

Thoracic Myelopathy & Ossification of the Ligamentum Flavum (OLF)

A patient guide

In brief: Thoracic myelopathy is a disturbance of the spinal cord within the thoracic spine, the long mid-section of the back behind the chest. It is far less common than the cervical myelopathy of the neck or the stenosis of the lower back, and it is one of the more frequently delayed diagnoses in spinal practice, partly because its symptoms in the legs and bladder can closely imitate problems arising lower down. The commonest single cause, especially in East Asian populations, is ossification of the ligamentum flavum, a condition in which one of the spine's stabilising ligaments gradually turns to bone and presses on the cord from behind. The thoracic cord sits in a narrow canal with a famously precarious blood supply, and these two facts together explain why this region behaves so differently from its neighbours and why surgery here is approached with particular care.

What thoracic myelopathy is (and why the thoracic spine is different)

The spinal cord is the great cable of nervous tissue that runs down from the brain inside the protective tunnel of the spine. Myelopathy is the word for any disturbance of the cord itself, as distinct from disturbance of the individual nerve roots that branch off it. Thoracic myelopathy, then, is dysfunction of the cord at the level of the thoracic spine, the twelve vertebrae of the upper and middle back to which the ribs attach.

The first and most important idea is that the cord does not run the whole length of the spine. It tapers and ends at around the first lumbar vertebra, just below the bottom of the thoracic spine. Below that point the canal contains only a brush of nerve roots, the cauda equina, and no cord at all. This single anatomical fact separates the thoracic spine from the lumbar spine beneath it. Compression in the thoracic region squeezes the cord, an injury to the central nervous system that tends to produce stiffness, spasticity and brisk reflexes in the legs. Compression in the lower lumbar spine, by contrast, squeezes nerve roots, which tends to produce the opposite picture of slackness and the cramping leg pain of walking known as claudication. The two can feel alike to the person experiencing them, yet they are mechanically and neurologically distinct.

The thoracic spine is also different in its dimensions and its shape. The canal here is the narrowest in relation to the cord it carries, with the least room to spare, so that a degree of intrusion which might be tolerated in the neck can become critical in the thorax. The thoracic spine also carries a natural forward curve, the kyphosis, which drapes the cord gently against the front wall of the canal. Where disease arises from the front, this curve presses the cord onto it.

The final distinguishing feature, and perhaps the most consequential, is the blood supply. The middle portion of the thoracic cord is a watershed, an area fed by the thinnest and least redundant supply of any part of the cord, depending heavily on a single dominant feeding vessel that usually enters in the lower thoracic region. A watershed, in this sense, is a frontier territory served by the end of a supply line, and like all such territories it is the first to suffer if the supply is disturbed. This precarious blood supply runs through everything that follows, and it is the reason the thoracic cord is regarded as uniquely intolerant.

How it differs from its neighbours

Because thoracic myelopathy borrows symptoms from the regions above and below it, it is worth setting out plainly how it differs from the conditions with which it is most often confused.

It differs from **cervical myelopathy**, the cord compression of the neck, chiefly in what it spares. Cervical myelopathy disturbs the cord high up and characteristically affects the hands and arms, producing clumsiness, loss

of fine movements and difficulty with buttons or handwriting, alongside changes in the legs. Thoracic myelopathy arises below the level of the arms, so the hands are typically untouched, and the disturbance is confined to the legs, the trunk and the bladder. A person with heavy, stiff legs but entirely normal hands is showing a pattern that points below the neck.

It differs from **thoracic radiculopathy**, the irritation of a single nerve root as it leaves the thoracic spine. A radiculopathy produces a band of pain or altered sensation running around the chest or abdomen along the line of one nerve, often described as a girdle or a tight strap, but it does not disturb the cord and so does not cause spasticity, imbalance or a sensory level. Myelopathy involves the long pathways of the cord itself and therefore adds these features.

It differs from **thoracic disc herniation** in that a herniated disc is one of the causes of thoracic myelopathy rather than a separate thing. A disc that protrudes and hardens at the front of the canal is one way the cord can be compressed from in front, whereas ossification of the ligamentum flavum compresses it from behind.

