Scholars Academic Journal of Pharmacy
Abbreviated Key Title: Sch Acad J Pharm
ISSN 2347-9531 (Print) | ISSN 2320-4206 (Online)
Journal homepage: http://saspublishers.com

OPEN ACCESS

Radiology

Intra- and Extra-Medullary Tuberculoma: Case Report and Review of the Literature

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DOI: 10.36347/sajp.2024.v13i04.004 | **Received:** 09.01.2024 | **Accepted:** 15.02.2024 | **Published:** 30.04.2024

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Abstract **Case Report**

Spinal cord tuberculoma is an extremely rare presentation of human tuberculosis in general, and of central nervous system involvement in particular. Only a dozen cases have been described in the literature. We report the case of a 62-year-old man who initially presented with neurological deficit in a context of febrile altered general condition. Diagnosis was made on MRI, which revealed necrotic lesions with medullary contrast.

Keywords: Tuberculome, intra and extra medullary, medullary compression.

INTRODUCTION

Tuberculosis is a real public health problem worldwide, but particularly in African and Asian countries [2-18]. In addition, the AIDS pandemic has led to an upsurge in tuberculosis cases in recent decades. Localization in the nervous system (CNS) is rare, accounting for around 0.5-2% of tuberculosis patients [5-16]. CNS tuberculoma is even rarer, most often developing in the brain parenchyma. Intramedullary location of tuberculoma accounts for 0.02% of CNS tuberculosis cases [1-16].

Intramedullary tuberculoma (IMT) has been little reported in the literature [9]. It usually manifests itself as a slowly compressing spinal cord, and advances in MRI now allow us to better define its radiological characteristics.

CASE REPORT

The patient was 62 years old and had undergone undocumented gastric surgery. He presented with peripheral facial paralysis and motor deficits in both lower limbs, as well as a chronic cough with worsening AEG that had been present for 2 months in a subfebrile setting. His medical history was unremarkable. There was no evidence of tuberculosis infection. Clinical examination revealed a relatively well-preserved general condition, spastic paresis rated 4/5 on the right and 3/5 on the left, and hypoesthesia at T10 level. Patellar and achilles reflexes were sharp, and there was a bilateral Babinski sign.

Biological workup showed normocytic normochromic anemia, a slightly accelerated sedimentation rate, renal workup was unremarkable, and HIV serology was negative. An initial thoracic-abdominal-pelvic CT scan showed a widely excavated left apical lung parenchymal mass on emphysematous lung, associated with bilateral distal pulmonary embolism (figure 1). MRI of the dorso-lumbar spine showed intramedullary nodular signal abnormalities at D5, D6, D12, L1 and L2 in T1 iso signal, T2 hypersignal, enhanced by PDC, with nodular thickening of the dorso-lumbar meningeal envelope, the site of micronodules in T1 and T2 hyposignal and enhanced by PDC. Associated nodular thickening of the horsetail roots and medullary cone, with the same signal and enhancement characteristics as the lesions described above (figure 2).

The diagnosis of intramedullary tuberculoma was evoked by the clinico-biological and radiological picture and the indication of microsurgical excision was retained. The patient was put on anti-tuberculosis treatment for 10 months and functional rehabilitation sessions were prescribed. Progress was satisfactory, with autonomous resumption of walking.

