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Tabetic Neuroarthropathy of the Lumbar Spine (Charcot Spine): A Case Report and Review of the Literature

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Abstract | **Case Report**

Tabetic neuroarthropathy of the spine, or Charcot spine, is an uncommon and severe osteoarticular complication of neurosyphilis (*tabes dorsalis*). It stems from the combined loss of deep proprioception and nociceptive pain sensation, subjecting the vertebral column to unperceived repetitive microtrauma that leads to marked structural disorganization and exuberant osteolysis. We report the case of a 51-year-old male patient followed for neurosyphilis complicated by tabetic arthropathy since 2024, presenting with left-sided lumboradiculopathy without motor deficit. Lumbosacral computed tomography (CT) demonstrated multi-level destructive disco-vertebral lesions predominating at L4–L5, combining somatic collapse, multiple subchondral pseudocysts, marginal reactive sclerosis, paraspinal detached osseous debris, and multi-level spinal canal and foraminal stenosis. This observation illustrates the characteristic CT semiology of Charcot spine and emphasizes the central role of CT in differentiating this entity from infectious spondylodiscitis or spinal malignancies.

Keywords: Tabetic arthropathy; Charcot spine; Neurosyphilis; *Tabes dorsalis*; Computed Tomography; Lumbar spine.

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INTRODUCTION

Neurogenic arthropathy, originally characterized by Jean-Martin Charcot in patients with *tabes dorsalis*, is defined as progressive joint destruction secondary to underlying proprioceptive and nociceptive sensory deficits. While diabetic neuroarthropathy of the peripheral foot and ankle represents the most common presentation in modern clinical practice, axial involvement—known as Charcot spine—remains an emblematic manifestation of tertiary syphilis.

Pathophysiologically, the selective degeneration of the dorsal spinal nerve roots and posterior columns eliminates protective neuromuscular reflexes. The resulting lack of pain perception permits repetitive axial loading and microfractures, while neurovascular dysregulation induces local hyperemic bone resorption. The lumbar spine and the lumbosacral junction, being subject to maximum physiological mobility and mechanical stress, represent the primary predilection sites.

Given the re-emergence of syphilis, radiologists must be familiar with these extensive destructive changes to prevent misdiagnosis as tuberculous spondylodiscitis (Pott's disease), pyogenic discitis, or osteolytic spinal metastasis. We report the characteristic computed tomography findings of advanced multi-level lumbar Charcot spine in a patient with documented neurosyphilis.

CASE PRESENTATION

Anamnesis and Clinical Examination

A 51-year-old male patient with a confirmed history of neurosyphilis complicated by tabetic arthropathy since 2024 was referred to the radiology department for the evaluation of left-sided lumboradiculopathy without motor deficit. Clinical examination revealed spinal stiffness without fever or local inflammatory signs, in the setting of a preserved general condition. Neurological evaluation showed impaired lower-extremity vibration and position sensation, consistent with underlying *tabes dorsalis*.

Laboratory Investigations

Laboratory tests showed no systemic biological inflammatory syndrome, with normal C-reactive protein (CRP) levels and leukocyte counts. Syphilitic serological markers corroborated the diagnosis of tertiary neurosyphilis.

Computed Tomography (CT) Findings

An unenhanced helical multi-detector CT acquisition of the entire spine was performed with contiguous millimetric reconstructions:

- **Cervical and Thoracic spine:** Maintained vertebral alignment and body heights without destructive osteolysis or soft-tissue collections, showing only mild degenerative disco-vertebral changes.
- **Lumbosacral spine:**
  - Straightening of the lumbar lordosis with left-convex lumbar scoliosis apexed at L4.
  - Diffuse decrease in osseous mineralization.
  - Multi-level destructive osteolytic disco-vertebral lesions extending from L2 to L5, with maximal destruction at the L4–L5 and L5–S1 segments.
  - Multiple subchondral and intra-somatic pseudocysts/geodes along the vertebral endplates.
  - Extensive osteolysis involving the posterior vertebral arches (pedicles and laminae).
  - Marked vertebral collapse, most pronounced at L4–L5, without marked retro-pulsed posterior wall fragment displacement.
  - Multiple detached bone fragments and debris scattered within the anterior and lateral paravertebral spaces, sparing the spinal canal.
  - Exuberant, dense marginal hyperostosis and productive osteophytosis, most marked at L4–L5 and L5–S1.
  - Multi-level intervertebral space narrowing, predominant at L4–L5 and L5–S1.
  - Narrowing of the osseous spinal canal diameter to 11 mm at the L4–L5 level.
  - Multi-level disc protrusions responsible for bilateral foraminal stenoses (notably left L2–L3, left L3–L4, right L4–L5, and bilateral L5–S1) causing disco-radicular impingement.
  - Well-circumscribed Lodwick IA osteolytic lesion of the right ilium, condensing bone islands in the pelvic bone, and partial fatty replacement of the lower lumbar paravertebral muscles.

