

Osteomalacia and the Spine

A patient guide

In brief: Osteomalacia is a disorder in which newly formed bone fails to mineralise properly, so that the protein scaffold of the skeleton, the osteoid, is laid down but is not adequately hardened with calcium and phosphate. The result is bone that is soft rather than scarce. In the spine this matters in a particular way, because the vertebrae carry the weight of the body and depend on a hard mineral shell to do so. When that shell is soft, the vertebrae yield under load, the back aches diffusely, the bone is tender, and over time the vertebral bodies can deform into the smooth biconcave shape long known to radiologists as the "codfish" or "fish" vertebra. The commonest cause by far is a lack of vitamin D, but a shortage of phosphate, including the rare tumour-driven and inherited forms, produces the same softening. The single most important idea, and the one that sets osteomalacia apart from osteoporosis, is that this is a problem of bone quality rather than bone quantity: the bone is present but poorly hardened. That distinction carries real weight when spinal surgery and metalwork are being considered, because soft, under-mineralised bone holds screws and cages poorly until the mineralisation is restored.

What osteomalacia is, and why soft bone matters in the spine

Bone is built in two stages. First the cells of the skeleton lay down a soft protein framework made largely of collagen, called osteoid. Then, over the following days and weeks, that framework is mineralised, meaning it is impregnated with crystals of calcium and phosphate that turn the soft scaffold into hard, load-bearing bone. Osteomalacia, whose name means literally "soft bone", is a failure of that second stage. The osteoid is laid down, but it is not properly hardened, so the skeleton accumulates a layer of unmineralised, rubbery bone. Mineralisation depends on having enough calcium and phosphate at the bone surface, and on the machinery that drives the process working correctly; when any of these is lacking, mineralisation lags behind and soft bone is the result.

In the spine this defect has a direct mechanical consequence. The vertebral bodies are the load-bearing pillars of the back, stacked one upon another and bearing the compressive weight of the trunk, the head and anything carried. Each vertebral body relies on a dense outer shell and a hard internal lattice to resist that load. When mineralisation fails, both the shell and the lattice become soft, and the vertebra behaves less like a firm block and more like something that can be slowly compressed. This is why the spine is one of the places where osteomalacia declares itself, through diffuse pain, tenderness and the gradual reshaping of the vertebrae.

It is worth being clear that the bone in osteomalacia is not missing; it is poorly hardened. This is the opposite of the intuitive picture many people hold, in which weak bone means too little bone. In osteomalacia there can be a normal or even increased amount of bone tissue, but a large part of it is soft osteoid that does its job badly. That single fact runs through everything that follows, and it is the reason osteomalacia must be thought about differently from the more familiar osteoporosis.

How osteomalacia shows itself in the spine

The spinal picture in osteomalacia tends to be diffuse rather than focal. The most characteristic feature is widespread, dull, aching bone pain, often felt across the lower back and through the pelvis and hips, which is different in character from the sharp, localised pain of a single trapped nerve. The pain of osteomalacia is the pain of the bone itself being soft and overstressed, and it is frequently accompanied by tenderness, so that pressure over the bones of the spine and pelvis can be uncomfortable. Many people also describe a more general aching that is hard to pin to one spot.

Alongside the bone pain, osteomalacia is associated with muscle weakness, particularly in the muscles around the hips and the trunk. This proximal weakness can make rising from a chair, climbing stairs and standing upright more effortful, and a waddling pattern of walking has long been described. Because the muscles that support and stabilise the spine are involved, the back can feel weak and easily fatigued as well as painful. The combination of soft bone and weak supporting muscle is part of why the spine struggles to hold itself upright comfortably.

Over a longer period, the softened vertebrae begin to deform. Under the steady downward pressure of the body's weight, the upper and lower surfaces of each vertebral body are gradually pushed inwards by the cushioning discs above and below, hollowing out the bone into a smooth, symmetrical, double concave shape. This is the appearance radiologists call the biconcave, "codfish" or "fish" vertebra. Because osteomalacia is a generalised metabolic process rather than a one-off injury, several neighbouring vertebrae tend to take on this same shape, which is one of the features that helps distinguish the metabolic pattern from a single traumatic fracture.

