enhancement around the effusion should shift the diagnosis towards an abscess.[3,6,7,9,10]

Treatment and Outcome
ACTLC resolves spontaneously over several weeks as macrophages and giant cells phagocytose the calcium hydroxyapatite crystal deposits.[6] Due to the self-resolving nature of this disease, symptomatic treatment with non-steroidal anti-inflammatory drugs (NSAIDs) has been the mainstay of treatment with corticosteroids and opioids being reserved for more severe cases. Antibiotics, local analgesic therapy, extracorporeal shock wave therapy, and surgical treatments are not indicated. Local recurrence has not been reported.[1,9,10]

CONCLUSION

ACTLC is a rare and often misdiagnosed disease of the head and neck. Clinical presentation and laboratory findings are non-specific; however, a CT scan revealing a calcific deposition in the retropharyngeal space confirms the diagnosis. Spontaneous resolution over several weeks warrants conservative treatment with analgesia and anti-inflammatory medications. Awareness of ACTLC must be increased in order to avoid diagnostic errors and prevent the usage of unnecessary medical interventions.

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