

## ACUTE 'SLIPPED DISC'

### DEFINITION

Acute extrusion of the nucleus pulposus of an intervertebral disc.

### EPIDEMIOLOGY

- Common.
- Incidence – acute cervical myelopathy related to disc: 2 (95% CI: 0.2–6) per 100 000 per year.
- Age: young adults or middle-aged.

### PATHOLOGY

- The prolapse is usually posterolateral rather than central.
- The lumbar discs are most often affected (L4/5 or L5/S1), cervical discs occasionally (C7 root involved in 70% of cases of cervical disc protrusion, C6 root in 20% of cases, C5 and C8 roots in the remaining 10%), and thoracic discs rarely.

### ETIOLOGY AND PATHOPHYSIOLOGY

- Acute slipped disc may be spontaneous or precipitated by sudden spine movement (e.g. sudden hyperextension of the neck, diving, forceful chiropractic manipulations), lifting and straining.
- A prolapsed cervical disc compresses the nerve root and spinal cord at that level (e.g. a laterally situated disc protrusion between the sixth and seventh cervical vertebrae compresses the seventh cervical root) (676), whereas a prolapsed disc in the lumbar region (e.g. at L4/5), where nerve roots are angulated, usually compresses the root passing to the foramen below (e.g. the L5 root) (685).
- Nerve root compression usually involves only one nerve root on one side (perhaps two) but a central lumbar disc prolapse may compress most of the cauda equina, causing multiple lumbosacral root lesions and sphincter disturbance.
- Spinal cord compression by central cervical or thoracic discs (at or above the level of the conus medullaris, which is at the level of the L1/2 intervertebral disc) causes upper motor neuron signs.

### CLINICAL FEATURES

- Acute pain, tenderness and paraspinal muscle spasm at the level of the prolapse. N.B. Central cervical disc herniations, compressing the spinal cord, may be painless.
- Nerve root (radicular) pain, which is sharp, burning or 'electric', and severe; combined with sensory disturbance; in the dermatomal distribution of the compressed nerve (677, 678, Table 45, 686).
- Pain: exacerbated by coughing, sneezing, straining, moving (bending and stooping), straight leg raising (low lumbar and sacral root compression), and femoral stretch test (upper lumbar roots); relieved by rest and particular postures.
- Muscle wasting and weakness and depressed tendon reflex in myotomal distribution (see Tables 46, 47).

### HISTORY

- Neck/back pain: location (radicular), onset (usually acute, ?precipitating factors), timing (?nocturnal), nature (sharp), intensity (severe), radiation (dermatomal), duration

(seconds-minutes-hours), associated symptoms (e.g. limb weakness or sensory disturbance, bladder or bowel dysfunction, weight loss, fever), exacerbating factors (e.g. cough, sneeze, strain) and relieving factors and body positions.

- History of trauma, weight loss, malignancy, other systemic illness.
- Medication/drug history (cf. ?analgesic abuse).
- Explore secondary gain phenomenon such as litigation, worker's compensation issues, psychiatric problems, narcotic abuse.

### EXAMINATION

- Gait: examine a few steps while walking 'normally', on toes, on heels, heel-to-toe; hopping on either leg, arising from a squat, Rhombert's sign.
- Spinal tenderness or spasm.
- Limited range of motion of the spine.
- Aggravation of pain with movement of the spine (particularly hyperextension of the neck and downward pressure on the head in the hyperextended position with cervical disc protrusion).
- Straight-leg raising (Lasegue's sign).
- Limb muscle bulk, tone, power, deep tendon reflexes, sensation (including perianal sensation and anal tone if a cauda equina syndrome is suspected).
- Signs of systemic disease, infection or malignancy.

### DIFFERENTIAL DIAGNOSIS

#### Painless cervical myelopathy (see p.545)

- Subacute degeneration of spinal cord.
- Motor neuron disease.

