



Case Report

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Paralytic hyperalgetic lumbosciatic of tuberculosis origin: about 3 cases in West Africa-Senegal

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Abstract

Extrapulmonary tuberculosis (TB) is uncommon but not rare. Bone and joint involvement constitute about 10% of extrapulmonary TB cases, with the spine being the most frequently affected site. Spinal TB patients typically present with back pain but other constitutional or pulmonary symptoms may be absent, rendering the diagnosis difficult. Standard radiographs only speak at a late stage of the disease, hence the interest of modern imaging, in particular computed tomography (CT) and magnetic resonance imaging (MRI), which allow for early diagnosis. We report three cases of paralytic hyperalgetic lumbosciatic of tuberculosis origin. The diagnosis was based on the sectional imaging associated with biological elements of high presumption and a favorable evolution under anti-tuberculosis treatment.

Keywords: Paralytic, Hyperalgetic, Lumbosciatic, Tuberculosis.

INTRODUCTION

Lumbosciatic are very common diseases. They can be common (disc or osteoarthritis) or symptomatic (inflammatory, infectious, tumor). Their diagnosis remains clinical and is made very easy with the advent of medical imaging. The etiologies mainly of mechanical origin but can sometimes relieve infectious diseases in particular tuberculous and realize of classic table of lumbosciatic symptomatic. We report three cases of hyperalgesic and disabling fibroblasts of tuberculous origin.

CASE REPORT

Cases 1

Mr. B M, 35 years old, with no history and no notion of tuberculous contagion, was received for intense lumbar pain rated at 8 on the visual analog scale (VAS) radiating to the left lower limb and preventing walking. This picture evolved for more than a month in a context of weight loss and sweats nocturnes without true syndrome infectious.

The examination found:

- a lumbar spinal syndrom
- a L5 root syndrome
- a sign of Lasègue at 15 ° on the left

The lumbar tomodensitometry found a spondylodiscite L4-L5 with an epiduritis and paravertable abscess.

The biological assessment found a biological inflammatory syndrome with a CRP at 96 mg/L, a SV at 90-110 m/s, a predominantly lymphocytic hyperleukocytosis and polyclonal hypergammaglobulinemia. The tuberculin skin test was negative.

Under Tuberculosis quadritherapy (Rifampicin, Pyrazinamide, Ethambutol and Isoniazide) combined with analgesics (paracetamol + codeine) and tricyclic antidepressants (Amitriptyline) during the first month of treatment and vitamin therapy B1 and B6, the evolution was favorable after 12 months of treatment with

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total analgesia, weight gain.

Case 2

Mrs. T M, 26 years old, with no history but with a notion of tuberculous contagion, was admitted for severe chronic and distorting low back pain, making it difficult to stand and walk. This table evolved for 3 months without improvement under analgesics and non-steroidal anti-inflammatory associated with weight loss and a nocturnal fever with sweats.

The examination found:

- a lumbar scoliosis with left convexity
- a lumbar spinal syndrom with L3-L4 gibbosity
- a bilateral L4 root syndrom
- a positive sign of Leri on the left and on the right

Computed tomography showed spondylodiscite L3-L4 with epiduritis and diffuse paravertebral abscess.

The biological check-up showed a biological inflammatory syndrome with CRP at 66 mg/L, SV at 35-51 m/s without leukocytosis, polyclonal hypergammaglobulinemia and a positive tuberculin skin test at 22 mm.

An anti-tuberculosis treatment (Rifampicin, Pyrazinamine, Ethambutol and Isoniazid) was initiated, combined with analgesics (paracetamol + codeine + caffeine) with physiotherapy and lumbar corset.

At the end of 6 months of treatment there was a total amendment of the pain with an initial correction of the scoliosis.



Figure 1: CT scan, Spondylitis L5



Figure 2: Lumbar CT scan, spondylodiscite L4-L5 (parenchymous window)

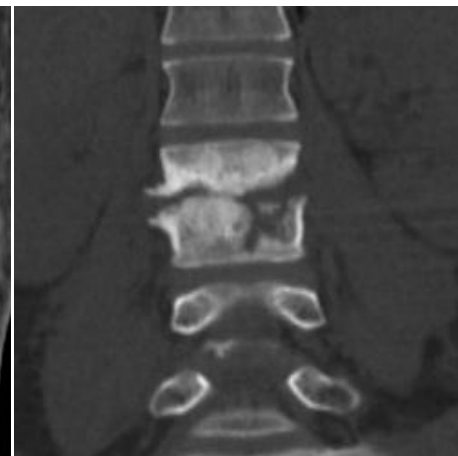


Figure 3: Lumbar CT scan, spondylodiscite L4-L5 (bone window)

DISCUSSION

In tuberculous lesions, Koch Bacillus (KB) are present both in the extracellular milieu where they multiply, and in macrophages where they are able to survive and multiply. Some of the intracellular tuberculosis bacilli are called dormant bacilli because they can survive for decades in quiescent form and reactivate with decreased immune defenses or anti-TNF- α treatment.

Vertebral tuberculosis, like other Osteoarticular tuberculosis, is a paucibacillary tuberculosis characterized by the small amount of KB in the lesions, and in which the majority of KB has a slow multiplication. It may be part of a recently acquired infection, but most often it appears to be related to the reactivation of latent tuberculosis acquired in the past, whether the initial infection was symptomatic or not^[1].

