

Axial Spondyloarthritis and the Spine

A patient guide

In brief: Axial spondyloarthritis, usually shortened to axSpA, is a chronic inflammatory condition that centres on the spine and the sacroiliac joints, the paired joints where the base of the spine meets the pelvis. It is best understood as an umbrella term covering a spectrum of disease. At one end lies radiographic axial spondyloarthritis, which is the condition long known as ankylosing spondylitis, in which damage to the sacroiliac joints is visible on plain X-rays. At the other lies non-radiographic axial spondyloarthritis, in which the inflammation is present and often visible on MRI, but the characteristic structural damage has not yet appeared on plain films. The two are now regarded as parts of one continuous disease rather than separate illnesses. The hallmark of axSpA is inflammatory back pain that begins in early adult life, eases with movement and worsens with rest, and over many years the inflammation can drive new bone to form across the joints of the spine, gradually stiffening and in some people fusing it. From the perspective of a spinal surgeon, the features of greatest interest are the late mechanical consequences of that fusion: a spine that has knitted into a single rigid column behaves quite differently from a normal one, becoming both prone to a particular pattern of fracture after seemingly minor injury and, in some people, fixed in a stooped forward posture. This page describes axSpA as a whole and its effect on the spine; a separate page deals specifically with ankylosing spondylitis.

What axial spondyloarthritis is, and how it relates to ankylosing spondylitis

Axial spondyloarthritis is a form of inflammatory arthritis that preferentially affects the axial skeleton, meaning the spine and the sacroiliac joints, rather than the peripheral joints of the limbs. It belongs to a wider family of related conditions, the spondyloarthritides, which share certain features such as a tendency to inflammation at the points where ligaments and tendons attach to bone, an association with the tissue type HLA-B27, and a recognised overlap with psoriasis, inflammatory bowel disease and inflammation of the eye.

The word axial points to the location of the trouble. Unlike rheumatoid arthritis, which characteristically attacks the small joints of the hands and feet, axSpA concentrates on the spinal column and on the sacroiliac joints at its base. These sacroiliac joints are very often the first site to be affected, and inflammation there, termed sacroiliitis, is one of the central features of the condition.

The single most useful idea for understanding the terminology is that axial spondyloarthritis is an umbrella. Beneath that umbrella sit two recognised forms. Radiographic axial spondyloarthritis is the established disease in which definite structural damage to the sacroiliac joints can be seen on plain X-rays; this is the entity that has historically been called ankylosing spondylitis, and the older name remains in wide use. Non-radiographic axial spondyloarthritis describes those in whom the clinical picture and, frequently, the appearances on MRI indicate the same disease process, but in whom the plain X-rays do not yet show the definite structural change required to label it ankylosing spondylitis.

The reason this distinction was introduced is largely one of timing. Structural damage visible on a plain radiograph can take years to develop after symptoms begin, and waiting for it once contributed to long delays in recognising the condition. The umbrella term, and the modern classification that sits behind it, allowed the disease to be recognised earlier, at a stage when only inflammation, rather than fixed structural change, is present. Because a dedicated page on this website addresses ankylosing spondylitis in detail, the present page treats the broader axSpA concept and concentrates on the spine itself, its inflammation, its progression towards stiffening, and the surgical considerations that follow from a fused spinal column.

How the classification works, and why it is not the same as a diagnosis

The framework most often referred to in this field is the classification developed by the Assessment of SpondyloArthritis international Society, generally abbreviated to ASAS. These criteria apply to people with back pain lasting three months or more whose symptoms began before the age of forty-five. Within that group, the criteria are satisfied along one of two routes. The first, the imaging route, requires sacroiliitis on imaging, whether definite structural change on X-ray or active inflammation on MRI, together with at least one additional feature of spondyloarthritis. The second, the clinical route, requires the presence of HLA-B27 together with at least two additional features.

It is worth being clear that these are classification criteria rather than diagnostic criteria. They were designed to define reasonably uniform groups of people for research and for cohort studies, not to serve as a checklist by which any individual is diagnosed. A diagnosis in practice rests on the overall clinical assessment, of which the classification features form only a part. This is a distinction frequently emphasised in the literature, and it matters because the criteria can be met by people who do not have the condition and missed in some who do.

The additional features that the criteria draw upon include inflammatory back pain, inflammation of an entheses or a peripheral joint, inflammation of the eye known as uveitis, the skin condition psoriasis, inflammatory bowel disease, a good response of symptoms to anti-inflammatory medication, a family history of related conditions, the presence of HLA-B27, and a raised level of inflammation in the blood. The accumulation of these features, set against the imaging findings and the tissue type, is what the classification weighs.

