Case report

Retropharyngeal tendinitis

A. KIER DC & P. McCARTHY PhD
Welsh Institute of Chiropractic, University of Glamorgan, Pontypridd, UK

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Retropharyngeal tendinitis of the longus coli muscle is a clinically significant condition that is only moderately documented in the literature. Symptoms can mimic pathologies such as meningitis or retropharyngeal abscess. Radiologically, it can also be mistaken for cervical fracture or a calcified stylohyoid ligament. It is characterized by insidious onset of severe progressive neck pain with restricted cervical range of motion. The disease is thought to resemble the same histophysiology as calcific tendinitis of the shoulder and involves the deposition of hydroxyapatite crystals. There appear to be two phases: a non-symptomatic deposition phase and a symptomatic resorptive phase. The underlying mechanism is not fully understood. Various authors have proposed that this clinical syndrome is more prevalent than previously considered.

Introduction

The pathological process underlying calcific tendinitis of the longus coli muscle is not fully understood. According to Fam *et al.* (1979), calcified peri-arthritis was first mentioned around 1870. Many names have been used to describe it, for example: 'peritendinitis calcarea', emphasizing the site; 'peri-articular apatite deposition', indicating the nature of the calcified deposit; 'calcifying tendinitis', indicating the active process and possibly explaining the deposition (Faure & Daculsi 1983); and hydroxyapatite deposition disease (HADD; Yochum & Rowe 1987). The presence of apatite in the tendon sheaths was first mentioned in the 1960s by McCarthy & Gatter (1966). Calcific tendinitis is associated with, and presumably caused by, hydroxyapatite crystal deposition in musculotendinous tissues, characteristically occurring near peri-articular tendinous insertions. A rare link to metabolic disorders such as diabetes, thyroid disease and tuberculosis has been noted, but only when the calcification occurred in the shoulder. Calcified tendinitis, although most frequently recognized in the shoulder (supraspinatus and deltoid tendons), can occur in almost any tendon, including areas such as the hip (gluteus maximus and vastus lateralis tendons), ankle (peroneus longus tendon), wrist (flexor carpi ulnaris and abductor pollicus longus tendons) and the neck (longus coli tendon) (Archer *et al.* 1992).

The objectives of the present paper are to present the characteristics of retropharyngeal tendinitis and, in so doing, increase awareness of this disorder. It is proposed that this entity may be more prevalent than previously suspected.

Correspondence: Annabel Kier DC, Welsh Institute of Chiropractic, University of Glamorgan, Pontypridd CF37 1DL, UK.

Case report

A 50-year-old female presented experiencing acute neck pain of one week in duration. The onset had been insidious, but increasing in both frequency and severity. The pain, which was sharp and continuous in nature, was experienced in the upper cervical spine with radiation into the suboccipital muscles, and down to both the right trapezius muscle and the inter-scapular region. The case history revealed no recent trauma or illness of any kind, and no previous episodes of this complaint. It was noted that the patient was experiencing great stress and emotional distress related to family circumstances. Dysphagia was not initially admitted, but confirmed upon further questioning.

On physical examination, the subject's blood pressure, pulse, temperature and neurological assessment were unremarkable. Her head was held in a slightly antalgic

