

Spine Infection After Surgery

A patient guide

In brief: A spine infection after surgery is a surgical site infection that develops in the tissues of the operated spine — in the skin and fat above the deep covering layer, or in the deeper muscle, bone, disc and any metalwork below it. It covers a wide range, from a minor, superficial wound infection that settles with simple measures, through to a deep infection around an instrumented fusion that threatens both the stability of the construct and the nearby nerves. Because the early picture can be hard to separate from an ordinary non-infected wound problem, and because the consequences of a missed deep infection are serious, postoperative spinal infection is a condition that the literature approaches with a deliberately low threshold of suspicion.

What a postoperative spine infection is (and why it sits apart)

A surgical site infection of the spine is, by definition, an infection introduced into or seeded around the operated tissues at or after the time of surgery. It is conventionally divided by depth. A **superficial** infection lies above the deep fascia, the tough sheet that covers the spinal muscles, and involves the skin and the fatty layer beneath it. A **deep** infection lies below that fascia and involves the muscle, the bone, the disc space and, where present, the implanted metalwork. This distinction by depth runs through the whole subject, because depth largely determines how serious the infection is and how it tends to be approached.

Postoperative discitis, sometimes called postoperative spondylodiscitis, is a particular form of deep infection in which the organism takes hold within a disc space that has been operated upon, typically after a discectomy. From the disc it can spread to the adjacent vertebral bone. It is recognised as a distinct entity precisely because it occurs in a disc that surgery has already disturbed, in contrast to the spontaneous disc and bone infections discussed below.

What sets postoperative spinal infection apart from its neighbours is that it must be carefully distinguished from several look-alikes, while at the same time never being dismissed too readily. It can resemble a simple non-infected wound problem such as a seroma or a haematoma; it can be confused with the spontaneous, blood-borne infections of the spine that arise without any operation; and, when it is low-grade, it can masquerade as one of the purely mechanical reasons that pain sometimes returns after spinal surgery. Each of these distinctions is examined in turn, because together they explain why this condition is described in the literature with such care.

Distinguishing it from a non-infected wound problem (seroma and haematoma)

After spinal surgery, fluid can collect beneath the healing wound. A **seroma** is a collection of clear, straw-coloured tissue fluid; a **haematoma** is a collection of blood. Both can cause the wound to swell, feel tense and sometimes leak, and both can be uncomfortable. In their early stages they can look very much like an infection, and this overlap is one of the central difficulties of the postoperative period.

The difficulty is compounded by imaging. A scan performed soon after surgery shows swelling, fluid and tissue reaction as a matter of course, and these expected changes are not reliably separated from infection by imaging alone. Oedema, a haematoma, a seroma and an early infection can all produce similar appearances. For this reason the literature emphasises that a fluid collection seen on a scan does not by itself establish, or exclude, infection.

A further subtlety is the difference between colonisation and true infection. A wound or fluid collection may grow bacteria on culture and yet not be truly infected; what marks a genuine infection is the presence of pus and of inflammatory white cells, rather than simply a positive culture. Reports of colonised seromas that closely mimic wound infections illustrate how careful this distinction has to be. The practical consequence described in the

literature is that separating a non-infected wound problem from a true infection often rests on the overall clinical picture and on the examination of fluid or tissue, rather than on any single test. None of this is guidance for any individual; it is the explanation for why the early postoperative wound is approached so attentively.

Distinguishing it from spontaneous (haematogenous) spine infection

The spine can also become infected without any operation at all. In **spontaneous, or haematogenous, vertebral osteomyelitis and discitis**, bacteria travel through the bloodstream from a distant source and settle in the spine, seeding the disc, which has no blood supply of its own, and the bordering bone. This is a recognised condition in its own right, with its own established framework for diagnosis and treatment, including the use of magnetic resonance imaging and, where the responsible organism is not already known from blood tests, an image-guided biopsy.