The "codfish" vertebra and the loss of vertebral shape

The biconcave vertebra deserves a description of its own, because it is one of the signatures of metabolic softening of the spine. A healthy vertebral body, seen from the side, has roughly flat or gently curved upper and lower surfaces, the endplates, against which the intervertebral discs sit. When the bone is soft, those endplates lose their rigidity, and the discs, which are themselves firm and pressurised, gradually press into the yielding bone from above and below. The endplates bow inwards, and the whole vertebral body takes on a concave-topped, concave-bottomed shape, narrow in the middle and flared at the edges. The discs, expanding into the space they have created, appear correspondingly tall and biconvex.

This deformity was historically described in osteomalacia and other softening conditions, and the term "codfish" reflects the resemblance to the shape of the vertebrae of bony fish. It is not unique to osteomalacia, since osteoporosis, certain blood disorders, prolonged steroid use and other states can produce a similar look, but in osteomalacia it is often striking and tends to be smooth, symmetrical and present across multiple adjacent levels, in keeping with a process that affects the whole skeleton at once. The deformity usually appears first in the lumbar vertebrae, where loads are greatest.

As several vertebrae are reshaped in this way, the overall sagittal balance of the spine can shift. Loss of vertebral height and a tendency for the front of the vertebrae to compress more than the back can tip the spine into a stooped, forward-bent posture, a kyphosis, and reduce a person's standing height. These changes are gradual and reflect the slow, plastic deformation of softened bone under habitual loads rather than a sudden collapse, which is part of what gives osteomalacic spinal deformity its characteristic smooth, rounded quality.

Insufficiency fractures and Looser zones

Soft bone is not only prone to slow deformation; it is also prone to fracture under loads that would not damage healthy bone. These are called insufficiency fractures, meaning fractures that occur because the bone is insufficient to bear ordinary stresses, rather than because an extraordinary force was applied. In the spine and pelvis, insufficiency fractures can cause a sudden worsening of pain against the background of the chronic ache, and because the underlying bone is soft throughout, such fractures may be multiple and may heal slowly and imperfectly.

The most distinctive radiological sign of osteomalacia is a particular kind of incomplete fracture called a Looser zone, also known as a pseudofracture or, after the radiologist who described them, a Milkman line. A Looser zone is a narrow band running across the bone, usually at right angles to its surface, where the bone has partially given way and the gap has filled with soft, unmineralised tissue rather than healing into solid bone. On an image it appears as a thin lucent line, often with a faint rim of denser bone alongside it. Looser zones characteristically occur at sites of mechanical stress, such as the pubic bones of the pelvis, the necks of the thigh bones, the ribs and the outer borders of the shoulder blades, and they are regarded as close to diagnostic of osteomalacia when present.

These pseudofractures are important in the spinal context for two reasons. First, their presence anywhere in the skeleton points strongly to a generalised mineralisation defect, which has implications for the spine even when the Looser zones themselves are in the pelvis or limbs. Second, the same process of incomplete, poorly healing fracture in soft bone can affect the vertebrae and the bone of the sacrum and pelvis around the spine, contributing to pain and to the slow loss of structural integrity in the region.

How osteomalacia differs from osteoporosis, and why it matters

Osteomalacia and osteoporosis are frequently confused, partly because both produce fragile bone and back pain, and partly because the words sound similar. They are, however, fundamentally different problems, and the distinction is not academic.

The core difference is one of quality versus quantity. Osteoporosis is a disorder of bone quantity: the amount of bone is reduced and its internal architecture is thinned, but the bone that remains is normally mineralised and properly hardened. Osteomalacia is a disorder of bone quality: the amount of bone may be normal, but a large part of it is soft, unmineralised osteoid. In plain terms, in osteoporosis there is too little bone, while in osteomalacia there is enough bone but it is not hard enough. One is a problem of how much; the other is a problem of how good.

