Acute prevertebral calcific tendinitis: A nonsurgical cause of prevertebral fluid collection

Todd A. Kupferman, MD; Clifford H. Rice, MD; Linda Gage-White, MD, PhD

Abstract
Calcific tendinitis of the prevertebral muscles is a rare clinical entity. Its nonspecific presenting symptoms (e.g., retropharyngeal space infection) may mimic an infectious etiology. Treatment is based on the administration of a nonsteroidal anti-inflammatory drug (NSAID) for 2 to 3 weeks and cessation of symptom-provoking activity. Most patients will improve greatly within the first 48 to 72 hours after the initiation of an NSAID, and complete resolution generally occurs within 3 weeks. We describe a new case of prevertebral calcific tendinitis, and we review the literature on this condition.

Introduction
Calcium deposition within a muscle tendon exposed to repetitive trauma can lead to symptoms of tendinitis. The tendons most often affected by calcification are in the shoulder, but calcification can develop in any tendon about an articulation.1-4 In cases that involve the cervical paraspinal musculature, patients may present with nuchal rigidity, neck pain, odynophagia, and low-grade fever.2,5 Because these symptoms are rather nonspecific—for example, they may accompany a deep neck space infection, neoplasm, etc.—radiologic imaging must be obtained to distinguish calcific tendinitis from other possible clinical entities.5

In this article, we describe a case of acute calcific tendinitis that occurred in the prevertebral muscles—a circumstance that posed a diagnostic dilemma. This report is intended to provide the clinician with information on making a proper diagnosis based on this case presentation and to present a succinct review of the literature.

Case report
A 34-year-old man presented to our emergency department with complaints of a stiff neck and odynophagia. His symptoms had begun suddenly; they first manifested when the patient awoke in the morning 36 hours prior to presentation. They were characterized by intense pain on swallowing and movement of the neck; in the latter case, the pain was present only in the setting of active or passive neck motion in any direction. The pain did not radiate. The patient denied trauma to the area and any history of similar episodes. He also denied photophobia, headache, myalgias, and other prodromal symptoms. Also of note, he had no dysphagia, otalgia, hoarseness, voice changes, shortness of breath, or chest pain.

The patient’s medical history was negative for arthritis and other possibly contributory illnesses, hospitalizations, and surgery. He was taking no medication, and he had no known drug allergies. His social history was significant for smoking 1 pack of cigarettes per day for the previous 16 years and for occasional alcohol use; he denied any illicit drug use. The patient reported that he had been employed as an electrician for the previous 16 years and, as such, he spent a considerable amount of time bending his neck to look upward while working. The family history was noncontributory.

The patient was alert, oriented, and appeared to be non-toxic. Other than an oral temperature of 101.0°F, his vital signs were within normal limits. He was uncomfortable lying on his back, but he exhibited no audible stridor, and his voice was normal. There were no external signs of trauma. The results of eye, ear, nose, oral cavity, and cranial nerve examinations were normal. Minimal erythema was noted in the mucosa of the posterior pharyngeal wall. Examination of the neck revealed nuchal rigidity with pronounced pain on both passive and activemotion. No palpable lymphadenopathy was noted, and no masses were detected. Findings on cardiovascular, pulmonary, abdominal, and neurologic examinations were normal, including negative Kernig’s and Brudzinski’s signs. Laboratory findings were significant for an elevated white blood cell count (15,000/mm³) and an elevated C-reactive protein level (7.0 mg/L; normal:
The erythrocyte sedimentation rate was normal. A lateral cervical spine roentgenogram revealed a loss of natural lordosis, a thickening of the retropharyngeal soft tissues from C1 through C7 that measured approximately 11 mm anterior to the body of C2, and an amorphous mass anterior to the body of C2 in the substance of the prevertebral space (figure 1). Contrast-enhanced computed tomography (CT) of the neck detected a non-ring–enhancing fluid collection within the prevertebral space anterior to the bodies of C1 through C7 (figure 2, A). A dense calcification was seen within the prevertebral musculature at the level of C2 (figure 2, B). Flexible nasopharyngoscopy revealed a slight fullness and mild erythema in the posterior pharyngeal wall; otherwise, findings were normal (figure 3).