Postoperative infection differs from this in its origin and therefore in its whole context. Here the organism is introduced in relation to the surgery rather than carried in by the blood from elsewhere, the operated tissues and any metalwork form the setting in which it develops, and the imaging is complicated by the changes that surgery itself produces. The two conditions may share a name in everyday language — both are infections of the spine — but they are separate clinical entities, and the literature treats them as such. Recognising which of the two is present matters, because the spontaneous form is investigated and understood through one body of guidance and the postoperative form through another.

Distinguishing it from mechanical causes of recurrent pain

Pain that returns or persists after spinal surgery has many possible explanations that have nothing to do with infection. The bone may fail to fuse, a state known as pseudarthrosis or non-union; the metalwork may loosen or, rarely, break; the segment next to a fusion may become symptomatic; or a disc may herniate again. These mechanical problems are common reasons for recurrent symptoms, and each is investigated and understood in its own right.

The complication is that a low-grade, smouldering infection can imitate these mechanical problems closely. When an infection is caused by a low-virulence organism, especially one that forms a film on the surface of metalwork, it may produce little more than gradually worsening pain, without fever, redness or obvious wound trouble.

Loosening of the implants seen on a scan, which might at first be read as a purely mechanical failure, can in some cases be the only outward sign of an indolent infection sitting on the metal. Because of this overlap, the literature describes a deliberate awareness that recurrent or unexplained pain after spinal surgery may, in a minority of cases, have an infective explanation that the obvious mechanical one would otherwise conceal.

Why a low threshold of suspicion matters

The reason all these distinctions are drawn so carefully is the asymmetry of the stakes. On one side lie the benign mimics — a seroma, a haematoma, a colonised but not truly infected wound, an ordinary mechanical cause of pain — which are common and, in themselves, far less serious. On the other side lies a missed deep infection, which is uncommon but carries real consequences.

A deep infection around an instrumented fusion threatens two things at once. It threatens the **construct**, because infection can interfere with the bone healing that the metalwork is there to support, and can ultimately loosen the fixation before fusion is achieved. And it threatens the **neural elements**, because infection in the spinal canal can form a collection that presses on the spinal cord or nerve roots. It is the combination of these two threats — to stability and to the nerves — that explains why the literature favours a low threshold for considering, investigating and acting upon the possibility of deep infection, even though most early wound concerns turn out to have a more innocent explanation. This is a description of the reasoning found in the published literature, not advice directed at any individual.

How common it is, and how the procedure influences the risk

Surgical site infection is an uncommon but well-recognised complication of spinal surgery, and its frequency depends heavily on the type of operation. Across a broad systematic review and meta-analysis, the pooled rate of infection after spine surgery was in the region of three per cent overall, but this single figure conceals a wide spread.

The presence of **metalwork** raises the rate. The same body of evidence reported infection in around four to four and a half per cent of instrumented operations, compared with roughly one and a half per cent of operations without instrumentation. Simple, clean procedures such as a lumbar discectomy carry among the lowest rates, often well under one per cent. The **approach** also matters: minimally invasive surgery, which works through smaller corridors, has been associated with lower infection rates than equivalent open surgery, on the order of one and a half per cent against close to four per cent in the pooled data. In broad terms, the risk rises with longer and more complex operations, with longer instrumented constructs, with deformity correction and with revision surgery, and falls with simpler, shorter, non-instrumented procedures.

Timing: early infection and delayed, low-grade infection

Postoperative spinal infections are often grouped by how soon after surgery they appear, although the exact time boundaries differ between studies, a recognised inconsistency in the literature.

Early, or acute, infection typically declares itself within the first few weeks, commonly cited as up to around six weeks. It tends to behave in a recognisable way, with features such as fever, redness or warmth around the wound, prolonged or pus-like wound drainage, and increasing pain. In some cases there may be neurological disturbance. These are the infections most likely to announce themselves clearly.