It differs most importantly, and most treacherously, from **lumbar spinal stenosis**, the narrowing of the canal in the lower back. Both can cause leg symptoms, walking difficulty and bladder change, and the leg complaints of thoracic myelopathy are frequently attributed to the lumbar spine, where narrowing is so common on scans that it is easily assumed to be the culprit. The distinction lies in the type of signs: lumbar stenosis tends to produce reduced reflexes and claudication eased by sitting and bending forward, whereas thoracic myelopathy produces brisk reflexes, spasticity and a sensory level on the trunk. This false localisation is a recognised reason why thoracic myelopathy is diagnosed late.

Ossification of the ligamentum flavum (OLF)

The ligamentum flavum is one of the spine's quiet workhorses. It is a short, elastic ligament, yellow in colour, that links the bony arches of adjacent vertebrae along the back wall of the canal and helps hold the spine together while allowing it to bend. Ossification of the ligamentum flavum, abbreviated to OLF, is the process by which this ligament gradually loses its elasticity and turns to bone. As it thickens and hardens, it bulges forwards into the canal and presses on the cord from behind.

OLF is the commonest single cause of thoracic myelopathy, and it is markedly more frequent in people of East Asian heritage, in whom it has been studied most extensively. In Japanese populations the average prevalence of thoracic OLF has been reported at around one in eight, more often in men than in women, although only a minority of those affected ever develop symptoms. It is associated in the literature with diffuse idiopathic skeletal hyperostosis, with diabetes, and with disorders of calcium and phosphate handling, conditions that share a tendency for soft tissues to mineralise.

The location of OLF is one of its most characteristic features. It clusters strongly at the **lower thoracic levels**, particularly around the tenth, eleventh and twelfth thoracic vertebrae and the junction with the lumbar spine. In one large surgical series of thoracic myelopathy, ossification of the ligamentum flavum accounted for roughly half of all cases, and around two thirds of those lay at these lower thoracic levels. The mechanical stresses concentrated at the relatively mobile thoracolumbar junction are thought to drive this distribution. The ossified ligament has been described in several shapes, from small lateral nodules to larger fused masses that bridge across the midline, and the larger and more bridging the ossification, the more it tends to narrow the canal.

A particular surgical concern with OLF is that the ossification can extend into the dura, the tough membrane that wraps the cord. When the dura itself ossifies, it becomes fused to the ligament and to the cord's covering, which makes removal more demanding and raises the chance of a leak of the fluid that surrounds the cord. This dural ossification is discussed further under assessment, because recognising it in advance is part of understanding the condition.

Thoracic OPLL, calcified disc, and the double crush

Although ossification of the ligamentum flavum is the leading cause, the thoracic cord can be compressed from the front as well as from behind, and the distinction between front and back disease shapes everything about how the condition is understood.

Ossification of the posterior longitudinal ligament, abbreviated to OPLL, is the front-of-canal counterpart to OLF. Here it is the posterior longitudinal ligament, which runs down the back of the vertebral bodies on the inside of the canal, that turns to bone. Because it lies in front of the cord, thoracic OPLL presses the cord backwards, and the natural forward curve of the thoracic spine tends to drive the cord onto it. Front-of-canal disease of this kind is technically more demanding to address, because the cord lies between the surgeon and the problem.

A **calcified thoracic disc** is another cause of front-of-canal compression. A disc that herniates in the thoracic spine often hardens and calcifies over time, forming a firm, sometimes broad lump that indents the cord from in front. These calcified central discs behave quite differently from the soft discs of the lower back and are recognised as one of the more challenging problems in spinal surgery.

The **double crush**, or tandem ossification, describes the situation in which the cord is squeezed from more than one direction or at more than one place. The ligamentum flavum behind and the posterior longitudinal ligament in front may ossify at the same level, gripping the cord from both sides at once. Alternatively, ossification in the neck may coexist with ossification in the thoracic spine, so that the cord is compromised at two separate levels. Coexisting thoracic ligament ossification has been reported in a substantial proportion of people with ossification in the neck, which is why the thoracic spine is given attention when widespread ossification is recognised. The practical importance of the double crush is that treating one site may not relieve symptoms if a second site is also at work, a recognised reason for persistent or unexplained myelopathy.