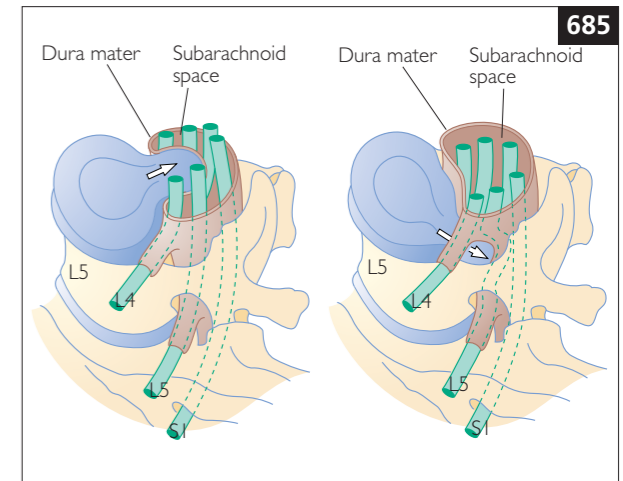
#### Back pain with or without radiculomyelopathy

- Discogenic or degenerative spinal stenosis:
  - Herniated intervertebral disc.
  - Degenerative spondylosis:
    - Central canal stenosis.
    - Lateral recess stenosis.
  - Synovial cyst of facet joint.
- Metabolic bone disease: osteoporotic compression fracture.
- Spinal trauma.
- Spinal tumor:
  - Primary intradural tumor of spinal cord, medullary cone or cauda equina.
  - Tumor of vertebral column or epidural space (or both):
    - Metastases.
    - Plasmacytoma or multiple myeloma.
    - Primary bone tumor.
  - Extraplural retroperitoneal malignant lesion.
- Vascular lesion:
  - Acute epidural hematoma.
  - Spinal cord infarction.
  - Arteriovenous malformation of the spinal cord.
  - Spinal dural arteriovenous fistula.
- Infection:
  - Intervertebral discitis or osteomyelitis.
  - Epidural or subdural abscess.
  - Urinary tract infection.
  - Herpes zoster or other viral-based radiculopathy.
- Intra-abdominal or intrapelvic pathology:
  - Abdominal aortic aneurysm.
  - Posterior perforating duodenal ulcer.
  - Pancreatic disease.
  - Endometriosis.

- Degenerative hip disease.
- Diabetic radiculoneuropathy.
- Acute inflammatory demyelinating polyradiculopathy (Guillain-Barré syndrome).
- Congenital:
  - Tethered cord.
  - Intraspinal lipoma.

### Radiculopathy

Mononeuropathy: e.g. ulnar nerve palsy may be mistaken for compression of the eighth cervical root by a disc protrusion at C7–T1.



**685** Illustration showing the effect of a lumbar disc prolapse at L4/5 which is central (illustration on the left), compressing many or all of the nerve roots below L4, particularly the most anterior roots (L5, S1, S2); and lateral (illustration on the right), compressing the L5 nerve root (and sometimes the S1 nerve root) and displacing the root pouch on the side of the prolapse.

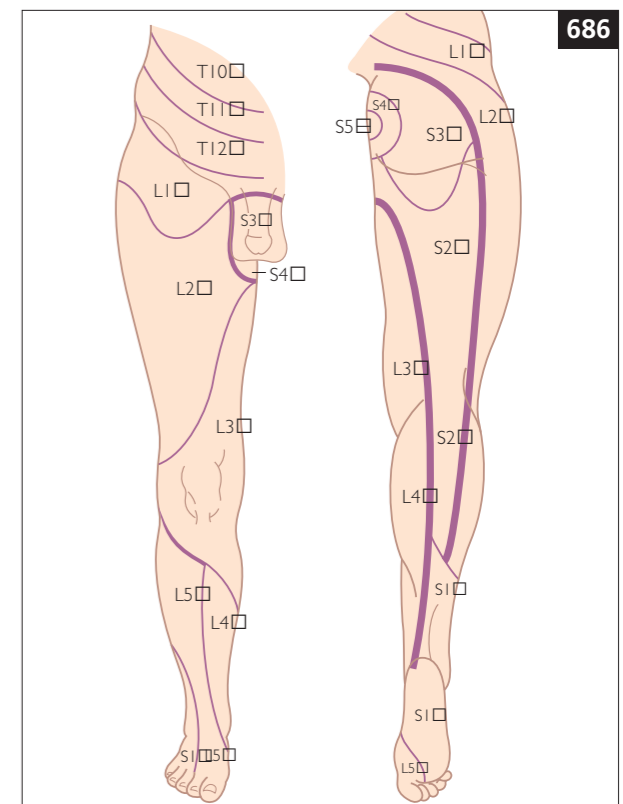
**Table 45 Nerve root (radicular) pain and sensory disturbance in the dermatomal distribution of the compressed nerve (677, 678 and see p.545)**

