Herniated Disc (Spinal Disc Herniation) — Symptoms and Diagnosis

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Spinal disc herniation is a common cause of pain syndromes involving the cervical and lumbar spine. It is the most common trigger of radiculopathies, which lead to pain and abnormal sensation of the dermatomes that may also be accompanied by pareses and loss of reflexes. A knowledge of radicular syndromes along with their diagnosis and treatment is highly meaningful in daily life as well as for examinations.

Definition of Spinal Disc Herniation

Spinal disc herniation: slipped disc

A spinal disc protrusion is a bulging disc with intact annulus fibrosus.

However, spinal disc herniation (also known as slipped disc) is characterized by the perforation of the annulus fibrosus and expulsion of the nucleus pulposus through the area of perforation. The posterior longitudinal ligament of the spine (lig. longitudinale posterius) may be intact or perforated in such cases.

Disc sequestration is marked by a perforated posterior longitudinal ligament with fragments of nucleus pulposus in the spinal canal.

Symptoms caused by spinal disc abnormalities are varied and may include lumbago (also called lumbalgia) and lumboischialgia as well as cervicobrachialgia.

Lumbago (‘low back pain’) is described as acute or chronic, non-radiating pain in the lumbar spinal region.

Lumboischialgia or cervicobrachialgia is associated with pain in the lumbar and
cervical spine, respectively, which radiates to the extremities. The segmental radiation of pain is caused by irritation of the nerve root.

**Radicular syndrome** (also known as *nerve root compression* or *radiculopathy*) describes the collection of symptoms that are triggered by compression or mechanical irritation of a spinal nerve root, which may include segmentally radiating pain seen in lumboischialgia and cervicobrahialgia.

**Note:** Spinal disc herniation is the most common cause of radicular syndrome. Acute inflammation, fractures as well as chronic degenerative, inflammatory and neoplastic processes can trigger radicular symptoms as well.

**Epidemiology of Spinal Disc Herniation**

**Common conditions in spinal disc herniation**

Low back pain (*lumbago*), which has a point prevalence of 37% and a lifetime prevalence of 87%, is the most important pain syndrome immediately following headache.

Pain in the cervical spine radiating to arms and shoulders (*cervicobrahialgia*) is another common concern.

Degenerative disc disease is a common cause of *vertebral pain syndromes*. The lumbar spinal region is most commonly affected (about 2/3 of all cases), followed by the cervical spine. Thoracic spinal disc herniation is quite rare.

Spinal disc herniation most commonly affects individuals aged 20–65 years with a maximum prevalence between ages 45 and 55 years. Men are affected more than women.

**Etiology of Spinal Disc Herniation**

**Causes of spinal disc herniation**

Spinal disc herniation is triggered by a fateful degeneration of the spinal disc with fissures of the *annulus fibrosus*. Conversely, the traumatic destruction of a previously intact spinal disc is extremely rare.

A prolapsing spinal disc causes myelocompression (myelopathy), *conus medullaris and cauda equina* (conus and cauda syndromes), or irritation of the respective spinal nerve roots (*radiculopathy*).
Spinal discs (discus intervertebrales) are responsible for spine movement and cushioning. With advancing age, the number of blood vessels reaching the discs decreases, which increases the risk of fibrosis of the jelly-like nucleus pulposus while the surrounding annulus fibrosus loses its taut texture. These changes are already observed histologically in early adulthood.

For example, tears in the annulus fibrosus under high exertion can lead to prolapse of the nucleus pulposus and spinal disc herniation. In case of spinal sequestration, fragments of nucleus pulposus are found in the spinal canal along with perforation of the posterior longitudinal ligament. This defect occurs predominantly in the lateral aspects of the posterior longitudinal ligament since it is most strongly attached to the annulus fibrosus at the midline.

Pathology and Pathophysiology of Spinal Disc Herniation

Affected structures in spinal disc herniation

The spinal disc tissue can shift medially, mediolaterally or laterally during spinal disc herniation. Most commonly, mediolateral herniations damage nerves in the cervical and lumbar region, which innervate the vertebra located beneath the disc.

