CASE REPORT

A rare cause of acute dysphagia: acute calcific tendonitis of the longus colli muscle

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SUMMARY

We are presenting a case of a 78-year-old female with multiple comorbidities and history of neck surgery, who presented with acute dysphagia, odynophagia and neck pain that has been progressively getting worse over the course of 2 days, with no recent injury or infection. The patient was afebrile. There were no visible signs of infection on routine oropharyngeal examination. Initial workup was unremarkable except for elevated erythrocyte sedimentation rate, C-reactive protein and creatinine. CT of the cervical spine ruled out any fracture or cervical spine injury but showed an area of calcification, prevertebral oedema and fluid collection inferior to the anterior arch of C1. MRI of the cervical spine also showed prevertebral oedema and fluid collection in the retropharyngeal space from the skull base to the C3 level without abnormal surrounding enhancement that supported the diagnosis of acute calcific tendonitis of the longus colli muscle rather than an infectious process. There was significant improvement of symptoms 48 hours after initiating treatment with systemic steroids. The patient remains asymptomatic 6 months after treatment.

BACKGROUND

Acute calcific tendonitis of the longus colli muscle is a rare clinical entity. Often, this condition is underdiagnosed due to the understated longus colli muscle. It typically presents with pain in the neck followed by dysphagia and odynophagia,^{1,2} which mimics presentation of retropharyngeal abscess and other infectious processes in the oropharyngeal area. Adults between 30 and 60 years of age are most commonly affected by this pathology.¹ In our case, we are presenting a 78-year-old female in whom dysphagia was the chief complaint followed by odynophagia and neck pain. We want to highlight the importance of considering some of the rarer pathologies when starting the work up for causes of dysphagia. Increasing awareness about this pathology as a cause of acute dysphagia might obviate the utilisation of unnecessary diagnostic tests and procedures that will help with administering appropriate treatment on time. Misdiagnosis of this entity may lead to unnecessary antibiotic use. With the appropriate diagnostic tests, imaging and proper treatment, improvement of symptoms may occur within a couple of days with complete resolution in 1–3 weeks.^{3,4}

CASE PRESENTATION

A 78-year-old female with history of type 2 diabetes mellitus, chronic kidney disease (CKD) stage III, history of anterior cervical discectomy and fusion of C4–C6, presented to the emergency department with a 2-day history of dysphagia, odynophagia and neck pain. The patient reported a sudden onset of dysphagia to both solids and liquids, along with odynophagia, followed by severe neck pain and stiffness the morning after. The patient denied any fever, nausea, vomiting, recent infection or trauma.

The patient was afebrile, and on routine oropharyngeal examination there was no signs of inflammation or infection. There was moderate restriction of the range of motion with cervical flexion and significant spasm of the bilateral sternocleidomastoid muscles. There were no signs of meningismus.

Initial laboratory tests showed elevated erythrocyte sedimentation rate (ESR) of 50 mm, C-reactive protein (CRP) of 15 mg/dL, creatinine of 1.5 mg/dL, blood urea nitrogen of 32 mg/dL and glomerular filtration rate of 40 mL/min/1.73 m². The rest of laboratory testing including complete blood count (CBC) and electrolytes were normal.

In the emergency department, a CT scan of the cervical spine was performed, and it showed prior anterior cervical discectomy and fusion from C4 to C6, bilateral foraminal stenosis at C6–7 and C7–T1, an area of calcification inferior to the anterior arch of C1 and mild to moderate prevertebral oedema and fluid collection as shown on [figure 1](#) and [figure 2](#).

After the CT scan findings were reviewed, neurosurgery was consulted, and they recommended admitting the patient to the hospital and performing an MRI of the cervical spine. MRI of the cervical spine showed anterior plate and screw fusion from C4 to C6 with no abnormal cord signal, prevertebral soft tissue oedema and fluid collection in the retropharyngeal space from the skull base to the inferior C3 level with no abnormal surrounding enhancement. As per neurosurgery, this finding goes along with the diagnosis of acute calcific tendonitis of the longus colli muscle [figure 3](#) and [figure 4](#).

Given the patient's severity of symptoms and history of CKD stage III, we preferred to avoid non-steroidal anti-inflammatory medications and treat her with systemic steroids. The patient reported significant improvement of her symptoms 48 hours after initiating therapy.

Patient was discharged on day 3 of hospitalisation. The patient was treated with a tapering dose of prednisone for 10 days. One week after treatment,

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Figure 1 Sagittal view of CT scan of the cervical spine shows an amorphous calcification (see arrow) anterior to the C1–C2 level.

the patient was doing well. She was able to tolerate a regular diet and move her neck without pain and continues to remain asymptomatic 6 months after treatment.

INVESTIGATIONS

The work up for dysphagia was initiated in the emergency department, including CBC with differential, comprehensive metabolic panel, CRP and ESR as basic workup for infection/inflammation and to assess the kidney function before ordering imaging studies with contrast.

