# Striking PET CT Findings in Infective Myelitis

Sir,

A 57 year--old previously healthy man from central kerala developed severe pain in the right leg which gradually increased upwards. Over the next few days, he developed an asymmetrical weakness of the right leg and presented to us. He had sustained a penetrating injury to the right foot 4 months earlier. On examination, he had fever, neck stiffness and an asymmetric paraparesis [right leg >> left] with dermatomal sensory loss in the right leg. He also had urinary retention and required an indwelling foley's catheter. HbA1c was 5.7% and routine blood tests were normal. MRI spine showed an intra- medullary enhancing lesion in the conus on right side. There was significant spinal cord edema with expansion from D11 to L1 level. Brain imaging was normal. A CSF study showed > 600 cells/cmm3 (N3L97), normal sugar levels and high protein [900mg%]. An extensive CSF pan-meningitis panel [XCyton, Bangalore] was negative. CSF cultures and AFB stains were also negative. Antibiotic therapy was commenced with Ceftazidime and vancomycin. However, his weakness progressed to a complete paraplegia with bowel and bladder retention with a high thoracic sensory level. At 14 days, a repeat CSF examination showed decreasing cell counts [300 cells with a lymphocytic predominance, normal sugar and high protein levels {460mg%}.

Repeat MRI showed extension of the spinal cord hyperintensities to the thoracic level. As the patient complained of intense backache, a whole body PET-CT was performed. The objectives were ostensibly to look for spondylodiskitis, a biopsiable lesion or a paraneoplastic lesion. The maximum standardized uptake value (SUVmax) was determined at the region of interest and compared with either the normal spinal cord or with normal reference values of the mediastinal blood pool. A diffuse lesion was defined as a single lesion that extended to > 2 vertebral levels.

PET CT showed diffuse FDG uptake in the spinal cord extending from D10 to L1 level without any evidence of spondylodiskitis or vertebral body uptake [Figure 1]. No other FDG active lesion was identified. However, the underlying diagnosis was still unclear. The differential diagnosis considered after this were an infective myelitis, inflammatory, demyelinating or neoplastic myelitis or an immune reconstitution syndrome. Due to concern for a possible inflammatory pathology, IV methylprednisolone 1 gm/day was added for 5 days. After a transient improvement for 5 days, he progressed further to quadriparesis, requiring intubation and mechanical ventilation. Over the next 5 days, he became drowsy and a repeat CT showed features suggestive of right cerebral swelling. A decompressive craniectomy was performed along with leptomeningeal and brain biopsy. He worsened 2 days after the surgery and expired after a total of 6 weeks. Post mortem, the brain and leptomeningeal biopsy grew Burkholderia pseudomallei and neuropathology showed features of a suppurative pathology with intense neutrophilic and histiocytic infiltration. [Figure 2] The Active meliodosis DetectTM rapid test [InBios, Seattle USA] and T3SS1 gene tests were positive on the tissue sample.

[18F]-Fluoro-deoxy glucose positron emission tomography (18FDG-PET) measures metabolic activity by regional uptake of glucose and is utilized in neurology for a wide variety of conditions including infections, neoplasms, inflammatory and paraneoplastic disorders. However, in contrast to brain or systemic disorders, PET-CT is under- utilized in spinal cord inflammatory or infective disorders. 18FDG-PET-CT of the spinal cord has significant limitations. It is hampered by a poor spatial resolution, the small size of the spinal cord (1-1.5 centimeters in diameter) and low 18FDG uptake in the spinal cord compared to the brain.[1] Moreover, intense or 'spillover' vertebral bone marrow activity may make it harder to differentiate marrow from pure spinal cord uptake.

Classically, spinal cord neoplasms show hypermetabolism on 18FDG-PET-CT and hence most literature pertains to its use in neoplastic disorders. Neuro-sarcoidosis is the one inflammatory myelopathy in which 18FDG PET-CT shows a hypermetabolic focus and is very useful [Table 1].[2] Most other inflammatory or demyelinating myelopathies do not show significant hypermetabolism and 18FDG PET-CT offers no extra information.[1] Hence it is not widely used for most cases of transverse myelitis. It has shown some utility in predicting the neurological outcome in cervical compressive myelopathy- where a high 18FDG uptake at the site of compression suggests an early and reversible myelopathy, compared to a low 18FDG uptake,

