Degenerative Spinal Disease

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Abstract: Degenerative spinal disease is the commonest spinal problem dealt with by a neurosurgeon. Degenerative spine disease takes up significant working hours for neurosurgeons throughout the world. It is a progressive deterioration of the elements of the spinal cord and includes disc abnormality, facet joint abnormality, osteophytes spondylolisthesis, spondylolysis, ligamentum flavum hypertrophy, etc. All the aforementioned conditions can result in spinal canal stenosis, which can lead to neural compression and can produce compression on the cord or root and lead to myelopathy, radiculopathy, myeloradiculopathy, and cauda equina syndrome. Most of the time, MRI, CT, and X-ray of the spine are enough for diagnosing these conditions. Surgical intervention is needed when there is myelopathy, radiculopathy, or instability. This chapter will briefly discuss the management of disc prolapses (lumbar, cervical, and dorsal); degenerative spinal canal stenosis (spondylosis with myelopathy and radiculopathy); spondylolisthesis; ossified posterior longitudinal ligaments; and ligamentum flavum hypertrophy. It will also discuss degenerative diseases of the craniovertebral junction with instability and myelopathy (atlantoaxial dislocation and atlanto-occipital dislocation).

Abbreviations

AAD	atlantoaxial dislocation	ADI	atlanto-dental interval
AOD	atlanto-occipital dislocation	AF	annulus fibrosis
ALL	anterior longitudinal ligament	AP	anterior–posterior
CCJ	craniocervical junction	CES	cauda equina syndrome
CT	computed tomography	CTA	CT angiogram
CSF	cerebrospinal fluid	CSM	cervical spondylotic myelopathy
CVJ	craniovertebral junction		
DISH	diffuse idiopathic skeletal hyperostosis	DVT	deep vein thrombosis
ECM	extracellular matrix	EP	endplate
HRQOL	health-related quality of life	ITF	intertransverse fusion
IV	Intravenous	IVD	intervertebral disc
LBP	low back pain	LDD	lumbar disc disease
LDH	lumbar disc herniation	LFH	ligamentum flavum hypertrophy
MDCT	multidetector CT	mJOAS	modified Japanese Orthopedic Association Score
MMP	matrix metalloproteinase		
MRI	magnetic resonance imaging	NP	nucleus pulposus
ODI	Oswestry disability index	OALL	ossified anterior longitudinal ligament
OPLL	ossified posterior longitudinal ligament	PEEK	poly ether ether ketone
PLIF	posterolateral interbody fusion	PLL	posterior longitudinal ligament
SLR	straight leg raising	UTI	urinary tract infection
VB	vertebral body		

1. Introduction

The human spine is made up of highly specialized structures and tissues that are designed to provide a wide range of movement and significant load-transmitting capacity for daily physical activity (Ferguson and Steffen 2003). The human spinal column is the body's main support structure. Along with the intervertebral discs and ligaments, it is supported by paraspinal muscles. The spine consists of 33 separate bones, called the vertebrae. With age, five sacral vertebrae become fused and give rise to one sacrum, and four coccygeal vertebrae become fused and become one coccyx. So, in adults, there are 26 vertebral bones. The flexibility of the discs between the vertebrae allows for a cushioning effect. The spine, which must be both stable and flexible, is particularly vulnerable to damage and can degenerate over time. The spinal column is composed of (1) the vertebral column, composed of vertebrae, intervertebral discs, and ligaments, and (2) the spinal cord, including nerve roots and the cauda equina, covered by the meninges.

In men, the spine is typically 60–65 cm long, while it is 45–50 cm long in women. Of this length, 1/4th–1/5th is contributed by discs. The diameter of the canal varies in different regions of spine. It is widest in the cervical

region (anterior–posterior AP diameter 17–18 mm), followed by the lumbar region (AP diameter average 11.5 mm) (Karantanas et al. 1998). It is narrowest in the thoracic region. The functions of the spine are weight transmission, acting as a shock absorber, like a cushion, and giving flexibility to the spine for movement. Bony structures protect the neural structures. The vertebral column is composed of vertebral bodies (VBs), pedicles, intervertebral discs, synovial facet joints, ligaments, and transverse and spinous processes. All these components can show signs of normal ageing as well as degenerative or regenerative changes (Pytel et al. 2006).

Spondylosis is an "umbrella" term to describe some type of degeneration in the spine. In the spine, the degenerative process involves the bony components (i.e., VBs), intervertebral discs, joints, and ligaments.

Degenerative spine disease is a progressive deterioration of the elements of the spinal cord and includes the following (Greenberg 2010):

1. Disc abnormality;
2. Facet joint abnormality;
3. Osteophytes;
4. Spondylolisthesis;
5. Spondylolysis;
6. Ligamentum flavum hypertrophy.

All the above conditions can cause spinal canal stenosis, which can lead to neural compression and can produce compression over the cord or root and lead to myelopathy, radiculopathy, myeloradiculopathy, and cauda equina syndrome.

2. Degenerative Disc Disease

2.1. Anatomy and Physiology of the Disc

The seven cervical, twelve thoracic, and five lumbar discs (twenty-three in all) are basically similar in structure, but they differ in size and shape according to the vertebrae in between which they are interposed (Easwaran 2012a). The nucleus pulposus (NP), a gelatinous inner core, the annulus fibrosus (AF), an outer rim, and cartilage endplates on both the upper and lower sides make up an intervertebral disc (Choi 2009; Easwaran 2012a). The outer AF, which is rich in type 1 collagen, is a circular layer that resists tensile tension because the collagen fibres run alternately between the lamellae of the annulus in an oblique fashion. The proteoglycan and water gel that make up the NP are connected by a loose network of type 2 collagen and elastin fibres (Karantanas et al. 1998). Aggrecan-1, the NP's most important proteoglycan, offers essential osmotic characteristics to resist compression, contributing to the shock-absorbing mechanism. One of the major avascular parts of the body is the intervertebral disc. Nutrients for the disc tissues are delivered by vessels in the subchondral bone close to the hyaline cartilage of the endplate. Small molecules like glucose and oxygen are carried over the endplate by passive diffusion. Proteoglycan and collagen, which make up a disc's basic skeletal framework, are produced by chondrocytes. The annulus fibrosus fibres, as well as the neighbouring dura and PLL, are innervated by the recurrent sinuvertebral nerve of Luschka, a meningeal branch of the spinal nerve that develops from the posterior ramus of each nerve root. Innervation is present in the outer annular regions, but not in the inner annular regions or the nucleus pulposus (Panigrahi and Reddy 2012).

2.2. Biomechanics of the Spine

The water content of the nucleus pulposus is vital for its biomechanical properties. On applying a compression force, water is expelled from the disc and it is reabsorbed into the disc once the force is removed. At low loads, the disc does not resist deformation but, beyond a certain load, the stiffness increases abruptly. This property allows the disc to remain flexible on minimal loading, while it contributes to stability as the loading increases. When the load limits are exceeded, the endplates fail first before the annulus fibrosus. This happens with compression loads of about 14,000 N in lumbar disc and 3000 N in the cervical disc (Kaiser et al. 2000). Water loss in the disc, associated with ageing, reduces the viscoelastic adaptability of the disc, making it prone to failure. The dehydrated disc behaves as a fibrous solid rather than a watery gel in terms of its biomechanical properties. Apart from compression loads, the other loads that the disc withstands are tension, shear, and torsion loads. During day-to-day activities, the loads acting on the disc are a combination of these pure loads (Easwaran 2012a).

2.3. Changes Caused by Ageing

With ageing, the water content in the nucleus pulposus (NP) becomes reduced. This dehydration is caused by a decrease in the proteoglycan content of the matrix in the NP. There is a reduction in collagen content with the replacement of type 2 collagen by type 1 collagen in the inner part of annulus (Singh et al. 2009). Type 1 collagen fibres become coarser and more cross-linked, thus increasing their stiffness. The collagen becomes non-enzymatically glycated, which further increases cross-linking. These biochemical changes are accompanied by histological changes such as the appearance of microstructural clefts, which have been observed as early as at the age of 15 years. There is fissuring and clumping of parts of the NP. The endplate (EP) undergoes changes like thinning, formation of fissures, and alteration of cell density, and there is subchondral bone sclerosis (Roberts et al. 2006). The changing microstructure and biochemistry alter the biomechanical characters of the ageing disc. As the NP becomes desiccated, the annulus fibrosus (AF) has to bear more of the compression load. The AF becomes stiffer but structurally weakened (Easwaran 2012a).

3. Lumbar Disc Herniation

3.1. Introduction

The term "lumbar degenerative disc disease" includes a spectrum of disorders like disc bulge, disc protrusion—which can be (i) central, (ii) paracentral, (iii) intraforaminal, or (iv) far lateral—disc extrusion (with or without a mitigated fragment), and internal disc disruption. Patients suffering from this condition present with low back pain (LBP) and/or radiculopathy of long duration or, acutely, with cauda equina syndrome (Panigrahi and Reddy 2012). Over 80% of the general public will experience LBP at some point in their lives, and it is responsible for about 15% of all sick days used at work. LBP is mostly brought on by lumbar disc disorders (LDDs), which include lumbar disc degeneration, including lumbar disc herniation. Studies have been conducted to identify the origins of LDDs. Genetic and environmental factors have been used to classify the causes (Kawaguchi 2018). Physical activity, sports, driving, and smoking can be classified as environmental risk factors (Kawaguchi 2018). Throughout the last century, mechanical load injury was proposed as the most important cause of disc prolapses (Luoma et al. 1998). Recent studies have revealed that genetic factors are a key factor in LDDs (Kawaguchi 2018).

The integrity and structure of both the nucleus and annulus in conjunction determine the mechanical characteristics of a disc. A change in each of these characteristics can have an impact on the disc's overall qualities. The disc gap is further strengthened by the anterior and posterior longitudinal ligaments (ALLs and PLLs). The ALL functions as a tension band to counteract pressures applied in extension because its attachment to the vertebral body margins is stronger than its attachment to the annulus. Even though it is weaker than the ALL, the PLL firmly adheres to the annulus fibrosus and functions as a tension band to fend against flexion stresses. In cases of free fragment disc herniation, it is usually ripped. The endplate's purpose is more biological than biomechanical in that it is crucial for the passage of nutrients into the disc (Easwaran 2012a).

3.2. Causes

The most frequent cause of disc herniation is disc degeneration, which is a slow, ageing-related wear-and-tear process. Discs lose flexibility as they age, making them more likely to rupture or rip at the slightest twist or strain. Most people struggle to identify the reason behind their ruptured disc. Twisting and turning when lifting can sometimes result in a herniated disc, as can using your back muscles rather than your leg and thigh muscles to raise heavy objects. Rarely is the cause a traumatic incident like a fall or a knock to the back.

3.3. Risk Factors

Factors that may increase the danger of a herniated intervertebral disc include the following:

- Weight. Weight is one factor that can make a herniated disc more likely to occur. Discs are put under additional strain due to excess body weight.
- Occupation. Those who have physically demanding occupations are more likely to experience back issues. Herniated discs are also more likely to occur if someone repeatedly lift, pull, push, bend laterally, or twist.
- Genetics. Herniated disc development is predisposed by genetics in some people.
- Smoking. Smoking is believed to reduce oxygen flow to the disc, hastening its degeneration.

3.4. Pathophysiology

Mechanical and metabolic disturbances both contribute to the complex processes of lumbar disc degeneration and herniation (Panigrahi and Reddy 2012). Twin studies have shown a genetic propensity to disc degeneration (Choi 2009). Disc herniation reaches a peak in the fourth decade of life, despite the fact that disc degeneration worsens with age (Panigrahi and Reddy 2012). The elastic modulus, which distributes stress to the most important areas of the disc, is aberrant and non-uniform in degenerated discs. They also have abnormal vascularity, collagen distribution, and collagen cross-linking. Repetitive or continuous axial overloading is the key determinant in the pathogenesis of lumbar degenerative disease. Obese individuals, manual labourers, truck drivers, and those involved in athletic activities like weightlifting and gymnastics are at risk of repetitive axial overloading of the spine (Panigrahi and Reddy 2012). Degenerated discs can no longer support a load hydrostatically. The endplate and annulus are where the load's stress is most concentrated (Choi 2009).

LDH is hypothesized to be caused by a number of alterations in the biology of the intervertebral disc:

1. Degradation of collagen and extracellular matrix (ECM) materials;
2. Upregulation of degradation systems like apoptosis and matrix metalloproteinase (MMP) expression;
3. Inflammation pathways;
4. Decreased water retention in the NP, increased type 1 collagen percentage in the NP and inner AF;
5. Degradation of collagen and extracellular matrix (ECM) materials (Amin et al. 2017; Kalb et al. 2012; Brayda-Bruno et al. 2014; Mayer et al. 2013).

The majority of disc herniations occur in a posterolateral direction, i.e., corresponding to the area of the spinal canal between the midline and the neural foramen, because the nucleus pulposus is situated somewhat posteriorly within the annulus and the PLL reinforces the annulus fibrosus in the midline posteriorly.

LDH symptoms are generated by several mechanisms. Among these are the presence and effects of *Propionibacterium acnes*, contributions from an acidic environment, microstructural alterations to the nerve root, and, most critically, the stretching effect of herniated disc material on the nerve root (Amin et al. 2017).

The types of disc herniation are as follows: (1) disc bulge; (2) protruded; (3) contained/entrapped; (4) extruded; (5) migrated,

The zones of lumbar disc herniation are as follows: (1) central canal zone— paramedian, midline, and subarticular zone; (2) foraminal zone; (3) extraforaminal zone (Greenberg 2010).

The most common spaces involved, according to frequency, are L4/5, L5/S1, L3/4, and others.

3.5. Clinical Features

3.5.1. Symptoms

An individual with a herniated lumbar disc can present with radiculopathy, neurogenic claudication, or cauda equina syndrome. The initial symptom in an individual with a disc herniation is pain, which may be in the back, buttock, thigh, leg, or foot and which may be present either in all or a few of these areas. Radicular pain is aggravated by bending, coughing, sneezing, and lifting a grounded object. The pain is usually relieved by lying down in a hip-and-knee-flexed posture.

The symptoms of lumbar disc herniation can be summarized as follows:

- Back pain that comes and goes. Moving about, coughing, sneezing, or standing for extended amounts of time may make it worse.
- Muscle spasms in the back.
- Sciatica, which causes pain to radiate from the back or buttock down the leg and into the calf or the foot.
- Leg muscles that are weakened.
- Numbness in the foot or the leg.
- Diminished knee- or ankle-level reflexes.
- Modifications in bowel or bladder function.

The level of disc herniation can be determined according to the motor and sensory functions of the respective nerve root. These are summarized in Table 1.

Patients who have large central disc herniation and resultant spinal canal stenosis present with neurogenic claudication which is asymptomatic at rest. They experience bilateral lower extremity pain after a variable duration of exertion, associated with numbness which is relieved with a brief period of rest. A central lumbar disc herniation may result in cauda equina syndrome with perineal numbness, loss of bladder and bowel control, and some degree of motor weakness in the legs (Panigrahi and Reddy 2012). It is important to look for spasms of

the paraspinal muscles and the range of movement of the spine and perform a rectal examination when cauda equina syndrome is suspected. It is important to interpret the straight leg raising test (Lasegue test)—a positive test implies a reproduction of radicular pain and not back pain.

Table 1. Common neurologic changes in herniated lumbar discs according to root level.

Root-Level	Motor Weakness	Sensory Loss	Reflex Depression	Muscle Wasting
L2	Hip flexion and abduction	Lat thigh	Nil	Thigh
L3	Knee extension	Patellar region	Knee	Thigh
L4	Knee extension, ankle dorsiflexion	Medial shin below knee	Knee	Thigh
L5	Extensor hallucis longus	Dorsum of foot, lateral calf		Calf
S1	Plantar flexion at ankle	Lateral border of foot, posterior calf	Ankle	Calf

Source: Authors' compilation based on data from Panigrahi and Reddy (2012).

3.5.2. Physical Findings in Radiculopathy

Nerve root impingement produces a set of symptoms and signs that help to identify the level of disc herniation. These include motor weakness, sensory changes, and reflex changes as described in Table 1. Nerve root tension signs also help to determine the level. These include the following:

1. Lasegue sign, also known as straight leg raising test (SLR): roots involved are L5 and S1 and, to small extent, L4.
2. Crossed SLR: more specific for the same roots than SLR.
3. Femoral stretch test, also known as reverse SLR: roots involved are L2, L3, and L4.

Some other tests, not practiced much, are the bowstring sign, cram test, sitting knee extension, the FABER test, etc. (Greenberg 2010).