Delayed, or late, infection appears later, sometimes many months or even years after the operation, and behaves very differently. It is almost always deep, and it is characteristically quiet. Rather than fever and an angry wound, it may present only as gradually worsening pain, or as loosening of the metalwork on a scan. Infections caused by the slow-growing organism *Cutibacterium acnes* are a notable example, with the interval between the first symptoms and the diagnosis often measured in months rather than weeks. This indolence is precisely what makes late infection difficult to recognise and is the reason it is approached with particular care.

The organisms involved, and the role of biofilm

The bacteria responsible for postoperative spinal infection are, for the most part, organisms that live on the skin. **Staphylococcus aureus**, including the meticillin-resistant form, is the single most common, followed by the **coagulase-negative staphylococci** such as *Staphylococcus epidermidis*, by *Cutibacterium acnes*, and by various Gram-negative bacteria from the bowel and elsewhere.

The timing tends to track with the type of organism. Early infections are more often caused by the more aggressive organisms, headed by *Staphylococcus aureus*, which provoke an obvious inflammatory response. Delayed infections are more often caused by the quieter, low-virulence organisms, the coagulase-negative staphylococci and *Cutibacterium acnes*, which provoke little reaction and are easily overlooked.

Central to the behaviour of infection on metalwork is the phenomenon of **biofilm**. Bacteria that reach the surface of an implant can attach to it and surround themselves with a protective slime layer, switching from a free-floating, active state to a dormant, shielded one. Within this biofilm they are far less exposed to the body's defences and to antibiotics, and they provoke little inflammation, which is part of why an implant-related infection can be so silent. Once a mature biofilm has formed on metalwork, as in delayed infection, the literature reports that clearing the infection by cleaning the tissues alone, without addressing the implant, is difficult, and that the chance of the infection returning is high if established biofilm on the metal is left in place. This biofilm behaviour is the single idea that does most to explain how postoperative spinal infection is approached.

What raises the risk

The recognised risk factors for surgical site infection after spinal surgery fall into two groups. The first relates to the person. Diabetes, obesity, smoking, long-term steroid or other immune-suppressing treatment, rheumatoid disease, poor nutrition, older age, a previous surgical site infection and a generally higher burden of illness have all been associated with increased risk in the published series. Several of these act through similar mechanisms: diabetes and smoking impair the small blood vessels and starve the healing tissues of oxygen, while obesity creates a thick, poorly supplied layer of fat that heals less readily.

The second group relates to the operation. A longer operating time, often cited as three hours or more, greater blood loss and the need for transfusion, the use of instrumentation, longer fusions, deformity surgery and revision procedures have all been linked to higher rates of infection. It should be noted that the meta-analyses are not entirely unanimous, and some pooled analyses have not confirmed every individual factor, which reflects the variation between studies rather than a settled disagreement. None of this identifies the situation of any particular person.

How it is assessed (blood markers, MRI with contrast, the metal problem, cultures)

The assessment of a possible postoperative spinal infection draws together blood tests, imaging and the sampling of tissue, each of which has recognised strengths and limitations in this setting.

The **inflammatory blood markers**, C-reactive protein and the erythrocyte sedimentation rate, follow a predictable course after uncomplicated surgery. C-reactive protein rises sharply, peaks within a day or two and usually settles within one to two weeks, while the sedimentation rate falls more slowly over several weeks. It is the departure from this expected pattern — a marker that fails to fall, or that rises again after starting to settle — that draws attention. C-reactive protein is regarded as the most useful single marker, both for raising suspicion and for following the response to treatment, where a steady fall is reassuring and a persistently high level is less so.

Magnetic resonance imaging with contrast is the imaging test of choice for deep infection and postoperative discitis, because it shows the disc, the bone, the surrounding tissues and any collection within the spinal canal. Its great difficulty after surgery is twofold. First, the operated level normally shows swelling and enhancement of the disc and adjacent bone as part of healing, so distinguishing expected post-surgical change from infection is genuinely hard. Second, **metalwork distorts the magnetic resonance image**, creating artefact that can obscure the very region of interest; specialised metal-reduction scanning techniques, and computed tomography for the bone and the implants, are used to work around this. Where the picture remains unclear, nuclear medicine scans are described as a further problem-solving tool.