In the case of herniation of the disc between C6 and C7, the C7 nerve root is affected as the nerve roots in the cervical spine extend above the assigned vertebral body. The cervical spine includes seven vertebrae but eight nerve roots. In case of a herniation between L5 and S1, the S1 is the affected root.
Lateral herniation in the **lumbar spine** damages the nerve root belonging to the higher vertebra as well. As shown in the anatomy of the lumbar spine, the nerve roots leave the spinal canal underneath the corresponding vertebra while the nerve roots of the next lower segment exit medially. Therefore, a lateral herniation at L3/4 can cause **L3 syndrome**, whereas a mediolateral herniation triggers **L4 syndrome**.

**Note:** Prolapse of the cervical spinal disc often occurs at the level of C5/6 and C6/7, while lumbar prolapse most often occurs at L4/5 and L5/S1 levels.

1. **Spinal cord**
2. Dorsal root
3. Spinal ganglion
4. Ventral root
5. Spinal nerve
6. Spinal disc
7. Spinal disc
8. Annulus fibrosus
Conus/cauda syndrome caused by spinal disc herniation

The adult spinal cord ends at about the L1 level near the conus medullaris. Underneath, the lumbar and sacral nerve roots run as cauda equina in the spinal canal. A marked, medial spinal disc prolapse in the lumbar region can cause compression of the conus medullaris or all the nerve roots of cauda equina at the corresponding level, which can lead to conus or cauda syndrome (see below). If cauda equina and conus medullaris are affected concurrently, it is referred to as conus-cauda syndrome.

Myelopathy caused by spinal disc herniation

Massive cervical or thoracic spinal disc prolapse can cause myelopathy by compressing the spinal column resulting in spastic pareses, pyramidal signs, and bladder control dysfunction.

Symptoms of Spinal Disc Herniation

Generalized inflammatory symptoms of spinal disc herniation

Lumbar pain is the predominant symptom of lumbar spinal disc herniation, which may be triggered by new or physiologically awkward movements. Pain is magnified by coughing, straining or sneezing. Nerve root irritation involving the L5/S1 region is associated with a positive Lasègue sign, also known as straight leg raise test (SLR) (i.e. the reversed Lasègue sign in the L3/4 area).

Cervical involvement also induces pain in the affected spinal area, often involving specific avoidance postures (caput opstipum = ‘wry neck’, abnormal straightening, scoliosis, torsion) as well as restricted movement and paravertebral myogeloses.

Radicular symptoms of spinal disc herniation

Most commonly, nerve root compression syndrome accompanies spinal disc herniation (= radiculopathies, radicular syndrome). Irritation of the exiting nerve roots leads to radiating pain and altered sensitivity in the corresponding dermatomes. In the case of cervical disc herniation, pain radiates into the arm (cervico-brachialgia). Lumbar disc herniation is characterized by lower back pain, which affects buttocks and legs (lumbo-ischialgia). Pareses and decreased muscle reflexes can occur depending on the severity and location of the herniation.

The key cervical radicular syndromes include:

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Paresis</th>
<th>Pain radiation, paresthesia, abnormal sensitivity</th>
<th>Weakened reflexes</th>
</tr>
</thead>
<tbody>
<tr>
<td>C5</td>
<td>Deltoideus</td>
<td>Lateral shoulder</td>
<td>Biceps reflex</td>
</tr>
<tr>
<td>C6</td>
<td>Biceps brachii, M. brachioradialis</td>
<td>Radial arm extending to thumb</td>
<td>Biceps reflex, radial reflex</td>
</tr>
<tr>
<td>C7</td>
<td>Triceps brachii</td>
<td>Regio antebrachii dorsalis, dorsal hand extending till 2nd-4th finger</td>
<td>Triceps reflex, biceps reflex</td>
</tr>
<tr>
<td>C8</td>
<td>Small hand muscles</td>
<td>Ulnar arm, lateral hand extending to 5th finger</td>
<td>Tromner reflex</td>
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</tbody>
</table>
These are important lumbar syndromes:

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Paresis</th>
<th>Pain radiation, paresthesia, sensitivity disturbance</th>
<th>Weakened reflexes</th>
</tr>
</thead>
<tbody>
<tr>
<td>L3</td>
<td>Deltoideus</td>
<td>Transverses anterior aspect of the thigh towards knee</td>
<td>Adductor reflex, perhaps patellar reflex</td>
</tr>
<tr>
<td>L4</td>
<td>Quadriceps, M. tibialis ant.</td>
<td>Anterior and medial lower leg</td>
<td>Patellar reflex</td>
</tr>
<tr>
<td>L5</td>
<td>Extensor hallucis longus (lifts big toe), perhaps M. tibialis ant. and post., M. gluteus med.</td>
<td>Lateral and anterior lower leg, dorsal foot extending to big toe</td>
<td>Tibialis posterior reflex</td>
</tr>
<tr>
<td>S1</td>
<td>Triceps surae (plantar flexion, test perhaps through standing on toes), M. glutaeus max., M. biceps femoris</td>
<td>Posterior and lateral thigh, lateral foot</td>
<td>Achilles reflex</td>
</tr>
<tr>
<td>S2 – S4</td>
<td>Bladder and rectal dysfunction (neurologic emergency, see cauda syndrome)</td>
<td>Posterior thigh, anal region (cauda syndrome: breech presentation)</td>
<td>Anal reflex</td>
</tr>
</tbody>
</table>

Symptoms of myelopathy

If medial spinal disc herniation causes spinal cord compression in the cervical or thoracic region, it can lead to spastic pareses of the lower extremities via damaged neural pathways (upper motoneuron). Reflexes are heightened and pyramidal signs occur. In addition, urinary incontinence due to bladder dysfunction and colon disease as well as a positive Lhermitte’s sign in case of cervical myelopathy may be observed.

**Note:** Typically, symptoms do not suggest complete paraplegia but only a unilateral compression of the spinal cord. Brown-Séquard syndrome: spastic paresis and hypesthesia below the lesion, contralateral pain and temperature sensation (dissociated sensitivity dysfunction).

Symptoms of Conus and Cauda syndrome
Marked medial spinal disc herniation involving the **conus medullaris** region may lead to loss of sensitivity in the so-called ‘breech anesthesia’ (**perianal** and medial thighs on both sides) and loss of polysynaptic reflexes, **anal reflex** and **bulbocavernosus reflex** (S3–S5 or S3/4). In addition to a flaccid anal sphincter, dysfunction of **urinary and fecal continence** and **sexual dysfunction** is observed.

Cauda syndrome is caused by a lesion below L1, which affects the **cauda equina** and leads to segmentally limited and limp paraparesis, limited reflexes as well as radicular sensitivity and dysfunction. Depending on the level of the lesion the **cremasteric reflex** (L2) may be lost.

**Note:** Paresis of the legs is not part of the isolated conus syndrome. Conus syndrome is accompanied by isolated damage of the conus medullaris (S3–S5) at the L1 level. In case of involvement of conus medullaris as well as cauda equina, the appropriate terminology is conus-cauda syndrome.

### Diagnosis of Spinal Disc Herniation

#### Anamnestic for spinal disc herniation

Anamnesis is an important part of diagnosing pain in the spine. Try to capture the pain characteristics and accompanying symptoms as well as previous illnesses, which may serve as warning signals for specific causes of pain with an urgent call for action. The following **red flags** warrant early detection and prevention of serious conditions:

<table>
<thead>
<tr>
<th>Fracture</th>
<th>Severe trauma (due to motor vehicle accidents, fall from great height, and sports injury), and minor trauma in elderly or patients potentially affected by osteoporosis; systemic steroid therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tumor</td>
<td>Advanced age, history of tumors, and generalized symptoms (fatigue, loss of appetite), and increased pain in supine position; severe nocturnal pain</td>
</tr>
<tr>
<td>Infection</td>
<td>Generalized symptoms (acute fever, chills, loss of appetite, and fatigue), a history of infection and associated general illness; drug abuse; immune suppression; strong pain at night</td>
</tr>
</tbody>
</table>
Radiculopathy

Pain radiating into the legs accompanied by paresthesia or weakness; Cauda syndrome: sudden bladder or rectal dysfunction, perianal and perineal loss of sensation; marked/increasing neurologic deficit; cessation of pain with increasing paralysis leading to complete loss of function of the reference muscle (nerve root death)

Clinical examination of spinal disc herniation

When radicular syndrome is suspected, a careful clinical examination should be conducted. Symptoms of pain radiation, possible paresis and reflex limitations as well as the loss of sensitivity suggest damaged nerve root.

**Note:** Loss of sensitivity in radiculopathy occurs in a striated fashion along the dermatomes. A differential diagnosis is based on segmented loss of sensitivity from peripheral nerve damage, which affects limited areas of skin.

Evaluation of **sharp or dull discrimination** can be used to trace sensitivity dysfunction to the corresponding segments because the pain sensation in the **dermatomes** does not overlap with touch sensitivity.

