Imaging and Management of the Charcot Spine Following Spinal Injury

Poster No.: P-0023  
Congress: ESSR 2012  
Type: Scientific Exhibit  
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Keywords: Musculoskeletal spine, Neuroradiology spine, Musculoskeletal joint, CT, SPECT-CT, Plain radiographic studies, Abscess delineation, Education, Abscess, Infection, Tissue characterisation  
DOI: 10.1594/essr2012/P-0023

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Purpose

Neuropathic arthropathy-Charcot spine (CS) is bone & joint destructive change that occurs secondary to loss of sensation with maintained mobility/loading. Evaluation of disease progression is a prognostic factor for operative success. We discuss the criteria for diagnosis, the main differential diagnoses, current interventional & surgical management, & the various clinical outcomes.

Methods and Materials

Charcot spine is associated with a variety of disorders, be primary or secondary to trauma. Post-traumatic Charcot spine is a silent disease that is could be diagnosed decades after the initial injury. The incidence of osteomyelitis is increased in patients with Charcot spine, is frequently occult & presents late. Suspected osteomyelitis warrants urgent intervention.

Results

We review the differential diagnosis & discriminatory imaging features of Charcot spine using various imaging modalities (plain radiographs, MRI, CT and nuclear medicine) & the spectrum of findings in various stages of the disease.

History:

Neuropathic osteo-arthropathy of the spine (Charcot spine) is named after Jean Martin Charcot who described it in a case of Tabes dorsalis. However, the first description of such an entity was made by John Kearsley Mitchel in 1831.

Mechanism:

Charcot spine is a destructive process that occurs as a result of reduced or absent protective pain and proprioceptive reflexes. Destructive changes occur in response to abnormal loading and repetitive micro trauma as the joints within the spine move beyond their natural limits, with both the micro trauma and the repair processes implicated in the pathological process. The altered muscle tone that occurs in neuropathic patients further reduces the stability of the spine and affects its alignment. The predisposing factors for the development of Charcot spine are preserved joint movement, mechanical stresses on a joint and decreased proprioception.
In the paralysed patient, passive movements of the spine during transfers are thought to be at least partially responsible. Repetitive movements without the protection of proprioceptive or pain reflexes, eventually result in chondral and osseous micro fractures. Secondary hyperaemia leads to oedema, effusions and ligamentous laxity. The bony changes that follow may be hypertrophic or atrophic. Hypertrophic changes are related to central cord lesions, and occur more frequently, resulting in sclerosis, osteophyte and pseudarthrosis formation and joint space narrowing. Atrophic changes are less common, are associated with peripheral nerve lesions, and are characterised by osteolysis. All components of the vertebral column and its supporting structures are affected, including vertebrae, discs, facet joints, ligaments and paraspinal musculature. The vertebral bone in neuropathic patients tends to be osteopaenic, further increasing the risk of micro fractures and fragmentation. Previous spinal surgery may be associated with additional instability. Charcot spine affects ambulant and non-ambulant patients. It may be isolated, or may be associated with neuropathic osteoarthropathy of other joints. The thoracolumbar junction and the lumbar spine are most frequently affected, and the pathological process may affect single or multiple vertebral segments.

**Pathology:**

Histopathological findings following biopsy include nonspecific chronic inflammatory cells and granulation tissue, reactive bone formation, cartilage fibrillation, and no evidence of infection in the tissue specimens obtained.

**Causes:**

The aetiology of Charcot spine may be primary (e.g. congenital insensitivity to pain) or it may be idiopathic. More commonly, Charcot’s spine is secondary, with causes including tabes dorsalis, diabetic polyneuropathy, syringomyelia, peripheral nerve injury, spinal AVM, paraplegia, polio, trauma /iatrogenic (including irradiation) or following direct spread from discitis. With the decline in the number of cases with tabes dorsalis due to diagnosed fall in the prevalence of syphilis over the years, trauma and diabetes are currently the leading underlying cause for Charcot spine in the developed world.

**Clinical findings:**

Charcot spine is frequently a silent process in the initial stages, and the diagnosis may be delayed by up to several decades.

1. Progressive kyphosis
2. Flexion instability
3. Reduced height
4. Back pain
5. Reduced spasticity
6. Change in bladder function
7. Grating and clicking noises in the spine on movement

**Imaging features (Plain radiographs and CT):**

As with neuropathic osteoarthropathy elsewhere within the body, the findings can be described by the 6 ‘D’s:

1. Destruction
2. Dislocation
3. Disorganisation
4. Debris
5. Distension
6. Density

Initially, plain radiographs maybe normal. Over time a progressive kyphosis, scoliosis and listhesis are seen (Figs 1,&2).
Vertebral body sclerosis and lysis may be mis-interpreted as changes secondary to advanced degenerative disease, metastatic disease or osteomyelitis.
Eventually, vertebral body collapse, fragmentation and subluxation is seen, frequently associated with paraspinal calcification (Figs 3-5). Large marginal vertebral body oetsophytes may form (Fig. 8). All three columns are involved in Charcot spine, with osseous destruction involving the vertebral body and the facet joints (Fig.6).
The vertebral end plates at an affected level may form a 'ball and socket' pseudarthrosis in late stages (Fig. 2).
A partially calcified mass may form in the paraspinal region due to chronic impaired fracture healing.
Affected disc spaces become narrowed and may contain vacuum phenomenon (Fig. 7).
Calcific debris may lead to central canal narrowing (Fig. 9, 11).