C5:	lateral shoulder and upper arm
C6:	lateral forearm, thumb and index finger
C7:	middle finger
C8:	ring and little fingers and medial forearm
T1:	medial forearm and upper arm
L4:	pain radiates down the leg to the thigh, knee, medial lower leg and medial malleolus and sometimes the groin
L5:	lateral lower leg; lateral malleolus and dorsum of the foot
S1:	heel, lateral border and sole of the foot
S2:	back of leg
S3,4,5:	perineal and perianal region (686).

Pain: sharp, 'electric', and severe, and exacerbated by coughing, sneezing, straining, moving (bending and stooping), straight leg raising (low lumbar and sacral root compression), and femoral stretch test (upper lumbar roots). Relieved by rest and particular postures

**Table 46 Innervation of spinal segments and muscles**

Spinal segment	Muscle	Action
C5, C6	Deltoid	Shoulder abduction
C5, C6	Biceps	Elbow flexion
C6, C7	Extensor carpi radialis	Wrist extension
C7, C8	Triceps	Elbow extension
C8, T1	Flexor digitorum profundus	Hand grasp
C8, T1	Hand intrinsic	Finger abduction
L1, L2, L3	Iliopsoas	Hip flexion
L2, L3, L4	Quadriceps	Knee extension
L4, L5, S1, S2	Hamstrings	Knee flexion
L4, L5	Tibialis anterior	Ankle dorsiflexion
L5, S1	Extensor hallucis longus	Great-toe extension
S1, S2	Gastrocnemius	Ankle plantar flexion
S2, S3, S4	Bladder and anal sphincter	Voluntary rectal tone



**686** Distribution of the lumbar and sacral dermatomes in the lower abdomen, buttocks and legs.

**Table 47 MRC grading scale for evaluation of muscle strength**

MRC grade	Muscle strength
5	Normal strength
4	Active power against both resistance and gravity
3	Active power against gravity but not resistance
2	Active movement only with gravity eliminated
1	Flicker or trace of contraction
0	No movement or contraction

**INVESTIGATIONS****Screen for non-discogenic causes of back pain**

- Plain radiography of the relevant part of the spine (anteroposterior and lateral projections).
- Full blood count.
- ESR.
- Urea and electrolytes, glucose, calcium, phosphate and liver function tests.
- Urinalysis.
- Prostatic specific antigen (in men older than 50 years of age).

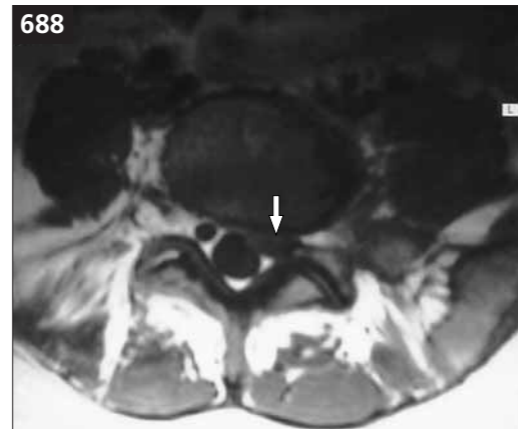
**Electromyography**

EMG can help confirm the clinical impression of the absence or presence, site and severity of a radiculopathy and may reveal an unsuspected polyradiculopathy or peripheral neuropathy.

**Imaging of lumbar spondylosis**

- Plain films may demonstrate narrowing of individual disc spaces and the spinal canal, either congenital or secondary to degenerative joint disease, but are rather non-specific and do not show the contribution from any soft tissue component.
- CT is very good and can be set up to cover the whole lumbar spine, by imaging through each disc space and the mid point of each vertebral body. Thus osteophytes, disc herniations, ligament hypertrophy, facet joint hypertrophy, lateral recess stenosis and exit canal narrowing can be demonstrated at all the lumbar levels. When disease is suspected at a specific level, then images of the corresponding disc space plus the one above and below are usually obtained. If there has been previous disc surgery the examination should be done before and after intravenous contrast.