3.6. Differential Diagnosis

Although back pain is the commonest symptom of a herniated lumbar disc, in making the diagnosis, it is also the least useful symptom. The following conditions also present with symptoms similar to those of a herniated lumbar disc:

1. Tumours involving the nerves (neurofibroma, Schwannoma, ependymoma) or metastatic deposits in the pedicle;
2. Peripheral neuropathy in diabetes mellitus and entrapment neuropathies involving the sciatic nerve in the pelvis;
3. Osteoarthritis of the hip;
4. Fractures involving the vertebra caused by trauma, osteoporosis, or metastatic deposits;
5. Arachnoid cyst and Tarlov's cysts of the spinal region;
6. Vascular claudication.

Some serious conditions involving low back pain should be excluded by the "red flag signs" depicted in the Table 2.

3.7. Investigations

3.7.1. Plain Radiograph

Plain X-rays are the first-line imaging tools utilized in low back pain. A diagnosis of lumbar disc herniation cannot be made from a plain radiograph, but it can help by providing indirect evidence, like features of degenerative changes including narrowing of intervertebral disc space, disc calcification, marginal osteophytes, and sclerosis of the nearby vertebral body endplate. Standard anteroposterior and lateral radiographs exclude other aetiologies of back pain. Dynamic extension and flexion views are needed to rule out associated spondylolisthesis.

Table 2. Red flag signs for individuals with low back problems.

Condition	Red Flags
Infection or cancer	1. Age > 50 or <20 years 2. History of malignancy 3. Immunosuppression 4. Unexplained wt. loss 5. IV drug abuse, UTI, fever, or chills 6. Back pain not relieved with rest
Spinal fracture	1. History of significant trauma 2. Prolong utilization of steroids 3. Age > 70 years
Severe neurologic compromise or cauda equina syndrome	1. Acute onset of overflow incontinence or urinary retention 2. Loss of sphincter tone or faecal incontinence 3. Progressive or global weakness in lower extremities

Source: Authors' compilation based on data from Greenberg (2010).

3.7.2. Magnetic Resonance Imaging (MRI)

MRI (Figure 1) is the preferred initial study for the assessment of lumbar degenerative diseases including disc herniations and lateral recess, central canal, and neural foraminal stenoses. It is the gold standard of neuroimaging in diagnosing suspected lumbar disc herniation, with an accuracy of about 97% (Roberts et al. 2006).

MR imaging findings consistent with a degenerated disc are as follows (Panigrahi and Reddy 2012):

- Decreased signal intensity on T2W scans of the nucleus pulposus, compared with a normal disc, because of a desiccation of the degenerated disc and resultant diminished water content in the nucleus pulposus.
- Irregularity of the outline of the nucleus pulposus.
- Decrease in disc height.
- An intense dot-like high-intensity signal in the posterior annulus signifying an annulus tear.

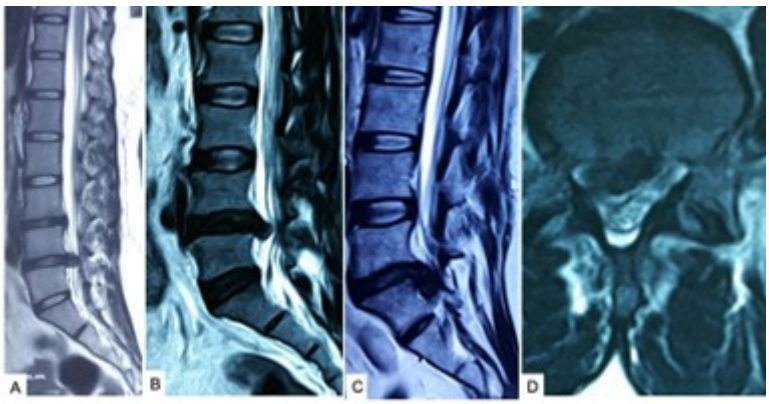


Figure 1. (**A**,**B**) MRI of lumbosacral spine (T2W sagittal images) showing PLID at L4 and 5. (**C**,**D**) MRI of lumbosacral spine (T2W sagittal and axial images) showing, respectively, PLID at L5 and S1 (left). Source: Figure by authors.

A disc herniation is best detected on axial images (either computed tomography or MR imaging) because in this plane, the focal, usually eccentric posterior extension of the disc material is readily visualized. The protruding disc obliterates epidural fat and displaces the nerve root sleeve, or both. Sagittal T1W and T2W MR images often demonstrate posterior bulging of the IVD. Sagittal T1W and moderately and heavily T2W images provide information regarding the level of herniation and the presence of extruded or sequestrated fragments, if any.

There are some contraindications to MRI. These include the following (Greenberg 2010):

(a) Cardiac pacemaker;
(b) Ferromagnetic aneurysmal clip;
(c) Metallic implants inside the body;
(d) Metallic FB within the eye;
(e) H/O of placement of coil, vascular stent, or filter in last 6 months;
(f) H/O of bullet of pellet within the body;
(g) Claustrophobia is a relative contraindication.

In these situations, CT scan and myelography are the solution.

3.7.3. Myelography

It is possible to delineate the dural sac, spinal cord, and exiting nerve roots on myelography. The myelographic signs of disc herniation include the following:

- Compression of the root of the nerve and thecal sac with an angular indentation on the anterolateral surface of the thecal tube.
- Compression of the nerve root with fusiform widening of the more distal end of the involved root.
- Central compression of the thecal tube by a centrally located herniated nucleus pulposus.

Myelographic findings are more appropriate for L4/5 than for L5/S1 because of the wider diameter of the epidural space at this level. Myelography cannot detect lateral or foraminal discs.

The disadvantages of myelography include the fact that it is an invasive procedure, occasionally needs overnight hospitalization, and cannot give information about lateral or foraminal disc herniation, as well as the issue of iodine allergy (Panigrahi and Reddy 2012; Greenberg 2010).

3.7.4. Computed Tomography

With multidetector CT (MDCT) substantially improving computed tomography (CT), which was previously regarded to be clinically inferior to MRI in LDH detection, the diagnostic level of CT is now almost on par with that of MRI. There are a number of scenarios where CT or myelography might be preferred over MRI, such as when MRI is unavailable or impractical (such as with pacemakers or cochlear implants) or when patients would feel too uncomfortable (intractable back pain or claustrophobia) (Amin et al. 2017).

3.7.5. Nerve Conduction Study and Electromyography

This study is sometimes required to exclude peripheral neuropathy and some conditions of the nerves.

3.8. Management

Successful management of LDH depends on the correct diagnosis of the problem and the selection of the appropriate mode of treatment. It is to be ensured that the symptoms are due to LDH. There are lot of causes of low back pain. For this purpose, careful history-taking is very important. The patient should be thoroughly examined, and scans, films, and reports should be carefully interpreted.

The management options for a patient with degenerative disc disease are as follows:

(1) Nonoperative;
(2) Operative.

3.8.1. Nonoperative Management

Initial nonsurgical treatment is warranted except in cases of absolute indications for surgery (discussed later on). It includes the following:

(a) Bed rest: for severe radicular discomfort, a brief (4-day) duration of bed rest is advised. Many patients find respite from their problems by lying flat, which is one justification for bed rest. Using the supine position to reduce intradiscal pressure is another justification.
(b) Nevertheless, a Cochrane review of nine trials with 1435 patients that compared bed rest with other therapies or different lengths of bed rest came to the conclusion that bed rest in comparison to staying active has, at best, no effect on low-back pain and, at worst, may have slightly harmful effects (Hagen et al. 2000). In patients with low back pain of varying durations, both with and without radiating pain, there was no discernible difference between the effects of bed rest compared to exercises, or between 7 days and 2–3 days of bed rest, in the management of acute low back pain.
(c) Activity modification: patients should temporarily restrict heavy weightlifting, prolonged sitting, and bending and twisting of the back.
(d) Physiotherapy: in the acute phase, this includes the use of hot packs, short-wave diathermy, and microwave therapy. After the acute phase, a graduated regime of back exercises is instituted.
(e) Analgesics.
(f) Muscle relaxants.

(g) Spinal manipulation therapy.
(h) Epidural injection.
(i) Patient education: the condition should be explained to the patient, and the patient should be reassured. The correct posture during work and sleep should be explained (Greenberg 2010; Panigrahi and Reddy 2012).

3.8.2. Operative Management

(a) Open techniques,
(b) Microdiscectomy,
(c) Minimally invasive surgery, including endoscopic techniques,
(d) Minimally invasive techniques:

- Chemonucleolysis;
- Automated percutaneous lumbar discectomy;
- Laser assisted percutaneous discectomy;
- Arthroscopic microdiscectomy;
- Intradiscal electrothermal therapy;
- Percutaneous nucleoplasty.

Operative therapy of LDH has previously been linked to increased short-term benefits and inconsistent value in the medium- to long-term range (Amin et al. 2017; Weinstein et al. 2006; Atlas et al. 1996) in a number of sizable investigations. A recent randomized Finnish study comparing nonoperative care with microdiscectomy in LDH (Amin et al. 2017; Österman et al. 2006) supported this conclusion. A subgroup analysis which revealed that microdiscectomy of L4-5 LDH produced better patient-reported outcomes than nonoperative therapy, including subjective job ability, ODI (Oswestry disability index), and HRQOL (Health-Related Quality of Life) scores, was the study's most innovative discovery (Amin et al. 2017; Österman et al. 2006).

Indications for Surgery

(1) Failure of nonsurgical measure to control pain for 5–8 weeks.
(2) Emergency surgery—(a) cauda equine syndrome (CES) (b); progressive motor deficit; (c) intolerable pain despite adequate narcotic analgesics.
(3) Patients who do not want to wait or try out nonsurgical treatment (Greenberg 2010).

Surgery is recommended if symptoms persist after 6 weeks of supervised conservative management, although the optimal timing of surgery is still being debated. Quick pain alleviation is the primary benefit of early surgery, although similar clinical outcomes one year later allow for prolonged conservative treatment in some patients (Panigrahi and Reddy 2012).

Microdiscectomy

The microsurgical approach in lumbar discectomy is currently the gold standard in the management of herniated lumbar disc disease. The success frequency of microdiscectomy ranges from 88 to 98.5%, while the complication rate is around 1.5%. The biggest advantage is the shorter incision and hence reduced postoperative pain, which reduces the length of hospital stay.

The complication rate following microdiscectomy is 15–30%. Intraoperative complications include the following:

- Exploration of the wrong site or level;
- Dural tears resulting in postoperative CSF leak or pseudomeningocoele;
- Injury to the nerve root;
- Retroperitoneal injury to great vessels and bowel;
- Facet joint fracture;
- Haemorrhage.

Wrong-level exploration can be minimized by using perioperative C-Arm X-ray. Postoperative complications include the following:

- Discitis (septic or aseptic);
- Arachnoiditis;
- Soft-tissue infection;

- Failure of pain relief;
- Recurrence of pain due to failed back surgery.

Minimally Invasive Surgery

Over the past 15–20 years, minimally invasive techniques for spine surgery have been developed and are being used more frequently. These methods are linked to lesser soft-tissue and skeletal trauma, cheaper acute care costs, and shorter hospital stays. Interlaminar, posterolateral, transforaminal, and transiliac are a few acknowledged percutaneous endoscopic methods for treating LDHs (Amin et al. 2017; Bai et al. 2017; Tonosu et al. 2016). When compared to open discectomy, endoscopic discectomy is often associated with shorter operating times, less blood loss, and lower reoperation rates, with no rise in overall complications or wound infections (Amin et al. 2017; Phan et al. 2017). However, a double-blind randomized control trial with 325 patients was unable to distinguish between open and endoscopic surgery in terms of long-term patient-centred results (Amin et al. 2017; Overdevest et al. 2017).

3.8.3. Complications of Lumbar Disc Surgery

The most common complications of lumbar disc surgery are as follows:

(a) Discitis;
(b) Recurrent disc herniation;
(c) Failed back syndrome (failure to relieve symptoms);
(d) Wrong site of operation;
(e) Unintended durotomy: CSF fistula, pseudomeningocele;
(f) Nerve root injury;
(g) Postop urinary retention: usually transient.

Some rare complications are CES, DVT, great vessel injury (aorta, vena cava), compression neuropathy due to positioning, and postoperative visual loss.

Discitis

The nucleus pulposus is infected. Disc and vertebral body (VB) damage could begin in the cartilaginous endplate and progress from there. Postop discitis can develop following a variety of surgeries, but lumbar discectomy is the most prevalent one.

Failed Back Syndrome

This occurs when, after having back surgery, the low back pain or radiculopathy symptoms do not improve in a satisfactory manner. These individuals frequently need analgesics and cannot go back to work. Factors that may contribute to or cause failed back syndrome include the following (Greenberg 2010):

(a) Wrong initial diagnosis due to inadequate clinical or radiological (imaging) preop work-up.
(b) Continued cauda equina or nerve root compression due to residual or recurrent disc herniation, wrong site operation, adjacent-level pathology, adhesive scar formation, pseudomeningocele, or segmental instability.
(c) Deafferentation pain, which is typically continuous and scorching or ice cold, is a symptom of permanent nerve root injury brought on by the initial disc herniation or following surgery.
(d) Discitis.
(e) Adhesive arachnoiditis.
(f) Postoperative reflex sympathetic dystrophy.

These patients should be thoroughly evaluated, including a detailed history, clinical examination, and re-checking all investigation documentation prior to the operation. Then, total blood count, lumbar spine X-ray, MRI with contrast, and CT scan with contrast and with bone window view should be repeated to identify any infection, residual disc herniation, and spinal instability, as well as to ascertain whether surgery was performed at the correct site and to assess the condition of the previous lesion for which surgery was performed.

Treatment is mostly symptomatic if there is no radiculopathy and includes bed rest, analgesics, and physical therapy. Sometimes, a short course of steroids can be given. Patients who have radiculopathy for residual/recurrent disc herniation and spinal instability need reoperation. For instability, fusion and fixation is needed.

3.9. Cauda Equina Syndrome

The cauda equina, a group of nerves near the end of the spinal cord, is so named because it resembles a horse's tail. These nerve roots continue in the lumbar and sacral region as the cauda equina. The clinical condition CES is caused by the malfunctioning of several lumbar and sacral nerve roots within the lumbar spinal canal, typically as a result of cauda equina compression. The lumbar region's most severe herniated disc is the most frequent cause of CES. Cauda equina syndrome requires immediate surgical treatment because it is a neurosurgical emergency.

The causes of CES include the following (Greenberg 2010):

1. Compression of the cauda equina:
    (a) massive herniated lumbar disc;
    (b) neoplasm;
    (c) free fat graft after discectomy;
    (d) spinal epidural haematoma;
    (e) trauma: fracture fragments compressing the cauda equina.
2. Infection—epidural abscess;
3. Neuropathy—ischaemic or inflammatory;
4. Ankylosing spondylitis.

Patients with CES may have some or all of the following "red flag" symptoms:

- Urinary retention: the commonest symptom.
- Urinary and/or faecal incontinence.
- "Saddle anaesthesia"—sensory impairment that can involve the genitals, anus, and the buttock region.
- Paralysis or weakness of more than one nerve root that leads to weakness of both lower extremities.
- Back pain and/or leg pain (also known as sciatica).
- Numbness and altered sensation in the back and/or legs.
- Bilateral absence of Achilles reflex.
- Sexual dysfunction.

Symptoms of cauda equina syndrome can be of (a) acute onset, developing suddenly within 24 h, or of (b) gradual onset, developing within several weeks.

Surgical decompression is frequently required as soon as possible in cases of cauda equina syndrome to lessen or remove pressure on the damaged nerves. Although there are conflicting findings in the literature about the best time to start treatment, it is widely accepted that having surgery within 24–48 h has the highest chance of improving sensory and motor impairments.

Although lumbar laminectomy is the preferred method of treating cauda equina syndrome, lumbar microdiscectomy may be performed in some specific circumstances. The degree of nerve injury present at the time of surgery and the speed with which the nerve is decompressed are two variables that affect the prognosis for cauda equina syndrome. Physical therapy is needed for rehabilitation.

Depending on the cause of CES, antibiotics or a high dose of corticosteroids may also be needed. Radiotherapy and chemotherapy may be needed after surgery in case of a tumour, but radiotherapy may delay the recovery of nerve function.

4. Spondylolisthesis

4.1. Introduction

One vertebra being displaced over the following lower vertebra in the sagittal plane is known as spondylolisthesis. Since the superior vertebra is usually displaced anteriorly and since this most frequently occurs in the lumbar region, it is also referred to as the forward slipping of lumbar vertebra (Easwaran 2012b).

Spine instability, or spondylolisthesis, causes the vertebrae to move more than they should. L5 over S1 is the most frequent pairing, followed by L4 over L5 (Greenberg 2010).

