Retropharyngeal calcific tendinitis is an under-recognized cause of acute cervical pain produced by inflammation of the longus colli muscle. The clinical presentation includes neck pain and tenderness, limitation of motion, dysphagia, occasional mild fever, leukocytosis, and elevated erythrocyte sedimentation rate. This condition may mimic more serious disorders of the prevertebral and retropharyngeal region and may be misdiagnosed as retropharyngeal abscess, traumatic injury, or infectious spondylitis [1].

The diagnosis can be established radiographically by identification of pathognomonic findings of amorphous calcification anterior to C1-2 with associated asymmetric soft tissue swelling [2]. We present a patient with retropharyngeal calcific tendinitis and typical radiographic features in order to familiarize the physician with this condition.

**PATIENT DESCRIPTION**

A 28 year old woman with no underlying medical conditions was admitted with a 2 day history of a painful stiff neck, with limitation in the range of motion, as well as odynophagia and dysphagia. Her symptoms had begun suddenly and there was no associated fever or recent illness, and no history of fall or trauma. The patient denied shortness of breath or dyspnea.

On examination, the patient was alert, oriented, and appeared to be non-toxic. Her vital signs were within normal limits, but her neck was stiff and tender to palpation. There was reduced active range of motion in all directions and very mild cervical lymphadenopathy on the right lateral aspect of her neck. No meningeal signs were seen and she denied numbness or paresthesias in her neck or upper extremities.

Otolaryngology examination including flexible nasopharyngoscopy was normal. Laboratory tests revealed a slightly elevated white blood count of 11.6 with a slightly increased neutrophil count of 79%.

Based on a suspicion of retropharyngeal abscess, the patient was treated with intravenous antibiotics (amoxicillin/clavulanate potassium), and pain control with intramuscular Voltaren® (Novartis Pharma, Switzerland). Her symptoms subsided within a few hours.

A computed tomography scan of the neck identified an amorphous calcification anterior to C1, C2 and abnormal thickened prevertebral soft tissues secondary to a prevertebral effusion [Figure]. There was no suggestion of an abscess. These findings led to the diagnosis of retropharyngeal calcific tendinitis.

**COMMENT**

Acute retropharyngeal calcific tendinitis, also known as calcific prevertebral tendinitis and calcific tendinitis of the longus colli, is a clinical syndrome that was initially described by Hartley in 1964 [3].

The condition is a form of calcium hydroxyapatite deposition disease. It is caused by calcium hydroxyapatite deposition in the longus colli tendon and muscle. The longus colli tendon is an uncommon location for this disease, which typically involves large joints, especially the shoulders [3,4]. The lon-
gus colli muscle tendon is located on the anterior surface of the vertebral column extending from the atlas to the third thoracic vertebra. The calcium hydroxyapatite deposits invoke a painful inflammatory response with symptoms such as increasing neck pain, dysphagia and odynophagia. The pain is aggravated by head and neck movement.

Due to its rather non-specific presentation and rare occurrence, this condition can easily be overlooked or misdiagnosed. Knowledge of the characteristic clinical symptoms and imaging features of this disorder are crucial for a correct diagnosis of this uncommon cause of odynophagia and dysphagia [5].

The diagnosis is readily made with X-ray. The principal radiographic findings include amorphous calcification anterior to C1–C2, with associated swelling of the prevertebral soft tissues from C1 through C4, although it can extend as inferiorly as C6 [2]. CT, with its greater contrast resolution, is a more sensitive technique for the detection of the amorphous calcification, which can often appear quite faint on plain film.

Retropharyngeal calcific tendinitis is a self-limiting condition. Pain reaches a maximum at 2–5 days, and then gradually resolves after 1–2 weeks. Treatment with a short course of non-steroidal anti-inflammatory drugs rapidly alleviates symptoms and neck pain. Follow-up with plain films typically shows complete resolution of the characteristic findings [3].

References

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Capsule

Self-renewing macrophages without transformation have therapeutic potential

The capacity for self-renewal is associated with progenitor cell populations and is lost upon differentiation. Aziz et al. discovered an exception to this rule when mouse monocytes and macrophages deficient in the transcription factors MafB and c-Maf were cultured in the presence of the growth factor, macrophage colony-stimulating factor (M-CSF). Under these conditions, MafB/c-Maf-deficient cells were able to divide continuously while maintaining the phenotype and function of mature cells. Unexpectedly, when these in vitro-cultured cells were transferred into mice, they did not induce tumors, despite continuing to divide, but rather incorporated themselves into tissues and adopted normal macrophage functions. Suppression of two genes involved in the self-renewal capacity of inducible pluripotent stem cells, KLF4 and c-Myc, inhibited the ability of MafB/c-Maf-deficient macrophages to self-renew. Such long-term propagation of a differentiated cell population that does not result in transformation when reintroduced in vivo has exciting therapeutic potential.

Science 2009; 326: 867
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Capsule

Sleep and Alzheimer’s disease

Accumulation of amyloid-β (Aβ) in the brain is thought to be the initiating event in the pathogenesis of Alzheimer’s disease (AD). Aβ is a peptide secreted in a soluble monomeric form predominantly by neurons and its aggregation into toxic forms is concentration dependent. Somatic activity regulates the release of Aβ in vivo. However, how physiological and environmental processes are involved in regulation of Aβ levels is not understood. Kang et al., by performing sleep-wake studies in freely behaving animals concomitant with in vivo microdialysis, found that brain interstitial fluid levels of Aβ were significantly correlated with wakefulness and negatively correlated with sleep. Furthermore, relatively short-term (3 weeks) sleep deprivation markedly accelerated amyloid plaque deposition in amyloid precursor protein transgenic mice. Thus, sleep-wake behavior is linked to Aβ levels and abnormal sleep may be linked to AD pathogenesis.

Science 2009; 326: 1005
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“If you can’t explain it simply, you don’t understand it well enough”
Albert Einstein