**Imaging features (MRI):**

The abnormalities seen on plain radiographs and CT described in the previous section will be present.
Reduced vertebral body signal on T1 weighted sequences and increased signal on T2 weighted signal. These osseous signal intensities occur diffusely throughout the affected vertebral bodies in Charcot spine, but are seen more typically at the endplates in discitis.
Low grade diffuse post-IV gadolinium enhancement of the affected vertebral bodies typically occurs in Charcot spine, with rim-enhancement of the involved intervertebral disc. This helps to differentiate between Charcot spine and infective discitis, with the latter showing more avid uptake centred around the endplates and diffuse enhancement of the disc.
Disc spaces are reduced in height and may contain vacuum phenomenon. The discs may be isointense to muscle on T1 weighted sequences and isointense to fluid on T2 weighted sequences (Fig 10). A paraspinal fluid collection containing calcific debris
may form, but should not be associated with any significant enhancement on post-gadolinium imaging. A diffuse change in signal underlying vertebral bodies is often noted in Charcot spine (Fig 12) whereas in discitis, the signal change is often centred at the end plates.

**Nuclear Medicine:**

Nuclear medicine has a limited diagnostic role in cases of Charcot spine. Radionuclide bone scans typically show increased isotope uptake in Charcot spine and infection. Indium-111 white blood cell scans are positive in acute infection, but negative in chronic infection and Charcot spine.

**Charcot spine vs. infective spondylo-discitis:**

Imaging features that point towards a neuropathic rather than an infective cause include:

- Involvement of all three columns (infection tends to predominantly affect the anterior column)
- Little or no paravertebral collections.
- Slower rate of progression than infective arthropathy.
- Multilevel disease with normal appearance of the segments in between.
- Disc space vacuum phenomenon is associated with a non-infective pathology.

An image-guided biopsy of the vertebral body and disc space is often required in cases where infection cannot be excluded on imaging alone (Fig 13).

**Surgical Management:**

Charcot spine should be managed with immobilisation (rest, traction and bracing). Surgical treatment may be required if there is progressive loss of shape or instability of the spine at the affected level. The affected level is approached anteriorly and the disc, end plates and adjacent bone are excised until healthy bone is reached, and a cage with bone graft is inserted together with both anterior and posterior fixation to ensure adequate stability.

**Images for this section:**
**Fig. 1:** Figure 1A: Lateral Radiographs of the thoraco-lumbar spine showing increasing kyphosis over a 6 month period in a patient with Charcot spine, distal to spinal stabilisation following a T2 complete cord injury. Mild end plate irregularity and sclerosis can be seen on the initial image.

![Lateral Radiographs of the thoraco-lumbar spine showing increasing kyphosis over a 6 month period in a patient with Charcot spine, distal to spinal stabilisation following a T2 complete cord injury. Mild end plate irregularity and sclerosis can be seen on the initial image.](image)

**Fig. 2:** Figure 1B: Lateral Radiographs of the thoraco-lumbar spine showing increasing kyphosis over a 6 month period in a patient with Charcot spine, distal to spinal stabilisation following a T2 complete cord injury. Mild end plate irregularity and sclerosis can be seen on the initial image.
Fig. 3: Figure2A: Lateral radiographs of the lumbar spine. End plate irregularity and sclerosis is present in figure A. Progressive change over 18 months has resulted in further osseous destruction of the L1-L2 end plates, and the formation of a 'ball and socket' joint.
Fig. 4: Figure 2B (18 months after Figure 2A): Lateral radiographs of the lumbar spine. End plate irregularity and sclerosis is present in figure A. Progressive change over 18 months has resulted in further osseous destruction of the L1-L2 end plates, and the formation of a 'ball and socket' joint.
**Fig. 5:** Figure 3A: Sagittal and coronal reconstructions of the thoracic spine showing disc space vacuum phenomenon, end plate destruction and irregularity, reduced vertebral body height and sclerosis of the affected vertebrae.
**Fig. 16:** Sagittal T1-W, T2-W and STIR images of the lumbar spine in a patient with Charcot spine, showing fluid signal in the disc space, end plate destruction and sclerosis. New bone formation beneath the posterior longitudinal ligament is causing spinal canal narrowing. The T12 and L1 residual vertebral bodies are mildly oedematous. An aspiration of the fluid and bone biopsy showed no evidence of infection.
Fig. 17: Sagittal T1-W, T2-W and STIR images of the lumbar spine in a patient with Charcot spine, showing fluid signal in the disc space, end plate destruction and sclerosis. New bone formation beneath the posterior longitudinal ligament is causing spinal canal narrowing. The T12 and L1 residual vertebral bodies are mildly oedematous. An aspiration of the fluid and bone biopsy showed no evidence of infection.