Inflammatory back pain: the clinical hallmark

The symptom that most often brings axial spondyloarthritis to attention is a particular kind of back pain, described as inflammatory back pain to distinguish it from the far commoner mechanical back pain. Its character is reasonably distinctive. It tends to begin insidiously, in early adult life and usually before the age of forty. It is typically worse with rest and through the night, often waking the person in the second half of the night, and it characteristically eases with movement and activity rather than being aggravated by it. Morning stiffness in the lower back, lasting for a prolonged period after waking, is a common companion.

The international criteria for inflammatory back pain identify five features: an age at onset below forty, an insidious onset, improvement with exercise, no improvement with rest, and pain at night that improves on getting up. When four of these five are present, the pattern is reasonably specific for an inflammatory cause, although by itself it does not establish the diagnosis.

This pattern is almost the mirror image of ordinary mechanical back pain, which tends to be eased by rest and provoked by activity. The contrast is one of the threads that runs through the recognition of the condition, and it explains why the daily rhythm of the pain, rather than its mere presence, is given such weight.

Sacroiliitis and the role of imaging

The sacroiliac joints are very often where the disease declares itself first, and inflammation there is a defining feature. On MRI, the change regarded as most telling is bone marrow oedema, an accumulation of inflammatory fluid within the bone immediately beneath the joint surface, in a typical location. The international working groups that have defined this appearance have been at pains to require that the oedema be sufficiently clear and abundant, precisely so that the minor and incidental signal changes seen in many normal or simply ageing spines are not mistaken for disease.

Alongside active inflammation, MRI can also reveal the structural consequences of the process over time: erosions where bone has been eaten away, sclerosis where it has thickened, fatty change in the marrow, and eventually ankylosis where the joint has bridged with new bone. Plain X-rays, by contrast, show the later structural picture: the erosion, sclerosis and eventual fusion of the sacroiliac joints that define the radiographic form of the disease, and the changes in the spine itself.

The distinction between radiographic and non-radiographic forms turns precisely on these imaging modalities. Where MRI shows active inflammation but the plain X-ray remains free of definite structural damage, the disease falls into the non-radiographic category; where the X-ray shows established structural change at the sacroiliac joints, it falls into the radiographic category that corresponds to ankylosing spondylitis. The imaging is therefore not merely a confirmatory step but the very thing that separates the two ends of the spectrum.

Enthesitis, syndesmophytes and the path towards a fused spine

Understanding why the spine of someone with long-standing axSpA can become rigid requires following the disease through its sequence. The primary lesion is enthesitis, inflammation at the entheses, the points where ligaments, tendons and joint capsules anchor into bone. In the spine, these attachment sites lie at the corners of the vertebrae and around the discs and small joints, and it is here that the inflammatory process takes hold.

The sequence then runs from inflammation, through erosion of bone at the inflamed sites, to a reparative phase in which new bone is laid down. This reparative new bone is the feature that sets axial spondyloarthritis apart from many other inflammatory arthritides, which tend to destroy rather than to build. The new bone forms vertical bridges, called syndesmophytes, that grow upward from the corner of one vertebra towards the next and gradually span the disc space between them. As these bridges form at level after level, adjacent vertebrae become connected by bone, and the natural mobility between them is lost.

When this process is widespread, the spine takes on a characteristic appearance on X-ray that has long been described as a bamboo spine, the flowing vertical bridges and the ossified ligaments giving the column a smooth, segmented, cane-like outline. The endpoint, reached only in some people and usually after many years, is ankylosis, in which substantial portions of the spine have knitted into a single continuous block of bone. New bone formation of this kind tends to favour sites of mechanical stress, and the mediators driving the inflammation and the bone formation are the subject of considerable ongoing research.

It is this fusion, rather than the inflammation in isolation, that gives the condition its surgical relevance. A spine that has become a rigid column behaves mechanically in ways that a normal, segmented and flexible spine does not, and the consequences of that altered mechanics are the subject of the sections that follow.

HLA-B27 and the genetic background

One of the most strongly recognised associations in the whole of medicine is that between axial spondyloarthritis and the tissue type HLA-B27, a genetically determined marker on the surface of cells. The great majority of people with the radiographic form of the disease carry HLA-B27, and the proportion in the non-radiographic form is broadly similar though somewhat lower. HLA-B27 is widely regarded as the single largest genetic contributor to susceptibility, although it is far from the whole story, since most carriers of the marker never develop the condition and additional genes and environmental factors are involved.

