eNeurologicalSci 13 (2018) 49–50

Contents lists available at ScienceDirect

eNeurologicalSci

journal homepage: www.elsevier.com/locate/ensci

Letter to the Editor

MR T2 high image of longus colli muscle without tendinous calcification; A suggestive sign of cervical spinal infection

Dear Editor, acute calcific tendinitis of the longus colli muscle (LCM) manifests as acute neck pain and dysphagia or odynophagia [1] and shows MRI signal changes due to inflammation of the LCM with fluid collection in the prevertebral space. The diagnostic feature of this disorder is the calcification of the superior oblique tendons of the LCM [2]. We present a case displaying very similar clinical and radiological features, except with no calcification of the tendon. The patient showed discitis-osteomyelitis of the cervical vertebrae associated with inflammation of the LCM. MRI signal changes in the LCM without calcification of the tendon might represent an important sign reflecting cervical spinal infection.

1. Case report

A 46-year-old woman was referred for headache and left neck-to-shoulder pain. She had no history of trauma or ingestion of a foreign body. She showed fever (38.0 °C), but no cervical lymphadenopathy or swollen tonsils were noted. The patient described non-pulsating headache in the left occipital region and neck pain that worsened when bending head forward. Neck rigidity was evident, but differed from so-called meningeal irritation. Neurological examination revealed no other abnormalities. Laboratory examination showed increases in both white blood cell count (18,400/μl, 95.2% neutrophils) and CRP (6.15 mg/dl). CSF examination showed normal levels of protein, glucose and cells.

Odynophagia appeared on day 3 after admission, and chest pain appeared on day 5. Chest CT showed infiltrative shadows in both lungs, accompanied by pleural effusion. Methicillin-sensitive *Staphylococcus aureus* (MSSA) was isolated from cultures of both venous blood and catheter urine collected on day 2. Septicemia accompanied by pulmonary edema was therefore diagnosed. Intravenous aminobenzylpenicillin (ABPC, ampicillin)/sulbactam (SBT) at 4.5 g/day was administered for 10 days, and high-dose methylprednisolone (mPSL) was started at 1 g/day for 3 days.

MRI and MRA of the head were unremarkable. Cervical MRI on day 3 revealed a longitudinal signal change, appearing as hypointensity on T1-weighted imaging and hyperintensity on short tau inversion recovery (STIR) imaging, in the soft tissue of the prevertebral space anterior to C2–C4. This lesion was suspected to represent acute calcific tendinitis of the LCM, but no calcification was observed in the tendon of LCM anterior to C1–C2 on 3-dimensional CT (Figs. 1A–C).

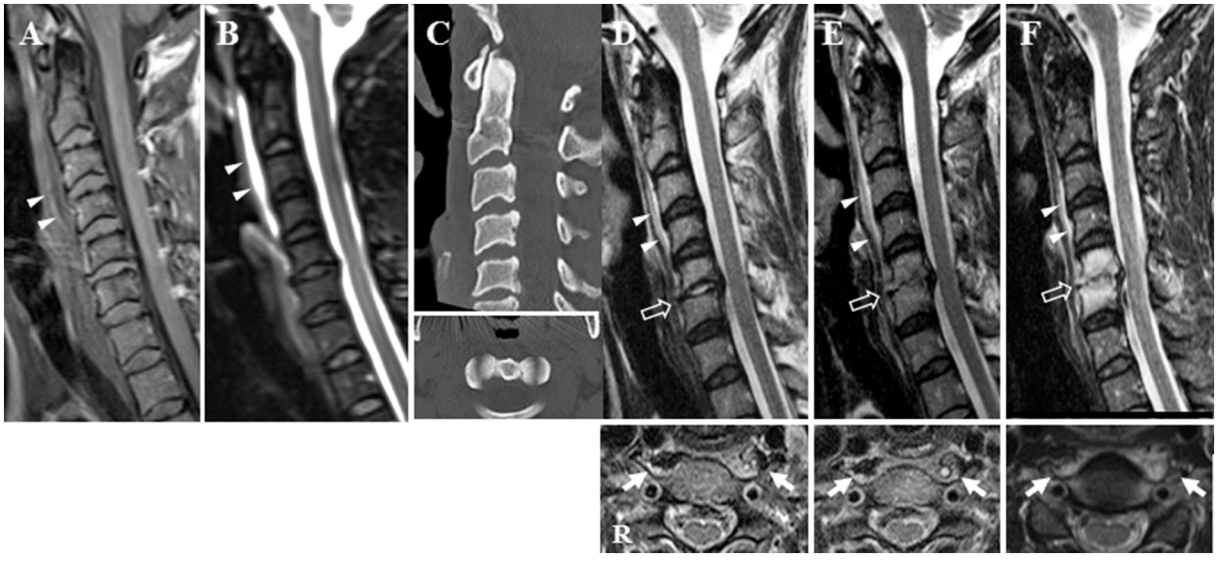

Fig. 1. MR and CT images of the cervical region. A–C) On day 3, cervical MRI reveals a longitudinal signal change, appearing as hypointensity on T1-weighted imaging (A) and hyperintensity on short tau inversion recovery (STIR) (B), in the soft tissue of the prevertebral space anterior to C2–C4 (arrowheads). No calcification was observed in the tendon of the LCM anterior to C1–C2 on 3-dimensional CT (C). D–F) T2-weighted images on day 10 (D) and day 16 (E), and fat-saturated T2-weighted image on day 26 (F) reveal hyperintense signals in both LCMs (arrows), soft tissue (arrowheads) and vertebral bodies of C5 and C6, along with bone destruction, and narrowing of the C5/C6 intervertebral disc (open arrows).