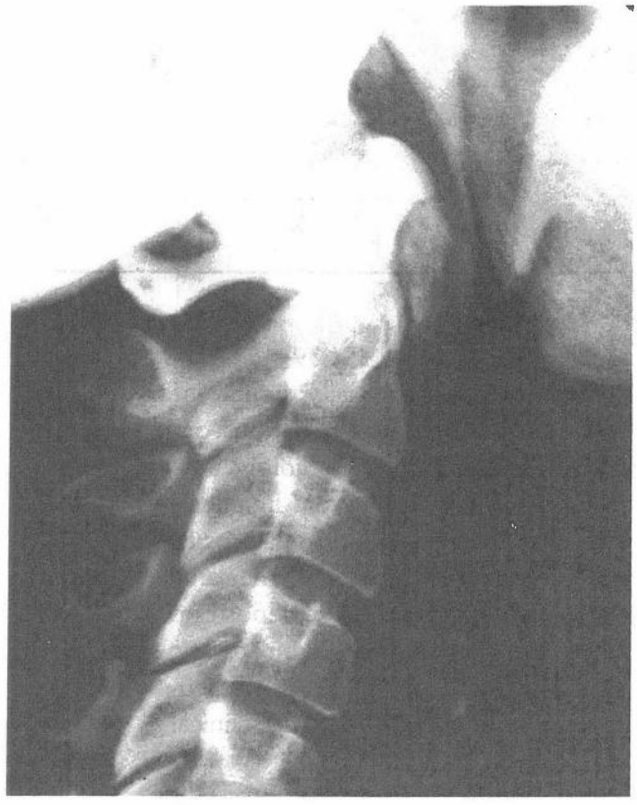

Figure 1. Radiolucent line separating the C1 anterior tubercle from a well-circumscribed amorphous calcification of the soft tissues.

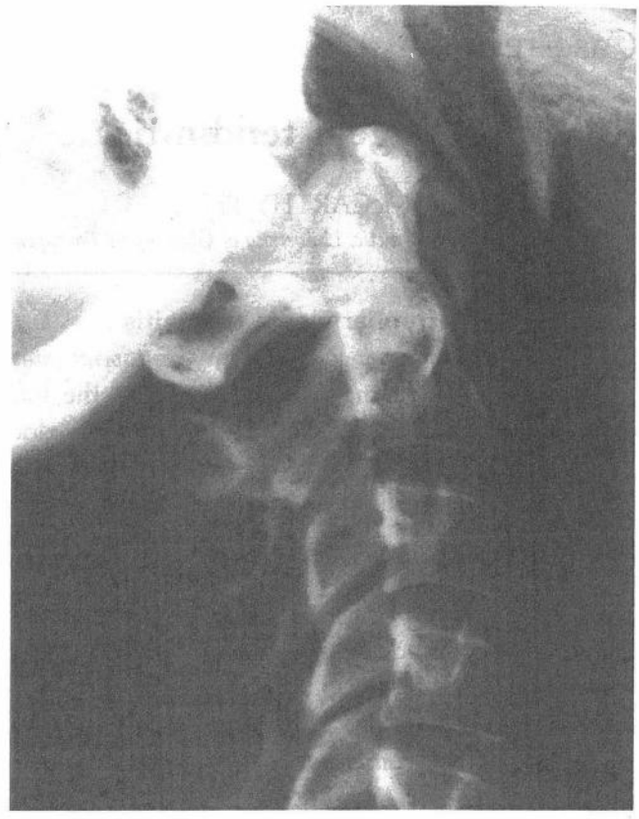

Figure 2. Calcified mass reduced in size 4 weeks after treatment.

posture to the left. All cervical ranges of motion (RoMs) were found to be severely restricted and painful. Plain-film X-rays (antero-posterior, antero-posterior-open mouth and lateral cervical views) were obtained by referral to a private hospital. A well-circumscribed amorphous calcification of the soft tissues, anterior to C1, in association with soft-tissue displacement of approximately 12 mm were noted. A radiolucent line separated the C1 anterior tubercle from this mass (Fig. 1). Interestingly, this finding was not mentioned in the initial radiological report accompanying the films. When bringing this information to the attention of the specialist in question, the suggested findings were dismissed. A working diagnosis of acute torticollis of the cervical spine (C2–3) was made. The subject's state of anxiety undoubtedly contributed towards her pain levels.

Treatment regime

The patient received five treatments. The initial three were within the first week and the latter two were spread out over the next 2 weeks. Treatment consisted of cryotherapy and Arnica (a homeopathic remedy for pain) as well as proprioceptive–neuromuscular facilitation therapy to the cervical musculature during the first visit. Subsequent visits included specific spinal manipulation of one or two of the following levels, i.e. C2, C4, C7 and T4, and soft-tissue therapy. Following the first three treatments, the subject had improved considerably with respect to both pain level and cervical RoM. By the fifth visit, the patient was asymptomatic. A retrospective radiological diagnosis of HADD of the longus coli muscle was made following further radiological consultation. Follow-up evaluations 4 weeks and 9 months later showed that she remained asymptomatic, and the subsequent lateral cervical radiographic views obtained during these visits demonstrated that the calcified mass not only reduced in size (Fig. 2), but was totally reabsorbed (Fig. 3).