At the level of the listhesis, lumbar disc herniation is uncommon; but, once the disc is exposed, it may "roll" out and produce MRI abnormalities that may mimic a herniated disc (Greenberg 2010). If the listhesis causes nerve root compression, it involves the root that exits below the pedicle of the slipped upper vertebra and causes low back pain with radiculopathy.

4.2. Classification and Aetiology

An aetiological classification that is widely accepted was proposed in 1976 by Wiltse, Newman, and MacNab (Table 3).

Table 3. Wiltse–Newman–MacNab classification of spondylolisthesis.

Type	Aetiology
Isthmic	Pars interarticularis (isthmus) defect
Subtype A	Bilateral chronic spondylolytic defect in the isthmus
Subtype B	Healed spondylolysis with elongated isthmus
Subtype C	Acute bilateral fracture of isthmus
Degenerative	Abnormal motion due to disc and facet joint degeneration
Dysplastic	Congenital abnormality of neural arch such as malformed L5 inferior or superior S1 facet, abnormal sacral surface, no pars defect
Traumatic	Acute fracture of the neural arch at a site other than the isthmus
Pathological	A. Generalized bone disease B. Localized bone disease disrupting the integrity of the neural arch

Source: Authors' compilation based on data from Wiltse et al. (1976).

Nowadays, this classification system has been rearranged into six types (with the inclusion of a new type).

Type 1: Dysplastic—the congenital arch of L5 or the upper sacrum permits spondylolisthesis. No pars defect. A total of 94% are associated with spina bifida occulta.
Type 2: Isthmic—a failure of the neural arch due to a defect in the pars interarticularis. There are three sub types: (a) lytic: fatigue fracture or insufficiency fracture of the pars; (b) elongated but intact pars; (c) acute fracture of the pars.
Type 3: Degenerative—as a result of long-standing intersegmental instability.
Type 4: Traumatic—as a result of fractures usually in areas other than the pars.
Type 5: Pathological—local or generalized bone disease.
Type 6: Postsurgical—caused by complications after surgery, e.g., laminectomy and discectomy.

Classification by the severity of slippage, calculated as the percentage of the width of the vertebral body (Massachusetts General Hospital 2016):

- Grade 1: 0–25%;
- Grade 2: 25–50%;
- Grade 3: 50–75%;
- Grade 4: 75–100%;
- Grade 5: >100%.

4.3. Presentation

Spondylolysis and spondylolisthesis may be entirely asymptomatic. Common presentations are as follows (Easwaran 2012b; Greenberg 2010):

(1) Low back pain: poorly localized, central, sometimes referred to the sacral and perineal region.
(2) Sciatica.
(3) Neurogenic claudication.
(4) Radiculopathy: commonly L4, L5, and S1.
(5) Postural abnormality: sagittal imbalance and scoliosis.

4.4. Investigations

Xray, MRI and CT scan are important in the management of spodylilisthesis (Figures 2–4).

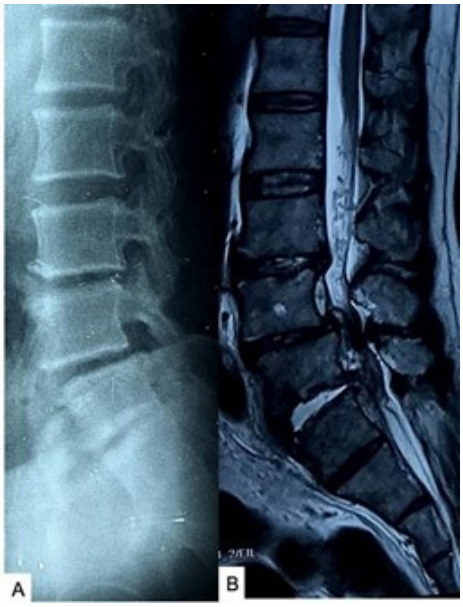


Figure 2. (**A**) X-ray of lumbosacral spine (lateral view) and (**B**) MRI of lumbosacral spine (T2W sagittal image) showing grade 1 spondylolisthesis at L4 and 5. Source: igure by authors.

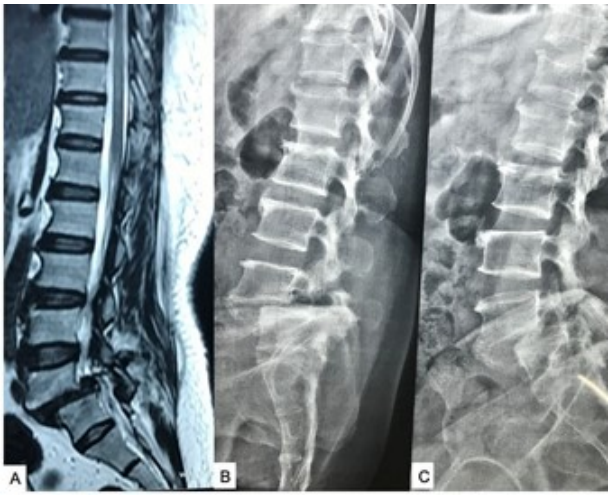


Figure 3. (**A**) MRI of lumbosacral spine (T2W sagittal image) and (**B**,**C**) dynamic X-ray of lumbosacral spine (lateral view) showing unstable grade 2 spondylolisthesis at L4 and 5 and fractured pars interarticularis with L5 sacralization. Source: Figure by authors.

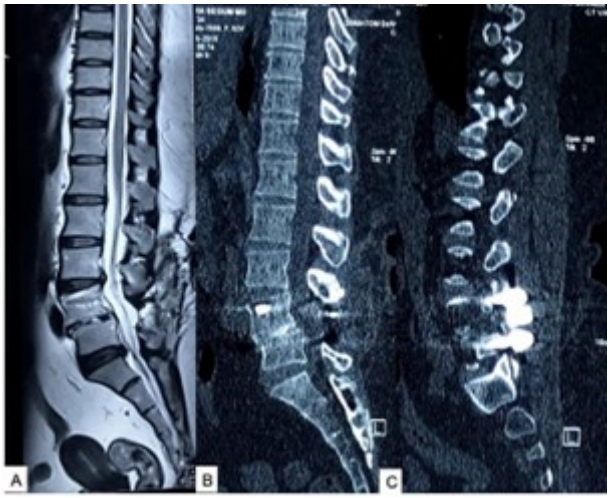


Figure 4. (**A**) MRI of lumbosacral spine (T2W sagittal image); (**B**,**C**) CT scan of lumbosacral spine (sagittal images) showing reduction, stabilization, and fusion of L4 and 5 in a case of spondylolisthesis. Source: Figure by authors.

4.4.1. Plain Radiographs

Plain X-rays are the best way to diagnose spondylolisthesis. The standard projections used are as follows: (1) The anteroposterior view, which also covers the sacroiliac joints and both hip joints and demonstrates scoliosis and sagittal balance; (2) The lateral view in the standing posture in a neutral position, flexion, and extension. The lateral neutral position shows the degree of slipping and the canal diameter. The flexion–extension dynamic film shows sagittal spinal instability. A forward movement of up to 2 mm in flexion is acceptable in healthy patients (Hayes et al. 1989). (3) The oblique view may demonstrate pars defects with the "Scottie dog collar" sign.

4.4.2. MRI

MRI provides details on soft tissues such ligaments, nerve roots, the synovium, and intervertebral discs (Easwaran 2012b). Loss of CSF signal on T2WI may be attributable to juxtafacet cysts, increased fluid in the facet joint, and vacuum discs, as well as lateral recess stenosis, central canal stenosis, and foraminal stenosis (Greenberg 2010).

4.4.3. Computed Tomography (CT)

A conventional CT scan or one conducted after water-soluble myelography typically reveals a "trefoil" channel (cloverleaf shaped, with three leaflets). Additionally, hypertrophied ligaments, AP canal diameter, facet arthropathy, and pars fractures, as well as, rarely, bulging annuli or herniated discs, are all visible on a CT scan (Greenberg 2010). CT scan is a *sine qua non* in preoperative planning (Easwaran 2012b).

4.5. Management

4.5.1. Conservative Management

Conservative management is warranted in individuals with no or minimal symptoms. This is offered to young patients with low-grade spondylolisthesis or spondylolysis and to older patients with non-disabling degenerative spondylolisthesis.

The modalities consist of 3–5 days of rest till the acute episode of back pain resolves. Longer periods of bed rest are counterproductive. Oral analgesics, bracing, various physical therapies, epidural/facetal steroid injections, and spinal flexion exercises are also prescribed (Easwaran 2012b).

4.5.2. Surgical Management

Indications for Surgery

Surgical management is warranted in (1) patients with disabling symptoms that are unrelieved by conservative management; (2) asymptomatic spondylolisthetic patients with a possible high risk of progression. The aims of surgery are halting the progression of neuro-deficit, pain relief, and, possibly, improving some presenting neurologic deficit (Greenberg 2010; Easwaran 2012b).

Surgical Options

These include (1) decompression by laminectomy, facetectomy, and foraminotomy; excision of thickened ligamentum flavum or hypertrophic synovium, drainage of synovial cysts, discectomy, and epidural scar release are the soft-tissue manoeuvres to relieve root compression; (2) reduction is seldom necessary to achieve the twin aims. Reduction is easier to achieve and becomes neurologically safer when it is attempted after decompression is completed. Distraction force applied to pedicle screws can help achieve reduction; (3) fusion: (a) interbody (PLIF); (b) interfacetal and (c) interspinous process; (d) intertransverse fusion (ITF); and (4) stabilization by instrumentation using a (a) transpedicular screw or a (b) transfacetal screw.

5. Dorsal Disc Prolapses

5.1. Introduction

Clinically symptomatic thoracic herniated discs are rare. Both sexes are affected equally. Most thoracic herniations usually happen centrally or posterolaterally, and less than 10% can be herniated laterally (McInerney and Ball 2000).

5.2. Clinical Features

Thoracic disc herniations are mostly asymptomatic. There is no typical pattern of clinical features that differentiates a herniated thoracic disc from other dorsal lesions. The usual signs and symptoms are pain (localized, axial, or radicular), motor impairment (myelopathy), hyper-reflexia and spasticity, and bowel and bladder dysfunction. Herniated thoracic disc myelopathy is usually progressive and often associated with sensory impairment down to the level of compression. Lateral thoracic disc prolapse is often associated with radicular pain in the dermatome of the root with or without paraesthesia or dysaesthesia. This radicular neuralgia is usually positional and nocturnal. Thoracic disc herniation is usually static or reduces in actual size in most cases (McInerney and Ball 2000; Shirzadi et al. 2013).

5.3. Neuroimaging

MRI of the thoracic spine is the primary imaging modality of choice for diagnosis, evaluation, and surgical planning. Plain CT is needed to evaluate the degree of calcification as thoracic herniated discs are often calcified (Figures 5 and 6).

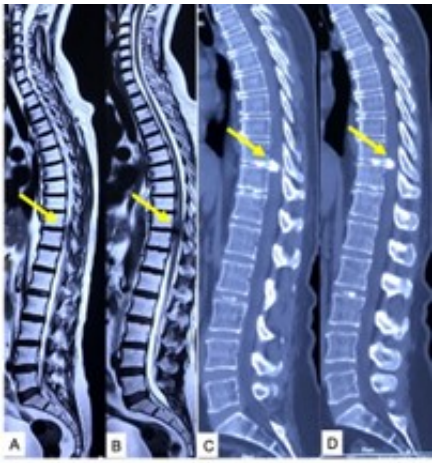


Figure 5. (**A**,**B**) MRI of spine (T2W sagittal images) showing thoracic disc prolapse at D10 and 11 with compressive myelopathy (marked with an arrow). (**C**,**D**) CT scan of spine (sagittal views) in the same patient showing the prolapsed disc was calcified (marked with an arrow). Source: Figure by authors.

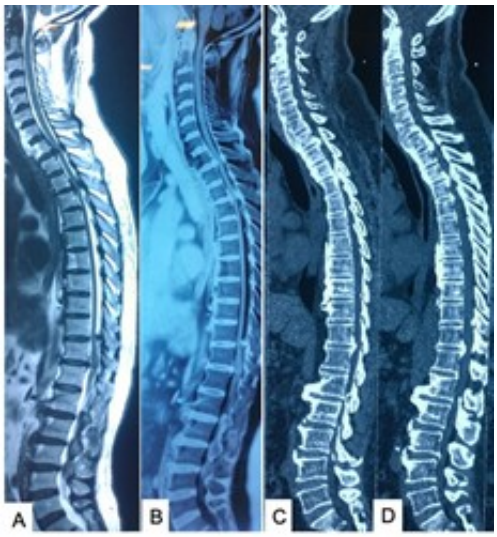


Figure 6. (**A**,**B**) MRI of the spine (T2W sagittal images) showing thoracic disc prolapse at D1 and 2 and D3 and 4 as well as ligamentum flavum hypertrophy (LFH) at the same levels with LFH at D10 and 11. (**C**,**D**) CT scan of the spine (sagittal views) in the same patient showing the prolapsed discs were calcified and LFH was also calcified. This patient also had an ossified anterior longitudinal ligament (OALL) and OPLL, indicating a case of diffuse idiopathic skeletal hyperostosis (DISH). Source: Figure by authors.

5.4. Treatment

5.4.1. Conservative

Conservative treatment is the first therapeutic option in most cases and usually consists of rest, analgesics, muscle relaxation, and movement restriction.

5.4.2. Surgical

Surgical indications are as follows:

(i) Severe myelopathy;
(ii) Progressive myelopathy;
(iii) Severe radiculopathy.

Surgical approaches include posterolateral (costotransversectomy, transpedicular, lateral extracavitary approach, lateral parascapular extrapleural approach) and anterolateral approaches (thoracotomy/thoracoscopic). The surgical treatment of herniated thoracic disc prolapses is associated with significant complications, such as neurological deterioration, wrong-level surgery, incomplete disc resection, postoperative instability, cerebrospinal fluid leaks, pulmonary complications, infection, intercostal neuralgia, etc. (McInerney and Ball 2000; Shirzadi et al. 2013).

6. Cervical Spondylosis and Myeloradiculopathy

The term "cervical spondylosis," which is occasionally used interchangeably with "cervical spinal stenosis," is typically used to describe cervical degenerative disc degeneration (Greenberg 2010). Age-related wear and tear in the cervical spine (neck), known as cervical spondylosis, can cause neck discomfort, stiffness, and other symptoms. Spondylosis usually implies more widespread age-related degenerative pathologies of the cervical spine, including intervertebral disc herniation, hypertrophy of the lamina, articular facets, and ligaments (lig. flavum, PLL), and subluxation of the spine. Common symptoms include neck pain or stiffness, nagging soreness in the neck, muscle spasms, dizziness, and headaches (Greenberg 2010). There are three main presentations linked to cervical spondylosis: (1) neck pain and brachialgia; (2) radiculopathy; (3) myelopathy (Deopujari and Kumar 2012).

6.1. Cervical Disc Herniation

6.1.1. Introduction

The nucleus pulposus is displaced as a result of cervical disc herniation, which may impinge on these crossing neurons as they leave the neural foramen or directly compress the spinal cord inside the spinal canal. When the nucleus pulposus partially or completely protrudes through the annulus fibrosus, disc herniation occurs. Acute or chronic occurrences of this mechanism are also possible. Chronic herniations are caused by the intervertebral disc degenerating and drying out as a result of normal ageing; these symptoms typically appear slowly or gradually and are usually less severe. Contrarily, acute herniations are typically brought on by trauma, with the nucleus pulposus protruding via a tear in the annulus fibrosus as a result (Caridi et al. 2011; Sharrak and Al Khalili 2021).

Cervical disc herniation is more common in women than in men as they age, and it is most commonly diagnosed in people between the ages of 51 and 60 (Sharrak and Al Khalili 2021).

6.1.2. Pathophysiology

A bulging nucleus pulposus is hypothesized to mechanically compress the nerve, contributing to the pathophysiology of herniated discs and to a localized rise in inflammatory cytokines (interleukin (IL)-1 and IL-6, substance P, bradykinin, TNF-alpha, and prostaglandins). Microvascular injury caused by compression forces can range in severity from a minor restriction of venous flow that produces congestion and oedema to a severe obstruction that can lead to arterial ischaemia (Sharrak and Al Khalili 2021; Rhee et al. 2007; Doughty and Bowley 2019). Posterolaterally, where the annulus fibrosus is thin and lacking the structural support of the posterior longitudinal ligament, herniations are more likely to happen (Dydyk et al. 2020).

