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Case Report

Intramedullary Thoracic Tuberculoma: A Male Case Report

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Abstract

Tuberculosis (TB) is a threat to global health. It is still prevalent in developing countries. TB of the Central nervous system (CNS) is the most serious and deadly form. The appearance of intramedullary tuberculoma is a rare form of CNS tuberculosis.

We report a typical case of intramedullary thoracic tuberculoma in a young Moroccan man, secondary to a pulmonary miliary tuberculosis.

The aim of this work is to familiarize radiologists and surgeons with this rare anomaly that must be mentioned among the differential diagnoses of intramedullary lesions, especially in countries endemic to tuberculosis.

Keywords: *Tuberculoma; Intra-Medullary; Thoracic Spine; MRI*

Introduction

TB is the oldest microbiologically documented human infectious disease and has probably killed more people in the history of human civilization than any other disease [1].

CNS tuberculosis is a serious complication of *Mycobacterium tuberculosis* (TM) infection that causes real public health problems worldwide. The World Health Organization (WHO) estimated that the incidence of tuberculosis worldwide was 10 million new cases with 1.5 million deaths in 2018. In Morocco, the average incidence of tuberculosis was 89 cases per 100,000 in 2015 [2].

Although tuberculosis is a disease that initially and mainly involves the lungs, extrapulmonary involvement occurs in about 14% of those affected, of which only 1% are neuromeningeal [1,2]. It includes tuberculous meningitis, which is the most common form, cerebral and spinal tuberculomas, and vertebral TM infection with spinal cord compression [1,3,4].

Intramedullary tuberculoma is a rare disease with an incidence of 2/100,000 of all tuberculosis and constitutes only 0.2 to 5% of all CNS tuberculomas [3,5-7]. First reported by Abercrombie in 1828, about 171 cases have been reported to date, and the most favorable site of intramedullary tuberculoma is the thoracic spine [4,8-10].

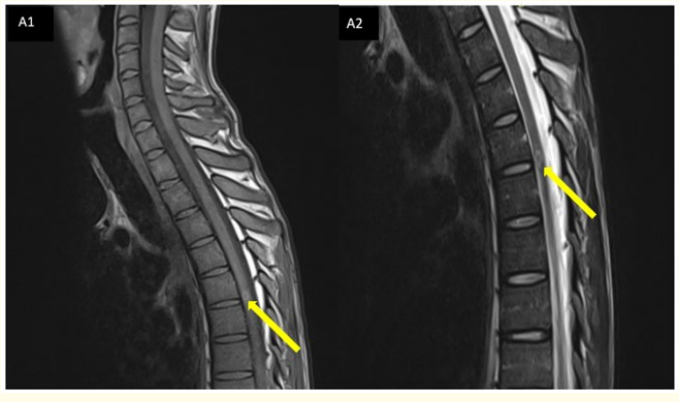
Case Report

A young 27 years old man, Moroccan, followed for pulmonary miliary tuberculosis under treatment since February 2023, revealed by signs of tuberculous impregnation; He presents since May 2023 (Three months) paresthesias types tingly in the lower limbs.

On clinical examination, the patient has a pyramidal syndrome in the two lower limbs without a motor or a sensory deficit. There were no signs of upper-limb involvement or encephalitis. He is a little embarrassed on the respiratory plan with a weight loss of 8 kg and a small fetish. The rest of the examination is without particularity.

Biologically, leukocytosis is at 12 300/mm^3 with accelerated SV. Mantoux's test and ELISA for HIV were negative. The pulmonary X-ray showed a TB pulmonary miliary tuberculosis. X-rays of dorso-lumbar spine showed no abnormalities.

The medullary MRI shows an abnormality of intramedullary signal lateralized left at the stage D5-D6, rounded, well limited, in central hypo signal, hyper peripheral signal T1et T2, raised annularly after injection of gadolinium, surrounded by edema in hyper signal T2, measuring 6 x 6 mm (Figure A1-A3 and B1 and B2). There were no abnormalities in the vertebral bodies or spinal soft tissues. This lesion, due to its characteristic location, size, and classical ring enhancement after gadolinium injection with surrounding edema, was considered typical of a tuberculoma.