**687, 688** T2W sagittal (**687**) and T1W axial (**688**) MRI of an L5/S1 disc (arrows) herniation.



- Myelography will demonstrate canal narrowing, nerve root displacement, thickening and cut off, disc herniations and the number of levels involved, but is invasive.
- Myelography with water-soluble contrast medium followed by CT (myelogram-CT) images the subarachnoid space and may reveal cauda equina tumors and other intradural lesions that can mimic lumbar disc herniation.
- MRI is excellent for imaging the lumbar spine and shows the features of disc herniation, canal stenosis and so on (**687–691**). As with CT, a pre- and post-contrast exam is required if there has been previous surgery. T1W and T2W midline sagittal followed by T1W axial images are routine. Absolute contraindications include the presence of a pacemaker or ferromagnetic implant (e.g. intracranial vascular clip); relative contraindications include claustrophobia, obesity and the need for advanced life support.

**DIAGNOSIS**

Diagnosis is based on findings of nerve root pain, objective neurologic signs of spinal cord and/or nerve root compression, and clear-cut evidence of a herniated disc on MRI or myelogram-CT that correlates precisely with the clinical findings.

**TREATMENT****Conservative**

- Reassure patients that they most likely have a benign, self-limited condition and that symptoms should rapidly resolve without active treatment. Hospitalization is usually not needed.
- Simple analgesia: aspirin, non-steroidal anti-inflammatory drugs, paracetamol (acetaminophen); narcotic analgesics are usually unnecessary.
- Muscle relaxants (e.g. cyclobenzaprine hydrochloride or methocarbamol) for a short term in selected patients with pronounced paraspinal muscle spasm.
- Bed rest: short term (2–3 days), on a firm mattress or board.
- Immobilization of the involved spine: avoid movements of the involved spine and strain on the spine; for cervical disc protrusions use a neck collar that is rigid enough to prevent neck movement, particularly if flexion and extension are painful (a soft collar may be more tolerable at night); for lumbar disc prolapses, use a firm lumbar corset. Use a chair with adequate back support, and change positions frequently. Control body weight.

- Begin a physiotherapy programme: aerobic exercise as tolerated but avoiding heavy lifting and repetitive bending and twisting of the back. Traction, manipulation, ultrasound therapy, transcutaneous electric stimulation, and application of heat and cold can be effective.
- Lumbar epidural corticosteroid injections may be of benefit in selected patients.
- *Reassess the few patients who have not experienced improvement within 6 weeks.*

**Surgical decompression**

- Emergency:
  - Acute cord compression.
  - Acute bladder disturbance due to cauda equina compression by a central lumbar disc prolapse.
- Urgent: severe or progressive loss of motor function due to a monoradiculopathy; trivial root signs such as absent tendon reflex are not an indication for surgery.
- Elective: continuing nerve root pain with a mild to moderate neurologic deficit which has not responded to about 6 weeks of conservative treatment.

Lumbar discectomy with magnified vision is the 'gold standard' operation for lumbar disc disease; various closed or percutaneous procedures, such as chemonucleolysis, percutaneous automated discectomy, and endoscopic discectomy are being trialled. Chymopapain is more effective than placebo for lumbar disc prolapse, but discectomy may lead to better clinical outcomes with fewer second procedures than chymopapain.

Surgical complications include great vessel and cauda equina injury and deep spinal infection but are uncommon.

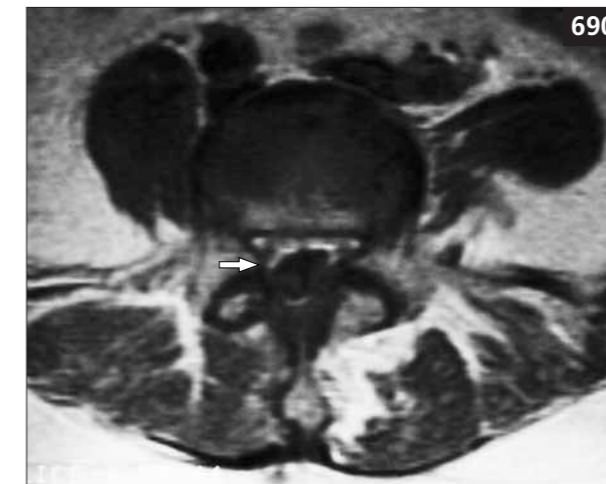
**PROGNOSIS**

- Acute neck and low back pain, in the absence of tumor and other serious underlying disease, usually resolves rapidly within 4–6 weeks.
- Lumbar discectomy is successful in 80–90% of patients, if properly selected.
- Patients with severe cauda equina syndrome due to massive midline disc herniation have a guarded prognosis for neurologic recovery, even with prompt disc removal and neural decompression.