BUN 32 mg/dL, creatinine 1.5 mg/dL, glucose 239 mg/dL, GFR of 40 mL/min/1.73 m² and electrolytes within normal range. White blood count 9.5 k/mcl, haemoglobin 13.1 g/dL, hematocrit 41%, platelets 172 k/mcl, ESR 50 mm and CRP 15.

Given the severity of dysphagia, a CT cervical spine without contrast was done in the emergency department to rule out fractures and other infectious processes of the neck. CT showed status postanterior cervical discectomy and fusion from C4 to C6. The instrumentation was intact. There was evidence of osseous fusion from C4 to C6. No acute vertebral fracture. There was an area of calcification inferior to the anterior arch of C1. No facet subluxation or dislocation. Multilevel cervical spondylosis with disc space narrowing, endplate osteophytes and facet arthropathy was seen. Moderate prevertebral oedema and fluid collection was seen.

Figure 2 CT of the axial neck shows a calcific density (see arrow) at the superior tendons of the longus colli at the C1–C2 level.

Figure 3 Sagittal T2-weighted fast-recover fast spin-echo sequence image demonstrates prevertebral soft tissue oedema and fluid collection in the retropharyngeal space from the skull base to the inferior C3 level with no abnormal surrounding enhancement (see arrow) with calcification in the proximal fibres of the longus colli tendon.

MRI cervical spine with or without contrast was ordered since CT scan was done without contrast. A soft tissue infectious process needed to be ruled out.

MRI showed anterior plate and screw fusion from C4 to C6 present. There was stable C7–T1 anterolisthesis. There was prevertebral soft tissue oedema and fluid collection in the retropharyngeal space from the skull base to the inferior C3 level with no abnormal surrounding enhancement. No abnormal cord signal was identified.

As per the CT scan findings, the MRI results and the neurosurgery evaluation, the cause of dysphagia most likely was due to tendinitis. Infectious causes were ruled out. Improvement of symptoms within 48 hours confirmed the diagnosis of acute longus colli tendinitis. At this point, using other diagnostic examinations like laryngoscopy or esophagoscopy was not necessary.

DIFFERENTIAL DIAGNOSIS

Dysphagia/odynophagia with neck pain raised high suspicion for tonsillitis, peritonsillar abscess or retropharyngeal abscess

Figure 4 Sagittal view of T1-weighted MRI shows a bright retropharyngeal space focal effusion (see arrow) dissecting into the longus colli muscles bilaterally from the anterior C1–C3 level with absence of wall enhancement in the surrounding soft tissue.

as a most likely diagnosis, followed by acute spine injury, acute cervical spine fracture, acute disc prolapse, malignancies or drug dystonic reactions. These would justify a decision by the emergency department attending physician to skip plain films and proceed with CT scan.

The absence of symptoms and signs of infection along with a normal white cell count, normal oropharyngeal examination and presence of calcifications on the CT scan helped to rule out infectious processes as the cause of the patient's symptoms and made the diagnosis of acute calcific longus colli tendonitis high on the differential.

TREATMENT

The first line of treatment is non-steroidal anti-inflammatory drugs (NSAIDs) with high rate of response. In severe, painful episodes, patients may also benefit from corticosteroids. NSAIDs are known for gastrointestinal, neurological, renal and allergic side effects that are responsible for approximately 12% of hospital admissions related to adverse effects. They also increase the risk of new cardiovascular events in patients with established heart disease.

For the above reasons and given the patient's multiple comorbidities and the severity of her symptoms, we preferred to treat our patient with a tapering dose of prednisone 40 mg for 10 days along with a high dose of acetaminophen as needed. The patient was placed on a soft diet and was monitored for improvement of her symptoms. A significant improvement of symptoms was reported 2 days after initiating therapy.

Treatment success was based on clinical improvement of symptoms, pain scale score and the ability to swallow. The patient was discharged on the third day of hospitalisation to finish the rest of her treatment at home and follow-up with her primary care physician.

OUTCOME AND FOLLOW-UP

The patient was discharged after 3 days of hospitalisation with significant improvement of her symptoms on the second day after initiating treatment. The patient had complete resolution of her symptoms in 1 week after starting treatment.

The symptoms of acute longus colli tendinitis usually improve within 2–3 days and resolve in 1–3 weeks after initiating treatment.¹ Follow-up on response to treatment relies on improvement in the symptoms. There is no need for repeat imaging studies to confirm resolution of the disease.

DISCUSSION

The longus colli muscle is located between the atlas and the third thoracic vertebra on the anterior surface of the vertebral column and contains three parts: the superior oblique, inferior oblique and vertical fibres. The longus colli muscles on both sides of the neck are responsible for cervical flexion, ipsilateral side flexion and a certain degree of cervical rotation.