The frequency of HLA-B27 in the general population varies considerably between different parts of the world, and the prevalence of axial spondyloarthritis tends to track that variation, being higher in populations where the marker is common. Carriage of HLA-B27 is also associated with an earlier onset of disease and with a greater tendency to inflammation of the eye. Its presence forms one strand of the clinical assessment, particularly within the clinical route of the classification, but like every other single feature it is neither necessary nor sufficient on its own.

Fractures in the fused, ankylosed spine

This is among the most important spinal aspects of axial spondyloarthritis, and it is where the surgeon's attention is most sharply focused. When the spine has fused into a long, rigid column, it ceases to behave like a flexible chain of independent segments and instead behaves much more like a single long bone. A long bone, struck with sufficient force, snaps; and a fused spine, deprived of the cushioning and the give that its many discs and joints once provided, behaves similarly when it is loaded beyond its tolerance.

The consequence is a distinctive and dangerous pattern of injury. The ankylosed spine can fracture after what would, in a normal spine, be a wholly trivial event: a fall from standing height, a stumble, sometimes a mechanism so minor that the injury is at first not suspected at all. Compounding this is the fact that the bone in long-standing disease is frequently osteoporotic, having been weakened both by the chronic inflammation and by the loss of the normal mechanical loading that comes with a mobile spine, so that less force is needed to break it.

The fractures that result are characteristically unstable. Because the rigid column transmits forces across long lever arms, the break commonly extends through all three columns of the spine, front, middle and back, rather than involving only part of it, and such three-column injuries are inherently unstable. They occur most often at the lower neck and at the junction between the neck and the chest. They are, regrettably, frequently missed or recognised late, because the underlying stiffness and the pre-existing deformity disguise the new injury, because the person may report only modest pain, and because plain X-rays of a fused and deformed spine are notoriously difficult to interpret.

The dangers of these fractures are considerable and well documented. They carry a high rate of associated injury to the spinal cord, and they are prone to bleeding around the cord, a spinal epidural haematoma, which can cause neurological deterioration even some time after the original injury. The published series describe rates of spinal cord injury, of secondary neurological worsening and of mortality that are all markedly higher than for comparable fractures in a normal spine. For these reasons the literature emphasises detailed cross-sectional imaging, in the form of CT to define the bony injury and MRI to assess the cord and to look for haematoma, since plain films alone are unreliable in this group.

Surgical considerations for fractures of the ankylosing spine

Because fractures through a fused, rigid spine are typically unstable three-column injuries occurring in a long lever-arm column, the published experience favours surgical stabilisation in most cases. The reasoning is mechanical: a fracture through a long rigid beam is subjected to large bending forces, and the short fixations that might suffice in a normal spine are generally insufficient to control them.

The approach most often described is long-segment posterior instrumented fusion, in which screws and rods span several levels above and below the fracture so as to neutralise the long lever arms on either side. Where the spinal cord is being compressed, whether by displaced bone or by an epidural haematoma, decompression is added. In the neck, a posterior approach is frequently favoured, with combined front-and-back surgery reserved for selected injuries, and there is growing interest in less invasive percutaneous techniques that achieve the same long-segment fixation with reduced blood loss. The literature has reported that surgical stabilisation is associated with improved survival compared with non-operative treatment in this particular group, a reflection of how poorly these unstable injuries are tolerated when left unsupported. These are descriptions of the patterns reported in published series, not recommendations for any individual, whose management is always a matter for the treating team.

Spinal deformity: fixed kyphosis and the chin-on-chest posture

The second major surgical theme is deformity. As the spine fuses over many years, it may fuse not in a neutral, upright posture but in a position of forward flexion, with a loss of the normal inward curve of the lower back, the lumbar lordosis, and an increase in the forward curve of the upper back, the thoracic kyphosis. Because the fusion is rigid, this stooped posture becomes fixed and cannot be corrected by the person's own effort.

In its more advanced forms this produces a markedly forward-bent stance, and in the most severe cases a chin-on-chest deformity in which the head is carried so far forward and downward that the person struggles to look ahead. The consequences extend well beyond appearance. The line of sight is directed at the ground, making walking and many daily tasks difficult; swallowing and breathing can be affected; and the whole body is thrown out of balance, with the centre of gravity carried forward of where it should sit.

In assessing this deformity, the literature describes particular measurements. The chin-brow vertical angle, the angle between a line drawn from the chin to the brow and the true vertical, captures the degree to which horizontal gaze has been lost and is used both to gauge severity and to plan correction. Measurements of the overall forward balance of the trunk and of the relationship between the spine and the pelvis are used alongside it. The aim of any correction described in the literature is to restore a near-horizontal gaze and a balanced posture, and overcorrection is recognised as a pitfall to be avoided.