The thoracic cord's blood supply and why decompression carries risk

The reason thoracic myelopathy is treated with such caution lies less in the bones than in the blood. The spinal cord is fed by a network of small arteries, but this network is not even along its length. The neck and the lowest part of the cord are relatively well supplied, with several contributing vessels. The middle of the thoracic cord is not. It is a watershed zone, the frontier where two supply territories meet and where the feeding vessels are at their thinnest and fewest.

The cord in this region leans heavily on a single dominant feeding artery, known by the name of the anatomist who described it, the artery of Adamkiewicz, which most often enters the canal in the lower thoracic or upper lumbar region, usually on the left. Because so much depends on this one vessel and on a slender front artery running down the cord, even a small disturbance to the blood supply in this territory can tip the cord into a shortage of oxygen. This vulnerability is intrinsic to the anatomy and is present before any surgeon arrives.

The consequence is that thoracic decompression carries a higher risk of neurological deterioration than the equivalent surgery in the neck or the lower back. There are several reasons, and they compound one another. The canal is narrow and the cord has little reserve space, so there is little margin for any swelling or manipulation. The cord is already compressed and therefore already working at the edge of its tolerance. And the blood supply is precarious, so any drop in perfusion is poorly compensated. For all these reasons the published literature on thoracic decompression documents a recognised, if modest, rate of worsened neurological function around the time of surgery, higher than for cervical or lumbar procedures, and this risk is one of the defining facts of the condition rather than a reflection on any particular operation or operator. It is the reason techniques have evolved specifically to minimise handling of the cord, and the reason monitoring of cord function during surgery is widely used.

How it tends to present

Thoracic myelopathy characteristically announces itself slowly and quietly, which is part of why it is so often recognised late. The onset is usually insidious, and the course is one of gradual decline, sometimes in steps, over

months or even years.

The earliest and most consistent complaints involve the legs. People describe heaviness, stiffness, weakness or a sense that the legs no longer obey readily, and above all a disturbance of walking and balance. The gait may become stiff and effortful, with a tendency to stumble or to feel unsteady, particularly on uneven ground or in the dark. Because the cord rather than the nerve roots is involved, the legs tend towards spasticity, a velvety resistance and over-readiness of the muscles, with brisk reflexes, rather than the slackness seen with nerve-root problems.

Sensation is frequently affected. Numbness or altered feeling commonly begins in the feet and ascends, and many people notice a band of tightness or altered sensation around the trunk, the so-called girdle sensation, marking the level of the compression. A sensory level of this kind, a line on the trunk below which feeling is altered, is a hallmark of cord disease and points towards the thoracic spine rather than the limbs.

In more advanced disease the bladder and bowel may be affected, with urgency, hesitancy or difficulty with control. The arms and hands are characteristically normal, which, as described above, helps distinguish the thoracic from the cervical pattern. Back pain or a band of girdle pain may accompany these features but is variable and is not the dominant complaint. None of this describes any particular individual, and the presence or absence of any feature in a given person is a matter for direct assessment.

How it is assessed (MRI and CT)

The assessment of thoracic myelopathy rests on imaging, because the diagnosis depends on showing both that the cord is compressed and what is compressing it.

****Magnetic resonance imaging, the MRI scan****, is the central investigation. It shows the cord directly, reveals where and how severely it is compressed, and identifies the level responsible. It also shows changes within the cord itself: a bright signal within the compressed segment reflects strain or damage to the cord tissue, a finding that carries weight when judging how established the problem is. Importantly, MRI of the thoracic spine is often arranged precisely because leg or bladder symptoms were not explained by scans of the neck or lower back, the thoracic spine being the region that is easy to overlook.

****Computed tomography, the CT scan****, is the complementary investigation and is particularly important here because the commonest causes are made of bone. CT shows ossification of the ligamentum flavum, ossification of the posterior longitudinal ligament and calcified discs far more clearly than MRI, defining their shape, their density and their extent. Where MRI shows the effect on the cord, CT shows the nature of the offending tissue.