Because imaging often cannot give a definitive answer, the **sampling of deep tissue for culture** holds a central place. Tissue taken at operation is regarded as the most reliable way to identify the responsible organism, supported where appropriate by blood cultures. Several deep samples are usually taken, and cultures may be held for a prolonged period to detect the slow-growing *Cutibacterium acnes*. Examination of the tissue under the microscope, looking for inflammatory cells, helps to separate true infection from mere colonisation. Aspiration of fluid through the skin is sometimes used but is anatomically difficult in the spine and less well established.

What the evidence shows about management — impersonal

It is important to state at the outset that the evidence on managing postoperative spinal infection rests largely on retrospective series, cohort studies and reviews, with few randomised trials. The principles that follow are therefore consensus-based and series-driven, and decisions in practice are individualised. What follows is a description of what the literature reports, not guidance for any person.

Superficial infections, confined to the layers above the deep fascia, are often described as manageable with local wound care and a course of antibiotics, sometimes with a minor procedure to clean the wound, and they

frequently settle without the need for major surgery.

Deep infections are generally addressed by surgical **debridement and irrigation** — a thorough cleaning of the infected tissues with copious washout, and the taking of deep samples — sometimes repeated, and sometimes combined with negative-pressure dressings. Around this sits the central question of what to do with the metalwork, and here the literature draws a clear general distinction based on timing.

In **early or acute deep infection**, the prevailing principle reported in the literature is **implant retention**. The reasoning is structural: the metalwork is still needed to hold the spine while the bone fuses, and removing it early would sacrifice that stability. The described approach is therefore debridement and irrigation with the implants left in place, combined with a prolonged course of antibiotics. Several large series report that retaining the implants in this way is effective in the majority of acute cases presenting within around three months, provided the metalwork is stable.

In **late or chronic infection**, by contrast, removal of the metalwork is more often required, particularly once the spine has fused and the implants are no longer mechanically essential. The reason is biofilm: a mature film on the metal cannot reliably be cleared while the metal remains, and the literature reports a high rate of recurrence — up to around half of cases in some reports — when established late infection is treated with the implants retained. Removal, exchange or staged revision of the metalwork is described in this setting, and the literature notes that removing implants once fusion is solid is generally safe and can improve healing and reduce pain.

Antibiotics are described as prolonged, commonly six weeks and often twelve weeks or longer where implants are retained, and ideally chosen to be active against organisms growing within biofilm; combinations including rifampicin for staphylococcal infection on retained metal are reported to improve outcomes. Where infection cannot be fully cleared and the metalwork cannot be removed, long-term **suppressive** antibiotics may be used to keep the construct working until the bone has fused. Reported predictors of treatment failure include delayed presentation, resistant or multiple organisms, loose implants and a high burden of other illness. The overall message of the literature is one of individualised, staged decision-making rather than fixed rules.

Outlook

The outlook after a postoperative spinal infection varies as widely as the infections themselves. Superficial infections, recognised and treated, generally resolve well and without lasting consequence. Deep infections are more demanding, but the literature is broadly encouraging about acute deep infection treated promptly with debridement, implant retention and prolonged antibiotics, with the majority of such cases controlled and the construct preserved while the bone fuses.

The more difficult situations are the delayed, low-grade infections, where the diagnosis may have been slow to emerge and where established biofilm on the metalwork often dictates a more involved course, sometimes including removal of the implants. Even here, the literature describes good rates of eventual control, particularly once fusion has been achieved and the metalwork is no longer essential. Across the whole field, the consistent themes are that early and accurate recognition improves the chances of a good result, that the timing of the infection and the behaviour of biofilm shape the approach, and that recovery is best understood as a gradual and individualised process. Any particular outlook is a matter for assessment by the treating team rather than something that can be read from a category alone.