The most commonly affected roots are C5 and C6. This can be explained by Sunderland's (Sunderland 1974) observation that the C4, C5, and C6 roots have a strong attachment to the vertebral column, while the others are

relatively free (Deopujari and Kumar 2012). Patients under 55 are much more likely to present with herniated nucleus pulposus-related radiculopathy, whereas patients over 55 are more likely to develop canal or foraminal stenosis as a result of osteophyte formation (Deopujari and Kumar 2012).

6.1.3. Clinical Features

The C5–C6 and C6–C7 vertebral bodies are the ones where cervical disc herniations most frequently happen. Symptoms will then develop at C6 and C7, respectively, as a result of this (Table 4). Axial neck ache or ipsilateral arm pain or paraesthesia in the concomitant dermatomal distribution are the most frequent subjective symptoms (Sharrak and Al Khalili 2021).

Table 4. Features of nerve root involvement as a result of compression by a herniated intervertebral disc in the cervical spine (radiculopathy).

Root Involved	Distribution of Pain	Distribution of Paraesthesia	Weak Muscles	Reflex Change
C2	Ear or eye pain, headache			
C3, C4	Ill-defined neck pain		Neck muscle spasm	
C5	Lateral arm, shoulder, neck	Lateral arm	Deltoid, supraspinatus, infraspinatus	Biceps reflex
C6	Lateral arm, forearm, thumb	Lateral arm and forearm, thumb and index finger	Biceps, brachioradialis, wrist extensors	Biceps, brachioradialis
C7	Dorsal arm and forearm, interscapular area	Middle and index finger	Triceps and wrist extensors	Diminished triceps
C8	Medial forearm and hand, 5th digit	Medial forearm and hand, 5th digit	Intrinsic muscles of hand	Finger flexor

Source: Authors' compilation based on data from Deopujari and Kumar (2012); Sharrak and Al Khalili (2021).

Cervical myelopathy occurs from the compression of the cervical spinal cord. This compression may be discogenic or spondylotic. When a herniated disc (large central disc), also called a soft disc, causes compression over the cord, this is called discogenic myelopathy. When the bony spur of an osteophyte also called a hard disc or an ossified posterior longitudinal ligament, causes a narrowing of the cervical spinal canal and results in the compression of the spinal cord, this is called spondylotic myelopathy.

The symptoms of cervical myelopathy may include the following:

- Neck pain;
- Reduced motion range;
- Stiffness;
- Weakness in the arms and hands;
- Difficulty handling small objects, such as coins or pens;
- Tingling or numbness in the arms and hands;
- Poor coordination and clumsiness of the hands;
- Balance issues.

The Spurling test, Lhermitte sign, and Hoffman test are examples of provocative tests. Acute radiculopathy can be diagnosed with the Spurling test. To detect spinal cord compression and myelopathy, the Hoffman test and the Lhermitte sign can be employed (Sharrak and Al Khalili 2021). The clinical pattern of myelopathy is characterized by the existence of long tract signs, which include hyporeflexia of deep tendon reflexes at the level of affection and hyper-reflexia below the level of affection in the upper and lower limbs, increased muscle tone or clonus, and the presence of pathological reflexes, including Hoffman's sign or Babinski's sign (UMNL below the level of the lesion and LMNL at the level of lesion) (Deopujari and Kumar 2012).

6.1.4. Classification of Cervical Myelopathy

Nurick Classification, founded on ambulatory function and gait (Derek 2021; Nurick 1972; Lasanianos et al. 2015):

Grade 0: Normal or root symptoms only;
Grade 1: Signs of spinal cord compression; normal gait;
Grade 2: Gait difficulties; however, fully employed;
Grade 3: Gait difficulties deter employment, walks unassisted;
Grade 4: Unable to walk without assistance;
Grade 5: Bed- or wheelchair-bound.

6.1.5. Investigation

Radiologic Evaluation

The diagnostic workup includes dynamic and static cervical spine X-ray, CT scan, and MRI. Cervical X-rays should be taken in the anteroposterior, lateral neutral, flexion, and extension, and oblique views, and may show loss of disc space height, foraminal osteophyte, spondylotic bars, kyphosis, posterior compression from facet arthropathy, subluxations, or late auto fusion of adjacent cervical segments. Flexion–extension lateral X-ray films may be helpful to evaluate significant instability. Oblique views can also demonstrate foraminal osteophytes.

MRI

MRI (Figure 7) is the preferred diagnostic modality for cervical disc herniation and spondylosis. MRI is helpful in assessing the spinal cord, spinal canal diameter, and various components responsible for stenosis and compression, viz., intervertebral discs, ligamentum flavum hypertrophy, and vertebral ligaments. Individuals with cervical spondylotic myelopathy frequently have greater increases in signal intensity changes on T2W MRI images at the level of spinal compression. This could be a sign of gliosis, myelomalacia, ischaemia, inflammation, or oedema (Deopujari and Kumar 2012; McCormick et al. 2003).

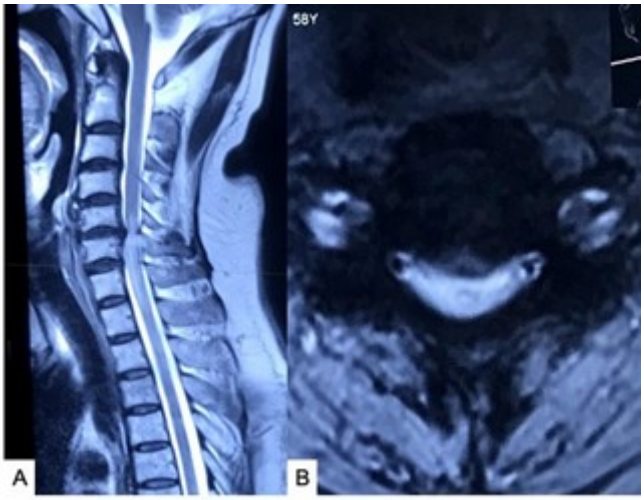


Figure 7. MRI T2W images of the cervical spine ((**A**) sagittal and (**B**) axial) showing prolapsed cervical intervertebral disc at C5 and 6 causing compression of the spinal cord with changes in signal intensity. Source: Figure by authors.

CT Scan

CT is required for better delineation of bone anatomy and OPLL. CT is also helpful in assessing the transverse foramina, facets, and the size, shape, and diameter of the spinal canal, as well as uncovertebral joints. Dynamic CT scans are the most helpful in the assessment of instability. CT myelography provides excellent visualization of radicular or cord compression in patients in whom MRI cannot be done for some reasons (Deopujari and Kumar 2012).

Neurophysiology

Patients with ambiguous symptoms or imaging results may benefit from nerve conduction and electromyography tests, which can also be used to rule out peripheral neuropathy. Brachial plexitis, carpal tunnel syndrome, and thoracic inlet syndromes can be accurately diagnosed with the help of neurophysiological studies (Deopujari and Kumar 2012; McCormick et al. 2003).

6.1.6. Treatment

A frequent degenerative condition known as cervical disc disease or spondylosis is characterized by the intervertebral disc's progressive degradation and ensuing alterations to the bones and soft tissues. Medical therapy and surgery are the two potential therapeutic treatments for cervical spondylosis and disc herniation.

(i) Medical or nonsurgical treatment: The majority of patients (75–90%) will recover from acute cervical radiculopathies brought on by a herniated disc; therefore, nonsurgical procedures are commonly used to treat them. The modalities that can be utilized include the following:

- Collar immobilization.
- Traction: intermittent cervical traction.
- Pharmacotherapy: NSAIDs and short courses of steroids, anti-neuralgic drugs, and muscle relaxants can help.
- Physical therapy: includes range-of-motion exercises, ice, strengthening exercises, heat, electrical stimulation, and ultrasound therapy.
- Cervical spine manipulation.

(ii) Interventional treatment: Injections of spinal steroids are a typical nonsurgical option. With pathological confirmation by MRI, perineural injections (translaminar and transforaminal epidurals, selective nerve root blocks) are an alternative. These procedures ought to be carried out with radiologic supervision (Sharrak and Al Khalili 2021; Eubanks 2010; Childress Marc A. 2016).

Surgical

Surgical intervention is indicated for patients with spondylosis and cervical disc herniation with the following presentations:

- disabling pain intractable to conservative therapy;
- acute spinal cord compression;
- progressive neuro-deficit and progressive muscular weakness or sensory disturbance.

Decompression of the nerve root and spinal cord and the removal of anteroposterior flattening and cervical cord distortion are the three main objectives of surgical care of patients with radiculopathy or myelopathy (Deopujari et al. 2012).

Surgical Options:

1. Anterior approach:

    - Anterior cervical discectomy without fusion (it is rarely performed nowadays).
    - Anterior cervical discectomy with fusion.

The anterior cervical discectomy with fusion procedure continues to be the gold standard because it enables the pathology to be removed and prevents recurring neural compression by conducting a fusion. Fusion can be performed by bone grafts taken from the iliac crest or cadaveric irradiated bone or using artificial devices like cages made of PEEK or titanium filled with osteogenic material. These bone grafts or devices may need stabilization with plates and screws. The disadvantage of anterior approaches is that immobility at fused level may elevate stress on adjacent disc spaces, leading to disc herniation at those levels.

Without performing fusion, mobility can be restored by artificial disc placement. This is called dynamic fusion. Normal biomechanics are restored when motion is maintained at the damaged disc level, and this has a number of benefits, including the prevention of adjacent segment degeneration and donor site morbidity and achieving early postoperative mobilization. Instability, prior fusion at an adjacent level, elderly patients (>60 years old), and severe facet arthrosis at the afflicted level are among the exclusion criteria for artificial discs (Deopujari et al. 2012).

The purposes of spinal internal fixation techniques are to establish anatomical alignment, safeguard neuronal components, and mechanically stabilize the spine while making an effort to maintain the motion of healthy spinal segments (Deopujari et al. 2012).

Hoarseness, paralysis of the tongue, difficulty swallowing, Horner's syndrome, oesophageal perforation and fistula, spinal cord/root injury, and vertebral artery injury are only a few of the potential side effects of anterior cervical surgery. A postoperative infection or haemorrhage may also result (Deopujari et al. 2012).

Graft-related complications are infection, collapse, extrusion, and donor site complications like pain and infection followed by failure of fusion (Deopujari et al. 2012; Hafez and Crockard 1997).

2. Posterior approach:

- Cervical laminectomy with or without lat. mass fixation.
- Keyhole laminotomy and foraminotomy.
- Laminoplasty.

The advantages of posterior surgical techniques are numerous. They commonly do not need stabilization, fusion, or instrumentation, and they typically take less operative time. Under direct observation, the nerve roots are decompressed with minimum risk to significant arteries and tissues. Disadvantages include postoperative neck pain, spinal instability stimulating further bone spur formation, and an inability to evaluate ventral canal osteophytes (Deopujari et al. 2012).

Foraminotomy

Root compression by extreme lateral disc herniation in the absence of cord compression is best managed by foraminotomy. It can sometimes be performed along with laminotomy.

Recommendations of the WFNS Spine Committee 2019 for Cervical Spondylotic Myelopathy

Guidelines for cervical spondylotic myelopathy (CSM) clinical presentation (WFNS Spine Committee 2019):

- Clonus, Hoffmann sign, Babinski's sign, inverted brachioradialis reflex, hyper-reflexia, and other myelopathic symptoms are essential to the clinical diagnosis of cervical myelopathy. They may, however, be absent in 20% of myelopathic individuals and are not highly sensitive.
- While not all patients may exhibit every myelopathic sign, a severe myelopathy will exhibit at least one of these.
- A patient's history and physical examination, together with other indicators, play a major role in the clinical diagnosis of CSM. These indicators and symptoms then trigger additional research using cervical spine imaging.
- In severe myelopathy patients, after laminoplasty, major recovery and improvement in myelopathic signs occur during the first 6 months and thereafter plateau.
- Treatment recommendations for individuals exhibiting myelopathic signs must be based on a mix of imaging examinations and clinical complaints, provided that no other plausible causes exist. Myelopathic indications do not always indicate CSM, and their successful surgical treatment is not impeded by their absence.

Suggestions for the natural course of cervical stenosis:

- Patients exhibiting indications of myelopathy and cervical stenosis may have a wide range of natural outcomes.
- Although it's conceivable for the disease to advance, the prognosis for those patients is unknown. Some people with severe disabilities may progress on their own without treatment, while others may remain stagnant for extended periods of time.
- The annual chance of developing myelopathy with cervical stenosis is about 3% for patients with substantial stenosis but no symptoms (pre-myelopathic).

Recommendations for electrophysiology:

- The following electrophysiological tests, in order of benefit, should be used on patients with CSM: electromyography (EMG), motor evoked potential (MEP), spinal cord evoked potential (SCEP), and somatosensory evoked potential (SEP).
- Regular electrophysiological studies help distinguish CSM from other neurological disorders in the differential diagnosis process. However, differential diagnosis is exceedingly challenging, particularly

in the early stages of the disease; specific testing are required, and it may be difficult to distinguish between moderate types of polyneuropathy and ALS.

- While it has been determined that MEP and SEP are useful tests for predicting the results of CSM surgery, there is no proof that they are more useful than clinical indicators.
- MR alterations may not be as good in predicting results as electrophysiological testing.
- Monitoring lower extremity power with electrophysiological tests is not very useful, and its usefulness during ACDF surgery is debatable.
- During CSM surgery, it has been discovered that EMG and MEP monitoring are helpful in reducing C5 root palsy.
- Clinical deterioration is not always evident in instances with intraoperative MEP/SEP worsening, and it is not specific. Modifications to the MEP/SEP during surgery may not always prevent brain damage and enhance results.

Recommendations for canal diameters on CT and MRI:

- Despite contradictory data, the preoperative workup should incorporate magnetic resonance imaging (MRI) morphometric examination of the spine, as it plays a major role in the assessment and prognostication of CSM.
- Compression ratio (CR), maximal canal compromise (MCC), and transverse area (TA) are the three factors measured by MRI that have the strongest correlations with the functional outcomes of patients with CSM after surgery. Since each parameter has advantages and disadvantages of its own, an evaluation of the MR parameters as a whole yields a more accurate prediction.

Suggestions for MRI signal intensity variations:

- A poorer prognosis in CSM may be associated with intense spinal cord T2 hyperintensity on cervical MRI.
- Patients should not be denied surgical treatment for cervical stem cell disease (CSM) if their cervical MRI shows less T2 signal abnormalities.
- More research is required to establish new grading schemes or to validate the ones that have been suggested.
- T1 hyposignal should be interpreted as an indication of a more severe illness with less hope for recovery.
- Additional research is required to determine how variations in the sagittal and axial extensions of the T1 signal affect the result.

Guiedlines for recent imaging techniques for CSM:

- Other than conventional MRI, diffusion MRI, MR spectroscopy, and dynamic MRI (dMRI) may be a part of MR examinations in a CSM imaging protocol. We recommend using them in outcome research. We will be better able to prognosticate and identify patients before the alterations and lasting harm set in with data gathered from clinical and imaging findings.

The following are recommendations for CSM, both surgical and nonsurgical:

- The WFNS Spine Committee supports Fehlings and colleagues' guidelines. Following consensus, the revised and modified WFNS Spine Committee Recommendations are outlined below.
- Surgical surgery is advised for people with moderate to severe CSM. To categorize CSM as severe, moderate, or mild, we advise utilizing the modified Japanese Orthopedic Association (mJOA) scale or its regional variants.
- For patients with mild CSM (mJOA score of 15–17), we advise providing surgical surgery or rehabilitation. If nonoperative therapy was used initially, we advise operative intervention when symptoms start to worsen quickly. For an illness that progresses slowly, nonoperative treatment may be taken into consideration.
- Prophylactic surgery should not be recommended for non-myelopathic individuals who have radiologic evidence of cord compression but do not exhibit radiculopathy symptoms. These individuals ought to receive clinical follow-up on a regular basis, counseling regarding the possibility of deterioration, and education regarding the indications and symptoms of advancement. Patients should be made aware of the possibility of neurological impairments following minor injuries.
- Non-myelopathic patients who exhibit clinical signs of radiculopathy and radiologic evidence of cord compression are high-risk candidates who should receive counseling since they may worsen. It is advised that these people have surgery, or if they decline, be under close observation and get rehabilitation. If they start exhibiting myelopathic symptoms, they should get surgery as soon as possible. Patients should be informed about neurological deficits that may follow trivial injury.