This difference shows up in several ways. The blood chemistry usually differs: in osteomalacia from vitamin D or phosphate deficiency, measures such as calcium, phosphate, vitamin D and the bone enzyme alkaline phosphatase are frequently abnormal, whereas in uncomplicated osteoporosis these are typically normal. The pain pattern often differs, with osteomalacia producing more diffuse, generalised bone pain and tenderness alongside muscle weakness, and osteoporosis often being silent until a fracture occurs. The most certain way to separate the two is a bone biopsy examined under the microscope, where osteomalacia reveals its characteristic wide seams of unmineralised osteoid; but in practice the picture is usually pieced together from the clinical story, the blood tests and the imaging. The two can also coexist in the same person, each adding to the fragility of the bone.

The reason this distinction matters so much is that the two conditions behave differently and are corrected differently. Crucially, osteomalacia is, in large part, reversible: when the missing vitamin D or phosphate is restored, the accumulated osteoid mineralises, the bone hardens, and much of the strength returns. That is a very different prospect from the slower business of rebuilding lost bone in osteoporosis, and it is central to how osteomalacia is approached before any spinal surgery.

Why recognising osteomalacia before spinal surgery is so important

This is the point at which the distinction between soft bone and scarce bone becomes most consequential for the spinal surgeon. Much of spinal surgery, particularly the fusion operations used for instability and deformity, relies on anchoring metalwork into the vertebrae. Screws are driven into the bony pillars of the vertebrae, the pedicles, and into the vertebral bodies; cages and spacers rest against the bony endplates; rods connect the screws to hold the spine in its corrected position. Every one of these depends on the bone being hard enough to grip the metal and to bear the concentrated loads at the points where implant meets bone.

Soft, under-mineralised bone holds metalwork poorly. A screw placed into osteomalacic bone has less to bite into and is more liable to loosen or pull out; a cage resting on a soft endplate is more liable to sink into the vertebral body, a process called subsidence; and the whole construct is more vulnerable to the bone giving way around it before the intended fusion has had time to solidify. The published experience with poor bone quality in spinal surgery, much of it drawn from osteoporosis, consistently links low bone quality to higher rates of screw loosening, implant failure, subsidence and failure of the bones to fuse. Osteomalacia adds its own particular problem to this picture: because the defect is one of mineralisation, the bone may be especially poor at holding metal until the mineralisation is corrected.

The encouraging counterpart to this concern is that osteomalacia is correctable, and often substantially so. Where the cause is vitamin D or phosphate deficiency, restoring the missing nutrient allows the accumulated soft osteoid

to mineralise, so that bone which held metal poorly can become much firmer over a period of treatment. This is why recognising osteomalacia before, rather than after, spinal instrumentation carries such weight in the literature on bone health and spine surgery. Correcting the mineralisation defect first means that any subsequent metalwork is anchored in bone that has been allowed to harden, rather than in bone that is still soft. The broad theme running through the bone-health literature is that the quality of the bone is the backdrop against which the success of spinal instrumentation is played out, and that mineralisation defects are best identified and addressed as part of preparing the spine.

The causes behind the soft bone

Although the spinal consequences of osteomalacia look similar whatever the cause, the underlying reasons fall into a few groups, and understanding them helps explain why the condition arises and why it is correctable.

By far the commonest cause is a deficiency of vitamin D, the substance the body uses to absorb calcium and phosphate from food and to drive bone mineralisation. Vitamin D is made in the skin under sunlight and obtained from a few foods, and deficiency can arise from limited sun exposure, darker skin, covering clothing, certain diets, problems absorbing fat and nutrients from the gut, and disorders of the liver or kidneys that prevent vitamin D being activated. When vitamin D is lacking, calcium and phosphate are in short supply at the bone surface, mineralisation falters, and soft bone accumulates.