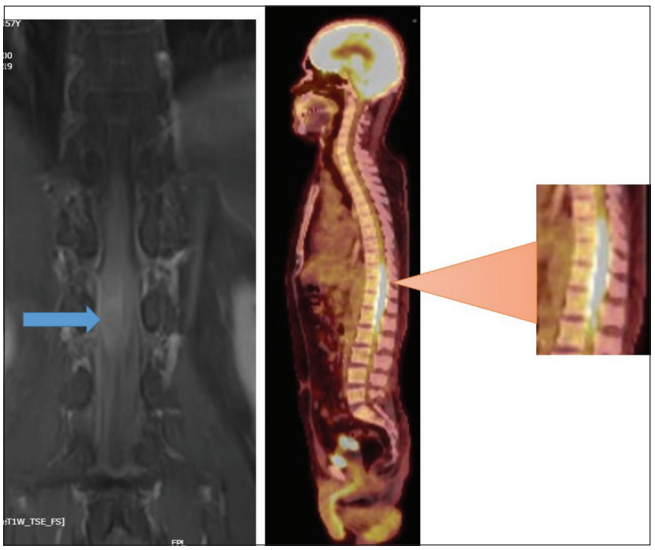

**Figure 1:** Panel on the left-MRI contrast ; Coronal T1 weighted images of the Lumbo-sacral region show a predominantly right sided contrast enhancing conus myelitis [blue arrow] Panels on the right show an FDG avid long segment hyperintensity in the lower thoracic cord with a magnified image showing the intramedullary avidity without local spillover.

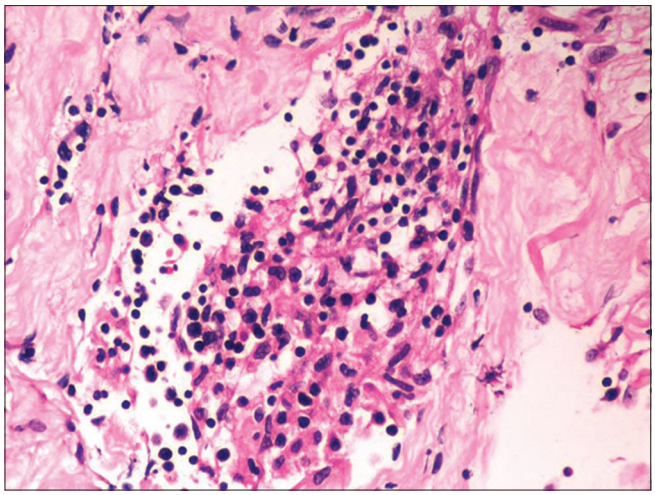

**Figure 2:** High power histopathology picture shows a dense neutrophilic infiltrate with a micro-abscess

**Table 1: Conditions associated with Spinal cord hypermetabolism on PET-CT**

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|---|
| Neuro-sarcoidosis |
| Spinal cord lymphoma |
| Intramedullary spinal cord metastases |
| Primary spinal cord tumors |
| Pediatric spinal cord tumors |
| Leptomeningeal metastases (2) |
| Early radiation myelopathy |
| Infective arachnoiditis |
| Intraspinal canal neurolymphomatosis |
| Leptomeningeal gliomatosis |
| Early cervical compressive myelopathy |
| Infective myelitis |

which suggests gliosis and poor post-operative neurological function.[3]

Infective myelitis is a rare entity and there is a paucity of data in this entity. Although one article has reported spinal cord hyper-metabolism in infective arachnoiditis, PET-CT findings have not been reported in pure intramedullary fulminant infective myelitis [Table 1].[4] Indolent infections like HTLV-1 myelopathy on the contrary show thoracic cord 18FDG-PET-CT hypo-metabolism.[5]

In our patient, the area of hypermetabolism corresponded with the area of active myelitis on contrast enhanced MRI. The SUVmax values were able to clearly delineate a long segment of intrinsic cord hypermetabolism. As our case illustrates, spinal cord hypermetabolism on 18FDG-PET-CT in the appropriate context should arouse the suspicion of an active or ongoing spinal cord intramedullary infective myelitis. Penetrating trauma is a well known mode of infection in meliodosis. The unusual mode of presentation as well as the need for repeated biopsies in meliodosis to establish the diagnosis contributed to delayed diagnosis.

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## Conflicts of interest

There are no conflicts of interest.

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