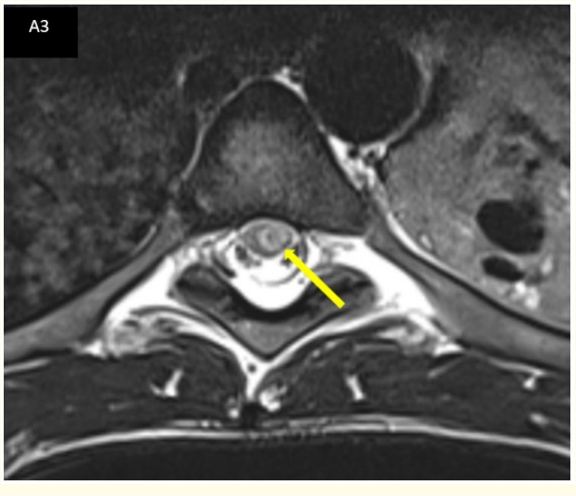


Figure A1-A3: *MRI of the thoracic marrow. T1 (A1) sagittal slices, T2 (A2) sagittal slices, and T2 axial (A3): left-sided intramedullary signal anomaly on stage D5-D6, rounded, well limited, in central hypo signal, discrete peripheral hyper signal T1 and T2, surrounded by lesional peri edema in hyper signal T2, measuring 6x6mm (Yellow arrow).*

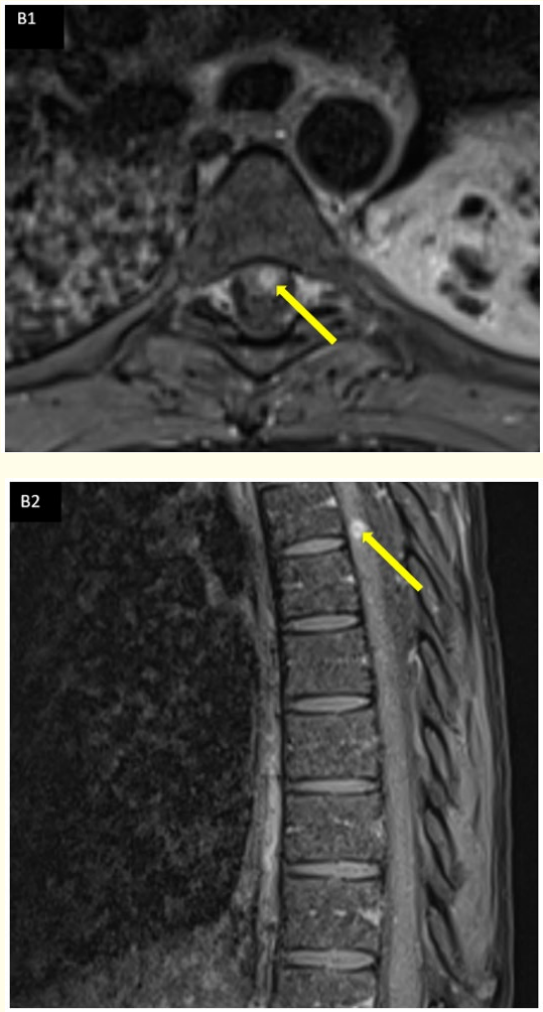


Figure B1 and B2: *MRI of the thoracic marrow. T1 FATSAT after gadolinium injection, axial section (B1) and sagittal section (B2): annular enhancement of the lesion after gadolinium injection (Yellow arrow).*

The patient was treated with anti-tuberculosis chemotherapy with a combination of four drugs [streptomycin, rifampicin, isoniazid, and pyrazinamide] for a period of two months, which was followed by rifampicin and isoniazid for an additional 10 months. He was also given a bolus of 16 mg dexamethasone, followed by 4 mg three times daily for three days. Dexamethasone was reduced over the next four days.

Within a week, He began to show signs of recovery in the form of pyramidal syndrome regression. He continued to improve her neurological condition regularly after her discharge.

Discussion

Intramedullary tuberculoma is mainly caused by active lesions of generally pulmonary tuberculosis that spread through blood [3,5,6], or tuberculosis of the brain that spreads through cerebrospinal fluids; it is rarely caused by the direct spread of local tuberculosis of the spine [7]. In our case, the patient presented with pulmonary TB combined with intramedullary tuberculoma.

