

## Sympathetic Chain

A number of diseases cause abnormalities in the vasculature of the hand (e.g. Raynaud's disease and Reflex Sympathetic Dystrophy). One of the modalities used in an attempt to relieve the symptoms include a sympathectomy of the inferior cervical ganglion. This can be done surgically or chemically, in which the latter is via injection of a chemical to disrupt the ganglion.

Horner's syndrome is the effect seen when the sympathetic chain is disrupted in the head/neck. It results in a 'para-sympathetic overload', namely:

- Miosis (constricted pupil)
- Anhidrosis (skin dryness)
- Ptosis (eyelid drooping)

This can be caused by upper thoracic disc prolapse, apical lung malignancy, brachial plexus trauma or iatrogenically.

## Disc Herniation

Disc herniations can be described according to either the **direction** (Slide 1, Slide 2 and Slide 3) or the layer they occur in. The descriptions are those of disc protrusion, extrusion and sequestrations, all of which can be visualized well on an MRI scan.

Before maturation, the disc herniation can occur between the ring 'apophysis' and the vertebral body proper, also known as '**Ring Apophysis Separation**'. Discal herniation into the gap between the annular ring apophysis and the underlying body results in no or partial union of the ring apophysis in adult life. The defect is contiguous with the nucleus.

End-plate irregularities (**Schmorl's nodes**) occur owing to local discal herniations at sites of previous vascular channels or cortical defects. The defect is usually well corticated and can be seen on the plain film, tomogram, CT scan, discogram (Slide 1 and Slide 2) and with MRI scanning. The discogram is generally not painful.

Defects at end-plates also occur after trauma, usually vertebral compressive in nature. Here, a cortical flake is depressed into the adjacent body; the lesions are painful in life and at discotomy, where contrast medium enters the vertebral body, before healing takes place.

## Spinal Infections

Infections involving the spinal column are an important group of conditions, which can present at any stage of life. Despite advantages in microbiology, surgical techniques and public health, there is a significant morbidity and mortality rate from these conditions.

They have traditionally been described according to the effected anatomical region, as well as the microorganisms found, and the host response. Rather than using the anatomical separation between disc infection ('discitis') and bony infection ('osteomyelitis') the term 'Infectious Spondylitis' is used to include all infections that involve the vertebral column. It has been shown in a number of series that although there are differences in clinical behaviour, the basic pathology is the same.

Intra-canal infections are described separately, as their presentation and course are distinct from vertebral column infections.

Irrespective of the site of infection or microorganism involved, there is only a small number of ways that the spine can be infected.

1) Septic embolic spread; this may be via the arterial, venous, or lymphatic routes, entering the vertebral body, or seeding in the epidural plexus. In nearly all adults, and occasionally in children, a primary source of infection can be identified. The most common ones being from the genito-urinary tract, soft tissue infections, and the respiratory tract.

- 2) Latrogenic; from investigative as well as therapeutic procedures, and finally,
- 3) Contiguous spread from adjacent tissue.

## ***Imaging***

### ***Plain Radiographs***

The plain film appearances lag behind changes demonstrated with radionuclide bone scanning and MRI. Local loss of bone density is followed by cortical destruction, especially well seen by comparison with normal end-plates elsewhere. Disc destruction follows and the disc height is lost (**Slide 1** and **Slide 2**). In malignant disease, loss of disc height follows herniation into the diseased body without actual loss of discal mass. Similarly, end-plate depressions in Scheuermann's disease also result in loss of disc height, but the end-plates are sclerotic, even if irregular.

Subsequent extension into the adjacent vertebral bodies causes further bone destruction, involving first the cortex and then the medulla. Overlying gut gas may obscure these changes. Linear tomography shows the lesions more clearly and gives a more accurate estimation of the area of destruction, often showing it to be larger than the area seen on the plain film.

### ***Radionuclide Studies***

While the radiographs may be normal initially, the radionuclide bone scan will be abnormal early on in the disease. Gallium scans, when used in conjunction with technetium scans, have an accuracy of about 94%. However, gallium and technetium scans may be falsely negative in leukopenic patients and patients that suffer from relative ischaemia.

### ***Computed Tomography***

Computed tomography (CT) scanning with sagittal and coronal reconstruction is of similar value in assessing cortical, **medullary and disc destruction**. Soft tissue masses around infected vertebrae are also clearly seen in axial scans.

### ***Magnetic Resonance Imaging***

Magnetic resonance imaging (MRI) is the modality of choice for diagnosing spinal infection (**Slide 1** and **Slide 2**) and exceeds any other modality currently available, as it is 95% accurate. MRI demonstrates both anatomic change and local features of inflammation. Trabecular and end-plate destruction, and marrow and discal inflammatory changes are seen. If marrow is mainly red, inflammatory changes are best seen as an increase in signal on T2-weighted sequences or as an area of signal loss in fat on T1-weighted images. Gadolinium results in an increase in signal on T1-weighted sequences. T1-weighted sequences have decreased signal intensity, with an indistinct margin between the two.

Infection may arise directly in the disc, especially in adolescents, as blood vessels still penetrate the end-plates to enter the discs. Subsequently the disease spreads to adjacent end-plates and vertebral bodies.

Subligamentous spread beneath the anterior longitudinal ligament causes anterior erosions of adjacent vertebral bodies. Here, soft tissue masses are especially prominent and enhance with gadolinium. In all cases, posterior extension into the spinal canal is demonstrated with CT and MR imaging.

The presence of a vacuum phenomenon indicates an absence of inflammatory change; when a vacuum phenomenon vanishes and adjacent end-plates lose density or clarity, infection in the elderly patient is quite likely.