Pain along the sciatic nerve can be due to many causes. Sciatica of radicular origin caused by compression of L5 or S1 roots usually

Case 3

Mrs RM, aged 56, with a history of high blood pressure without the notion of tuberculous contagion, was received for a flaccid paraparesis with intense lumbosacral pain that gradually settled over a month in a context of vespero-nocturnal fever, anorexia and slimming.

The examination found:

- a lumbar-sacral spinal syndrom
- a flaccid paraparesis of 3/5 with mild amyotrophy without Babinski sign

The lumbar computed tomography showed spondylodiscite L5-S1 with diffuse paravertebral abscess.

Biology showed predominantly neutrophilic leukocytosis, CRP at 47 mg/L, SV at 60-90 m/s, polyclonal hypergammaglobulinemia. The tuberculin skin test was negative.

Tuberculosis treatment (Rifampicin, Pyrazinamine, Ethambutol and Isoniazide) combined with analgesics (paracetamol + codeine) and gabapentin with motor rehabilitation and physiotherapy, the evolution was favorable after 10 months of treatment with a disappearance of the pain and a resumption of the walk with a walker.

In all three cases, after 12 months of anti-tuberculosis treatment, biological and scannographic controls had normalized.

separated from truncular or peripheral sciatica. Radicular sciatica is divided into mechanical sciatica and the so-called inflammatory sciatica. In the majority of cases radicular pain is of mechanical origin and due to discal herniation in L4-L5 or L5-S1. The herniation can be visualized by lumbar computerized tomography or by radiculography. However, discal herniation alone does not fully account for the pain suffered, and inflammatory processes around the disc and the nerve root play an important role. Posterior intervertebral osteoarthritis and lumbar canal stenosis also are frequent causes of sciatic pain. Truncular sciatica is much less frequent and should incite clinicians to investigate for pelvic tumoral infiltration.

In our cases, all patients presented first pain in lumbar spine and secondly radicular sciatica. In most of publications in the spinal stenosis and herniated disc are common etiologies of radicular sciatica as in younger patients than in older patients^[2,3]. In African countries, the sciatica

pain of inflammatory schedule with fever should be investigated first, bone and joint tuberculosis^[4].

As in our cases the main symptom of vertebral tuberculosis is spinal pain, present in 80% to 95% of cases. In a recent prospective study^[5], the rachialgia schedule was inflammatory in 68 % of cases, mechanical in 16 % of cases and mixed in 11 % of cases. The general signs (asthenia, anorexia, weight loss) are present in almost 50 % of cases^[6,7]. A fever is reported in 31 % to 42 % of cases and night sweats in about 20 % of cases^[6,8].

The existence of an extravertebral localization of tuberculosis is very important to seek and its frequency is variously appreciated, from 10% to 25% depending on the series. The frequency of associated extravertebral osteoarticular tuberculosis varies from 8% to 20%^[6,9].

Neurological involvement is common in vertebral tuberculosis. Neurological signs are reported in 30% to 60% of cases^[6,7]. These can be radiculalgia, which are more frequent in the lumbar spine, including cruralgia or sciatica^[6].

The diagnosis of spinal TB has been based upon a combination of clinical and radiological findings. MRI is considered to be the most accurate as it allows for identification of not only bone destruction but also granulomatous tissue and tuberculomas, which maybe not be apparent on plain radiographs or CT. There are several imaging findings suggestive of spinal TB. Decreased signal intensity of affected bone and soft tissues on T2-weighted images with an associated thin rim enhancement of increased intensity is a pathognomonic sign for caseating necrosis or a cold abscess in TB^[10]. Regardless of imaging, confirmation of the disease requires biopsy demonstrating acid-fast bacilli on microscopy or isolated culture of the organism. In contrast with pulmonary TB, extra pulmonary TB lesions have a lower amount of bacilli, resulting in less accurate results from microscopy^[11]. PCR has been an effective diagnostic tool for pulmonary TB and is now thought to have high sensitivity and specificity for extra pulmonary TB as well. Compared to culture, PCR allows for a more rapid diagnosis and greater sensitivity even when small amounts of bacilli are present, as is the case with vertebral biopsies^[12].

Spinal TB has a rather insidious course which often leads to greater diagnostic delay. The absence of fever, inflammatory changes, and constitutional symptoms further leads clinicians to prematurely exclude TB from their differential. Later diagnosis of the disease has been associated with a worse prognosis and a greater need for surgical intervention. Although advances in MRI should expectedly improve diagnosis time, the diagnostic delay for spinal TB has remained stable. Additionally, MRI, though valuable^[11,13], does not help to differentiate between infection and malignancy^[12].

As with most other forms of extra pulmonary TB, antituberculous chemotherapy is the main stay of treatment for spinal TB. However, there is no standardized regimen or known optimal duration of treatment. Therapy should initially include isoniazid, rifampin, pyrazinamide, and either ethambutol or streptomycin and can be modified based on results of susceptibility testing. Varying treatment durations ranging from 6 to 18 months have been reported^[13].

CONCLUSION

The TB is still a common condition in developing countries. Early presentations are pain around lumbar spine. Later the patient presents with deformities, shortening of limb and restriction of movements. The constitutional symptoms may or may not be present in all the cases. Diagnosis is mainly clinicoradiological, however, supportive blood investigations and imaging modalities like CT scan or MRI are helpful. Histological proof may not be necessary in all the cases in the endemic

zones for TB. The management depends upon the stage of clinical presentation and the severity of destruction as visible radiologically.

Conflicts of Interest:

The authors do not state any conflict of interest.

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