**689, 690** T1W axial pre- (**689**) and post contrast (**690**) MRI of the lumbar spine in a patient with fibrosis following previous lumbar disc surgery. Note the enhancing tissue around the right hand side of the spinal theca (arrow).



**691** T2W sagittal MRI of thoracic discs (arrows).



## SPINAL EPIDURAL HEMATOMA

### DEFINITION

Hemorrhage in the spinal epidural space.

### EPIDEMIOLOGY

- Uncommon.
- Age: any age.
- Gender: M=F.

### PATHOLOGY

- Blood in the spinal epidural space.
- Direct compression of the spinal cord or roots by the mass effect of the hematoma.
- Spinal cord/nerve root pressure necrosis.

### ETIOLOGY AND PATHOPHYSIOLOGY

- Trauma.
- Spinal extradural arteriovenous malformation (692).
- Vertebral body hemangioma.
- Epidural metastases.
- Arteritis.
- Hypertension.
- Bleeding diathesis due to a coagulopathy.
- Anticoagulant therapy.
- Cocaine injection.
- Epidural anesthesia.
- Lumbar puncture in patients with a bleeding diathesis.
- Idiopathic.

### CLINICAL FEATURES

Sudden and severe neck or back pain, followed in minutes to hours by progressive motor, sensory and sphincteric disturbances referable to radicular spinal cord or cauda equina origin. Other clinical features are determined by the underlying cause (e.g. vasculitis causing associated skin rash [693–695]).

### DIFFERENTIAL DIAGNOSIS

- Hemorrhage into the spinal cord (hematomyelia) or spinal subarachnoid space (693).
- Intervertebral disc prolapse.
- Vertebral body collapse (spinal angulation: tuberculosis, tumor).
- Trauma.
- Spinal cord infarction.
- Epidural abscess.

### INVESTIGATIONS

#### Blood

- Full blood count.
- ESR.
- Coagulation profile.
- Serum chemistry profile.
- VDRL/RPR and TPHA.
- Antinuclear antibody titer.
- Urine test for cocaine/amphetamine metabolites.

### MRI spine

MRI is the primary method of diagnosis (693). The findings depend on the timing of the MR scan after the onset of the hemorrhage. At about 36 hours after onset, T1W MRI of the spine usually shows an isointense collection in the epidural space, usually anterior, with or without cord or nerve root compression. An underlying spinal vascular malformation may be seen.

### CT spine

CT may also show an epidural mass, which does not tend to enhance with contrast.

### Chest x-ray

Hilar lymphadenopathy, pulmonary nodules or interstitial lung disease may be seen in patients with arteritis causing spinal epidural hemorrhage.

### Spinal angiography

May identify the source of the hemorrhage and even distinguish between the several types of vascular malformations and hemangioblastomas, and localize them accurately to the vertebral bodies, epidural or subdural space, or spinal cord.



**692** Autopsy specimen of spinal cord showing an epidural spinal arteriovenous malformation (a common cause of spinal epidural hematoma), containing dark clotted blood. (Courtesy of Professor BA Kakulas, Royal Perth Hospital, Western Australia.)



**693** MRI cervical and upper thoracic spine, T1W image, sagittal plane, showing a linear streak of high intensity due to hemorrhage in the spinal subarachnoid space in a patient with arteritis due to Churg–Strauss syndrome (allergic angitis and granulomatosis).

## SPINAL EPIDURAL ABSCESS

### DEFINITION

Pus or infected granulation tissue in the spinal epidural space.

### EPIDEMIOLOGY

- Rare.
- Age: any age; children or adults.
- Gender: either sex.

