NSAIDs Regimen in the Management of Acute Longus Coli Calcific Tendinitis

Abstract

Acute calcific longus colli tendinitis, which is also known as retropharyngeal tendinitis or acute calcific prevertebral tendinitis, is a reactive self-limiting inflammatory response to the acute or subacute deposition amorphous calcium hydroxyapatite crystal on the tendon of the longus colli muscle anterior to C1-C2 disk space. A 53 year old male presented with few days of neck pain and odynophagia. Blood work showed mild leukocytosis and elevated CRP CT scan showed mild edematous prevertebral thickening involving the retropharyngeal space, predominantly on the left side with no appreciable surrounding peripheral enhancement. These findings are associated with small amount of linear calcifications/ossifications involving the superior fibers of the left longus collis muscle anterior to C1 arch. These findings could be mistaken for retropharyngeal abscess; however the symptoms improved subsequently on NSAIDs. The aim of this study was to present the characteristic radiological features of retropharyngeal calcific tendinitis, since it’s basically a radiological diagnosis, in order to help clinicians clinch an accurate diagnosis.

Keywords: Neck pain; Odynophagia; Acute longus colli calcific tendinitis; Acute calcific prevertebral tendinitis; Retropharyngeal tendinitis

Introduction

Acute longus colli calcific tendinitis, which is also known as retropharyngeal tendinitis, is self-limiting condition resulted from deposition amorphous calcium hydroxyapatite crystal typically anterior to C1-C2 disk space, however it can be seen at different levels (C4-C5 or C5-C6). The clinical presentation is usually mistaken with other serious conditions, such as retropharyngeal abscess, disk herniation, neck tumor or trauma. Symptoms include neck stiffness, odynophagia, limitation of rotational cervical ROM, mild fever, trismus and/or neck pain/swelling. It is presumed to arise from a noninfectious inflammatory response of the superior oblique tendons of the longus colli muscle secondary to the calcium hydroxyapatite crystal deposition. The arrangement of these muscle tendons, which extend from upper mediastinum at the level of T2 to the anterior tubercle of Atlas vertebra, makes these muscles in the vicinity of prevertebral cervical space.

The importance of recognizing this pathology lies in preventing the misdiagnosis of retropharyngeal abscess, which could result in unnecessary surgical intervention rather than conservative measures.

Case Report

A 53 year old gentleman, presented to emergency with sudden onset progressively worsening of left sided neck pain and odynophagia that didn't improve initially on flexrill (cyclobenzaprine 10mg TID), Percocet and Cefixime 400mg BID for a week course, he revisited ED after 2 days with same issue and enhanced CT scan done. It demonstrate small amount of linear calcifications/ossifications involving the left longus collis muscle adjacent to C1 arch as well as the base of the odontoid process of C2 (arrows at Figure1 A,B & C). No evidence of adjacent intervertebral disc space narrowing or bony destruction to suggest underlying diskitis changes. Slightly more inferiorly, there is mild prevertebral thickening and edema involving the retropharyngeal space predominantly at the left side (Figure 2A), which tapers smoothly inferiorly at C3/C4 level. There is obliteration of the left para-pharyngeal space with mild adjacent mass effect on the nasopharynx anteriorly and the ipsilateral carotid sheath vessels laterally. No evidence of venous thrombosis demonstrated within the internal jugular veins bilaterally. The described edematous changes extends to the right retropharyngeal space as well, with no central gas locules or surrounding peripheral enhancement demonstrated to suggest underlying phlegmon or abscess formation (arrows at Figure 2B).