- The literature consistently shows a deficiency of data regarding the effectiveness of nonoperative treatment for cervical myelopathy. Therefore, in most circumstances, nonoperative treatment may not be the best option.
- Circumferential cord compression on axial MRI, decreased CSF space diameter, hypermobility of spinal segments, angular edged deformity, instability, greater angle of vertebral slip, lower segmental lordotic angle, and presence of OPLL are predictive factors that suggest a potential deterioration during nonoperative management. Prolonged MEPs and SEPs, symptomatic radiculopathy, and EMG indications of anterior horn cell lesions are significant predictors of the development of myelopathy (poor evidence).
- The duration of symptoms has the greatest impact on outcomes. Subpar results are the result of significant delays in surgical care. Put another way, patients who experience less symptoms for a shorter period of time following surgery are more likely to have better outcomes (poor evidence).
- The WFNS Spine Committee strongly recommends randomized controlled trials comparing surgical versus nonsurgical therapies in mild CSM, as there is still clinical equipoise between surgery and conservative treatment.
- There is also a need to analyse the cost-effectiveness and standardized methodology of long-term follow-up in mild CSM.

The following are recommendations for surgical indications for the treatment of CSM:

- Patients with CSM who have progressive neurological deficit, recurrent or persistent radiculopathy that is not responding to conservative treatment (after three years), static neurological deficit with severe radicular pain when accompanied by confirmatory imaging (CT, MRI), and clinical–radiological correlation are candidates for surgery.
- Patients with CSM who have anterior surgery indications include a straightened spine or kyphotic spine with a compression level below three.

Suggesations for comparison of anterior surgical techniques for CSM:

- There are numerous alternatives for anterior decompression, including anterior cervical discectomy and fusion (ACDF), anterior cervical corpectomy and fusion (ACCF), oblique corpectomy, skip corpectomy, and hybrid surgery.
- A corpectomy is a good option for a ventral compression of fewer than three vertebral segments in patients with CSM in whom a single-level disc and osteophyte excision are insufficient to decompress the cord. A corpectomy can correct a cervical spine kyphotic deformity and return the lordotic curvature alignment to normal.
- Alternate-segment discectomy/osteophyte removal with preservation of the intervening vertebra's body is biomechanically more stable than a total corpectomy with contiguous segment discectomy in situations of multi-segment illness with contiguous multi-segment thecal compression.

Recommendations for endoscopic as well as partial corpectomy interventions:

- The sagittal canal diameter can be significantly increased with an oblique partial corpectomy. However, in cases of bilateral radiculopathy, this surgery could be challenging to carry out. It is not advisable to choose an oblique corpectomy if there is a lot of instability.
- Some surgical method adjustments have reduced the incidence of Horner's syndrome (caused by unilateral disruption of the sympathetic chain) to less than 5%.

The following are recommendations for CSM in the elderly:

- In patients with osteophytes at C5-6-7 causing bony ankylosis, CSM may appear at lower levels, like the C7-T1 level, or at higher levels where mobility segments are intact, such as the C3-4 level.

Suggestions regarding complications from anterior procedures for CSM:

- There is a wide range of reported complications from anterior surgeries for CSM. Compared to neurologic and implant-related consequences, approach-related issues (dysphagia, dysphonia, oesophageal damage, respiratory distress, etc.) are more frequent. Surgical issues should be extremely infrequent when using careful surgical techniques and the right implants.

Suggestions for improving the success rate of anterior surgeries for CSM include:

- Improvements in 70% to 80% of patients have been documented following anterior surgery for CSM.
- Recoveries from JOA typically range from 60% to 70%.
- The success rates for ACDF, ACCF, and oblique corpectomy are not significantly different.

- Compared to ACCF, ACDF is typically linked with less intraoperative blood loss and fewer surgical complications. Functional outcomes are found to be the same when use the Neck Disability Index (NDI), JOA, and Odom's criteria.

Recommendations for choosing a surgical method:

- Patients with CSM should take into account a variety of factors when choosing a surgical strategy, including patient comorbidities, the number of levels implicated, the location of the compressive pathology, and the sagittal curvature.

Suggestions for posterior surgical techniques for CSM include:

- Posterior surgical decompression is a useful method for enhancing patients' neurological function.
- For CSM, the three posterior surgical approaches are laminectomy, laminectomy with fusion, and laminoplasty. If there are three or more levels of anterior compression, these methods are frequently applied. However, posterior decompressive procedures are required in cases when there is considerable posterior compression at one or both levels.
- It is unclear how beneficial each posterior decompression technique is in comparison to the others. When a patient has kyphosis, laminectomy and posterior fixation with fusion are the best options, particularly if the kyphosis is flexible. On the other hand, anterior surgery in conjunction with posterior decompression is the optimum treatment for rigid kyphosis. Laminotorosis preservation can benefit from laminoplasty. Laminoplasty cases with severe axial neck pain should not be considered. Nonetheless, there are always cases that fall into the gray area, such patients with a straightened cervical spine, where it's difficult to determine which course of action is preferable.
- When treating patients with substantial dorsal and ventral osteophytic compression, which cannot be addressed comprehensively with a single anterior or posterior operation, a combination approach should be used.
- Selecting the right procedure for a given patient requires consideration of a number of factors. Surgeons should customize their preoperative counseling to make patients aware of these details.

Recommendations for complications of posterior surgeries for CSM:

- Complications resulting from posterior surgeries for CSM include injury to the spinal cord and nerve roots, implant-related complications, C5 palsy, spring-back closure of the lamina after laminoplasty, and postlaminectomy kyphosis.

Recommendations for the success rate of posterior procedures for CSM include:

- A tendency indicates that laminoplasty is superior to standard laminectomy but about equal to the more recent, minimally invasive skip laminectomies.

Suggestions for the future of surgical approaches:

- The current body of knowledge is inadequate, particularly when it comes to weighing the costs and benefits of different surgical approaches, comparing the effectiveness of different surgical approaches using different techniques, and conducting long-term follow-up to ascertain results. Therefore, ongoing study on the results of cervical spine surgery is crucial.
- Prospective registries with long-term follow-up will be crucial for our future decisions, as doing randomized controlled studies in spine surgery is exceedingly challenging.

Suggestions for CSM outcome measures include the following:

- There are numerous outcome measures available. We suggest Nurick's grade, the Myelopathy Disability Index (MDI), and the modified Japanese Orthopedic Association (mJOA) scale as functional measures.
- Walking tests are useful for quantitative assessments, and the Short Form 36 (SF-36) is a useful tool for assessing functional quality of life.

Suggestions for clinical factors influencing results:

- Age, length of symptoms, and severity of myelopathy upon presentation are the three clinical factors most frequently associated with CSM. More unfavorable outcomes can be anticipated following surgery the older the patient is, the longer the symptoms have persisted, and the more severe the symptoms were at presentation.

- Nevertheless, further research is needed to confirm the impact of examination results on surgical outcomes. The following predictive characteristics have been researched and appear to influence the results in CSM: clonus, leg spasticity, hand atrophy, and Babinski's sign.

The following are recommendations for radiological characteristics that impact outcomes:

- There is a correlation between the severity of myelopathy and overall health scores and cervical alignment metrics. One of the most crucial factors has been determined to be the cervical spine's curvature.
- Worse results are predicted by cervical spine kyphosis. Notably, those with normal cervical lordosis experience significant neurological improvement.
- Results are predicted by cervical spine instability. Longer symptom duration, a worse preoperative JOA score, and more preoperative physical indicators are substantially predictive of a poor surgical outcome in patients with single-segmental CSM with instability.
- An important consideration in the prognosis of CSM is the spinal cord compression ratio. The spinal canal's AP diameter, however, is not clinically significant.
- The results of spinal cord atrophy cannot be predicted.
- On T2-weighted MR images, high signal intensity is a poor prognostic indicator.

Suggestions for surgical factors influencing results:

- If the disease is localized (affecting one or two levels), surgery should be done from the anterior or posterior.
- Posterior decompression need to be selected if the anterior compression is diffuse-narrowing or consists of more than two levels.
- When making decisions in cases involving many levels (more than two) CSM, the cervical sagittal vertical axis is the most crucial consideration.

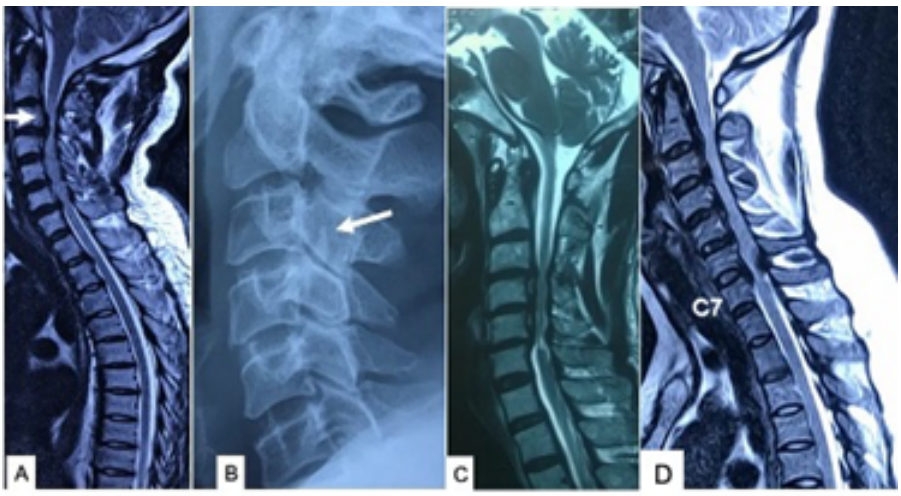
7. Ossification of the Posterior Longitudinal Ligament (OPLL)

7.1. Introduction

Although OPLL (Figure 8a,b and Figure 9) occurs in many ethnic groups, patients of Asian heritage frequently experience it. Patients with OPLL are between the ages of 32 to 81 (mean: 53), with a small male predominance. With age, the prevalence rises (Greenberg 2010). OPLL's precise pathophysiology is not entirely established (Saetia et al. 2011). The posterior longitudinal ligament first undergoes fibrosis, then calcifies, and finally ossifies (Greenberg 2010).

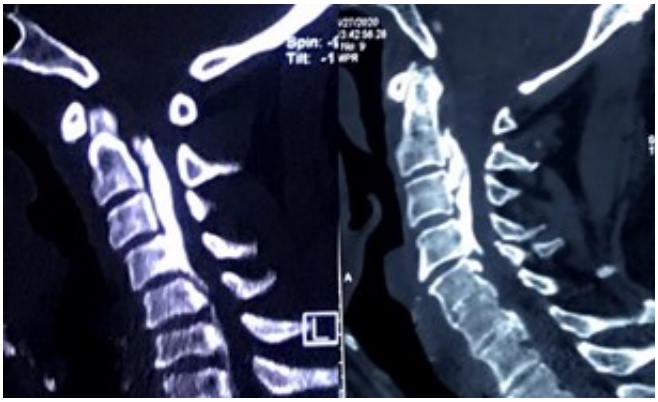
Location:

- Cervical: 75%.
- Thoracic: 15%.
- Lumbar: 10%.



(**a**)

Figure 8. *Cont.*



(**b**)

Figure 8. (**a**) MRI of cervical spine (T2W sagittal images) showing OPLL at C3-6, with cord compression mostly at C3 and 4 (**A**). X-ray of cervical spine of patient in Figure 8A (lateral view) showing calcification of OPLL at C3 and 4 (marked with an arrow) (**B**). MRI of cervical spine (T2W sagittal images) showing OPLL with cord compression at C4 & 5 and C6 & 7, respectively (**C**,**D**). (**b**) CT scan of cervical spine showing C2-4 level OPLL in two different patients. Source: Figure by authors.

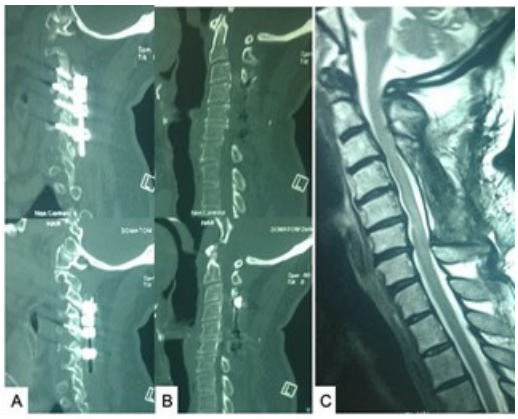


Figure 9. (**A**,**B**) CT scan of cervical spine (sagittal views) and (**C**) MRI of cervical spine (T2W sagittal view) in the same patient showing cord decompression by laminectomy at C3-6 with bilateral lateral mass fixation in a patient with OPLL. Source: Figure by authors.

7.2. Stages of Spinal Cord Damage Due to OPLL

- Stage 0: normal or mild compression of the anterior horn, no neuronal loss.
- Stage 1: mild compression of the anterior horn plus partial neuronal loss.
- Stage 2: marked deformity of anterior horn with severe neuronal loss.
- Stage 3: serious spinal cord damage.

Due to the spinal cord's ossification and aberrant signal intensity, T2W sequences are thought to be the most useful in evaluating spinal cord compression (Hirai et al. 2001).

7.3. Pathologic Classification

1. Segmental: limited to the region beneath the vertebral bodies, avoids disc gaps.
2. Continuous: spans the disc space from VB to VB(s).
3. Mixed: incorporates components of the first two while skipping areas.
4. Other variations: one of these is a rare form of OPLL that is restricted to the disc space and is continuous with the endplates and features focal PLL hypertrophy with punctate calcification (Greenberg 2010).

The majority of patients exhibit no symptoms or only minor subjective complaints (Greenberg 2010). The majority of OPLL patients who do have symptoms have them due to neurological impairments such radiculopathy, myelopathy, and/or bowel and bladder complaints (Saetia et al. 2011). The evaluation of OPLL can be achieved via plain X-ray and via CT scan of the spine. MRI of the spine will demonstrate the degree of spinal cord involvement.

7.4. Treatment Decisions Based on Clinical Grade

1. Class I: radiological proof devoid of clinical symptoms or indications. The majority of OPLL patients have no symptoms. Unless severe, conservative management is recommended.
2. Patients in Class II have myelopathy or radiculopathy. Expectantly, a minimal or stable neuro-deficit may follow. Surgical intervention is necessary when there is a significant deficiency or when progression is seen.
3. Class IIIA myelopathy ranges from moderate to severe. Surgery is typically necessary.
4. Class IIIB involves severe to total quadriplegia. For partially quadriplegic patients who are slowly getting worse, surgery is a possibility. A worse prognosis is linked to rapid worsening or total quadriplegia, old age, or poor health (Greenberg 2010).

Approaches to OPLL may be either anterior or posterior. When the OPLL involves one or two segments, an anterior approach is best; this may involve corpectomy and removal of the ossified posterior longitudinal ligament followed by fusion of the spine using a bone graft or cage. If more than two vertebrae are involved, a posterior approach is used. Laminectomy or laminoplasty with or without lateral mass fixation with rods and screws are the choices. The "K-line", which is a virtual line between the midpoints of the anteroposterior canal diameter at C2 and C7, is a useful prognostic indicator for sufficient decompression by laminoplasty for OPLL with kyphosis and/or thick ossification foci. The K-line can reflect both the alignment and the thickness of the OPLL, which determine the surgical outcomes (Fujiyoshi et al. 2008).

8. Degenerative and Inflammatory Craniovertebral Junction (CVJ) Instability: Atlantoaxial Dislocation (AAD)

8.1. Introduction

If not treated promptly and effectively, atlantoaxial dislocation (AAD), a relatively uncommon and potentially catastrophic disturbance of the normal occipital–cervical anatomy, may lead to permanent deficits or sagittal deformity (Yang et al. 2014).

Traumatic, inflammatory (including infectious), idiopathic, or congenital diseases can cause the atlantoaxial joints to become unstable (Subin et al. 1995; Chowdhury et al. 2011).

8.2. Craniocervical Junction: Anatomy

Ligaments (Figure 10) attaching the axis and atlas to the clivus, occipital bone, and occipital condyle secure the connection of the skull and cervical spine. Numerous motions of the craniocervical junction (CCJ) necessitate the stabilization of numerous ligaments. The atlas' superior articular facet and the occipital condyle combine to form the atlanto-occipital joint, which is stabilized by the articular capsule.

