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Tuberculous surgical site infections: Diagnosis, management, and prevention in the modern era

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Abstract

Tuberculosis surgical site infections (TB SSIs) represent a rare but significant complication in diverse surgical contexts, accounting for less than 1% of all TB cases. They present unique diagnostic and management challenges due to their insidious clinical course, which differs markedly from typical bacterial infections. TB SSIs arise either through direct inoculation of mycobacteria into surgical wounds or reactivation of latent TB at sites of surgical trauma. Risk factors include immunosuppression, malnutrition, and the presence of surgical implants. Clinical presentation is characterized by chronic, nonhealing wounds with minimal acute inflammation, often weeks to months after surgery. Diagnostic approaches include microbiological testing (acid-fast bacilli smear, mycobacterial culture, molecular tests), histopathological examination revealing granulomas, and supportive imaging. Intraoperative findings typically include granulomatous tissue, caseous necrosis, and well-formed sinus tracts. Management requires extended antitubercular therapy (9–12 months) combined with adequate surgical debridement and often removal of infected implants. With appropriate treatment, outcomes are generally favorable, with most patients achieving complete wound healing and infection resolution. Prevention strategies include preoperative TB screening in high-risk patients, rigorous sterilization protocols, careful allograft screening, and surveillance for unusual SSI patterns. Recent outbreaks linked to contaminated bone grafts highlight the importance of vigilance and robust tissue banking protocols. Early recognition and multidisciplinary management are essential for optimizing outcomes in these challenging infections.

Keywords: Antitubercular therapy; Granulomatous inflammation; Surgical site infection; Tuberculosis; Wound debridement

Abbreviations: AFB, acid-fast bacilli; ATT, antitubercular therapy; IGRA, interferon-gamma release assay; LTBI, latent TB infection; MDR-TB, multidrug-resistant tuberculosis; NAAT, nucleic acid amplification test; NPWT, negative pressure wound therapy; PCR, polymerase chain reaction; SSI, surgical site infection; TB, tuberculosis

1. Introduction

According to the World Health Organization Global Tuberculosis Report 2024, 16% of 8.2 million newly diagnosed and notified TB cases worldwide in 2023 were extrapulmonary.[1] Cutaneous and wound TB infections are rare, accounting for under 1% of all TB cases,[2] but they pose a significant diagnostic and management challenge when they occur in surgical sites. Typical surgical site infections (SSIs) are caused by pyogenic bacteria (eg, *Staphylococcus aureus*), whereas *Mycobacterium tuberculosis* is an uncommon culprit.[3] Nevertheless, TB SSIs have been increasingly recognized in diverse surgical contexts, from orthopedic implant infections to port-site infections after laparoscopy.[4,5] Recent years have seen notable developments—outbreaks of TB SSIs due to contaminated bone grafts have been reported, prompting improved tissue donor screening.[6] There have also been advances in diagnostic methods and case reports describing successful management strategies for TB SSIs.

This review provides an analysis of TB SSIs targeted to surgeons. We discuss the pathophysiology, clinical presentation, diagnosis, intraoperative findings, and current treatment strategies. The key features of pathophysiology, presentation, and management of TB SSIs are summarized in Table 1. We also summarize outcomes and recommend preventive measures to mitigate TB SSIs. The information is drawn from research in recent years to reflect the most up-to-date knowledge in this evolving field.

2. Pathophysiology and risk factors

Tuberculous SSIs arise via two principal mechanisms: (1) direct inoculation of TB bacilli into the surgical wound or (2) reactivation of latent TB infection (LTBI) in surgical tissues. In the first scenario, an exogenous source introduces *M. tuberculosis* to the wound. This has been documented in cases of contaminated surgical instruments or materials—for example, lapses in instrument sterilization have caused laparoscopic port-site TB infections,[4] and contaminated allograft bone material led to dozens of spinal TB SSI cases in recent outbreaks.[7] In the second scenario, the patient harbors dormant TB that localizes and reactivates at the surgical site due to local tissue injury or altered immunity.[5] Surgical trauma and the presence of foreign bodies create an environment of altered local immunity that may allow latent bacilli to proliferate. "Implant tuberculosis" has been described, defined as TB developing after surgical implantation of tissue or devices, without other active TB focus.[8]

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Table 1.