### PATHOLOGY

- An abscess containing pus or infected granulation tissue in any part of the spinal epidural space, most commonly the dorsal aspect of the thoracic spinal cord, where the loose attachment of the dura permits rapid spread of infection.
- Direct compression of the spinal cord or roots by the mass effect of the abscess.
- Spinal cord infarction secondary to pressure and thrombophlebitis of the local venous channels.

### ETIOLOGY

- *Staphylococcus aureus*: most common.
- Streptococci.
- Brucella (a Gram-negative coccobacillus which may infect people in close contact with infected animals and carcasses [camels, cows, goats, sheep] or after drinking infected milk).
- Salmonella.
- Fungus.
- Tuberculosis (696) (complications include nerve root and/or spinal cord compression, arteritis and spinal cord infarction, spinal arachnoiditis, and spinal cord tuberculoma).
- Anaerobic organisms.

### CSF

Blood and xanthochromia are present in the spinal fluid, if there has been subarachnoid hemorrhage or hemorrhage into the spinal cord (hematomyelia).

### DIAGNOSIS

MRI spine or spinal angiography in a patient with appropriate clinical syndrome.

### TREATMENT

- Prompt neurosurgical decompressive laminectomy, particularly if acute onset and neurologic function is deteriorating.
- Microsurgical resection or embolization of the cause (e.g. if a vascular malformation is present).

### PROGNOSIS

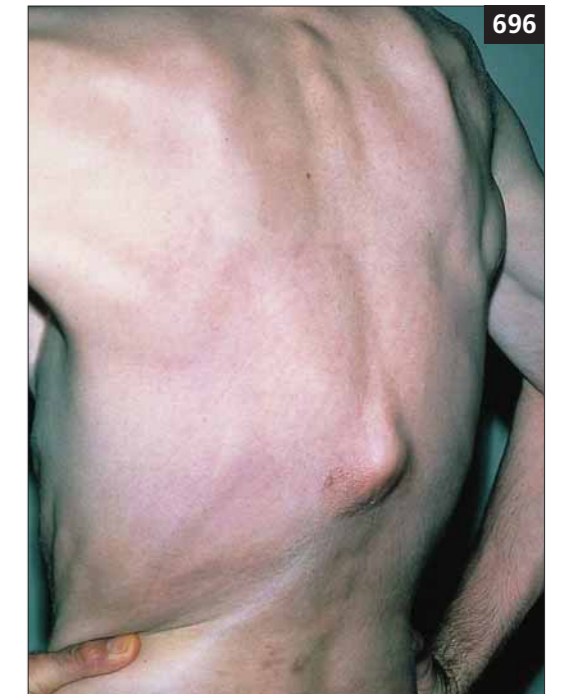
Depends on the site, duration and degree of spinal cord and nerve root compression, and the underlying cause of the hematoma.



**694, 695** Hands (694) and shin (695) of the patient with Churg–Strauss syndrome (693) showing the typical skin rash.



**696** Spine of a patient with a tuberculous spinal epidural abscess due to localized spinal meningitis (Pott's disease of the spine) with vertebral body collapse and destruction of the adjacent intervertebral discs.



**PATHOPHYSIOLOGY**

Seeding of the spinal epidural space or a vertebral body with micro-organisms occurs under the following circumstances:

- Septicemia complicating a chronic medical illness (e.g. diabetes, infective endocarditis, immunocompromise) with seeding of the spinal epidural space or of a vertebral body.
- Vertebral osteomyelitis.
- Infected intervertebral disc.
- Penetrating or non-penetrating back injury at the time of furunculosis or other skin or wound infection.
- Local spinal surgery: laminectomy.
- Spinal or epidural anesthesia.
- Lumbar puncture.

**CLINICAL FEATURES**

- Subacute onset.
- Systemic upset: fever, headache, malaise.
- Back pain and tenderness to palpation/percussion: local and severe.
- Neck/spine rigidity.
- Nerve root pain.
- Nerve root and/or spinal cord compression/infarction: progressive paraparesis, sensory loss and sphincter paralysis (urinary and fecal retention).

**DIFFERENTIAL DIAGNOSIS**

- Epidural hematoma (see p.554).
- Acute intervertebral disc prolapse (see p.550).
- Subdural abscess: spinal imaging often reveals a less sharp margin and a greater vertical extent of the abscess than an epidural abscess.
- Transverse myelitis (see p.561).
- Spinal cord abscess.
- Spinal cord infarction (see p.558).