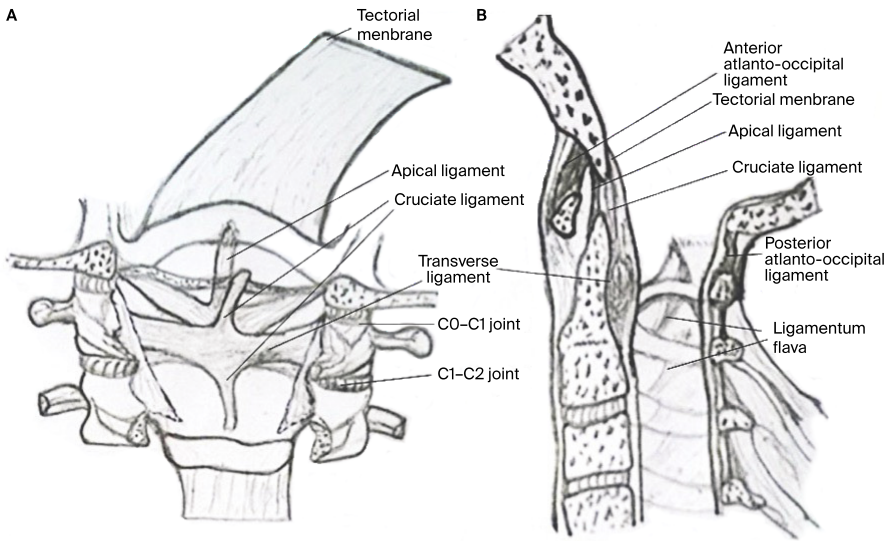


Figure 10. Schematic hand drawings of craniovertebral junction, coronal cut view (**A**) and sagittal cut view (**B**), showing anatomical organization of ligaments connecting the occiput (C0), C1, and C2. Source: Figure by authors.

This joint allows for 5° of axial rotation and 25° of flexion and extension (Hall et al. 2015; Tubbs et al. 2011). The three joints that make up the atlantoaxial segment allow for 30° of axial rotation and 15° of axial flexion. These include an atlanto-dental joint and two lateral mass articulations. The latter limits excessive extension and allows for only 10° of extension in an average individual. From the anterior arch of the C1 to the anterior side of the clivus, the anterior atlanto-occipital membrane attaches. It prevents excessive neck extension by extending the anterior longitudinal ligament. The medial occipital condyle is connected to the lateral aspect of the odontoid process via the alar ligaments. Each side has an atlanto-occipital displacement. At the atlanto-occipital joint, these ligaments prevent flexion and axial rotation on the opposite side. The apical ligament connects the tip of the odontoid process to the occipital bone, the anterior-to-superior band of the cruciate ligament, and behind to the alar ligaments. In 20% of cases, this ligament may be congenitally missing. It is frequently a rudimentary structure that contributes nothing to the mechanical stability of the CCJ. The Barkow ligament, which runs anterior to and parallel to the alar ligaments, joins the tip of the dens to the occipital condyle. This ligament might help stop overly extended neck postures. The medial occipital condyles are where the transverse occipital ligament attaches and spans the foramen magnum. This ligament may help to limit excessive lateral bending, flexion, and axial rotation because it occasionally links with the alar ligaments (Chowdhury et al. 2017; Hall et al. 2015; Tubbs et al. 2011). The superior, transverse, and inferior bands that make up the cruciform or cruciate ligament are positioned directly behind the odontoid in the middle. The odontoid to the basion is stabilized by the superior band. The cruciform ligament's transverse band, which is its strongest component, stabilizes the odontoid to the lateral masses of the C1. It avoids posterior displacement of the dens and restricts C1's lateral mobility in relation to the dens, keeping anterior C1-2 subluxation to 3 to 5 mm. The superior band is carried over into the inferior band, further solidifying the connection between the body of axis and the bason. Immediately behind the cruciate ligament is where the tectorial membrane is located. It joins the clivus laterally to the hypoglossal canals and continues as the posterior longitudinal ligament via the spinal canal. Extra flexion and extension are prevented by this ligament. The lateral masses of C1, which are located anterior to the tectorial membrane, are connected to the auxiliary atlantoaxial ligament by the posterior portion of the C2's body. This ligament's function is unknown. The occipital bone and the posterior atlas arch are connected by the posterior atlanto-occipital membrane. It carries the ligamentum flavum further. The ligamentum nuchae connects the external occipital protuberance to the spinous process of C7 and is a continuation of the supraspinous ligament. The purpose of this ligament is to limit excessive neck flexion (Chowdhury et al. 2017; Hall et al. 2015; Tubbs et al. 2010; Tubbs et al. 2011).

8.3. Aetiology

The aetiology of AAD can be grossly divided into congenital, traumatic, or inflammatory, though the cause is usually multifactorial (Yang et al. 2014).

8.3.1. Traumatic Causes

A purely traumatic AAD in the absence of a background risk factor is extremely uncommon (Venkatesan et al. 2012). It is discussed in the Chapter of spinal trauma.

8.3.2. Congenital Causes

Certain congenital diseases are linked to anomalies in the craniocervical region, which puts these populations at risk for atlantoaxial dislocation (Yang et al. 2014; Menezes et al. 1980). These are as follows:

- Down syndrome (trisomy 21) (Figure 11).
- Skeletal dysplasias (spondyloepiphyseal dysplasia, Goldenhar syndrome, and Morquio syndrome) (Figure 12) (Song and Maher 2007).
- Spondyloepiphyseal dysplasia (Miyoshi et al. 2004).
- Congenital osseous abnormalities (Wang et al. 2013) [failures in segmentation, like os odontorium (Figure 13), C2–C3 fusion, occipitalized atlas, basilar invagination (Figure 14), and asymmetrical occiput–C3 facet joints]

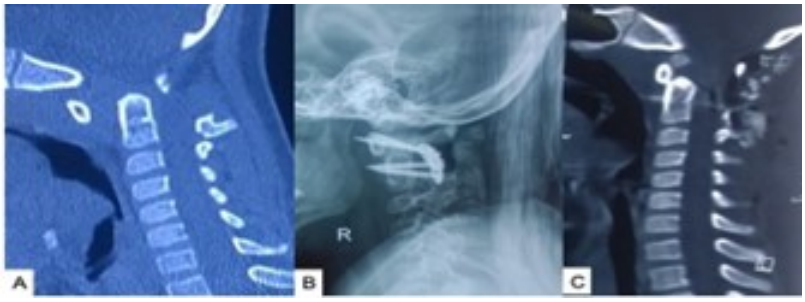


Figure 11. (**A**) CT scan of CVJ (sagittal view) showing AAD in 8-year-old boy with Down syndrome. (**B**) Postoperative plain X-ray of CVJ (lateral view) and (**C**) postoperative CT of CVJ (sagittal view) showing reduction, fixation, and fusion of AAD with lateral mass screws and plates and with facet joint fusion. Source: Figure by authors.

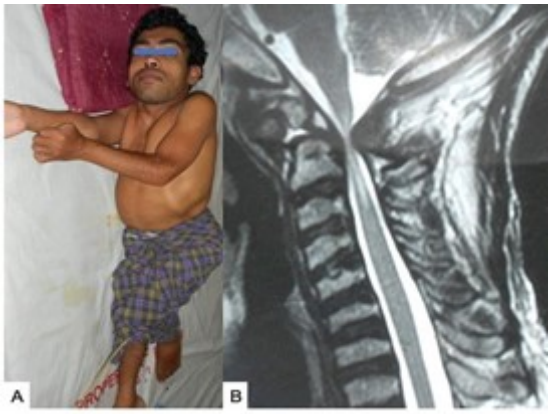


Figure 12. (**A**) A patient with skeletal dysplasia with quadriparesis. (**B**) MRI of the patient's cervical spine showing high cervical "pencil tip" compressed cord due to AAD. Source: Figure by authors.

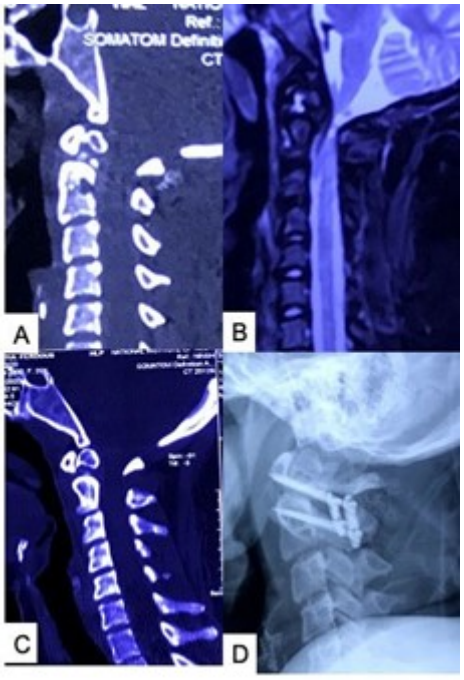


Figure 13. (**A**) CT scan of CVJ (sagittal view) showing "os odontorium" with anterior AAD. (**B**) MRI of CVJ (T2W sagittal view) showing severe cord compression. (**C**) Postoperative CT scan of CVJ in the same patient showing reduced AAD. (**D**) Postoperative X-ray (lateral view) of CVJ in the same patient showing reduction and stabilization with lateral mass screws and plates. Source: Figure by authors.

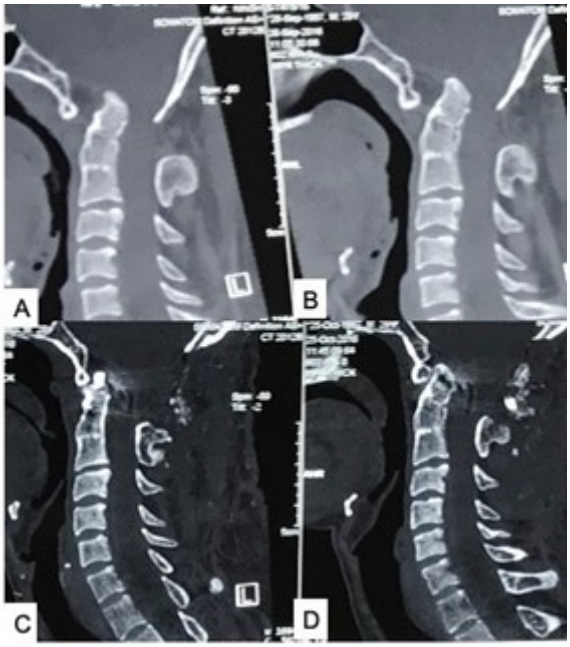


Figure 14. CT scan of CVJ (sagittal images). (**A**,**B**) Preoperative images showing AAD with basilar invagination (BI). (**C**,**D**) Postoperative images showing vertical and horizontal reduction, stabilization, and fusion. Source: Figure by authors.

8.3.3. Inflammatory Causes

Chronic inflammation in rheumatoid arthritis causes chronic synovitis, which results in bone degradation and ligament elasticity, which can cause instability and atlantoaxial dislocation (Figure 15) (Yang et al. 2014).

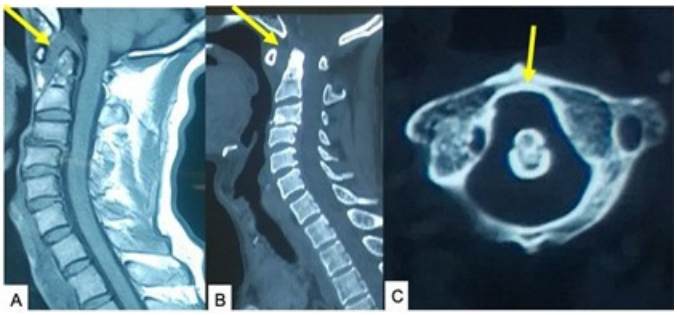


Figure 15. (**A**) MRI of CVJ in a rheumatoid arthritis patient showing atlantoaxial instability with cord compression and soft tissue mass due to pannus formation (marked with an arrow). (**B**,**C**) CT scan of CVJ (sagittal and axial views, respectively) showing AAD in the same patient. Source: Figure by authors.

8.3.4. Degenerative Causes

Osteoarthritis of C1C2 joints.

8.3.5. Infective Causes

Tuberculosis and hydatidosis.

8.3.6. Neoplastic Causes

Metastasis, lymphoma, myeloma, etc.

8.4. Clinical Presentation

Neck pain and restriction of neck movement (50%), weakness and numbness (70%), pyramidal signs (90%) (Passias et al. 2013; Yin et al. 2013), sphincter disturbances, respiratory distress. lower cranial nerve palsy,

myelopathy, respiratory failure, neurologic compromise, vertebral artery dissection, and, rarely, quadriplegia or death if left untreated (Yang et al. 2014; Panda et al. 2010).

8.5. Differential Diagnosis

Torticollis (Figure 16), atlantoaxial rotatory fixation, odontoid fractures without atlantoaxial dislocation (Yang et al. 2014).

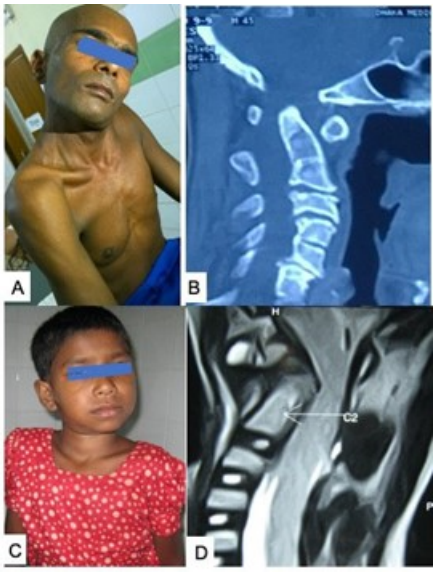


Figure 16. Adult patient with torticollis (**A**) and AAD (**B**). Paediatric patient with torticollis (**C**) and AAD (**D**). Source: Figure by authors.

8.6. Investigations and Diagnosis

8.6.1. X-Ray

Utilizing the atlanto-dental interval, X-ray radiographic measurements of the atlantoaxial joint articulation determine atlantoaxial dislocation (ADI is a small, slit-like space between the posterior aspect of the anterior atlas ring and the anterior aspect of the odontoid process). Radiographs of the neck's dynamic flexion and extension allow for the measurement of the ADI and the definition of joint reducibility (Yang et al. 2014). When the head moves, the ADI typically remains static and does not exceed 3 mm in adults and 5 mm in children (Yang et al. 2014; Passias et al. 2013).

Anterior dislocations are the primary cause of the majority (70%) of symptomatic AAD cases. The space for the spinal cord is limited as a result of anterior dislocation increasing the ADI (Yeom et al. 2013). Less than 14 mm of spinal cord space predicts the beginning of paralysis and has been linked to the severity of neurological deficiency (Yang et al. 2014; Yurube et al. 2012). Neutral and dynamic X-rays have a low diagnostic sensitivity.

8.6.2. Magnetic Resonance Imaging (MRI)

With high sensitivity and specificity, MRI is highly helpful for learning about joints, soft tissues, and the spinal cord. It can also occasionally reveal early warning signals of instability (Yeom et al. 2013).

8.6.3. Computed Tomography (CT)

Computed tomography (CT) has higher specificity for the diagnosis of CVJ instability (Yang et al. 2014). It provides skeletal details and provides essential information for surgical planning. Dynamic CT scan of the CVJ with 3D reconstruction and CTA of the vertebral arteries are warranted.

8.7. Classification of Atlantoaxial Dislocations

AAD was classified early into two subcategories by Greenberg (Yang et al. 2014):

1. Reducible;
2. Irreducible.

Hawkins and Fielding's classification system according to the direction of dislocation is as follows:

1. Posterior;
2. Anterior;
3. Lateral;
4. Rotational (Fielding and Hawkins 1977).

The Wang classification is as follows:

Type 1: instability;
Type 2: reducible dislocation;
Type 3: irreducible dislocation;
Type 4: bony dislocation (Wang et al. 2013).

The Wang classification is used for the classification and treatment strategy of AAD (Wang and Wang 2012; Wang et al. 2013). Here, preoperative assessment is conducted using dynamic X-ray, reconstructive CT, and a skeletal traction test.

8.8. Treatment

The aims of AAD treatment are as follows:

(i) Sagittal alignment correction of the upper cervical spine;
(ii) Fixation close to the anatomical alignment (Ferguson and Steffen 2003).

8.8.1. Surgical Treatment

Indications for surgery:

- Symptomatic atlantoaxial dislocation (to prevent possible respiratory failure, progressive neurological deficit, and death) (Finn et al. 2008).
- Asymptomatic AAD (to avoid myelopathy from persistent instability). In this case, though, there are some controversies (Yang et al. 2014; Panda et al. 2010).

Surgical approaches:

A. Posterior

The main surgical procedures are posterior. Posterior surgical procedures include the following:

(i) C1–C2 reduction, fusion (including facets joint), and stabilization by Goel's/Harm's technique;
(ii) Reduction and transarticular screw fixation;
(iii) Occipitocervical/C1–C2 fusion (periodontoid tissue release or/after transoral odontoidectomy).

The main complications of posterior approaches are vertebral artery injury and mobility reduction (Yeom et al. 2013). The overall complication rate in transoral surgery is 9.4% and includes CSF leakage, wound infection, wound dehiscence, pneumonia, and death (Yang et al. 2014).

