

Yellow Ligament Calcification Complicated by Cervical Myelopathy: A Case Report and Review of the Literature

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Abstract

Case Report

Calcification of the yellow ligament (CLJ) of the cervical spine is a rare condition, described mainly in Japanese. We report a new case of CLJ of the cervical spine causing cervical myelopathy in a 62-year-old Moroccan patient. The myelopathy was revealed by spastic tetraparesis and posterior cord syndrome. The diagnosis of spinal cord compression was made by magnetic resonance imaging (MRI). The CT scan showed massive calcification of the yellow ligament at C4-C5 compressing the right posterolateral aspect of the spinal cord. Treatment consisted of laminectomy of C4-C5 with resection of the calcified yellow ligament. The evolution was rapidly favorable, with complete regression of the symptomatology. CLJ is an extremely rare disease. Its diagnosis is generally easy, its treatment is always surgical, but its pathogenesis remains hypothetical. A review of the literature concerning this pathology is presented.

Keywords: Calcification, yellow ligament, cervical myelopathy, Imaging.

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INTRODUCTION

The first cases of myelopathy secondary to calcification of the yellow ligament (CLJ) at the dorsolumbar level were described by Polgars in 1929 [11]. It was not until the late 1970s that the first observations of cervical myelopathy due to JLC of the cervical spine were reported [4, 9]. The majority of cases were described in Japanese patients as a more or less slowly evolving cervical myelopathy.

We report a new case of cervical myelopathy due to CLJ in a Moroccan patient. The epidemiological, clinical and radiological aspects as well as the pathogenesis of this pathological entity will be discussed.

OBSERVATION

This was a 62-year-old patient, without any particular pathological history, who complained, for two months before his hospitalization, of a sensation of heaviness and weakness of the four limbs, more marked on the right side, with difficulties to perform the usual fine gestures, especially of the right hand. He did not present, on the other hand, any painful symptomatology, nor paresthesias.

The clinical examination revealed spastic tetraparesis, sharp osteotendinous reflexes in all four limbs, a bilateral Babinski sign and a posterior cord syndrome. These clinical signs were clearly more marked on the right side. The rest of the clinical examination was normal and, in particular, there were no superficial sensitivity disorders or amyotrophy. The MRI of the cervical spine showed a posterior indentation in hyposignal, both in T1 and in T2 (Figure 1). The whole is responsible for a compression of the medullary cord, at the level of C4-C5, which is the site of a T2 hyper signal in relation to a suffering. The CT scan of the cervical spine showed an oval image, spontaneously hyperdense, not changing after injection of contrast medium, and corresponding to a voluminous calcification of the yellow ligament between C4 and C5. This calcification compressed the right posterolateral aspect of the medulla (Figure 2), and appeared to be independent of the lamina of the posterior arch of C4. The biological workup was normal, especially the calcemia and the phosphoremia. The patient underwent surgery: laminectomy of C4 and C5, with resection of the calcified yellow ligament which was obviously compressive, leaving a visible imprint on the external surface of the dura mater at the end of the operation. Histological examination confirmed the calcification of the yellow ligament,

showing that the latter was the site of abundant deposits of microcrystals with square ends, refractive to polarized light, corresponding to calcium pyrophosphate crystals. The postoperative course was

simple and the evolution was rapidly favorable. Twenty-eight months after the operation, the patient was asymptomatic and the clinical examination revealed only signs of residual pyramidal irritation.