Discussion

Pathophysiology and histopathology of calcific tendinitis

Calcified peri-arthritis comprises a syndrome characterized by acute peri-articular inflammation associated with transient juxta-articular radiological calcified deposits. The calcified deposits are composed of masses of

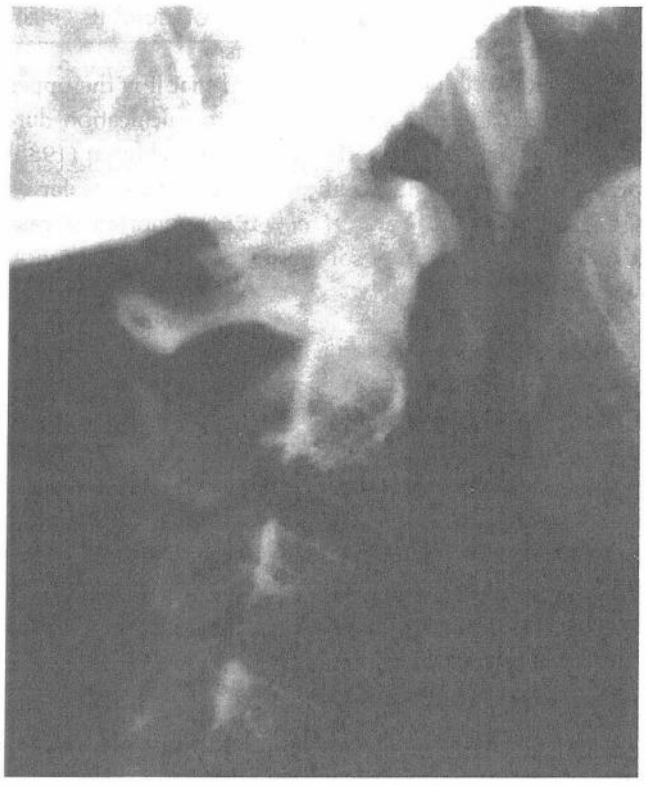

Figure 3. Calcified mass totally reabsorbed 9 months after treatment.

hydroxyapatite crystals which have been identified in synovial fluid samples despite the crystal size being very small (approximately 50–200 nm in length and 5–20 nm in width) (Fam *et al.* 1979). The actual underlying pathophysiological mechanism of the calcification and its spontaneous resorption is not completely understood, but several hypothetical mechanisms have been proposed (Archer *et al.* 1992).

One suggestion is that repetitive motion or chronic overload of the muscle insertion influences the development of calcified tendinites. This indicates a process of degeneration, i.e. necrosis and fraying of the tendon fibres. Such a change leads to formation of a fibrinoid mass with leukocytes and histocytes, which replace any necrotic tissue. Calcification follows in an unknown manner (Kaplan & Eavey 1984). Another school of thought believes that hypoxia in the muscle insertion leads to chondroid metaplasia and subsequent calcification. However, neither the typical clinical history nor the histological features of calcifying tendinites appear to suggest an underlying degenerative process.

Morphologically, evidence suggests both the formative and resorptive phases may coexist in the calcified deposit. Several of the calcified foci noted during investigation into rotator cuff tendinitis of the shoulder were partially or completely surrounded by mononuclear and multinucleated cells with phagocytosed material in the cytoplasm. Many thin-walled blood vessels were found in areas of resorptive activity, but polymorphonuclear or plasma cells were not. Uhthoff *et al.* (1976) suggested that persistent hypoxia causes parts of the tendon transform into fibrocartilage, where chondrocytes mediate the deposition of calcium at multiple foci. In the course of time, phagocytosing cells accumulate around individual foci, with concomitant vascular proliferation. The vascular channels not only provide a pathway for resorption, but also restore normal perfusion and oxygen tension to the tissue. The above authors initially postulated that, following resorption of calcium, the tendon in all probability regains its original architecture through new matrix synthesis, so that no functional impairment remains. This theory was later challenged by Faure & Daculsi (1983), who questioned the possible existence of a resorptive phase, based on the small number of cells previously observed. Josza (1980) also found no indication of necrosis or inflammation, and proposed a hypoxaemic aetiology.