Intramedullary tuberculomas of the spine were observed in relatively young patients (mean age, 28.6 years) and more frequently in women (63% of cases) than in men [7,8,10,11].

The most common area affected by intramedullary tuberculosis is the thoracic region, which occurs in about 70% of cases. The probable reason why intramedullary tuberculosis involves the thoracic spinal cord is mainly because this region receives about 45% of the total blood supply to the spine, and it is known that TM is aerobic and therefore develops better in a region where the partial pressure of oxygen is high [8-10]. Our patient is young and had an intramedullary tuberculoma at D5-D6 level.

Intramedullary tuberculoma is most often manifested by the sub-lesional appearance of spastic paraplegia (61%) and flaccid paraplegia (33%) due to compression of the spinal cord, including progressive weakness of the lower limbs, sensory abnormalities, disorders of urination and defecation, and symptoms of tuberculous impregnation, namely mild fever, night sweats, weight loss, and fatigue [7,9,12]. This is consistent with our patient, who presented with a pyramidal syndrome in both lower limbs without a motor or sensory deficit and signs of tuberculous impregnation.

It can be difficult to differentiate intramedullary tuberculomas from other medullary lesions such as primary medullary tumors (astrocytoma, ependymoma, hemangioblastoma and lymphoma) and metastatic, inflammatory lesions, demyelinating diseases (multiple sclerosis), vascular diseases (malformations, congestion), and chronic granulomatous diseases such as sarcoidosis, brucellosis, and histiocytoma [5-7,9].

However, MRI has revolutionized tuberculoma imaging, and the diagnosis can be made with reasonable certainty, thus avoiding the need for an invasive procedure [5], as was the case in our patient.

The MRI aspect of intramedullary tuberculomas was first described by Rhoton., *et al*. and subsequent descriptions of the characteristics of this lesion and the different stages of tuberculoma formation (non-caseating, caseating with a solid center, and caseating with a liquid center) have been published by many authors [4].

The MRI characteristics of intramedullary tuberculomas vary according to the stages of tuberculoma formation. They appear as nodular lesions in hypo signal with or without the central hyper signal in T2 (due to the variable amount of broken necrosis) and as nodular lesions in hypo-to-iso signal in T1 with edema, whose extent can vary from one to nine vertebrae [3,8,9,12,13]. After injection of gadolinium, note at the early phase a homogeneous enhancement; at the late phase, note an annular enhancement (tissue of peripheral infectious granulation); and if the breaking necrosis appears at the center of the lesion, we can observe the “target sign” which is a valuable way to differentiate tuberculosis from other intramedullary lesions [7,9]. The MRI aspect in our patient suggested a solid caseous tuberculoma that appeared in central hyposignal, peripheral hypersignal T1 and T2, raised annularly after injection of gadolinium.

Evidence-based guidelines for the treatment of neurotuberculosis are missing [1]. In principle, in the treatment of CNS tuberculosis, drugs that enter the central nervous system, such as isoniazid, rifampicin, ethambutol, and pyrazinamide, should be administered [3,9].

WHO recommends 2 months of intensive treatment using the combination of isoniazid, rifampicin, pyrazinamide, ethambutol, or streptomycin, followed by a combination of isoniazid and rifampicin for 7 to 10 months. Initial adjuvant corticosteroids (dexamethasone or prednisolone) are recommended, with discontinuation for 6 - 8 weeks [1,4,9].

In the management of intramedullary tuberculosis, medical, surgical, or combined approaches have been used with good results in different series, although there is no consensus on the ideal treatment [4,9].

Although some authors only recommend medical treatment, surgery is usually indicated when the diagnosis is uncertain, the lesion is large and is accompanied by rapid deterioration of neurological functions, or there is no adequate response to antituberculosis chemotherapy [4,8,9]. Our case proves that medical treatment can achieve a good clinical result and that surgery is not always indicated.

Conclusion

Although intramedullary tuberculoma is a rare condition, it must be kept in mind in differential diagnoses of spinal cord injury, especially in countries with endemic tuberculosis.

MRI is the exam of choice to diagnose and monitor intramedullary tuberculomas without invasive procedures.

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