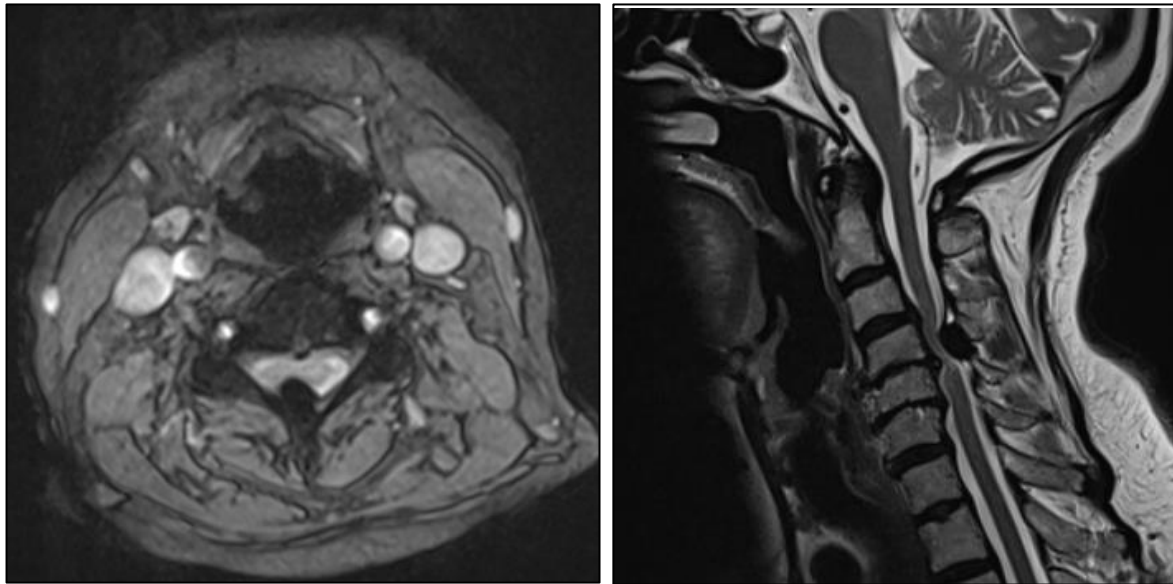


Fig 1: MRI of the cervical spine (axial and sagittal section in T2 sequence): hypertrophy and calcification of the right yellow ligament at C4-C5, responsible for compression of the spinal cord

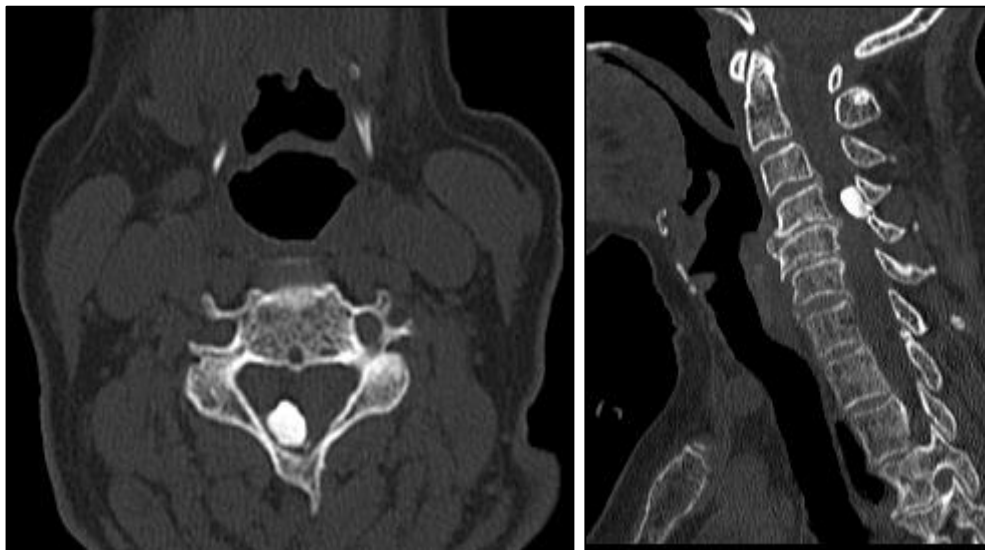


Fig 2: Spinal CT scan (axial and sagittal sections): showing calcification of the yellow ligament at C4-C5, compressing the right posterolateral aspect of the cord

DISCUSSION

Calcification of the yellow ligament of the cervical spine is an exceptional condition. Only 92 cases have been reported in the literature to date, either as isolated clinical cases or in short series [2, 8]. It is thought to be the prerogative of Japanese subjects. Indeed, only two cases have been described in Caucasians [2, 6] and seven cases in black subjects [3].

To the best of our knowledge, our observation is the first case of isolated calcification of the yellow ligament of the cervical spine reported in a Moroccan

patient. It is a pathology essentially of the woman: indeed, the epidemiological analysis of the literature finds a majority female sex ratio (79 women/13 men). The average age of onset is 65.3 years (extremes: 30-89 years) [1, 3, 7]. The usual mode of presentation is, like that of our patient, a cervical myelopathy of rapidly progressive installation, by compression of the medullary cord by the calcific masses developed in the yellow ligament. The time to onset of the disorders varies according to the series: from 6 months for Cabre *et al.*, [3] to 10.5 months in the series of Imai *et al.*, [7]. This delay is only 2 months in our observation.

The clinical symptomatology is that of a cervical spinal cord compression with a lesion syndrome in the upper limbs and a sublesion syndrome in all four limbs; however, there is usually no spinal syndrome. Cervical pain and radiculalgia are rarely reported [7]. The posterior cord syndrome found in our patient is rarely reported in the literature, which seems to be in contradiction with the location of the calcified masses that directly compress the posterior cords of the spinal cord. The interest of biology is limited: it can show at most an inflammatory syndrome (not found in our patient).

The positive diagnosis of CLJ can be made on X-rays of the cervical spine, but their profitability is inconsistent [8, 9]. The key examination is the CT scan of the cervical spine. Calcifications appear as hyperdense oval masses developing in the posterior arch, independent of the vertebral laminae. The acute angle of connection of the oval mass to the lamina and the interlaminal location differentiate the calcification from yellow ligament ossification. Indeed, the latter connects to the lamina at an obtuse angle. This distinction between calcification and ossification of the yellow ligament are important to consider because they are two different pathologies. Calcification is due to the deposition of crystals in the yellow ligament, crystals which, due to their mass effect, will cause medullary compression. Calcification is most often part of the larger condition of articular chondrocalcinosis.