Based on the history, physical examination, and radiologic findings, acute prevertebral calcific tendinitis was placed at the top of the differential diagnosis because this condition, although rare, typically manifests in this fashion. However, an infectious etiology could not be entirely ruled out. Rather than performing an intraoperative incision and drainage of a possibly sterile fluid collection and risk exposing the retropharyngeal, “danger,” and prevertebral spaces to oropharyngeal flora, we admitted the patient for close observation and treated him with an intravenous non-steroidal anti-inflammatory drug (NSAID) and intravenous broad-spectrum antibiotics. Within 1 hour of receiving the NSAID, he reported a significant alleviation of his neck pain. By the next morning, his nuchal rigidity had resolved and the amount of pain with passive and active neck motion had decreased markedly.

Over the next few days, the patient’s white blood cell count, C-reactive protein level, and fever trended downward. Repeat CT with intravenous contrast performed 24 hours after admission showed only a slight reduction in the fluid collection. But more important, it still showed no ring enhancement around the fluid collection, which reinforced our decision not to perform surgery. On hospital day 3, the patient was discharged home on a 2-week course of an NSAID. In addition, because this is such a rare diagnosis, he was given a 2-week prescription for a third-generation penicillin. At the 4-week follow-up, he was asymptomatic and doing well.

Discussion

Acute prevertebral calcific tendinitis is rare, as fewer than 40 cases have been reported in the medical literature. The age of previously reported patients ranged from 21 to 81 years; most were between 30 and 60 years. No predilection for either sex has been noted. Typically, 2 to 7 days passed before these patients sought medical attention. The most common presenting symptoms were nuchal pain with an associated limitation of neck movement and dysphagia or odynophagia. Patients typically had no systemic signs of toxemia, although fever was occasionally present. Mild leukocytosis was common, as was an elevated erythrocyte sedimentation rate. No positive throat cultures were reported. Our patient had an elevated C-reactive protein level, but this finding has not been associated with previous cases of prevertebral calcific tendinitis.

The pathogenesis of calcific tendinitis is not entirely understood. The condition is thought to result from the sudden deposition of calcium salts within the intima of blood vessels, leading to the formation of a dense calcification. This calcification is thought to be responsible for the symptoms and signs associated with acute prevertebral calcific tendinitis.
understood. One postulated cause is repetitive-use injury. Repeated insults are believed to lead to fibrocartilage transformation, possibly secondary to a hypoxemic event. Initially, focal deposits of calcium are separated by strands of fibrocartilage. This is followed by a resorptive, phagocytic phase in which mononuclear cells and multinucleated giant cells surround the fibrocartilage and calcium, accompanied by an associated inflammatory response. The symptoms seem to be associated with the resorptive phase.

The differential diagnosis of the presenting symptoms of prevertebral calcific tendinitis is extensive. The most obvious diagnostic dilemma is whether to treat such an acute clinical presentation as an inflammatory process or as an infectious process (figure 4). It would be a great disservice to a patient if a physician failed to drain an evolving abscess, just as it would be to perform an incision and drainage on a sterile effusion that had arisen secondary to tendinitis. A radiologic finding of an amorphous soft-tissue calcification in the longus colli muscle at the level of C1 or C2 is considered pathognomonic for prevertebral calcific tendinitis. The associated sterile effusion is not ring-enhancing with intravenous contrast. Given that an abscess in the early stages of formation may also be nonenhancing, a repeat CT of the neck is needed to exclude this possibility. If the fluid becomes ring-enhancing with intravenous contrast, incision and drainage should be performed, despite the presence of the pathognomonic calcification in the prevertebral soft tissues.

Treatment is based on the administration of an NSAID for 2 to 3 weeks and cessation of symptom-provoking activity. Most patients will improve greatly within the first 48 to 72 hours after the initiation of an NSAID, and a complete resolution generally occurs within 3 weeks.

If symptoms persist unabated beyond 2 weeks or if they worsen after a period of initial improvement, the patient should be promptly reevaluated.

In retrospect, the use of antibiotics in our patient was probably not necessary, but given the rarity of this disease and the potential catastrophic consequences of not treating an infection of the retropharyngeal space, we felt the better part of valor would be to err on the side of overtreatment. For the practicing otolaryngologist, the importance of recognizing this rare condition lies in the potential for avoiding an unnecessary surgical procedure.

References