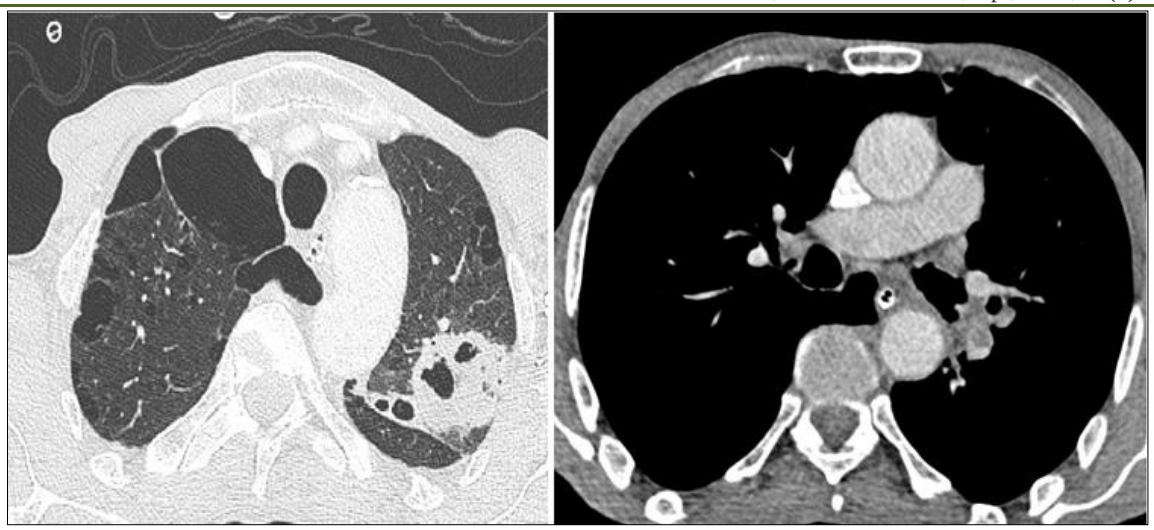

Fig. 1

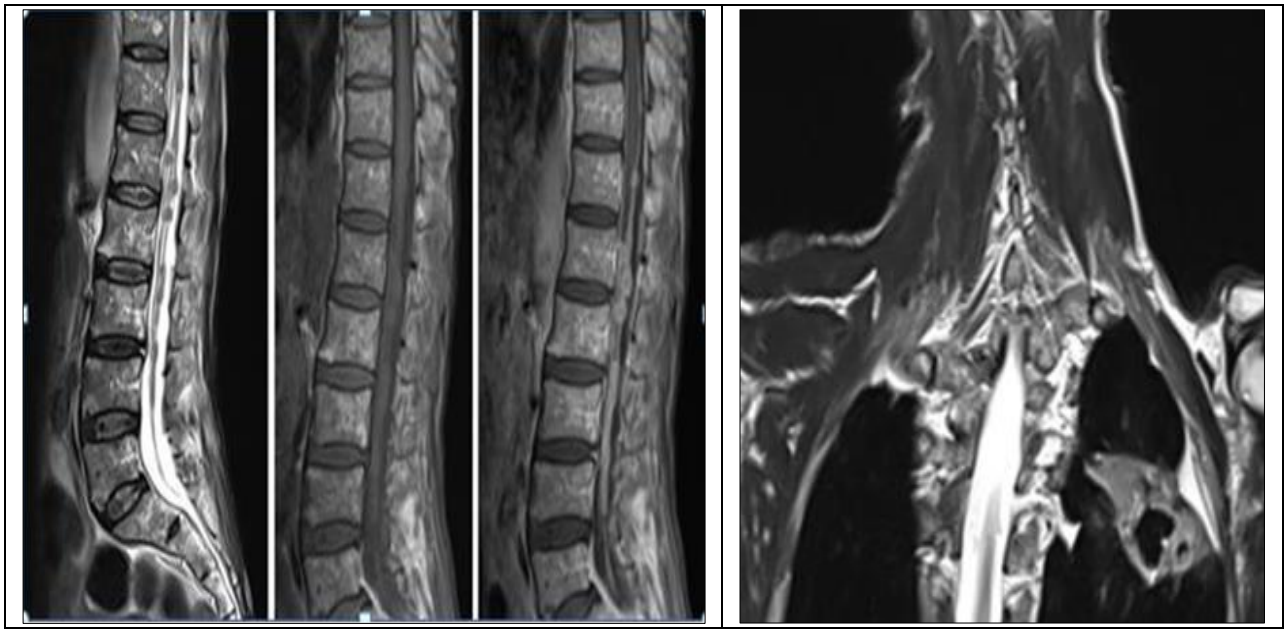

Fig. 2

DISCUSSION

Spinal cord involvement in tuberculosis is most often secondary to vertebral localization, in the classic form of BK or POTT spondylodiscitis. In addition, Mycobacterium tuberculosis can directly affect nerve tissue. Tuberculous lesions develop either in the epidural or subdural space, by diffusion through the meninges, or in the cerebral or medullary parenchyma. This leads to a local inflammatory reaction that develops into a granuloma [7-13]. Tuberculoma localization in brain parenchyma is more frequent than in medullary tissue, with a ratio of 1/42 [2-18]. The weight ratio, and the difference in vascularization, which is greater in the brain, could explain this disproportion [3-17].