Calcifying tendinitis is analogous to skeletal matrix vesicle-initiated calcification (Anderson 1988). Nodular calcific deposits are seen to occur in areas which have already undergone fibrocartilaginous metaplasia. Tenocytes in the affected regions assume the electron-microscopic features of cartilage cells and become surrounded by a metachromatic, cartilage-like matrix containing matrix vesicles. The latter undergoes hydroxyapatite mineralization, at first within and then at the surface, in a fashion closely resembling that of normal mineralization in the growth plate. Grossly, the crystals change from a granular form in the chronic phase to a milky emulsion in the acute phase (Gaartner & Simons 1990; Figler 1993). Gaartner & Simons (1990) demonstrated that the macroscopic difference in calcific deposits is not reflected in their mineralogical structure because neither a chemical composition change nor an alteration in their crystal lattice takes place. Therefore, the above authors concluded that no chemical dissolution process of the inorganic material is responsible for the resorption activity in the acute phase. The bonding of all hydroxyapatite deposits seems to be governed by organic constituents, as indicated by the results of spectroscopy. The histopathological nature of retropharyngeal calcified tendinitis is uncertain because of a lack of biopsy material and the self-limiting nature of the disease. However, it is generally agreed that it represents the same pathology as that seen most often in the vicinity of the glenohumeral joint (Figler 1993).

Pain patterns

There appears to be a relationship between pain onset or intensification, and the degree of resorptive activity (Pinals & Short 1966). Out of 28 patients with severe pain undergoing surgery, 21 showed histological evidence of resorptive activities in comparison with only four out of the 18 with moderate pain (Uhthoff *et al.* 1976). The pain and tenderness may be related to rupture of the calcified deposits from their sheaths causing an acute local inflammatory reaction (Bernstein 1975; Wildus 1985; Benanti *et al.* 1986).

Anatomy of the retropharynx

Regardless of the varying opinions concerning the histopathology of retropharyngeal tendinitis, all authors appear to agree that the tissue involved is the longus coli muscle of the anterior cervical spine. The anatomical possibilities for alternative tissue locations of the calcific deposits are limited. The only structures in this area which might also be considered are the anterior surface of the vertebral body, the tendinous attachments of the longus coli muscle (with only a few associated muscle fibres extending up to this level) and the mucous membrane of the posterior pharynx (Haun 1978). The retropharyngeal space lies between the bucco-pharyngeal fascia and pre-vertebral fascia, the latter consisting of the anterior longitudinal ligament and the longus coli muscle. This can be seen immediately anterior to the cervical vertebrae on a lateral cervical spinal radiograph (Benanti *et al.* 1986).

The longus coli muscle extends from the anterior tubercle of the atlas into the superior mediastinum. It is divisible into three components: inferior oblique, vertical and superior oblique. The inferior oblique section ascends laterally from the front of the first three thoracic vertebrae to the anterior tubercles of the transverse processes of the fifth and sixth cervical vertebrae. The vertical, intermediate, part ascends from the front of the bodies of the upper three thoracic and lower three cervical vertebrae to the front of the bodies of the second, third and fourth cervical vertebrae. The superior oblique part passes from anterior tubercles of the transverse processes of the third, fourth and fifth cervical vertebrae upwards, medially attaching to the anterolateral surfaces of the anterior tubercle of the atlas. The nerve supply to this muscle group derives from the ventral rami of the second to seventh cervical spinal nerves. The action of the longus coli is to bend the neck forwards in flexion; the oblique portions may be involved in lateral flexion and contralateral rotation. Its main antagonist is the longissimus cervicis.