Iconography and Figure Captions

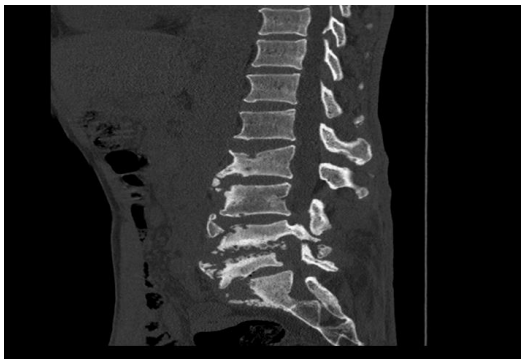


Figure1

Sagittal slice illustrating extensive disco-vertebral destruction centered on the L4–L5 level, showing vertebral collapse, complete intervertebral disc height loss, endplate pseudocysts, and marked subchondral sclerosis. Massive anterior marginal osteophytic bridging is present without significant focal retropulsion of the posterior wall.

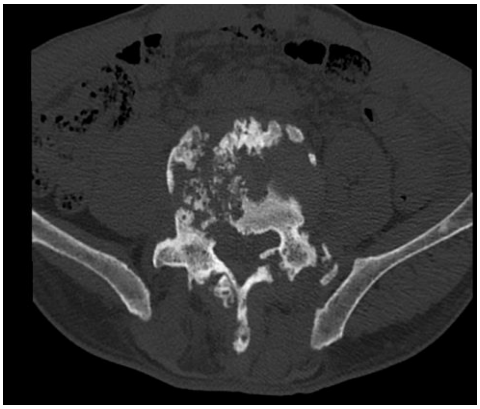


Figure 2

Severe architectural breakdown of the vertebral body characterized by central osteolysis, surrounding sclerosis, and multiple detached bone fragments (debris) extruded into the peri-vertebral soft tissues. The osseous spinal canal is narrowed to 11 mm with concomitant foraminal compromise.



Figure 3

Coronal view demonstrating left-convex lumbar scoliosis with vertebral tilt.

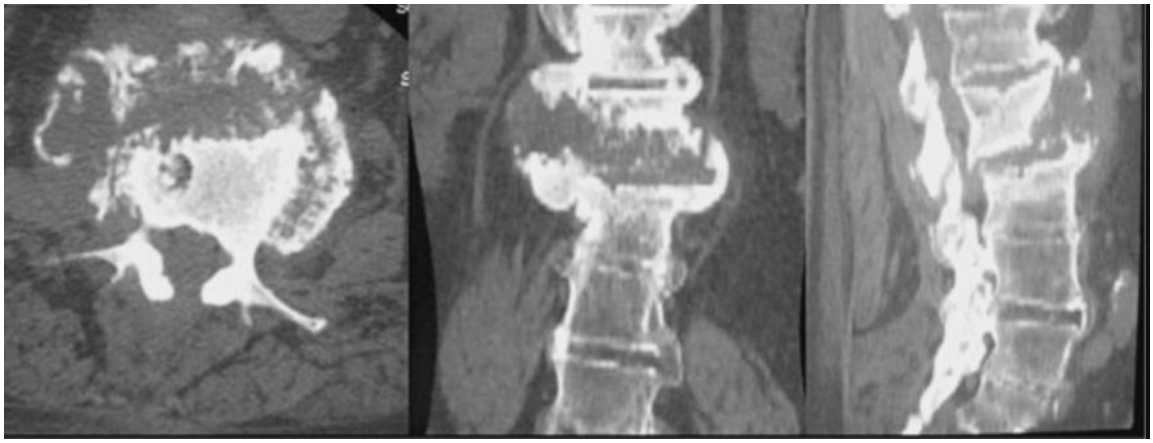
The collapsed L4–L5 space is surrounded by extensive detached bone detrition and dense reactive hyperostosis.