Key Features of Tuberculous SSIs: Pathophysiology, Presentation, and Management

Category	Key Features	References
Pathophysiology	• Direct inoculation from contaminated instruments/grafts • Reactivation of latent TB at surgical site • Granulomatous inflammation with caseation • "Cold abscess" formation unlike acute pyogenic infections	[4,5,7,21]
Risk factors	• Immunosuppression (HIV, diabetes, steroids) • Implants/foreign bodies • Malnutrition/hypoalbuminemia • Endemic TB regions • Prolonged surgery and large wounds	[12,15,16]
Clinical presentation	• Delayed onset (weeks to months postsurgery) • Chronic drainage or nonhealing wound • Sinus tract formation • Minimal acute inflammation • Variable constitutional symptoms • Failure to respond to standard antibiotics	[3,5,8,16,22,26]
Diagnostic tests	• Mycobacterial culture (gold standard) • Molecular tests (NAAT/PCR) • Histopathology showing granulomas • Imaging for extent assessment • AFB smear (low sensitivity)	[4,8,26,34]
Treatment	• Extended antitubercular therapy (9–12 months) • Thorough surgical debridement • Foreign body removal when feasible • Wound management with NPWT or flap reconstruction • Nutritional support	[3,7,16,21,51]
Prevention	• Preoperative TB screening in high-risk patients • Rigorous instrument sterilization • Tissue/allograft donor screening • Surveillance for unusual SSI patterns • Treatment of latent TB in select cases	[6,53,54]

NAAT, nucleic acid amplification test; NPWT, negative pressure wound therapy; PCR, polymerase chain reaction; SSI, surgical site infection; TB, tuberculosis.

Several risk factors predispose to TB SSIs. Immunosuppression is important—patients with HIV, diabetes, or on chronic corticosteroids have impaired TB containment.[9–11] Rare cases of extensive cutaneous TB infection have occurred in lupus patients on steroids.[12] Malnutrition and low serum albumin also increase SSI risk[13,14]; a recent study of spinal tuberculosis procedures identified low albumin to be a significant risk factor for postoperative wound infection.[15] Prolonged surgeries and large wounds may further raise risk by increasing tissue devitalization. The presence of implants or grafts provides a surface for TB to adhere to. In prosthetic joint TB infections, the prosthesis often must be removed for cure.[16] High TB endemicity plays a role as well: patients in regions with high TB prevalence are more likely to harbor latent infection or be exposed to TB during surgical care.

Mechanistically, once *M. tuberculosis* is present in a wound, it elicits a chronic granulomatous inflammation rather than acute pus formation. The bacteria typically trigger macrophage activation and granuloma formation in the wound tissues.[17,18] Over weeks, this can lead to caseous necrosis in the affected tissue. Unlike pyogenic infections, a tuberculous SSI may form a so-called "cold abscess"—a localized collection with granulomas and necrotic debris but without the intense heat, pain, and erythema of typical abscesses.[19,20] This pathologic process explains the often indolent, insidious course of TB SSIs. The infection tends to smolder, causing delayed healing, sinus tract formation, and chronic discharge, rather than the fulminant sepsis of an acute SSI.[5,21] TB bacilli can also spread along tissue planes, creating multiple sinus tracts or involving deeper structures adjacent to the surgical site.

3. Clinical presentation

Tuberculous SSIs typically present as chronic, nonhealing wound infections rather than the acute fever and purulence seen in ordinary postoperative infections. The time from surgery to symptom onset is often longer. Patients may present weeks to months after surgery with persistent wound drainage, a sinus tract, or poor incision healing.[16] In one series of tuberculous prosthetic joint infections, the median onset was ~10 months postimplantation (range: 1–132 months)[16]—highlighting that TB SSIs can have a substantially delayed presentation. Another report described port-site TB appearing approximately 3–4 weeks after laparoscopic cholecystectomy,[22] which is later than typical bacterial port infections. Thus, an SSI that arises late or fails to resolve with standard therapy should raise suspicion for TB.[5]

Local signs of a TB SSI are often subtle. Patients may note a persistent serous or bloody discharge from the incision, or a slowly enlarging sinus tract that does not respond to conventional antibiotics.[5,8] The wound edges might be indurated and bluish or violaceous, and granulation tissue may be visible in the tract. Pain is variable—some patients have mild discomfort, while others report chronic pain at the site. Classic inflammatory signs (redness, warmth, and acute tenderness) are usually blunted; these infections are often described as "cold" abscesses or sinuses.[5] For example, in a case of TB infection in a cesarean section wound, the patient had a painless sinus that intermittently discharged material but lacked high fever or acute swelling.[8] In sternal wound TB after cardiac surgery, patients may present with wound dehiscence and a draining sinus on the chest wall, sometimes long after the initial sternotomy has healed.[3]