Infection of the bone marrow usually occurs via the hematogenous route, from a tuberculosis focus that has developed remotely in the body. Usually, the disease is progressive pulmonary tuberculosis [4-18].

In this case, the bone marrow involvement was isolated. TIM occurs most frequently in young subjects between the ages of 20 and 30 [3-17], with a higher frequency in HIV-positive subjects, or those undergoing immunosuppressive therapy [1-18]. Our patient's HIV serology was negative. Such seronegativity has also been found in various patients in other studies [4-16]. The thoracic medullary segment is the most frequent site of TIM. The other cervical and lumbar segments are more rarely involved [1-9]. The clinical picture of IMT is similar to that of other intramedullary tumors [17]. It is generally a syndrome of slow spinal cord compression, the clinical expression of which depends on the site of the lesion [5-16]. The occurrence of rapidly worsening motor deficit has been reported by some authors [9-18].

The association of signs of tuberculosis impregnation such as fever, night sweats, and the notion of tuberculosis contagion can be found on questioning [5]. In our case, it was a motor deficit of slow onset in a context of weight loss and night fever. MRI provides a more accurate picture of the radiological features of TIM, enabling an easier diagnostic approach [2-16].

Rhoton [12], was the first to describe these features in 1988. Today, with advances in MRI, two aspects of TIM are described depending on the stage of evolution [3-17]. In the initial stage, there is an inflammatory reaction with more or less extensive peripheral edema, the capsule is poor in collagen, and the tuberculoma appears is ointense in both T 1 and T2 sequences, with homogeneous enhancement after injection. At a later stage, the tuberculoma capsule becomes enriched with collagen, and the circular inflammatory reaction diminishes in intensity or disappears. The lesion appears hypointense on t1 and iso a hypointense on T2, with annular contrast in the center of a hypointense image. The center of the lesion becomes hyperintense on T2 with the appearance of caseum. The peripheral part of the granuloma may appear hypointense to hyperintense on T2, depending on the stage of evolution. Peri-lesional edema appears as a T2-hyperintense image. In our case, the lesions were multi-stage intramedullary nodular lesions with T1 iso signal, T2 hypersignal, enhanced by PDC with nodular thickening of the dorsolumbar meningeal envelope. Several authors report the existence of multiple tuberculomas on MRI, as well as the simultaneous presence of intracerebral tuberculomas [9-18].YEN [18], recommends systematic brain MRI in people with multiple IMDs, as these sites are often asymptomatic.

There is as yet no clear consensus on the treatment of IMT [9-13].The use of corticosteroids is controversial, as their efficacy has not been proven [5, 6]. However, their action on peri- lesional edema, and the improvement in neurological disorders observed in some patients, justify their prescription [4-13]. Anti-tuberculosis treatment (ATT) should be the first-line treatment [3-14]. When the diagnosis of IMT is accepted, early administration of anti- tuberculosis treatment, beyond 6 months, usually results in a complete cure [8-16]. The indications for surgery are not clearly defined in the literature [7]. In any case, it should be reserved for certain conditions, notably in cases of diagnostic doubt, an increase in the size of the tuberculoma or a worsening of the neurological deficit despite well-administered medical treatment [2-5].

However, in practice, the discovery of a well-circumscribed intramedullary mass, associated with the presence of neurological disorders, usually motivates surgical removal of the lesion without delay, as was the case in our patient. The diagnosis of IMT is confirmed a posteriori by anatomopathological examination of the surgical specimen, and anti-tuberculosis treatment is immediately instituted [4-17].

CONCLUSION

Although IMT is a rare condition, it should be suspected in the presence of slow cord compression, especially in our context. The contribution of modern neuroradiological techniques to its diagnosis, the use of microsurgical techniques and the combination of appropriate anti-tuberculosis treatment now make it potentially curable.

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