Although Wildus (1985) suggested that it is the upper tendon insertion that appears prone to calcification during the tendinitis, it is worth noting that Fahlgren (1988) presented a case with calcification descending as far as C4–5. In addition, Yang *et al.* (1986) reported a case involving calcification of soft tissue anterior to all cervical vertebrae.

Radiological findings

Yochum & Rowe (1987) described the calcification as 'an amorphous calcification up to 20 mm in diameter anterior and inferior to the anterior tubercle of the atlas'. The calcification occurs in the retropharyngeal interspace and leads to soft-tissue swelling. The normal pre-vertebral width at C2 on a lateral cervical X-ray is <5 mm (Benanti *et al.* 1986); however, in the presence of calcific deposits, Artenian *et al.* (1989) reported up to 16 mm and Haun (1978) reported 20 mm. In addition to the radiological features, the lordosis can often be diminished as a result of the associated muscle spasm.

Chronic calcification tends to have a 'harder' appearance with more well-defined and somewhat denser margins than the 'softer' amorphous pattern associated with symptomatic calcified deposits (Haun 1978; Gaartner & Simons 1990).

Literature review

To the best of the present authors' knowledge, there are no more that 40 cases of retropharyngeal calcification reported in the English literature (standard Medline search with keywords). Kogstad *et al.* (1989) reported finding 60 cases, indicating the presence of reported cases in the Scandinavian literature which previously were not taken into account.

Attention was first drawn to this particular entity by Hartley (1964). The presentation of retropharyngeal tendinitis is characterized by the sudden onset of acute neck pain with restricted cervical RoMs and an amorphous calcified deposit of varying size is noted on a lateral cervical X-ray anterior to the atlanto-axial joint (Table 1).

The average age of onset ranges from 30 to 60 years (Artenian *et al.* 1989; Figler 1993), even though Benanti *et al.* (1986) reported a much wider age range (6–81 years). There appears to be no gender predisposition.

The usual course of retropharyngeal calcified tendinitis is self-limiting. With treatment, the acute phase lasts no

Table 1. Signs and symptoms noted in the literature

- Severe cervical pain, which may radiate to the occipital, inter-scapular and/or shoulder regions
- Severely restricted and painful cervical ranges of motion
- Insidious onset, sudden and progressive (2–7 days)
- History of trauma or recent illness is not a factor
- Throat culture negative
- Blood tests often normal; however, mild leucocytosis and mildly elevated erythrocyte sedimentation rate have been noted
- Normal or mildly elevated oral temperature
- Mild to severe dysphagia/odynophagia
- Nuchal pain and spasm
- No muscular wasting or fasciation
- Hypertonicity of the cervical musculature (both anteriorly and posteriorly)
- Little or no cervical lymphadenopathy
- Retropharyngeal soft tissue swelling at C1–4 may be present on X-ray
- Amorphous calcification anterior to C1–2 visible on plain lateral cervical X-ray in most cases

more than 7–14 days. All cases reported in the literature were eventually treated conservatively with non-steroidal anti-inflammatory drugs, at times combined with analgesics and immobilization of the cervical spine with a soft collar. The use of local corticosteroid injections should be judged according to the perceived risk of infection and local necrosis (Uhthoff *et al.* 1976). The calcification will most likely be reabsorbed within approximately 2 months (Weinberg *et al.* 1982), but cases have been reported where chronic asymptomatic retropharyngeal calcification was noted and no treatment was apparently warranted.

Careful case history taking may reveal a previous trauma or acute episode of insidious neck pain (Newmark *et al.* 1981; Warrington & Palmer 1983). It is important to emphasize that the onset of one reported case followed one hour after a chiropractic cervical manipulation (Kogstad *et al.* 1989). No specific explanation was given for the underlying pathological reactions, but the authors suggested that the patient may have had a calcific deposit prior to the chiropractic treatment. Therefore, the cervical manipulation could have released crystals, and thereby, caused an inflammatory reaction.