**INVESTIGATIONS****Blood**

- Neutrophil leukocytosis.
- Raised ESR.
- Serology may be positive for infectious organisms such as brucella.

**MRI spine**

The imaging method of choice (697–699). The findings are similar to those of epidural tumors (see p.567). Bone involvement varies as the abscess can simply sit as a cuff in the spinal canal with minimal bone involvement, though more commonly bone involvement is substantial. The cord appears normal width or narrowed, with the CSF obliterated around it at the level of the lesion, but visible above and below, and a mass around the outside plus or minus bone destruction. MRI not only demonstrates the extent of the symptomatic lesion, but also other lesions that may be at other levels and may be asymptomatic.

**Spine x-ray**

Bone changes of osteomyelitis.

**Myelography (with or without CT)**

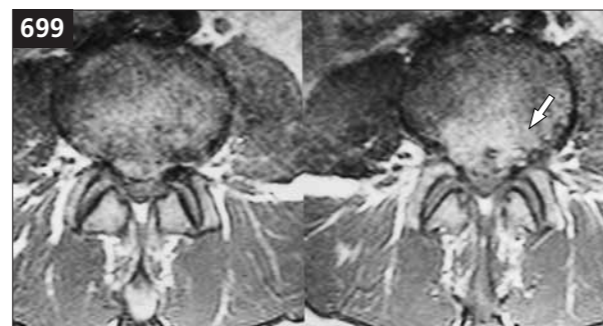
Shows spinal cord compression. It may only show up to the lower level of the lesion, as if the block is complete, the contrast will not pass it to outline the cord above.

**CSF**

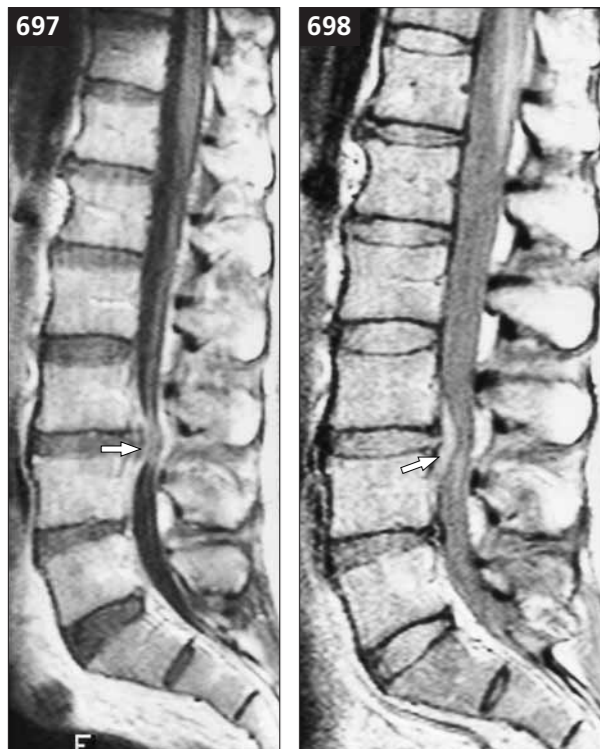
- Cells: mild pleocytosis: <100 white cells/mm<sup>3</sup>, both polymorphonuclear leukocytes and lymphocytes; frank pus if the needle penetrates the abscess.
- Protein: quite high: 1–4 g/l (100–400 mg/dl).
- Glucose: normal.
- Culture: often sterile.

**DIAGNOSIS**

The definitive pre-surgical diagnosis can only be made with an MRI scan, indicating the extent and localization of the lesion, in combination with clinical, serologic, microbiologic and/or pathologic information.



**697–699** MRI lumbar spine, T1W (697) and dual echo T2W (698) images in the sagittal plane from T11 to S3, and axial T1W (699) images at the L3/4 disc level showing at the L3/4 disc level an anterior epidural soft tissue mass which causes severe canal stenosis. The soft tissue mass (arrows) is of intermediate signal intensity on T1W images with increased signal on T2W images and minimal contrast enhancement. The differential diagnosis includes epidural abscess, tumor or hemorrhage.