Surgical considerations for spinal deformity: corrective osteotomy

Where a fixed kyphotic deformity is severe and disabling, the reconstructive procedure described in the literature is corrective osteotomy, in which the surgeon deliberately cuts through the fused bone in order to realign the column, most often in the lumbar spine. Two principal techniques recur in the published accounts.

The first is the Smith-Petersen osteotomy, also called a posterior column osteotomy, first described in the 1940s. Here the posterior elements of a vertebra are removed and the spine is hinged backward, opening at the front through a disc space. Each such osteotomy contributes a modest amount of correction, in the region of ten degrees, so that where a larger correction is needed several are performed at successive levels. The second is the pedicle subtraction osteotomy, a three-column closing-wedge osteotomy in which a wedge of bone is removed from a single vertebra and the gap is closed, hinging the spine backward at that level. A single pedicle subtraction osteotomy achieves a far greater correction at one level, commonly in the region of thirty to forty degrees, and the published series report substantial gains in lumbar lordosis and large improvements in the chin-brow angle and in overall balance.

The comparison between the techniques is a recurring theme in the literature. The pedicle subtraction osteotomy delivers more correction at a single level than the Smith-Petersen approach, but at the cost of greater blood loss and longer surgery, reflecting the more extensive bone removal involved. For the most severe deformities, osteotomy at more than one level is described. These are powerful but demanding operations, and the published accounts document their risks as well as their benefits, which is why the threshold for undertaking them is weighed against the severity of the disability. As with fracture care, these descriptions report what the literature observes; decisions in any individual case rest with the treating team.

How axSpA differs from the dedicated ankylosing spondylitis page

Because this website carries a separate page devoted to ankylosing spondylitis, it is worth being clear about how the two fit together rather than repeating the same ground. Ankylosing spondylitis is, in the modern framework, the radiographic form of axial spondyloarthritis, the established disease in which structural damage to the sacroiliac joints is visible on plain X-rays. Axial spondyloarthritis is the broader umbrella that contains both this radiographic form and the non-radiographic form, in which the same disease process is present and often visible on MRI but has not yet produced definite structural change on plain films.

The present page therefore takes the wider view, treating the inflammatory spectrum as a whole, the way the disease moves from inflammation through new bone formation towards stiffening and fusion, and the mechanical consequences for the spine that follow. The dedicated ankylosing spondylitis page addresses the established radiographic disease in its own right. Holding the two together, the umbrella and the established form beneath it, is the clearest way to understand where any individual sits along the spectrum.

Osteoporosis and vertebral fracture risk

A point that links the inflammatory and the mechanical aspects of the condition is the tendency for the bone itself to become weak. Low bone density and frank osteoporosis are common in axial spondyloarthritis, and they arise from two directions at once: the chronic inflammation itself promotes bone loss, and the gradual loss of spinal mobility removes the mechanical loading that normally keeps bone strong. The result is that the very spine that is stiffening and fusing is often, at the same time, becoming more fragile, which is part of why fractures occur after such minor trauma.

A particular difficulty arises in measuring this bone weakness. The standard bone density scan of the lower back, performed from front to back, can give falsely reassuring readings in advanced disease, because the syndesmophytes and the ossified ligaments lying over the vertebrae add to the apparent density and mask the true weakness of the bone within. For this reason the literature describes assessing bone density at the hip, or by other views and techniques, as more reliable in this setting. The factors associated with greater bone loss and fracture risk that recur in the published work include longer disease duration, higher levels of inflammation, advanced structural change in the spine and reduced spinal movement.

Outlook

The outlook in axial spondyloarthritis spans a very wide range, and much depends on where along the spectrum a given person sits and how the disease behaves over time. Many people have a condition that, while troublesome, never progresses to extensive fusion or fixed deformity, and the modern emphasis on earlier recognition and on suppressing inflammation is directed at limiting progression. The spinal consequences described on this page, the fragile fused column prone to fracture and the fixed kyphotic deformity, represent the more advanced and the more mechanically significant end of the spectrum rather than the universal course.

From the surgeon's vantage point, the consistent themes are clear. A spine that has fused into a rigid column behaves like a long bone and is vulnerable to unstable, easily missed fractures after minor injury, which is why imaging and a high index of suspicion are emphasised so strongly in this group. A spine that has fused in a stooped posture can leave a person fixed in a disabling forward-bent stance, for which corrective osteotomy is the reconstructive option described in the literature. And underlying both is bone that is frequently weaker than it appears. Any individual outlook, however, is a matter for assessment by the treating team, taking account of the form and activity of the disease, the structural changes present, the state of the bone and the person's circumstances as a whole, rather than something that can be read from a category alone.