https://doi.org/10.1016/j.ensci.2018.08.002
Received 10 July 2018; Accepted 20 August 2018
Available online 23 August 2018

On day 10, T2-weighted imaging revealed hyperintensity of both LCMs, higher intensity on MRI in the left muscle than in the right. Edematous regions of soft tissue and fluid collection gradually improved, and neck pain also resolved. On day 16, however, MRI showed narrowing of the C5/C6 intervertebral disc, and signal hyperintensity and destruction of the C5 and C6 vertebral bodies was evident (Figs. 1D–F). These findings indicated pyogenic spondylitis (discitis-osteomyelitis) of C5 and C6. From day 16, 750 mg/day of ampicillin/clavulanate was orally administered for 10 days. Furthermore, oral administration of cefalexin at 2 g/day was continued for 2 months. Although these vertebral changes were still recognizable on MRI after 1 year, the patient did not show any neurological manifestations and no recurrence of spondylitis was apparent.

2. Discussion

Acute calcific tendinitis of LCM is a clinical entity that has been demonstrated to be secondary to calcium hydroxyapatite deposition in the tendon [3]. The proposed pathophysiology is rupture of calcific hydroxyapatite crystals, provoking an aseptic inflammatory response in the surrounding LCM and leading to formation of reactive fluid in the prevertebral space [4]. As this disorder is caused by a benign inflammatory process, treatment usually involves a few weeks of nonsteroidal anti-inflammatory drugs. In the present case, however, inflammation of LCM without tendinitis was induced from a pyogenic process in the prevertebral space associated with discitis-osteomyelitis of the cervical vertebrae. Intensive antimicrobial therapy was thus necessitated. With early-stage discitis-osteomyelitis, radiological changes (including on MRI) to the vertebral body or intervertebral space might be subtle, and treatment could thus be delayed in some cases. Neurological complications have frequently been reported, especially cervical discitis-osteomyelitis [5,6]. We propose that MRI signal changes, as STIR or T2-weighted hyperintensity, of the LCM without calcification of the tendon could represent a potentially important sign suggestive of pyogenic inflammation in cervical spinal infections, including cervical discitis-osteomyelitis. Ledbetter et al. recently reported that psoas T2 hyperintensity, as the imaging psoas sign, correlates closely with discitis-osteomyelitis [7]. LCM hyperintensity without tendon calcification in the cervical region might have diagnostic value similar to psoas T2 hyperintensity in the lumbar region.

References

[1] J. Hartley, Acute cervical pain associated with retropharyngeaL calcium deposit. A case report, J. Bone Joint Surg. Am. 46 (1964) 1753–1754.
[2] H. Tomita, T. Yamashiro, H. Ikeda, et al., Fluid collection in the retropharyngeal space: a wide spectrum of various emergency diseases, Eur. J. Radiol. 85 (2016) 1247–1256.
[3] D. Ring, A.R. Vaccaro, G. Scuderi, et al., Acute calcific retropharyngeal tendinitis. Clinical presentation and pathological characterization, J. Bone Joint Surg. Am. 76 (1994) 1636–1642.
[4] C.W. Hayes, W.F. Conway, Calcium hydroxyapatite deposition disease, Radiographics 10 (1990) 1031–1048.
[5] M.C. McHenry, K.A. Easley, G.A. Locker, Vertebral osteomyelitis: long-term outcome for 253 patients from 7 Cleveland-area hospitals, Clin. Infect. Dis. 34 (2002 May 15) 1342–1350.
[6] W. Zimmerli, Vertebral osteomyelitis, N. Engl. J. Med. 362 (2010 Mar 18) 1022–1029.
[7] L.N. Ledbetter, K.L. Salzman, L.M. Shah, Imaging Psoas sign in lumbar spinal infections: evaluation of diagnostic accuracy and comparison with established imaging characteristics, AJNR Am. J. Neuroradiol. 37 (2016) 736–741.

Yuji Shiga[a,*], Masaru Kuriyama[a], Keisuke Tachiyama[a], Yuhei Kanaya[a], Makoto Takemaru[a], Shinichi Takeshima[a], Kazuhiro Takamatsu[a], Yutaka Shimoe[a], Akio Tanaka[b]
[a] *Department of Neurology, Brain Attack Center Ota Memorial Hospital, Fukuyama, Japan*
[b] *Department of Radiology, Brain Attack Center Ota Memorial Hospital, Fukuyama, Japan*
E-mail address: yushiga@hiroshima-u.ac.jp (Y. Shiga)

* Corresponding author.