

Intramedullary and Extramedullary Intradural Tuberculomas: About Two Cases

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Abstract

Case Report

Intramedullary and extramedullary intradural locations of tuberculosis are rare events. The MRI is currently the gold standard examination in terms of the diagnosis and follow-up.

Keywords: Intramedullary tuberculomas, extramedullary intradural Tuberculomas, MRI.

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INTRODUCTION

Intramedullary and extramedullary intradural locations of tuberculosis are rare events which clinically manifest as insidious spinal cord compression. Our goal is to illustrate these rare locations of central nervous system tuberculosis.

CASE REPORTS

Case 1

A 24 year old woman with common pulmonary tuberculosis confirmed by positive smear microscopy in whom the treatment failed and who presented with left hemiparesis and a spinal cord compression syndrome. This patient's cerebral CT showed supra and infra tentorial nodular lesions representing tuberculomas. Her spinal MRI showed intramedullary lesions at the level of the C7, T11 vertebra and the T11-T12 intervertebral space in relation with intramedullary tuberculomas. The patient was started on a post therapeutic failure tuberculosis treatment consisting of 3RHZE/5RHE and received 3 bolus injections of Solumedrol which enabled her to significantly improve clinically speaking.

Case 2

A 29 year old woman suffering from multifocal tuberculosis of digestive, neurological, meningeal, and pulmonary localizations, on an antituberculosis drug regimen, presented with a medullary compression syndrome consisting of paraparesis, sphincter disorders, hyperactive deep tendon reflexes. Her spinal MRI showed an intradural extramedullary mass within the vertebral canal at the

level of T12 associated with meningeal thickening and increased contrast enhancement at the levels of the cervical, thoracic and lumbar spine meninges related to her tuberculosis diagnosis.

DISCUSSION

Most of the time, spinal tuberculosis is secondary to tuberculosis of vertebral location, classically known as tubercular spondylodiscitis or Pott's disease. However, mycobacterium tuberculosis can also directly involve nervous tissue notably the spinal cord. Tubercular lesions in the epidural space or in the subdural space diffuse through the meninges and develop in the cerebral parenchyma or in the spinal cord producing a local inflammatory reaction which evolves into a granuloma.

Intramedullary tuberculoma

Intramedullary tuberculomas have rarely been reported in the literature. Spinal cord involvement is often the result of hematogenous spread of an active distal pulmonary tuberculosis focus. This was the case of our patient who presented with microscopically confirmed pulmonary tuberculosis and was started on antitubercular drugs at the time of diagnosis.

Clinical Presentation: Intramedullary tuberculomas often manifest with symptoms of progressive spinal cord compression which vary depending on the exact location of the lesion. Our patient presented with spinal cord compression syndrome manifesting as left hemiparesis.

Myelography and spinal computed tomography are seldom performed nowadays in the context of spinal tuberculomas.

The MRI does a better job at specifying the radiological characteristics of intramedullary tuberculomas and facilitates their diagnostic approach. Rhoton (4) was the first to describe these characteristics in 1988. Currently, two radiological characteristics of intramedullary tuberculomas are described according to their evolutionary stage (5).

At the initial stage: An inflammatory reaction with more or less significant peripheral edema. The tuberculoma appears iso-intense in the T1 and T2 sequences and homogeneously enhances after contrast injection.

At a more advanced stage: The tuberculoma's capsule becomes enriched in collagen and the circular inflammatory reaction decreases in intensity or disappears entirely. The lesion is hypo intense in the T1 sequence. In the T2 sequence the lesion is iso or hypo intense and is described as a central hypo intense image surrounded by ring enhancement following contrast injection.

The center of the lesion becomes hyper intense in the T2 sequence as the lesion becomes more caseous. The peripheral part of the granuloma may appear hypo or hyper intense on the T2 sequence depending on the lesion's stage of development. Peri-lesional edema presents as a hyper intense image on the T2 sequence.

In our patient's case, an intramedullary tuberculoma was located at the C7 vertebral body level, well circumscribed, oval shaped, measuring 3mm and extending over 7mm. It was isointense in the T1 sequence, with a hypointense center in the T2 sequence, and showed ring enhancement after contrast injection. This lesion was associated with three similar intramedullary lesions at the T11 vertebral body level and the T11-T12 intervertebral space level, the largest of which measured 5x8 mm.

Several authors report the presence of multiple simultaneous tuberculomas on MRI as well as the presence of a concomitant intracerebral tuberculoma which was our patient's case too (6). YEN (6) recommends systematically performing a brain MRI in patients presenting multiple intramedullary tuberculomas.

The first-line treatment consists in an anti-tubercular drug regimen. The use of corticosteroids is controversial as its efficacy has not been proven. However corticosteroids' action on peri-lesional edema and the improvement noticed in some patients' neurological disorders justify their prescription.

Prognosis: The combination of modern neuroradiological means that facilitate the diagnosis, the use of microsurgical techniques and the adequate use of anti-tubercular drugs, make intramedullary tuberculomas potentially curable nowadays.

Extra medullary intradural tuberculoma

Extramedullary intradural tuberculomas without associated bone lesions are a rare cause of spinal cord compression.

Bucy and Oberhill first described an extramedullary intradural tuberculoma in 1950 [7]. To our knowledge, less than twenty other cases have since been reported in the literature [8].

Clinical Presentation: the neurological symptomatology associated with the various locations of extramedullary intradural tuberculomas is non-specific. The clinical presentation can be that of a slowly progressive spinal cord compression evolving for several weeks or months, as was our patient's case, or more rarely, that of acute myelitis having developed within 24 hours.