DISCUSSION

Tabetic neuroarthropathy of the spine is a classical clinicoradiological entity that has become infrequent but remains clinically critical.

The CT imaging features combine the classical **"6 Ds"** of Charcot neuroarthropathy:

1. **Destruction:** Severe vertebral endplate erosion and somatic collapse.
2. **Debris:** Multiple fragmented, detached bone particles extruded into the anterior and lateral paravertebral spaces.
3. **Disorganization:** Major loss of spinal alignment with scoliotic tilting and facet articulation breakdown.
4. **Dislocation:** Multi-level segment instability and subluxation.
5. **Density increase:** Pronounced subchondral eburnation and hyperostotic sclerosis bordering the lytic defects.
6. **Distension:** Intervertebral vacuum phenomena and capsular laxity.



Computed tomography (CT) scan of Charcot spine in a paraplegic patient.

Axial, coronal, and sagittal views demonstrating the characteristic combination of extensive erosive disco-vertebral lesions and exuberant hypertrophic ossification at the L2–L3 level.

Computed tomography is superior to plain radiography in characterizing the exact extent of bone destruction, quantifying the residual canal caliber (11 mm in our patient), and demonstrating detached peri-vertebral bone debris.

The primary differential diagnosis is infectious spondylodiscitis (pyogenic or tuberculous/Pott's disease). Several key clinicoradiological features support tabetic neuroarthropathy:

- **Clinicoradiological dissociation:** The magnitude of structural osseous destruction

contrasts sharply with the absence of disabling pain or febrile muscular contracture, attributable to tabetic sensory loss.

- **Absence of abscess collections:** Unlike Pott's disease, Charcot spine exhibits detached osseous debris within surrounding soft tissues rather than true peripheral rim-enhancing cold psoas abscesses.
- **Productive bone formation:** Exuberant, dense marginal osteophytosis and marked sclerosis are characteristic of Charcot joints and absent in early destructive infectious spondylodiscitis.

Osteolytic primary or secondary spinal tumors are excluded by the direct centering on the intervertebral disc spaces, preservation of cortical continuity, and hypertrophic reparative sclerosis. While complementary MRI remains helpful to assess the thecal sac and conus medullaris, CT remains the modality of choice for bone detail and surgical planning.

Added Value of Multi-Modality Imaging in Charcot Spine : Modern diagnostic imaging provides complementary, indispensable contributions to the diagnosis and management of tabetic neuroarthropathy. While plain radiography remains a common initial modality revealing gross structural deformity, bone detrition, and prominent osteophytic bridging, it routinely underestimates the true degree of acute fragmentation and neurological impingement. Magnetic resonance imaging (MRI) provides superior contrast resolution of soft tissues, the thecal sac, and the nerve roots ; it is uniquely sensitive in distinguishing neuropathic fluid-filled disc spaces from infectious epidural phlegmons or cold psoas abscesses, although bone marrow edema can occasionally be misinterpreted as active spondylodiscitis. In this diagnostic triad, high-resolution multi-detector CT bridges the gap: it definitively depicts the micro-architecture of fragmented cortical bone, quantifies bone bridge density, confirms the presence of intra-somatic vacuum clefts, and provides millimeter-accurate canal cross-sections essential for stabilizing neurosurgical interventions

CONCLUSION

Tabetic neuroarthropathy of the lumbar spine (Charcot spine) is a severe complication of tertiary neurosyphilis. Computed tomography demonstrates the simultaneous coexistence of aggressive destructive osteolysis and productive reparative changes, including vertebral collapse, peri-vertebral osseous debris, and dense hypertrophic osteophytosis. Recognizing this specific CT semiology prevents diagnostic errors and avoidable invasive procedures when encountering spinal lesions mimicking infection or malignancy.

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