A second group of causes centres on phosphate rather than vitamin D. Phosphate is one of the two minerals, with calcium, that harden bone, and a sustained shortage of it produces osteomalacia even when vitamin D is adequate. Phosphate can be lost excessively through the kidneys, and one striking cause of this is a rare condition called tumour-induced or oncogenic osteomalacia, in which a small, often hard-to-find tumour secretes a hormone-like substance, fibroblast growth factor 23, that drives phosphate out of the body in the urine. Inherited disorders of phosphate handling, of which X-linked hypophosphataemia is the best known, do the same from early life and can, in adults, additionally cause thickening and ossification of the spinal ligaments that narrows the spinal canal. Certain long-term medications, including some anticonvulsants and the long-term heavy use of aluminium-containing antacids, can also interfere with vitamin D or phosphate and produce osteomalacia.

What these causes share is that they are, for the most part, identifiable and addressable. Restoring vitamin D, replacing phosphate, removing a causative tumour or stopping an offending drug each allows mineralisation to resume. This is the practical reason why pinning down the cause matters: it points the way to correcting the soft bone, which in turn changes the outlook for the spine, including its readiness to hold any metalwork that surgery may require.

How osteomalacia of the spine is assessed

Building a picture of osteomalacia draws together the clinical story, blood tests, imaging and, in uncertain cases, examination of the bone itself, because no single test captures the whole condition.

The clinical pattern of diffuse bone pain, tenderness and proximal muscle weakness raises the possibility, particularly when there are risk factors for vitamin D deficiency or phosphate loss. Blood tests are central, because in the common forms the chemistry is informative: low vitamin D, a low or low-normal calcium and phosphate, and a raised level of the bone enzyme alkaline phosphatase together point towards a mineralisation defect, and measuring phosphate and related markers helps separate the vitamin D from the phosphate-losing forms. These patterns are also what distinguish osteomalacia from osteoporosis, in which such tests are usually normal.

Imaging contributes in two ways. Plain X-rays may show the generalised loss of bone density, the biconcave vertebrae and, most tellingly, Looser zones at their characteristic sites, any of which strengthens the case for osteomalacia. Bone density scanning, the DEXA scan, measures the amount of mineral in the bone and is the standard test for osteoporosis, but it cannot by itself tell soft bone from scarce bone, which is one reason osteomalacia can be missed if it is not specifically considered. Where the distinction remains genuinely uncertain,

a bone biopsy examined under the microscope is the definitive test, revealing the wide seams of unmineralised osteoid that define the condition, though in practice this is seldom required because the clinical and biochemical picture is usually sufficient. In the spinal-surgical setting, the assessment of bone quality, through density scanning and through measures such as the density of the bone on a CT scan, has the added purpose of judging how well the bone is likely to hold metalwork.

Outlook

The outlook in osteomalacia of the spine is, in an important respect, more hopeful than that of many spinal conditions, precisely because the underlying defect is so often correctable. Where the cause is vitamin D or phosphate deficiency, restoring what is missing allows the soft osteoid to mineralise, the bone to harden and much of the lost strength to return, frequently with a marked easing of the diffuse bone pain and muscle weakness over a period of treatment. This capacity for genuine reversal sets osteomalacia apart from conditions in which bone, once lost, is rebuilt only slowly.

There are limits to this recovery. Deformity that has already developed, such as the biconcave reshaping of the vertebrae or an established kyphosis, reflects bone that has already changed shape and does not simply reverse when the bone hardens, even though the bone itself becomes stronger. Insufficiency fractures and pseudofractures tend to heal as mineralisation is restored, but the time course can be slow. Where a phosphate-losing tumour or an inherited disorder is responsible, the outlook is bound up with addressing that specific cause. And where osteomalacia coexists with osteoporosis, both contribute to fragility and both bear on the outcome.

For the spine in particular, the consistent themes across the literature are that osteomalacia is a defect of mineralisation rather than of quantity, that it is frequently reversible once the cause is identified and corrected, that the soft bone of the untreated condition holds metalwork poorly, and that recognising and correcting the mineralisation defect before spinal instrumentation is therefore of real importance. Any individual outlook depends on the cause, the degree of deformity already present, the bone's response to correction and the wider circumstances, and is a matter for assessment by the treating team rather than something that can be read from the diagnosis alone.