

MR Imaging of Intraspinal TB - A Misguide in Disguise

Asutosh Dave¹, Jitendra Kumar Singh², Vishal Patel³

^{1, 2, 3} Department of Radiology, GCS Medical College and Hospital, Ahmedabad, Gujarat, India.

PRESENTATION OF CASE

A 47-year-old female patient came with complaints of low back pain with progressive numbness and pain in left lower limb. She also had intermittent fever with significant weight loss of 15 kgs over the last 6 months. She has been taking anti-TB drugs since 1 year but discontinued 3 months back, after which her symptoms worsened. She complained of fever accompanied by weight loss along with low back pain and lower limb weakness 1 year back. She then underwent for MRI scan in our dept.

Corresponding Author:

*Dr. Jitendra Kumar Singh,
302, Kunj Vihar, Opp. Annexe House,
Near Madhuram Tower, Shahibaug,
Ahmedabad – 380004, Gujarat, India.
E-mail: doctorjitendra12@gmail.com*

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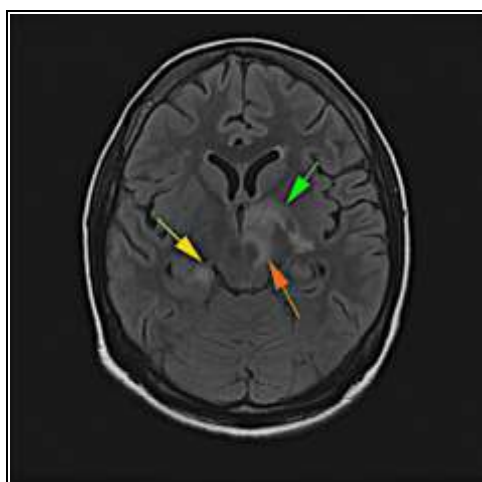


Figure 1.
Axial Flair Brain Image Showing
Hyper Intense Foci in Right Medial
Temporal Lobe (Yellow Arrow),
Left Sub Thalamic Nucleus (Orange
Arrow) and Left Putamen (Green
Arrow)



Figure 2.
T2W Sagittal Image of Spine
Showing Presence of Well-
Defined Intramedullary Nodule
with Hyper Intense Cord

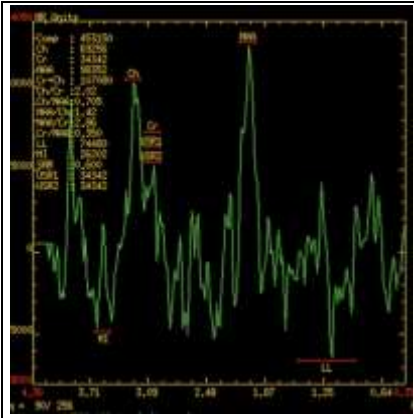


Figure 3.
Mutivoxel Spectroscopy Graph Revealing Elevated Lipid-Lactate Peak at the Point of Interest in Brain Parenchyma

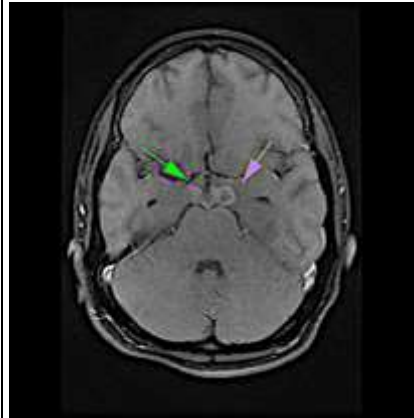


Figure 4.
Axial Brain Contrast Image Showing Presence of Peripherally Enhancing Lesions in Right (Green Arrow) and Left (Pink Arrow) Sub Thalamic Nuclei

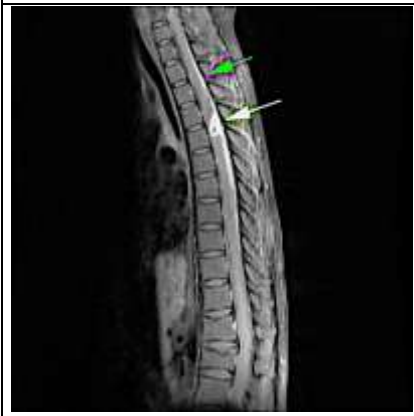


Figure 5.
Sagittal Spine Contrast Image Showing Ring Enhancing Intramedullary Lesions (Pink Arrow) with Leptomeningeal Enhancement (Green Arrow)

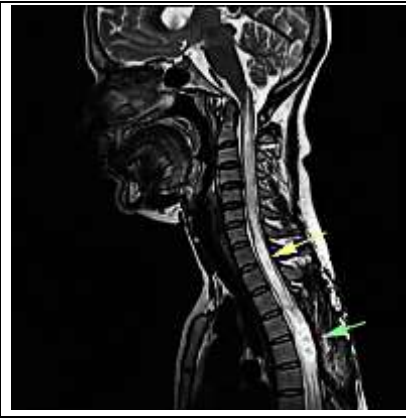


Figure 6.
T2W Sagittal Cervico-Dorsal Spine Showing Disseminated Intramedullary Hyperintensity (Yellow Arrow) along with Well Defined Disseminated Nodules Predominantly in the Dorsal Spine (Green Arrow)

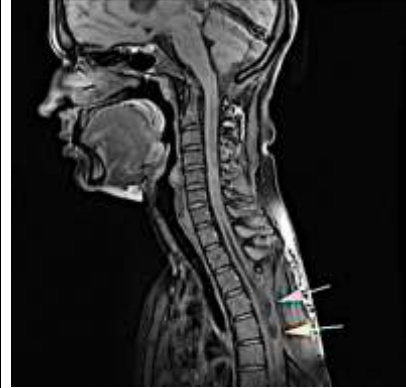


Figure 7.
Contrast Sagittal MRI of Spine Showing Diffuse Peripherally Enhancing Nodules in Dorsal Spine (Blue and Orange Arrow) in the Same Location as Seen One Year Ago. However, This Time Bigger in Size