At this point the patient was prescribed NSAIDs (Naproxen) and our service was consulted to rule out any acute surgical condition. His main issue when we saw him was the left sided rotational neck movement, and his odynophagia got better with pain control regimen, no airways symptoms or constitutional symptoms. On examination there was no neck masses/swelling, limited neck rotational ROM especially too left turn. No pain in flexion or extension. Oral cavity and oropharyngeal within normal limits. No vocal folds are within normal limits. Mild elevation of WBCs 13.74...
x10^9/L and C-reactive protein 8.22 mg/L with negative blood cultures and no other laboratory abnormalities. And based on the CT scan results, he was diagnosed with acute longus colli calcific tendinitis and was treated conservatively with Ibuprofen (NSAIDs) as needed for 10-14 days course. Patient was seen by his family physician in 2 weeks with complete resolution of neck pain but with residual left shoulder pain for which he scheduled him for US study.

Figure 1: Axial (A), coronal (B) and sagittal (C) enhanced CT neck at the bone window, left to right, which demonstrates linear calcifications “white arrows” adjacent to C1 arch as well as the odontoid process of C2. Note that there is no evidence of adjacent vertebral body bony destruction or intervertebral disc space narrowing demonstrated to suggest underlying diskitis.

Figure 2: Sagittal (A) and axial (B) enhanced CT neck at soft tissue window, left to right, which demonstrate mild prevertebral thickening with edema “DOTTED white arrows” measuring up to 1.1 cm in thickness. There is mild obliteration of the left para-pharyngeal space with mild adjacent mass effect on the nasopharynx and the left carotid sheath vessels laterally “smooth white arrow”. No evidence of peripheral enhancement or venous thrombosis demonstrated to suggest underlying infection.
Discussion

Acute longus colli calcific tendinitis is a reactive self-limiting inflammatory response to the acute or subacute deposition amorphous calcium hydroxyapatite crystal on the tendon of the longus colli muscle at the anterior C1-C2 disk space or different spaces (C4-C6) [1-3]. In 199, Ring et al. [4] described 5 cases of calcific retropharyngeal tendinitis and biopsies of these lesions showed an inflammatory response to hydroxyapatite crystal deposition [4].

This pathology with no ethnic over expression and usually affects young adults between 30 and 60 years of age and the range distribution reported between 21 to 80 year old [5]. There is no specific etiology revealed but some of the postulated mechanism involve excessive mechanical trauma in conjunction with an existing degenerative cervical spine disease, collagen vascular disorders, renal failure, or osteoarthritis can result in the deposition of calcium crystals in the muscle tendons [6,7].

In a literature review done by Park et al, they found the most common symptoms were neck pain in 94%, limited neck range of motion and odynophagia in 45%, neck stiffness in 42%, dysphagia in 27%, sore throat in 17% and neck spasm in 11%. Laboratory findings include a slightly elevated white blood cells count, erythrocyte sedimentation rate, and C-reactive protein [6,8,9].

CT scan is the gold standard for diagnosing this condition as it can detect both the prevertebral edema and calcium hydroxyapatite crystals deposition at the longus colli tendon, in contrast to MRI, which can show the prevertebral edema and fluid effusion but not the calcific deposits, which makes CT scan more sensitive than prevertebral calcification than MRI. Furthermore, most sensitive radiological test for distinguishing retropharyngeal abscess from retropharyngeal tendinitis is the CT scan [4,8,10,11]. The typical radiological findings are calcific deposits anterior to C1-C2 and prevertebral soft tissue swelling. Although diffuse prevertebral soft tissue thickening extends from C1 to C4, it can extend as inferiorly as C6 [12].

Calcific retropharyngeal tendinitis is a self-limiting, benign disease that rarely requires admission, and successful treatment can be achieved by NSAIDs and supportive care. Usually patient will notice an instant improvement in 48-72hrs but sometimes recovery could take up to 2 weeks, while calcium deposit should resolve in a couple of weeks. If symptoms are sever, a course of corticosteroid can be implemented [13,14].

Conclusion

In conclusion, this disease is thought to be a self-limiting condition that resolves spontaneously after 1-2 weeks with anti-inflammatory medications, analgesics and rest. For that, accurate diagnosis should be made, invasive examinations and surgical intervention should be avoided and cautiously rule out life-threatening condition especially retropharyngeal abscess.

References