B. Anterior

Anterior surgical approaches include the following:

(i) Transoral or endonasal odontoidectomy (posterior fixation is needed with it);
(ii) Anterior C1-C2 transarticular screw fixation or screw and plate fixation (retropharyngeal) (Padua et al. 2013).

A C1 lateral mass screw approach combined with a C2 pedicle screw fixation connected by rods is utilized in C1 lateral mass screw and C2 pedicle screw fixation to stabilize the atlantoaxial joint; screws and plates can also be employed (Harms and Melcher 2001; Goel and Laheri 1994; Abumi et al. 1994). When called upon, the approach permits extension to the occiput or subaxially (De Iure et al. 2009; Deen et al. 2003). Additionally, it encourages intraoperative reduction following screw fixation (Harms and Melcher 2001).

As an alternative, a crossing screw method through the C2 lamina was reported for the C1 lateral mass screw and C2 laminar screw fixation technique in 2004. But it cannot prevent lateral bending (Finn et al. 2008; Wang 2007; Lapsiwala et al. 2006) and it has a high frequency of hardware failure (Yeom et al. 2013).

8.8.2. Nonoperative Treatment

In adults who are symptomatic and there are no surgical contraindications, conservative treatment is typically not advised. Even in cases of asymptomatic AAD, stabilization by posterior arthrodesis is necessary because chronic instability frequently results in myelopathy (Healey et al. 2002).

Prior to ambulatory orthotic immobilization with active range-of-motion exercises until free motion is restored, cervical halter traction in the supine position for 24 to 48 h is recommended (Koval and Zuckerman 2006); this is the case in paediatric transverse ligament disruption and in patients with Grisel syndrome (Yang et al. 2014).

9. Atlanto-Occipital Dislocation (AOD)

The main cause of AOD is injury to the ligaments connecting the occiput to the upper cervical spine, which is frequently without comorbid fractures. As a result, it is more likely to go unnoticed than traumatic cervical spine fractures. A better comprehension of the anatomy of the craniocervical junction (CCJ) is necessary for the accurate diagnosis and management of this injury (Chowdhury et al. 2017). Different traumatic processes can cause AOD, but they are all characterized by the transmission of too much force to the craniovertebral junction (CVJ), which causes widespread ligamentous disruption. These processes can be a combination of lateral flexion, hyperextension, or hyperflexion (Hall et al. 2015; Montane et al. 1991; Yüksel et al. 2008). In the presence of relatively moderate trauma, certain predisposing factors, including neoplastic, inflammatory, neoplastic, and congenital illnesses, may enhance the risk of AOD. The CVJ may be affected by rheumatoid arthritis, which can also weaken the transverse ligament, increasing the chance of C1 subluxation. Up to 30% of the time, Down syndrome is accompanied by laxity of the craniocervical ligaments. By producing a fulcrum-like action, congenital cervical vertebral fusion disorders may also predispose people to AOD (Chowdhury et al. 2017; Montane et al. 1991; Tubbs et al. 2011).

Numerous reports have shown that trauma-related atlanto-occipital instability frequently results in death, while patients who suffer from less severe injuries may live (Papadopoulos et al. 1991; Guigui et al. 1995; Hosalkar et al. 2005). Atlanto-occipital instability caused by non-trauma is uncommon. Non-traumatic AOD (Figure 17) may present with clinical features like those of AAD (Chowdhury et al. 2017). Investigations including dynamic X-ray/fluoroscopy of the CVJ, CT scan with VA-CTA, and MRI of the CVJ are needed for detailed assessment and surgical planning.

Surgical techniques for AOD (Chowdhury et al. 2017) include the following:

- Co–C1 wiring and fusion: a midline lower-occipital burr hole is made and then occipito-atlantal fixation is performed using an epidural wire followed by fusion.
- C0–C1 transarticular fixation: here, condylar joint transarticular screw fixation is performed under fluoroscopic guidance.
- Occipital condyle C1 lateral mass screw–rod fixation and fusion (C0-C1 fixation): condylar joint fixation with screws and rods with fusion.

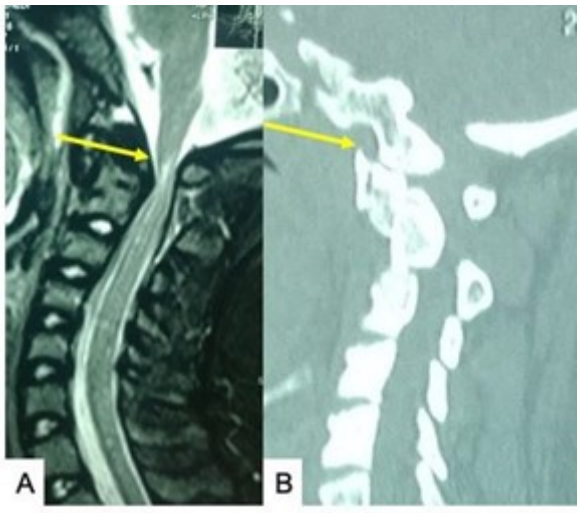


Figure 17. (**A**) MRI of CVJ showing high cervical spinal cord compression due to craniovertebral junction instability. (**B**) CT scan of CVJ in the same patient showing condylar joint or atlanto-occipital dislocation (AOD) (marked with an arrow). Source: Figure by authors.

10. Ligamentum Flavum Hypertrophy (LFH)

Ligamentum flavum hypertrophy (LFH) is a rare cause of compressive myeloradiculopathy. The anterior surfaces of the laminae of an adjacent neural arch are joined by a sequence of paired ligaments called the ligamentum flavum. The main cause of LFH is fibrosis, which develops as a result of the accumulation of mechanical stress due to trauma and age (Safak et al. 2010). Although the prevalence of LFH is unclear, several authors have put out numerous theories to explain the pathophysiology of the disease (Sairyo et al. 2007; Sairyo et al. 2005; Park et al. 2009).

The ligamentum flavum can enlarge due to degeneration, which can result in spinal canal stenosis and root discomfort (Liu et al. 2003). The two main causes of ligamentum flavum hypertrophy— injury and scar tissue—can both be widespread and occasionally unilateral. A portion of the elastic fibres is ruptured during trauma (whether slight or severe), which causes the ligamentum flavum to expand to some extent.

After repair, further hypertrophy occurs due to reparative scar formation. The disease progresses slowly, with repeated cycles of hypertrophy with calcification by repeated trauma resulting in myeloradiculopathy (Ambulgekar and Kulkarni 2021). It can occur in any part of the spinal column. The patient may present with clinical features of slowly progressing myeloradiculopathy. MRI demonstrates a hypertrophied ligamentum flavum with extension and a degree of myeloradicular compression. CT scan shows calcification of LFH (Figure 18).

Surgical decompression by laminectomy and excision of LFH is the treatment of choice in symptomatic cases. During the removal of calcified LFH, careful drilling under a microscope is needed.

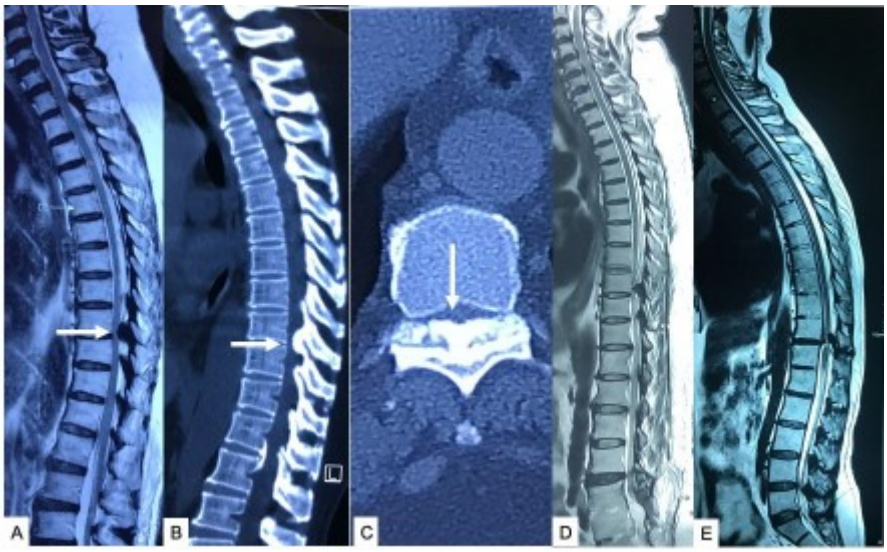


Figure 18. (**A**) MRI of spine (T2W sagittal image) showing ligamentum flavum hypertrophy (LFH), most prominent at D9 and 10. (**B**,**C**) CT scan of spine (sagittal view and axial view, respectively) showing calcification of LFH in the patient in Figure 18A. (**D**,**E**) MRI of spine (T2W sagittal images) showing dorsal LFH with dorsal spinal cord compression in two different patients. Source: Figure by authors.

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References

Abumi, Kuniyoshi, Hajime Itoh, Hiroshi Taneichi, and Kiyoshi Kaneda. 1994. Transpedicular screw fixation for traumatic lesions of the middle and lower cervical spine: Description of the techniques and preliminary report. *Journal of Spinal Disorders* 7: 19–28. [CrossRef]

Ambulgekar, Rajesh K., and Pratik G Kulkarni. 2021. Compressive myelopathy due to Ligamentum flavum hypertrophy leading to spastic paraplegia: A care report. *International Journal of Orthopaedics Sciences* 7: 568–71. [CrossRef]

Amin, Raj M., Nicholas S. Andrade, and Brian J. Neuman. 2017. Lumbar Disc Herniation. *Current Reviews in Musculoskeletal Medicine* 10: 507–16. [CrossRef] [PubMed]

Atlas, Steven J., Richard A. Deyo, Robert B. Keller, Alice M. Chapin, Donald L. Patrick, John M. Long, and Daniel E. Singer. 1996. The Maine lumbar spine study part II: 1-year outcomes of surgical and non-surgical management of sciatica. *Spine* 21: 1777–86. [CrossRef]

Bai, Jiayue, Wei Zhang, Yapeng Wang, Jilong An, Jian Zhang, Yapeng Sun, Wenyuan Ding, and Yong Shen. 2017. Application of transiliac approach to intervertebral endoscopic discectomy in L5/S1 intervertebral disc herniation. *European Journal of Medical Research* 22: 4–13. [CrossRef]

Brayda-Bruno, Marco, Marta Tibiletti, Keita Ito, Jeremy Fairbank, Fabio Galbusera, Alberto Zerbi, Sally Roberts, Ellen Wachtel, Yulia Merkher, and Sarit Sara Sivan. 2014. Advances in the diagnosis of degenerated lumbar discs and their possible clinical application. *European Spine Journal* 23: 315–23. [CrossRef]

Caridi, John M., Matthias Pumberger, and Alexander P. Hughes. 2011. Cervical radiculopathy: A review. *HSS Journal* 7: 265–72. [CrossRef]

Childress Marc A., Blair A. Becker. 2016. Nonoperative Management of Cervical Radiculopathy. *American Family Physician* 93: 746–54.

Choi, Yong-Soo. 2009. Pathophysiology of Degenerative Disc Disease. *Asian Spine Journal* 3: 39–44. [CrossRef]

Chowdhury, Forhad H., Mohammod Raziul Haque, Sarwar Murshed Alam, S.M. Noman Khaled Chowdhury, Shamsul Islam Khan, and Atul Goel. 2017. Condylar Joint Fusion and Stabilization (by Screws and Plates) in Nontraumatic Atlanto-Occipital Dislocation: Technical Report of 2 Cases. *World Neurosurgery* 107: 54–62. [CrossRef]

Chowdhury, Forhad Hossain, Md. Raziul Haque, S. M. Noman Khaled Chowdhury Chowdhury, Md. Shafiqul Islam, Zahid Raihan, and Mainul Haque Sarkar. 2011. Surgery at Cranio-vertebral (CV) Junction: Our Experience of 32 Cases. *Journal of Bangladesh College of Physicians and Surgeons* 29: 78–84. [CrossRef]

De Iure, Federico, R. Donthineni, and Stafeno Boriani. 2009. Outcomes of C1 and C2 posterior screw fixation for upper cervical spine fusion. *European Spine Journal* 18: 2–6. [CrossRef] [PubMed]

Deen, H. Gordon, Barry D. Birch, Robert E. Wharen, and Ronald Reimer. 2003. Lateral mass screw-rod fixation of the cervical spine: A prospective clinical series with 1-year follow-up. *Spine Journal* 3: 489–95. [CrossRef]

Deopujari, C. E., and Rajiv Kumar. 2012. Cervical Disc Disease and Spondylosis—Clinical Features and Diagnosis. In *Ramamurthi and Tandon's Textbook of Neurosurgery*. Edited by Prakash Narain Tandon and Ravi Ramamurthi. New Delhi: Jaypee Brothers Medical Publication, pp. 1248–51.

Deopujari, C. E., Rajiv Kumar, and Rajan Shah. 2012. Cervical Disc Disease and Spondylosis Management. In *Ramamurthi and Tandon's Textbook of Neurosurgery*. Edited by Prakash Narain Tandon and Ravi Ramamurthi. New Delhi: Jaypee Brothers Medical Publication, pp. 1252–61.

Derek, W. Moore. 2021. Cervical Myelopathy. Available online: https://www.orthobullets.com/spine/2031/cervical-myelopathy (accessed on 14 November 2022).

Doughty, Christopher T., and Michael P. Bowley. 2019. Entrapment Neuropathies of the Upper Extremity. *Medical Clinics of North America* 103: 357–70. [CrossRef]

Dydyk, Alexander M., Ruben Ngnitewe Massa, and Fassil B. Mesfin. 2020. Disc Herniation. In *StatPearls [Internet]*. Treasure Island: StatPearls Publishing.

Easwaran, Ramakrishna. 2012a. Pathophysiology of Disc Degeneration. In *Ramamurthi and Tandon's Textbook of Neurosurgery*. Edited by Prakash Narain Tandon and Ravi Ramamurthi. New Delhi: Jaypee Brothers Medical Publication, pp. 1239–47.

Easwaran, Ramakrishna. 2012b. Spondylolisthesis. In *Ramamurthi and Tandon's Textbook of Neurosurgery*. Edited by Prakash Narain Tandon and Ravi Ramamurthi. New Delhi: Jaypee Brothers Medical Publication, pp. 1297–311.

Eubanks, Jason David. 2010. Cervical radiculopathy: Nonoperative management of neck pain and radicular symptoms. *American Family Physician* 81: 33–40. [PubMed]

Ferguson, Stephen J., and Thomas Steffen. 2003. Biomechanics of the aging spine. *European Spine Journal* 12: S97–S103. [CrossRef]

Fielding, J. William, and R. J. Hawkins. 1977. Atlanto-axial rotatory fixation. (Fixed rotatory subluxation of the atlanto-axial joint). *Journal of Bone and Joint Surgery American* 59: 37–44. [CrossRef]

Finn, Michael A., Daniel R. Fassett, Todd D. Mccall, Randy Clark, Andrew T. Dailey, and Darrel S. Brodke. 2008. The cervical end of an occipitocervical fusion: A biomechanical evaluation of 3 constructs. Laboratory investigation. *Journal of Neurosurgery: Spine* 9: 296–300. [CrossRef]

Fujiyoshi, Takayuki, Masashi Yamazaki, Junko Kawabe, Tomonori Endo, Takeo Furuya, Masao Koda, Akihiko Okawa, Kazuhisa Takahashi, and Hiroaki Konishi. 2008. A new concept for making decisions regarding the surgical approach for cervical ossification of the posterior longitudinal ligament: The K-line. *Spine* 33: E990–E993. [CrossRef]

Goel, Ajay, and Vishal Laheri. 1994. Plate and screw fixation for atlanto-axial subluxation. *Acta Neurochirurgica* 129: 47–53. [CrossRef]

Greenberg, Mark S. 2010. *Handbook of Neurosurgery*. New York: Thieme.