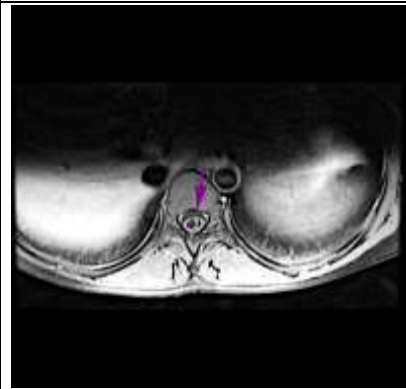


Figure 8.
Contrast Axial Image of Lumbar Spine Showing Peripherally Enhancing Well Defined Collection in Epidural Region (Purple Arrow)

Follow-Up Scan after 1 Year

Imaging Features

MRI showed FLAIR hyper intensity at different foci of brain (figure 1) multiple ring enhancing lesions in brain (figure 4) with elevated lipid-lactate levels (figure 3) in spectroscopy graph in the region of interest. There is also presence of focal intramedullary hyper intensity in dorsal spine (figure 2) which shows ring enhancement along with leptomenigeal enhancement (figure 5). Patient also had her CSF culture done which revealed elevated lymphocyte count and LDH levels suggesting tubercular aetiology. Patient then started with anti - TB drugs for one-year course, however she left the course in between and again presented with similar complaints one year later and then again underwent for MRI study of spine.

Follow up MRI scan revealed disseminated spinal nodular lesions (figure 6) which have been increased in size and shows peripheral enhancement with diffuse cord hyperintensity (figure 7). There was also presence of epidural collection in lumbar region (figure 8).

Radiological Features

Most intraspinal tuberculosis cases arise from a primary extraspinal source, with the lungs and intracranial region being the most common primary sites, as demonstrated in this study; these two primary tuberculous diseases disseminate to the intraspinal region through the blood and CSF, respectively.¹ The thoracic segments were most commonly involved in our series. This involvement pattern can be attributed to the distribution of blood supply to the spinal cord : the thoracic segment receives nearly 45 % of the spinal cord blood supply.^{2,3} Intraspinal tuberculosis cases were additionally accompanied by leptomenigitis, which showed isointense on T1 - weighted images and mixed iso - or slightly hyperintense on T2 - weighted images, and obvious homogeneous linear, mottled, or patchy enhancement in more than two segments on enhanced T1 - weighted images. The lesions had indistinct edges and appeared to integrate with each other, most showing obvious enhancement along with focal or diffuse spinal cord oedema and syringomyelia. The findings in patients with

tuberculous leptomeningitis included hydrocephalus, intramedullary tuberculoma, spinal arachnoiditis, en plaque appearance of the sacral nerve roots, focal or diffuse oedema in the spinal cord, and syringomyelia, which have also been reported by others.^{4,5,6} Tuberculoma appeared a regular shape of isointense on T1- and slightly hyperintense on T2-weighted images, with homogeneous nodular enhancement seen in the early stage, while ring enhancement observed in the late stage, presumably due to caseous necrosis in the lesion center.⁷

DIFFERENTIAL DIAGNOSIS

1. Sarcoidosis - unlike tuberculosis, sarcoidosis shows extra - axial homogenous enhancement with leptomeningeal spread. It shows resolution on steroid therapy and never shows ring enhancement.
2. Immunocompromised disseminated lymphoma - Unlike tuberculomas, lymphoma in immunocompromised patients show significant diffusion restriction with midline shift along with surrounding oedema. The condition resolves after steroid therapy and blood reports show significant lymphopenia.
3. Metastasis - Metastasis appears to be in non - uniform ill - defined lesions with surrounding significant perilesional oedema and mass effect. Spectroscopy shows elevated choline peak as well. Leptomeningeal spread may or may not be seen.

DISCUSSION

a. Pathology

Tuberculosis, caused by *Mycobacterium tuberculosis* is a chronic infectious disease characterized by the formation of granuloma and rarely of abscess in the infected tissue.⁸ is the central nervous system involvement very rare affecting 0.5 – 2 % of the cases. Intraspinal tuberculoma is almost always secondary to tuberculous involvement elsewhere in the body most common location being is the thoracic spinal cord. The higher incidence involving the thoracolumbar region is explained on the basis of the regional blood flow to the spinal cord. Intramedullary tuberculoma may be difficult to differentiate from space - occupying lesions such as primary and metastatic spinal tumours and other chronic granulomatous diseases such as sarcoidosis, brucellosis, histiocytosis. Generally, patients with intraspinal tuberculoma have a gradual onset of the disease over weeks to years with progressive involvement of motor and sensory pathways. Slow deterioration of neurological symptoms is caused by an expansion of the lesion causing pressure on the cord. Sometimes it is precipitated by heavy lifting or minor motor accidents. Abscess formation becomes manifest by the accumulation of the necrotic tissue, debris and caseous material as the disease progresses. Abscess frequently bursts through or passes around the anterior

or posterior longitudinal ligament and may spread to distant anatomical regions from the original site of the infection.

b. Management

The treatment of intramedullary abscess should include a combination of surgical and pharmacological therapies. Surgery is indicated for the evacuation of the pus and use of appropriate antibiotics and corticosteroids should be considered in the treatment.^{9,10,11}

FINAL DIAGNOSIS

Cerebral Tuberculosis with Intraspinal Dissemination

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