There was a noticeable pathognomonic calcification with possible soft-tissue swelling on the lateral cervical X-ray of the present subject. Calcification may have well-defined margins early in the course of the inflammation, but it becomes less well defined as the deposit is reabsorbed (Benanti *et al.* 1986). In some cases, only the soft-tissue swelling is visible since the calcification is not dense enough to be apparent on routine radiographs. The lack of demonstrable soft-tissue calcification should not detract from this diagnosis. In these cases, the utilization of computed tomography (CT) is helpful because CT has superior contrast resolution and axial display (Hall *et al.* 1986). The calcification tends to lie slightly off the midline, which is consistent with the superio-medial course of the superior oblique portion of the longus coli muscle (Artenian *et al.* 1989). It is important to recognize the signs and symptoms of this disease, and take the appropriate steps in the diagnostic phase. This will reassure the patient that she or he has been given the correct diagnosis while unnecessarily risky, expensive and painful procedures (e.g. antibiotics, hospitalization, biopsies or lumbar punctures) can be avoided (Benanti *et al.* 1986; Hall *et al.* 1986; Newmark *et al.* 1986; Archer *et al.* 1992).

It is of interest to note that Benanti *et al.* (1986) reported a retrospective diagnosis of retropharyngeal tendinitis 3 years after a number of patients had been discharged. These patients spent between 14 and 20 days in hospital. Although apparently uncommon, the possibility of recurrence has been reported (Haun 1978).

Differential diagnosis

The presentation of retropharyngeal tendinitis can mimic several conditions with respect to radiological findings, and signs and symptoms. Hall *et al.* (1986) emphasized that presentation with possible elevated erythrocyte sedimentation rate, mild leucocytosis and mild fever should raise suspicion of retropharyngeal infection. The differential diagnosis should also include potentially more serious conditions such as: meningitis, epidural haematoma, retropharyngeal cellulitis/abscess, streptococcal infection of the throat, mononucleosis, infectious spondylitis, traumatic or atraumatic disc hernation, subluxation, and acute torticollis.

With respect to the radiological findings, these have to be differentiated from the following: an accessory ossiclem, which would be present without soft-tissue swelling; and an avulsed fragment, i.e. an osseous mass rather than a calcified one, which would have a bony matrix. Spinal fracture is also a possibility, including a horizontal fracture of the anterior arch of the atlas, which may result from a hyper-extension injury to the neck (Warrington & Palmer 1983). Slight rotation of the neck on X-ray can cause the transverse process or lateral masses of the axis to project anteriorly. Styloid processes and calcified

stylohyoid ligaments can also be misinterpreted as tendinous calcification, as can an earlobe. The possibility of foreign body irritation should also be kept in mind (Weinberg *et al.* 1982; Wildus 1985; Benanti *et al.* 1986; Newmark *et al.* 1986; Artenian *et al.* 1989; Figler 1993). The fact that the soft-tissue swelling is even throughout may be used to differentiate it from the soft-tissue swellings seen in many tumours (Kulling *et al.* 1991).

Conclusion

The actual underlying mechanism of retropharyngeal calcified tendinitis is not fully understood. The assumption that it follows the same histopathology as supraspinatus tendinitis is generally accepted, as is the conviction that the tissue affected is the superior tendon of the longus coli muscle in the anterior cervical spine. An increase in the awareness and understanding of this perplexing condition can lead to fast and safe diagnosis without unnecessary risks and costs. The possibility of the use of CT in this scenario should not be overlooked.

It is important to note that there may be a possibility of provoking the acute reabsorptive phase presence of an asymptomatic or chronic calcific deposit in the cervical musculature. As such, this could contra-indicate the use of cervical manipulation; however, there is currently little evidence for or against such a possibility. Therefore, further investigation into this matter is warranted.

There is general agreement that, in all probability, this condition has been an infrequently described but consistent clinical syndrome. It is most likely more prevalent than previously suspected. In order to investigate this further, retrospective investigations will have to be performed in both the chiropractic and the hospital setting.

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