Guigui, P., M. Milaire, G. Morvan, B. Lassale, and A. Deburge. 1995. Traumatic atlanto-occipital dislocation with survival: Case report and review of the literature. *European Spine Journal* 4: 242–47. [CrossRef] [PubMed]

Hafez, R. F., and H. A. Crockard. 1997. Failure of osseous conduction with interbody B.O.P. graft. *British Journal of Neurosurgery* 11: 57–59. [CrossRef] [PubMed]

Hagen, Kåre B., Gunvor Hilde, Gro Jamtvedt, and Michael F. Winnem. 2000. The Cochrane review of bed rest for acute low back pain and sciatica. *Spine* 25: 2932–39. [CrossRef] [PubMed]

Hall, Graham C., Michael J. Kinsman, Ryan G. Nazar, Rob T. Hruska, Kevin J. Mansfield, Maxwell Boakye, and Ralph Rahme. 2015. Atlanto-occipital dislocation. *World Journal of Orthopedics* 6: 236–43. [CrossRef]

Harms, Jürgen, and Robert P. Melcher. 2001. Posterior C1-C2 fusion with polyaxial screw and rod fixation. *Spine* 26: 2467–71. [CrossRef] [PubMed]

Hayes, Mark A., Thomas C. Howard, Curtis R. Gruel, and Joseph A. Kopta. 1989. Roentgenographic evaluation of lumbar spine flexion- extension in asymptomatic individuals. *Spine* 14: 327–31. [CrossRef]

Healey, David, Merv Letts, and James G. Jarvis. 2002. Cervical spine instability in children with Goldenhar's syndrome. *Canadian Journal of Surgery* 45: 341–4.

Hirai, Toshinori, Yukunori Korogi, Mutsumasa Takahashi, and Osamu Shimomura. 2001. Ossification of the posterior longitudinal ligament and ligamentum flavum: Imaging features. *Seminars in Musculoskeletal Radiology* 5: 83–8. [CrossRef]

Hosalkar, Harish S., Eric L. Cain, David Horn, Kingsley R. Chin, John P. Dormans, and Denis S. Drummond. 2005. Traumatic atlanto-occipital dislocation in children. *Journal of Bone & Joint Surgery American* 87: 2480–88. [CrossRef]

Kaiser, M. G., A. T. Parsa, B. D. Birch, and P. C. McCormick. 2000. The Intervertebral Disc. In *Neurosurgery. The Scientific Basis of Clinical Practice*. Edited by Alan Crockard, Richard Hayward and Julian T. Hoff. London: Blackwell Science, pp. 1061–72.

Kalb, Samuel, Nikolay L. Martirosyan, M. Yashar S. Kalani, Guy G. Broc, and Nicholas Theodore. 2012. Genetics of the degenerated intervertebral disc. *World Neurosurgery* 77: 491–501. [CrossRef]

Karantanas, A. H., A. H. Zibis, M. Papaliaga, E. Georgiou, and S. Rousogiannis. 1998. Dimensions of the lumbar spinal canal: Variations and correlations with somatometric parameters using CT. *European Radiology* 8: 1581–5. [CrossRef] [PubMed]

Kawaguchi, Yoshiharu. 2018. Genetic background of degenerative disc disease in the lumbar spine. *Spine Surgery and Related Research* 2: 98–112. [CrossRef]

Koval, Keneth J., and Joseph D. Zuckerman. 2006. *Handbook of Fractures*. Philadelphia: Lippincott Williams & Wilkins.

Lapsiwala, Samir B., Paul A. Anderson, Ashish Oza, and Daniel K. Resnick. 2006. Biomechanical comparison of four C1 to C2 rigid fixative techniques: Anterior transarticular, posterior transarticular, C1 to C2 pedicle, and C1 to C2 intralaminar screws. *Neurosurgery* 58: 516–21. [CrossRef]

Lasanianos, Nick G., George K. Triantafyllopoulos, and Spiros G. Pneumaticos. 2015. Myelopathy: Nurick Grading System. In *Trauma and Orthopaedic Classifications*. Edited by Nick G. Lasanianos, Nikolaos K. Kanakaris and Peter V. Giannoudis. Berlin and Heidelberg: Springer, pp. 255–56.

Liu, L. M., Y. M. Song, and Quan Gong. 2003. Treatment of lumbarstenosis and root pain resulting from simple hypertrophy of lumbar Ligamenta flava. *ZhongguoXiu Fu ChongJianWai Ke Za Zhi* 17: 50–51.

Luoma, Katariina, Hilkka Riihimäki, Raili Raininko, Ritva Luukkonen, Antti Lamminen, and Eira Viikari-Juntura. 1998. Lumbar disc degeneration in relation to occupation. *Scandinavian Journal of Work, Environment & Health* 24: 358–66.

Massachusetts General Hospital. 2016. *Spondylolysis and Spondylolisthesis of the Lumbar Spine*. Boston: Massachusetts General Hospital.

Mayer, Jillian E., James C. Iatridis, Danny Chan, Sheeraz A. Qureshi, Omri Gottesman, and Andrew C. Hecht. 2013. Genetic polymorphisms associated with intervertebral disc degeneration. *Spine Journal* 13: 299–317. [CrossRef] [PubMed]

McCormick, William E., Michael P. Steinmetz, and Edward C. Benzel. 2003. Cervical spondylotic myelopathy: Make the difficult diagnosis, then refer for surgery. *Cleveland Clinic Journal of Medicine* 70: 899–904. [CrossRef] [PubMed]

McInerney, James, and Perry A. Ball. 2000. The pathophysiology of thoracic disc disease. *Neurosurgical Focus* 9: E1. [CrossRef]

Menezes, Arnold H., John C. VanGilder, Carl J. Graf, and Dennis E. McDonnell. 1980. Craniocervical abnormalities. A comprehensive surgical approach. *Journal of Neurosurgery* 53: 444–55. [CrossRef]

Miyoshi, Kota, Kozo Nakamura, Nobuhiko Haga, and Yoji Mikami. 2004. Surgical treatment for atlantoaxial subluxation with myelopathy in spondyloepiphyseal dysplasia congenita. *Spine* 29: E488–E491. [CrossRef]

Montane, Ismael, Frank J. Eismont, and Barth A. Green. 1991. Traumatic occipitoatlantal dislocation. *Spine* 16: 112–6. [CrossRef] [PubMed]

Nurick, S. 1972. The pathogenesis of the spinal cord disorder associated with cervical spondylosis. *Brain* 95: 87–100. [CrossRef] [PubMed]

Österman, Heikki, Seppo Seitsalo, Jaro Karppinen, and Antti Malmivaara. 2006. Effectiveness of microdiscectomy for lumbar disc herniation. *Spine* 31: 2409–14. [CrossRef] [PubMed]

Overdevest, Gijsbert M., Wilco C. Peul, Ronald Brand, Bart W. Koes, Ronald H. M. A. Bartels, Wee F. Tan, and Mark P. Arts. 2017. Tubular discectomy versus conventional microdiscectomy for the treatment of lumbar disc herniation: Long-term results of a randomised controlled trial. *Journal of Neurology, Neurosurgery and Psychiatry* 88: 1008–16. [CrossRef] [PubMed]

Padua, Mary R. A., Jin S. Yeom, So Y. Lee, So M. Lee, Ho-Joong Kim, Bong-Soon Chang, Choon-Ki Lee, and K. Daniel Riew. 2013. Fluoroscopically guided anterior atlantoaxial transarticular screws: A feasibility and trajectory study using CT-based simulation software. *The Spine Journal* 13: 1455–63. [CrossRef]

Panda, Samhita, S. Ravishankar, and D. Nagaraja. 2010. Bilateral vertebral artery dissection caused by atlantoaxial dislocation. *Journal of the Association of Physicians of India* 58: 187–89.

Panigrahi, M. K., and S. R. Reddy. 2012. Lumber disc disease. In *Ramamurthi and Tandon's Textbook of Neurosurgery*. Edited by Prakash Narain Tandon and Ravi Ramamurthi. New Delhi: Jaypee Brothers Medical Publication, pp. 1280–89.

Papadopoulos, Stephen M., Curtis A. Dickman, Volker K. H. Sonntag, Harold L. Rekate, and Robert F. Spetzler. 1991. Traumatic atlantooccipital dislocation with survival. *Neurosurgery* 28: 574–79. [CrossRef]

Park, Jong-Beom, Chae-Gwan Kong, Kyung-Hwan Suhl, Eun-Deok Chang, and K. Daniel Riew. 2009. The increased expression of matrix metalloproteinases associated with elastin degradation and fibrosis of the Ligamentum flavum in patients with lumbar spinal stenosis. *Clinics in Orthopedic Surgery* 1: 81–89. [CrossRef]

Passias, Peter G., Shenglin Wang, Michal Kozanek, Shaobai Wang, and Chao Wang. 2013. Relationship between the alignment of the occipitoaxial and subaxial cervical spine in patients with congenital atlantoxial dislocations. *Journal of Spinal Disorders and Techniques* 26: 15–21. [CrossRef]

Phan, Kevin, Joshua Xu, Konrad Schultz, Mohammed Ali Alvi, Victor M. Lu, Panagiotis Kerezoudis, Patrick R. Maloney, Meghan E. Murphy, Ralph J. Mobbs, and Mohamad Bydon. 2017. Full-endoscopic versus micro-endoscopic and open discectomy: A systematic review and meta-analysis of outcomes and complications. *Clinical Neurology and Neurosurgery* 154: 1–12. [CrossRef]

Pytel, Peter, Robert L. Wollmann, Richard G. Fessler, Thomas N. Krausz, and Anthony G. Montag. 2006. Degenerative Spine Disease Pathologic Findings in 985 Surgical Specimens. *American Journal of Clinical Pathology* 125: 193–202. [CrossRef]

Rhee, John M., Tim Yoon, and K. Daniel Riew. 2007. Cervical radiculopathy. *Journal of the American Academy of Orthopaedic Surgeons* 15: 486–94. [CrossRef]

Roberts, Sally, Helena Evans, Jayesh Trivedi, and Janis Menage. 2006. Histology and pathology of the human intervertebral disc. *Journal of Bone and Joint Surgery American* 88: 10–14.

Saetia, Kriangsak, Dosang Cho, Sangkook Lee, Daniel H. Kim, and Sang Don Kim. 2011. Ossification of the posterior longitudinal ligament: A review. *Neurosurgical Focus* 30: E1. [CrossRef]

Safak, Alp Alper, Merih Is, Ozdemir Sevinc, Cagatay Barut, Nesrin Eryoruk, Besir Erdogmus, and Murat Dosoglu. 2010. The thickness of the Ligamenta flava in relation to age and gender. *Clinical Anatomy* 23: 79–83. [CrossRef]

Sairyo, Koichi, Ashok Biyani, Vijay Goel, Douglas Leaman, Robert Booth Jr, Jean Thomas, Daniel Gehling, Lakshmi Vishnubhotla, Rebecca Long, and Nabil Ebraheim. 2005. Pathomechanism of Ligamentum flavum hypertrophy: A multidisciplinary investigation based on clinical, biomechanical, histologic, and biologic assessments. *Spine* 30: 2649–56. [CrossRef]

Sairyo, Koichi, Ashok Biyani, Vijay K. Goel, Douglas W. Leaman, Robert Booth Jr, Jean Thomas, Nabil A. Ebraheim, Ian A. Cowgill, and Suneeth E. Mohan. 2007. Lumbar Ligamentum flavum hypertrophy is due to accumulation of inflammation related scar tissue. *Spine* 32: E340–E347. [CrossRef]

Sharrak, Samir, and Yasir Al Khalili. 2021. Cervical Disc Herniation. In *StatPearls [Internet]*; Treasure Island: StatPearls Publishing. Available online: https://www.ncbi.nlm.nih.gov/books/NBK546618/ (accessed on 15 November 2022).

Shirzadi, Ali, Doniel Drazin, Sunil Jeswani, Leah Lovely, and John Liu. 2013. A typical presentation of thoracic disc herniation: Case series and review of literature. *Case Report in Orthopedics* 2013: 621476. [CrossRef]

Singh, Kern, Koichi Masuda, Eugene J-M. A. Thonar, Howard S. An, and Gabriella Cs-Szabo. 2009. Age-related changes in the extracellular matrix of nucleus pulposus and anulus fibrosus of human intervertebral disc. *Spine* 34: 10–6. [CrossRef]

Song, Debbie, and Cormac O. Maher. 2007. Spinal disorders associated with skeletal dysplasias and syndromes. *Neurosurgery Clinics of North America* 18: 499–514. [CrossRef]

Subin, Bill, Jin-Fa Liu, G. June Marshall, Hua-Yang Huang, Jin-Hua Ou, and Gue-Zou Xu. 1995. Transoral anterior decompression and fusion of chronic irreducible atlantoaxial dislocation with spinal cord compression. *Spine* 20: 1233–40. [CrossRef]

Sunderland, Sydney. 1974. Meningo neural relations in the intervertebral foramen. *Journal of Neurosurgery* 40: 756–63. [CrossRef]

Tonosu, Juichi, Yasushi Oshima, Ryutaro Shiboi, Akihiko Hayashi, Yuichi Takano, Hirohiko Inanami, and Hisashi Koga. 2016. Consideration of proper operative route for interlaminar approach for percutaneous endoscopic lumbar discectomy. *Journal of Spine Surgery* 2: 281–88. [CrossRef]

Tubbs, R. Shane, Joshua Dixon, Marios Loukas, Mohammadali M. Shoja, and Aaron A. Cohen-Gadol. 2010. Ligament of Barkow of the craniocervical junction: Its anatomy and potential clinical and functional significance. *Journal of Neurosurgery: Spine* 12: 619–22. [CrossRef]

Tubbs, R. Shane, Justin D. Hallock, Virginia Radcliff, Robert P. Naftel, Martin Mortazavi, Mohammadali M. Shoja, Marios Loukas, and Aaron A. Cohen-Gadol. 2011. Ligaments of the craniocervical junction. *Journal of Neurosurgery: Spine* 14: 697–709. [CrossRef]

Venkatesan, Muralidharan, Raj Bhatt, and Martyn L. Newey. 2012. Traumatic atlantoaxial rotatory subluxation (TAARS) in adults: A report of two cases and literature review. *Injury* 43: 1212–15. [CrossRef]

Wang, Michael Y. 2007. Cervical crossing laminar screws: Early clinical results and complications. *Operative Neurosurgery* 61: 311–15. [CrossRef]

Wang, Shenglin, and C. Wang. 2012. A novel surgical classification and treatment strategy for atlantoaxial dislocations based on 1106 cases over 14 years. Paper presented at Cervical Spine Research Society, Chicago, IL, USA, December 7.

Wang, Shenglin, Chao Wang, Ming Yan, Haitao Zhou, and Gengting Dang. 2013. Novel surgical classification and treatment strategy for atlantoaxial dislocations. *Spine* 38: E1348–E1356. [CrossRef]

Weinstein, James N., Tor D. Tosteson, Jon D. Lurie, Anna N. A. Tosteson, Brett Hanscom, Jonathan S. Skinner, William A. Abdu, Alan S. Hilibrand, Scott D. Boden, and Richard A. Deyo. 2006. Surgical vs nonoperative treatment for lumbardisk herniation. *JAMA* 296: 2441. [CrossRef]

WFNS Spine Committee. 2019. Cervical Spondylotic Myelopathy. Available online: https://wfns-spine.org/pdf/Recommendations-of-WFNS-Spine-Committee.pdf (accessed on 12 March 2024).

Wiltse, Leon L., P. H. Newman, and Ian Macnab. 1976. Classification of spon-dylolisis and spondylolisthesis. *Clinical Orthopaedics and Related Research* 117: 23–9.

Yang, Sun Y., Anthony J. Boniello, Caroline E. Poorman, Andy L. Chang, Shenglin Wang, and Peter G. Passias. 2014. A review of the diagnosis and treatment of atlantoaxial dislocations. *Global Spine Journal* 4: 197–210. [CrossRef]

Yeom, Jin S., Jacob M. Buchowski, Ho-Joong Kim, Bong-Soon Chang, Choon-Ki Lee, and K. Daniel Riew. 2013. Risk of vertebral artery injury: Comparison between C1-C2 transarticular and C2 pedicle screws. *Spine Journal* 13: 775–85. [CrossRef]

Yin, Yi-Heng, Guang-Yu Qiao, Xin-Guang Yu, Huai-Yu Tong, and Yuan-Zheng Zhang. 2013. Posterior realignment of irreducible atlantoaxial dislocation with C1-C2 screw and rod system: A technique of direct reduction and fixation. *Spine Journal* 13: 1864–71. [CrossRef]

Yüksel, K. Zafer, Murvet Yüksel, L. Fernando Gonzalez, Seungwon Baek, Joseph E. Heiserman, Volker KH Sonntag, and Neil R. Crawford. 2008. Occipitocervical vertical distractioninjuries: Anatomical biomechanical, and 3-tesla magnetic resonance imaging investigation. *Spine* 33: 2066–73. [CrossRef]

Yurube, Takashi, Masatoshi Sumi, Kotaro Nishida, Hiroshi Miyamoto, Kozo Kohyama, Tsukasa Matsubara, Yasushi Miura, Daisuke Sugiyama, and Minoru Doita. 2012. Incidence and aggravation of cervical spine instabilities in rheumatoid arthritis: A prospective minimum 5-year follow-up study of patients initially without cervical involvement. *Spine* 37: 2136–44. [CrossRef]