Fig. 18: Axial T2 and T1 weighted images showing a small fluid collection in the right psoas muscle (arrow). Note the new bone formation causing spinal canal narrowing.
**Fig. 19:** Figure 11A&B: Axial T2 and T1 weighted images showing a small fluid collection in the right psoas muscle (arrow). Note the new bone formation causing spinal canal narrowing.
**Fig. 20:** Adult patient with thoracic spine stabilisation following a complete T2 level cord injury. Sagittal T1-W, T2-W and STIR images of the whole spine showing neuropathic osteoarthropathy affecting the T10 and T11 vertebrae. Note the vertebral body oedema, end plate destruction and sclerosis. This has resulted in a mild localised kyphosis and reduced vertebral body height, most marked anteriorly. The axial T2-W image shows a spinal cord syrinx at T11.
**Fig. 21:** Figure12A,B,C&D: Adult patient with thoracic spine stabilisation following a complete T2 level cord injury. Sagittal T1-W, T2-W and STIR images of the whole spine showing neuropathic osteoarthropathy affecting the T10 and T11 vertebrae. Note the vertebral body oedema, end plate destruction and sclerosis. This has resulted in a mild localised kyphosis and reduced vertebral body height, most marked anteriorly. The axial T2-W image shows a spinal cord syrinx at T11.
**Fig. 22:** Figure12A,B,C&D: Adult patient with thoracic spine stabilisation following a complete T2 level cord injury. Sagittal T1-W, T2-W and STIR images of the whole spine showing neuropathic osteoarthropathy affecting the T10 and T11 vertebrae. Note the vertebral body oedema, end plate destruction and sclerosis. This has resulted in a mild localised kyphosis and reduced vertebral body height, most marked anteriorly. The axial T2-W image shows a spinal cord syrinx at T11.

![Figure 22 Image](image)

**Fig. 23:** Figure12A,B,C&D: Adult patient with thoracic spine stabilisation following a complete T2 level cord injury. Sagittal T1-W, T2-W and STIR images of the whole spine showing neuropathic osteoarthropathy affecting the T10 and T11 vertebrae. Note the vertebral body oedema, end plate destruction and sclerosis. This has resulted in a mild localised kyphosis and reduced vertebral body height, most marked anteriorly. The axial T2-W image shows a spinal cord syrinx at T11.
**Fig. 15:** Sagittal T1-W, T2-W and STIR images of the lumbar spine in a patient with Charcot spine, showing fluid signal in the disc space, end plate destruction and sclerosis. New bone formation beneath the posterior longitudinal ligament is causing spinal canal narrowing. The T12 and L1 residual vertebral bodies are mildly oedematous. An aspiration of the fluid and bone biopsy showed no evidence of infection.

**Fig. 14:** Axial images of the T12 vertebra showing calcified debris resulting in central canal narrowing
**Fig. 6:** Figure 3B: Sagittal and coronal reconstructions of the thoracic spine showing disc space vacuum phenomenon, end plate destruction and irregularity, reduced vertebral body height and sclerosis of the affected vertebrae.

![Image of the thoracic spine showing disc space vacuum phenomenon](image)

**Fig. 7:** Figure 4: Axial image through a thoracic vertebra in an adult patient with Charcot spine, showing calcific debris adjacent to the vertebral body and within the spinal canal.
**Fig. 8:** Axial CT image showing calcific debris in the paraspinal musculature (arrows)
**Fig. 9:** Figure 6A and B: Sagittal and coronal CT reconstructions showing bilateral pars defects (arrows) and facet joint subluxation
Fig. 10: Figure 6A and B: Sagittal and coronal CT reconstructions showing bilateral pars defects (arrows) and facet joint subluxation
**Fig. 11:** Sagittal CT reconstruction showing disc space vacuum phenomenon (arrow). Note the adjacent vertebral sclerosis.
**Fig. 12:** Coronal CT reconstruction showing the formation of large marginal osteophytes (red arrow), and osseous fragmentation in the paraspinal soft tissues (white arrow)

**Fig. 13:** Figure9A&B: Axial images of the T12 vertebra showing calcified debris resulting in central canal narrowing
**Fig. 24:** Figure 13: Lateral view showing a fluoroscopic-guided aspiration / biopsy of the disc space collection and vertebral end plate, in order to exclude infection prior to stabilisation surgery.
Conclusion

Knowledge of the radiological features of Charcot on all modalities is required to avoid mis-diagnosis and mis-management in this vulnerable group of patients.

Imaging can help to differentiate between Charcot spine, infective spondylo-discitis, advanced degenerative and metastatic disease. However, image-guided biopsy is frequently used prior to surgical planning to confirm the radiological diagnosis.

References


Personal Information

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