CT has a second, specific role in ossification of the ligamentum flavum, which is to look for signs that the dura, the cord's covering, has also turned to bone. Several recognised appearances on CT, described by names such as the tram-track sign and the double-layer sign, suggest that the ligament and the dura have ossified together. This matters because dural ossification is common in surgically treated cases and substantially raises the chance of a leak of spinal fluid if the ossified tissue is removed. Recognising it in advance is part of a complete picture of the condition. In some circumstances, imaging of the cord's blood supply may also be undertaken to locate the dominant feeding artery before surgery in this watershed territory.

What the evidence shows about management — impersonal

It is worth being clear at the outset that the evidence base for thoracic myelopathy is built on retrospective surgical series, systematic reviews of those series, anatomical and biomechanical studies, and expert consensus. There are no large randomised trials comparing one approach with another, and the condition is uncommon enough that decisions are individualised. What follows describes what the literature reports, not guidance for any person.

For established, progressive thoracic myelopathy from compression, the literature offers no effective medical or non-surgical cure; ****surgical decompression**** to relieve pressure on the cord is the mainstay once the condition is progressing. Mild ossification discovered incidentally, without any sign of cord disturbance, may simply be

observed.

For **ossification of the ligamentum flavum**, which compresses the cord from behind, the standard operation is a **posterior decompression**, removing the bony arches and the ossified ligament to give the cord room, sometimes with instrumented fixation. The literature increasingly favours lifting the ossified segment away in a single piece, a so-called floating or en bloc technique often performed with an ultrasonic bone-cutting instrument, in preference to removing it piecemeal. The reasoning is that the repeated passage of instruments beneath an already-compressed cord is itself a source of injury, and that single-piece removal disturbs the cord less, with less bleeding and shorter operating time reported.

For **front-of-canal disease**, namely thoracic OPLL and calcified discs, the pressure comes from in front of the cord, and the literature describes approaching it from the front or the side, through the chest or around the side of the spine, so that the offending tissue can be removed directly without retracting the intolerant cord. For central calcified discs in particular, anterior approaches have been reported to give better neurological improvement than working from behind, at the cost of a more involved operation, because reaching across or past the cord from behind to a hard central lump is hazardous.

Reported **outcomes** for ossification of the ligamentum flavum show that around half of people achieve meaningful neurological recovery, with pooled recovery rates of roughly fifty per cent across systematic reviews, though results vary widely. Better outcomes are associated in the literature with a shorter duration of symptoms, milder pre-operative deficit, fewer levels involved and the absence of established signal change within the cord; poorer outcomes with long-standing severe deficit, dural ossification and tandem disease. **Intraoperative monitoring** of cord function is widely used, and the literature reports that loss of monitoring signals without recovery correlates with lasting deficit. The recognised **risks** of thoracic decompression, set against these benefits, include the leak of spinal fluid, especially where the dura has ossified, and the neurological deterioration that follows from the cord's precarious blood supply and minimal reserve. All of this sits within a framework of individualised, shared decision-making rather than fixed rules.

Outlook

The outlook in thoracic myelopathy is shaped above all by how far the condition has advanced before it is addressed and by the precariousness of the thoracic cord. The recurring theme across the literature is that the state of the cord at the point of treatment is the strongest single influence on what follows.

Where the diagnosis is reached while the deficit is still mild, where the duration has been short and the cord has not yet developed established internal change, the reported prospects for neurological recovery are more favourable. Where the condition has been present for a long time, where the deficit is severe, or where the cord shows established signal change, recovery is less complete, and some loss of function may persist. This is the chief reason the condition is taken seriously and the reason its slow, easily overlooked onset is so often remarked upon.

It should be said plainly that thoracic myelopathy does not behave like a simple mechanical problem with a guaranteed mechanical solution. The combination of a narrow canal, an intolerant cord and a watershed blood supply means that outcomes are more variable and the margins narrower than in the neck or the lower back, and that even successful relief of compression may leave some residual disturbance, particularly when treatment follows a long history. Across the field the consistent messages are that early and accurate recognition offers the best chance of a good result, that the cause and the level of the compression shape both the treatment and the outlook, and that recovery is a gradual process. The variation between individuals is wide, and any individual outlook is a matter for assessment by the treating team rather than something that can be read from a category alone.