Positive Lasègue test for spinal disc herniation

The positive **Lasègue sign** is an indicator of nerve root irritation. A positive Lasègue test occurs when the stretching of the **N. ischiadicus** (roots L5/S1) elicits pain. The patient is in a supine position while the examiner lifts the patient’s extended leg. Radiating pain at about 45° is considered a pathological sign.

The reverse Lasègue test shows painful stretch of **N. femoralis** (roots L3/L4). In this test, the patient is in prone position while the leg is bent at the knee and the foot is guided toward the buttocks with a lifted thigh. Radiating pain and attempted evasive motion by the patient represent a positive test result.

Radiological diagnosis of spinal disc herniation

**MRI** (axial and sagittal) is the modality of choice to confirm spinal disc herniation. Conventional X-rays, as well as CT, can be performed in order to evaluate osseous structures. Further diagnostic clues can be obtained via **myelographic** imaging.

**Note:** Disc herniation may be asymptomatic, and conversely radicular dysfunction can occur without prolapse, which can be established diagnostically or surgically.
Neurophysiological examination

Under specific circumstances, electrophysiological evaluation can be used to distinguish between radiculopathy and peripheral nerve damage. In principle, no additional electrophysiological confirmation is necessary for monoradicular syndrome with appropriate imaging and corresponding proof of compression.

Differential Diagnosis of Spinal Disc Herniation

Spinal disc herniation in the lumbar region can be differentiated from unspecific back pain with pseudo-radicular radiating pain as well as other causes of the radicular syndrome.

Pseudoradicular syndrome of spinal disc herniation

Back pain that appears radicular in nature and radiates into arms and legs but is not immediately caused by nerve root compression is called pseudoradicular syndrome. Pain does not typically radiate strictly as in radiculopathy, and instead, it may appear as paresthesia, without a strong correlation with dermatomes. The pseudoradicular syndrome is usually orthopedic in nature, perhaps mediated by facet syndrome or iliosacral joint syndrome suggesting joint distortions (‘blockages’), which are attributed to degenerative changes or poor posture.

Peripheral nerve lesions of spinal disc herniation

Peripheral nerve lesions or plexus lesions can mimic nerve root damage clinically. In addition to accurate analysis of clinical symptoms, neurography can be used to facilitate the diagnosis.

Radiculitis of spinal disc herniation

Radiculitis caused by either a bacterial or viral infection (mainly borreliosis and herpes zoster) and autoimmune or cryptogenic inflammation should be considered in the differential diagnosis. Aside from spinal disc herniation, localized demand for space (tumor, bone metastases) can cause radiculopathy. Also, degenerative spinal changes, spinal abscesses or spondylodiscitis should be considered as well as the possibility of diabetic radiculopathy.

Treatment of Spinal Disc Herniation

Conservative treatment of spinal disc herniation

Radiculopathy should be treated conservatively initially. In case of cervical radiculopathy, limiting motion via a neck brace may be required along with mobilizing physiotherapy. Rest for patients with lumbalgia in its acute phase is no longer recommended. Further, patients with radicular syndromes are advised to seek physiotherapy and be active four days after the acute event at the latest.

Pain is treated with analgesics (acetaminophen, NSAID) and muscle relaxers. If non-opioid analgesics prove ineffective, weak opioid analgesics may be prescribed. Principally, treatment follows the step care recommended by the World Health Organization (WHO).
Note: Chiropractic treatment is contraindicated for spinal disc herniation.

Surgical treatment for spinal disc herniation

**Absolute indications** for surgical intervention in spinal disc herniation are:

- Damage to bladder and rectum
- Conus-cauda syndrome
- Noted paresis (3/5)
- Signs of myelopathy

**Relative indications** are spinal disc sequestration and failure with conservative therapy (persisting pain syndrome for weeks in spite of conservative therapy).

Spinal disc material can be removed via microsurgical **discectomy**: Under certain circumstances, there will be **intercorporal spondylodesis** (fusion, ‘stiffening’) following surgery.

Course and Prognosis of Spinal Disc Herniation

**Abatement of spinal disc herniation**

Appropriate and adequate conservative therapy can ameliorate up to 80% of all cases with spinal disc herniation and effects of the acute radicular syndrome. Success with conservative therapy is comparable to surgical intervention, following appropriate monitoring and observation.

The **rate of recidivism**, however, is rather large. Appropriate prophylaxis includes muscle building exercises, sufficient motion, and control of overweight.

References

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