Ossification of the yellow ligament, on the other hand, is a bone metaplasia that develops within a hypertrophied yellow ligament: it is an enchondral ossification leading to the formation of lamellar bone. Ossification of the yellow ligament is also a condition that almost exclusively affects Japanese subjects; it usually occurs in the low dorsal spine, causing dorsarthrosic myelopathy, but may occasionally involve the lower cervical spine [3, 5, 12].

Cervical MRI can demonstrate posterior spinal cord compression, but not calcifications, which have the same signal as a non-calcified yellow ligament. It also allows to appreciate the changes of the medullary signal on the T2 sequences, as well as a possible degenerative narrowing of the cervical canal, as it was the case in our patient, thus explaining the rapidity of the installation of the disorders. The spontaneous evolution can lead to a definitive tetraplegia in a few months [3], hence the interest of a rapid treatment consisting of a decompressive laminectomy with resection of the calcifications that are never adherent to the dura mater.

The treatment allows total recovery in the majority of cases [8, 12]. The pathogenesis of this entity remains obscure and hypothetical, apart from certain metabolic diseases [8]: the relatively advanced age of the patients argues for a degenerative component; the preferential location of CLJs in the low cervical spine,

subject to strong physical constraints, is in favor of a mechanical origin; the predominance in post-menopausal women suggests a hormonal factor; finally, the frequency of this pathology in the Japanese could have a genetic determinism [10].

CONCLUSION

Our observation of cervical myelopathy by CLJ adds to the admittedly small but increasingly reported series involving non-Japanese patients. The diagnosis of CLJ of the cervical spine is based on MRI, which determines the lesion level, and on CT scan, which directly objectifies the calcifications. The treatment is surgical, as soon as possible. The pathogenesis of this entity remains hypothetical.

REFERENCE

1. Baba, H, Maezawa, Y., Kawahara, N., Tomita, K., Furuzawa, N., & Imura, S. (1993). Dépôt de cristaux de calcium dans le ligamentum flavum de la colonne cervicale. *Spine*, 18, 2174-2181.
2. Berghausen, E., Balogh, K., Landis, W., Lee, D., & Wright, A. (1987). Cervical myelopathy attributable to pseudogout. Rapport de cas avec observations radiologiques, histologiques et cristallographiques. *Clin Orthop*, 214, 217-221.
3. Cabre, P., Pascal-Moussellard, H., Kaidomar, S., Bucki, B., Bardin, T., Smadja, D., & Arfi, S. (2001). Calcification des ligaments jaunes du rachis cervical chez l'antillais. *Revue du rhumatisme*, 68(3), 255-263.
4. Ellman, M. H., Vazquez, T, Ferguson, L., & Mandel, N. (1978). Calcium pyrophosphate deposition in ligamentum flavum. *Arthritis Rheum*, 21, 611-613.
5. Guesmi, H., Houteville, J. P., Martins, J., De Lucca, F., & Khoury, S. (1998). Myélopathie dorsarthrosique. À propos d'un cas et revue de la littérature. *Rhumatologie*, 50, 223-227.
6. Hankey, G., & Khangure, M. S. (1988). Myélopathie cervicale due à une calcification du ligamentum flavum. *Aust NZ J Surg*, 58, 247-249.
7. Imai, S., & Hukuda, S. (1994). Cervical radiculomyelopathy due to deposition of calcium pyrophosphate dihydrate crystals in the ligamentum flavum: historical and histological evaluation attendant inflammation. *J Spinal Disord*, 7, 513-517.
8. Iwasaki, Y., Akino, M., Abe, H., Tsuru, M., Tashiro, K., Miyasaka, K., ... & Ito, T. (1983). Calcification of the ligamentum flavum of the cervical spine: Report of four cases. *Journal of neurosurgery*, 59(3), 531-534.
9. Kamakura, K., Nanko, S., Furukawa, T., Mannen, T., & Toyokura, Y. (1979). Cervical radiculomyelopathy due to calcified ligamenta flava. *Ann Neurol*, 5, 193-195.
10. Matsunaga, S., Yamaguchi, M., Hayashi, K., & Sakou, T. (1999). Genetic analysis of ossification

- of the posterior longitudinal ligament. *Spine*, 24, 937-938.
11. Polgar, F. (1929). Über interakuelle Wirbelkalkung. *Fortschr Geb Röntgen*, 40, 292-298.
12. Yamagami, T., Kawano, N., & Nakano, H. (2000). Calcification du ligamentum flavum carvical. Case report. *Neurol Med Chir (Tokyo)*, 40, 234-238.