Acute calcific tendonitis of the longus colli is an inflammatory process that affects the superior oblique fibres. It was first described by Hartley in 1964.⁵ As per Ring *et al* that reported on five patients, misdiagnosis of this condition led to unnecessary treatment and in one case unnecessary biopsy.¹

The aetiology of this disease is not yet well understood. Commonly, the longus colli muscle is injured in rear end whip-lash injuries secondary to a motor vehicle accident. In the absence of trauma, it is assumed that excessive mechanical tension in the presence of a pre-existing injury to the cervical spine whether degenerative or traumatic can lead to the precipitation

of amorphous calcium hydroxyapatite crystals that trigger the inflammatory system leading to a non-infectious inflammatory reaction.⁶

The incidence of this underdiagnosed disease is unknown and was only reported in recent epidemiological studies done by Horowitz *et al* as 1.31 cases per 100 000 person-years.⁷ Usually it occurs in the third to sixth decade of life with slight female predominance.¹ Our case proves that it can present in any age.

Patients usually present with neck pain and stiffness progressing over a period of a few days as well as dysphagia, odynophagia and referred occipital headache.¹⁶ The presentation is not always typical. In our case, dysphagia (sensation of food and liquids getting stuck in the throat) was the main complaint followed by odynophagia and neck pain. In the setting of acute longus colli tendinitis, dysphagia is the result of the inflammatory reaction caused by deposition of amorphous calcium crystals leading to oedema of the retropharyngeal space that affects the nearby constrictor muscles, which play an important role in the process of swallowing by contracting on the bolus and propelling it down into the oesophagus.⁶ On physical exam, there may be limitation of neck movement, neck muscle spasm and low grade fever. In addition, the haematological laboratory tests may show mild elevation of the ESR, elevation of CRP and mild elevation of white blood cell count.

A careful physical examination is required to rule out life-threatening pathologies including retropharyngeal abscess, meningitis, epidural abscess, cervical artery dissection, cervical osteomyelitis or cervical disc herniation.

The high resolution contrast enhanced CT scan is the imaging modality of choice to diagnose acute calcific tendonitis of the longus colli muscle. Typical radiological findings on CT are prevertebral soft tissue swelling and amorphous calcification anterior to C1-C2 (figures 1 and 2). Prevertebral tissue thickening can extend as low as C6.² Subtle calcifications within the tendon can be missed on lateral plain radiographs.¹⁸ MRI with high contrast resolution has higher sensitivity in detecting soft tissue and retropharyngeal oedema and lower sensitivity in detecting prevertebral calcifications as compared with a CT scan.⁹ In our case, since the CT scan done in the emergency department was without contrast, we needed confirmation by MRI with contrast to finally differentiate between retropharyngeal oedema and retropharyngeal abscess.

Learning points

1. Acute calcific tendonitis of the longus colli muscle usually presents with neck pain and neck stiffness progressing over a period of few days followed by dysphagia, odynophagia or the other way around as shown in our case.
2. Usually it occurs in the third to sixth decade of life with slight female predominance.¹ Our case proves that it can present in any age.
3. Awareness of existence of longus colli tendonitis and recognition of its radiological features are key for appropriate diagnosis of this condition. This in turn would obviate unnecessary diagnostic procedures and inappropriate treatment.
4. Consider treatment with systemic steroids in patients with chronic kidney and cardiovascular disease or other situations where using non-steroidal anti inflammatory drugs are contraindicated.

The symptoms of acute longus colli tendinitis usually improve within 2–3 days and resolve in 1–3 weeks after initiating treatment. Follow-up on response to treatment relies on improvement in the symptoms. There is no need for repeat imaging studies to confirm resolution of the disease.^{1 9}

The first-line treatment for acute calcific longus colli tendinitis is with NSAIDs and rest, but steroids can be used in patients with severe neck pain, refractory pain and/or in cases where NSAID use is contraindicated.^{1 6 10} Given our patient's comorbidities and CKD, we decided to treat her with a short course of steroids.

Recognition of the anatomy of the longus colli muscle, awareness of the existence of longus colli tendinitis and recognition of its radiological features are key for appropriate diagnosis of this condition. This in turn would obviate unnecessary diagnostic procedures and inappropriate treatment.

Contributors HAD encountered this case while admitting and hospitalising this patient. Upon identifying the case as a potential paper, HAD consulted Dr FB to help research this case. As a joint effort, Dr FB and HAD researched similar cases to distinguish the case, phrased the case clearly and finalised the manuscript to be reviewed. To receive an objective and positive criticism to the authors' work, all authors consulted Drs CS and AS to review the work and provide the authors with sufficient feedback. Drs CS and AS reviewed and proofread the paper and handed it back to FB and HAD with scientific and language feedback.

Competing interests None declared.

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