**TREATMENT**

- Surgical decompression by laminectomy and drainage as soon as possible if the spinal cord is compressed.
- Antibiotics appropriate for the infecting organism must be given (e.g. for brucellosis: doxycycline, 200 mg orally, once daily, plus rifampicin (rifampin), 900 mg orally, once daily for at least 6 weeks, or tetracycline, 500 mg orally, four times daily for 6 weeks plus streptomycin, 1g daily i.m. for 3 weeks).

**PROGNOSIS**

- A fibrous or granulomatous reaction may develop at the site of apparently successful surgical drainage and give rise to a slowly progressive, then stabilizing syndrome of incomplete cord compression.
- If allowed to smoulder due to delayed diagnosis or inadequate therapy, a chronic adhesive meningomyelitis may evolve.

**SPINAL ARACHNOIDITIS****DEFINITION**

A non-infectious or post infectious aseptic inflammatory process of the leptomeninges which may or may not be associated with other disease of the spine or spinal cord and results in a progressive overgrowth of fibrous tissue in the subarachnoid space.

**EPIDEMIOLOGY**

- Uncommon.
- Age: any age; most commonly 40–60 years of age; rarely younger than 20 years.
- Gender: M=F.

**ETIOLOGY**

- Myelography using oil based contrast media (pantopaque, Myodil [iopendylate]). These substances had to be removed from the CSF space by aspiration through the lumbar puncture needle once the procedure had finished. Due to the oily nature it was impossible to remove all of it. The contrast excited an inflammatory reaction leading to arachnoiditis. One occasionally still sees plain x-rays of the spine with little radiopaque droplets overlying the spinal canal indicating a previous Myodil myelogram. Modern water-soluble contrast media rarely, if ever, cause arachnoiditis.
- Intrathecal administration of penicillin and other antibiotics, spinal anesthetics (a detergent that had contaminated vials of procainamide), and repeated corticosteroid injections.
- Repeated spinal surgery (e.g. for lumbar discs).
- Subarachnoid hemorrhage.
- Meningeal infection:
  - Viral: lymphocytic choriomeningitis.
  - Bacterial: acute: meningococcal, gonococcal, listeria; subacute or chronic: tuberculosis, syphilis, cryptococcus.
  - Worms: cysticercosis.
- Ankylosing spondylitis.

**PATHOGENESIS**

- The arachnoid is an avascular membrane which lies between the vascular pia and the vascular dura. It has a limited capacity to participate in an inflammatory response.
- In response to injury or irritation, a reactive inflammatory response begins in the vascular pia and dura and progresses to a chronic stage with fibrous thickening of the arachnoid and adhesions between the pia and the dura.

**PATHOLOGY**

- Opacification and thickening of the arachnoidal membranes.
- Adhesions between the arachnoid and dura due to proliferation of connective tissue in response to an antecedent arachnoidal inflammation.
- Obliteration of the subarachnoid space.
- Strangulation of nerve roots and the spinal cord by thickened connective tissue, causing compression and progressive vascular occlusion and ischemia of the spinal roots and cord, and obstruction of CSF flow at the foramen magnum, possibly causing hydrocephalus and syringomyelia.
- Variable destruction of the peripherally placed fibers of the spinal cord.
- Secondary degeneration of the dorsal and lateral columns of the spinal cord.
- Usually diffuse, with a predilection for the thoracic segments.
- Occasionally confined to relatively circumscribed sections of the cord or subarachnoid space, giving rise to loculated collections of fluid (meningitis serosa circumscripta).

**CLINICAL FEATURES****Asymptomatic****Myeloradiculopathy**

- Onset: concurrent with acute arachnoidal inflammation or delayed for weeks, months or even years.
- Dorsal sensory nerve root compression:
  - Spine and/or radicular pain in the distribution of one or more sensory nerve roots, initially on one side and then bilaterally. Persistent and burning, stinging or aching.
  - Depressed deep tendon reflexes.
- Ventral motor nerve root compression: muscle wasting and weakness (particularly with cauda equina involvement).

**Spinal cord compression**

Spastic ataxic paraparesis, slowly progressive (may follow months or years after radicular symptoms).

**Signs of the underlying cause**

Previous disc surgery, Myodil (iopendylate), infection, subarachnoid hemorrhage, malignancy.