The presence of additional general signs and of a secondary tuberculosis location are of great additional diagnostic value. Our patient was being followed for multifocal tuberculosis (digestive, neuro-meningeal and pulmonary) and was receiving an anti-tuberculosis treatment at the time of her diagnosis.

Although computed tomography provides scarce information in the context of tuberculomas, it can nonetheless shows a decrease of the perimedullary subarachnoid spaces with displacement of the medullary parenchyma, and does a better job at characterizing tuberculomas when coupled with myelography [9].

The MRI has become the gold standard as it not only allows one to precisely locate the expansive process within the vertebral canal but also enables one to identify an associated arachnoiditis. In some cases the MRI also orients the diagnosis towards an infectious etiology, especially if an image of a target sign is present.

Although the MRI aspect of intramedullary tuberculomas has been known since it was first described by Rhoton in 1988 [4], to our knowledge, its intradural location has never been described up until now.

According to Gupta [10, 9], the most common MRI aspect of intramedullary tuberculomas is the following: an enlarged and swollen isointense or slightly hypointense spinal cord in the T1 sequence and a hyperintense spinal cord in the T2 sequence. This aspect changes after injection of Gadolinium depending

on the tuberculoma's stage of development. It is homogeneously hyperintense at the initial inflammatory stage, followed by rim enhancement and hypointensity in the central lesion at a later stage of development. Finally it appears as a target sign with a central hyperintensity at the caseification stage.

In our patient's case, the MRI objectified an oval shaped extra-medullary intra-dural mass within the vertebral canal at the level of T12 measuring 3x1 cm. This mass was hypointense in the T1 sequence, discretely and heterogeneously hyperintense in the T2 sequence and homogeneously and intensely enhancing after gadolinium injection. It occupied the posterior epidural space and exerted a mass effect on the spinal cord leading to signs of spine damage at that level. Moreover, it was associated with a thickening and contrast enhancement of the cervical, thoracic and

lumbar spine meninges which was more pronounced at the level of the filum terminale. No intervertebral disc involvement of the disease could be detected.

The treatment is above all surgical. Its goal is to alleviate the mass effect on the spinal cord and to obtain histological and bacteriological certainty of the diagnosis.

The prognosis is generally good provided that the patient is treated early in order to avoid any permanent sequelae.

CONCLUSION

The MRI is currently the gold standard examination in terms of the diagnosis and follow-up of intramedullary and extramedullary tuberculomas.

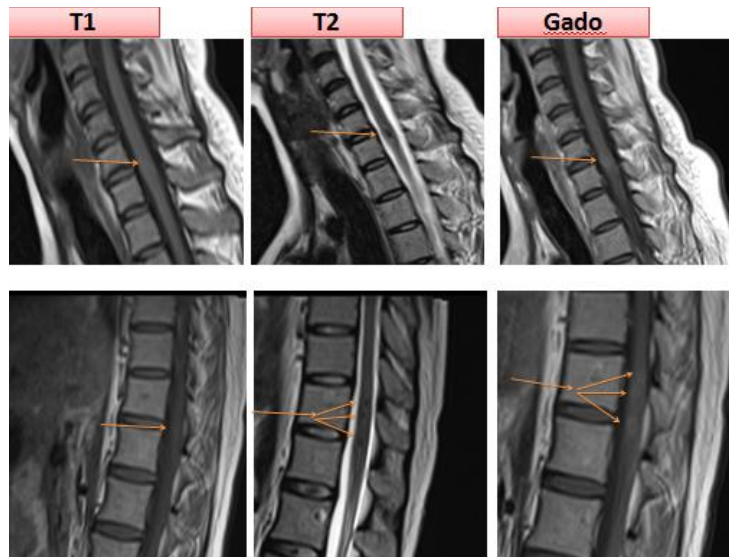


Fig-1-6

Intramedullary lesions at the level of C7 (n=1), well circumscribed oval shaped, at the level of T11 and the T11-T12 intervertebral space (n=3), isointense on

the T1 sequence, with a hypointense center on the T2 sequence and with ring enhancement after contrast injection.

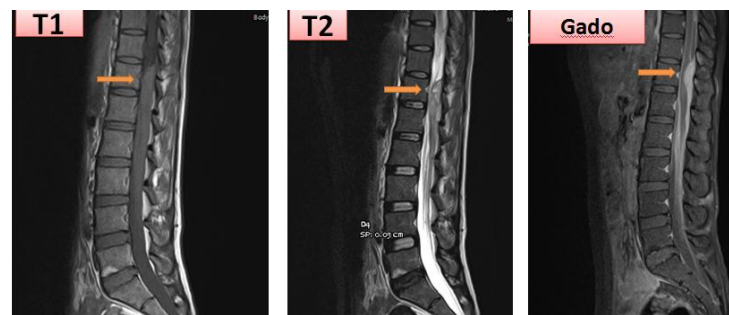


Fig-7-9

An oval shaped extramedullary intradural mass located within the vertebral canal at the level of T12. This mass is hypointense in the T1 sequence, slightly and heterogeneously hyperintense in the T2 sequence, and enhances homogeneously and intensely after

gadolinium injection. It occupies the posterior epidural space and exerts a mass effect on the spinal cord at that level. What's more it is associated with meningeal thickening and increased contrast enhancement at the levels of the cervical, thoracic and lumbar spine

meninges as well as at the level of the filum terminal where the enhancement is more pronounced. No intervertebral disc involvement is present.

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