Constitutional symptoms (fever, night sweats, and weight loss) are not prominent in many TB SSIs, but they can occur, especially if the infection becomes disseminated.[23–25] In a U.S. outbreak of spinal TB SSIs from contaminated bone grafts, 67% of patients eventually developed systemic symptoms such as fever and malaise.[26] However, 11% had no systemic signs and only localized wound issues.[26] Lymph node involvement may be present if TB spreads regionally.[27,28] In abdominal surgical wounds, underlying peritoneal TB can lead to persistent fistulae and poor healing of the laparotomy incision.[21]

The diverse presentations of TB SSIs are illustrated through several cases. A 38-year-old left ventricular assist device recipient developed a nonhealing subxiphoid wound and chronic sternal abscess 5 months postsurgery. Despite antibiotics and negative routine cultures, the wound persisted until TB was identified through molecular testing.[29] Similarly, a 34-year-old woman experienced nonhealing of her cesarean incision for weeks, eventually diagnosed with tuberculous infection that recurred during therapy, necessitating additional local treatment.[8] Another cluster of patients developed characteristic postlaparoscopy port-site ulcers—painless with granulomatous tissue on biopsy—later confirmed as TB infections. These cases share a common pattern: chronically draining or indurated surgical wounds with minimal acute inflammation that fail to respond to conventional wound care approaches.[5]

4. Diagnostic approaches

Diagnosing a tuberculous SSI requires a combination of clinical suspicion and targeted laboratory investigations. Definitive diagnosis rests on identifying *M. tuberculosis* in the wound tissue or exudate—either by culture or molecular methods—or characteristic histopathology. Given the often equivocal presentation, a multidisciplinary diagnostic approach is recommended, integrating surgical evaluation, microbiology, and pathology.[4,5]

4.1. Microbiological testing

- **Acid-fast bacilli (AFB) smear**: A simple AFB stain on wound fluid or tissue can sometimes reveal red mycobacterial rods. However, yield is low in TB SSIs due to the typically low bacillary load. Many cases report negative AFB smears[30]; for example, in one cesarean SSI case, the discharge smear was negative despite confirmed TB by other means.[8] Thus, a negative smear does not exclude TB.
- **Mycobacterial culture**: This remains the gold standard. Tissue or fluid from the wound should be sent for TB culture. Culture has high specificity and can detect as few as 10 viable organisms, but it is slow, often requiring 4–8 weeks for growth.[26] In one series of prosthetic joint TB infections, one patient's diagnosis was missed until autopsy because cultures turned positive only after the patient had died.[16] Nonetheless, culture is crucial not only to confirm TB but also to allow drug susceptibility testing.
- **Nucleic acid amplification tests (NAAT)**: Rapid molecular tests have become a cornerstone for early TB detection in wounds. The GeneXpert MTB/RIF assay can detect *M. tuberculosis* DNA and rifampin resistance within hours.[31–33] In TB SSIs, NAAT on tissue or pus can provide a quick answer when culture is pending. For example, in a nonhealing chest wound case, Cartridge-Based polymerase chain reaction (PCR) identified TB bacilli from wound exudate even when smears were negative, leading to timely treatment.[8] NAAT sensitivity in tissue is moderate (~80%–90% in some studies for extrapulmonary TB[34]), so false negatives can occur, especially if bacillary load is very low.
- **Other microbiology**: Routine aerobic and anaerobic cultures of the wound are usually negative or grow only secondary colonizers. In some TB SSI cases, a secondary bacterial infection co-exists, which can muddy the picture.[16] Mixed infections have been reported—one obstetric wound had *Kodamaea* yeast along with *M. tuberculosis*.[5]

4.2. Histopathological examination

Obtaining a surgical biopsy of the wound tissue is highly diagnostic. Caseating granulomas on histology are the hallmark of tuberculous infection.[35–37] Classic findings include granulomas with central necrosis and Langhans giant cells, with or without visible AFB. In many reported cases, biopsy provided the first evidence of TB, often before cultures resulted.[4] For example, in a port-site TB infection, histology showed multiple epithelioid granulomas with Langerhans giant cells.[4] Sometimes granulomas may be noncaseating or nonspecific. Pathology can also help rule out malignancies or other granulomatous diseases that could mimic TB. If available, tissue PCR or in-situ tests on the biopsy can be performed.[38] Ultimately, combining biopsy with culture yields the highest diagnostic sensitivity. In TB SSIs, multiple case reports underscore that an excisional biopsy was pivotal for diagnosis.[4,5]

4.3. Immunological tests

LTBI can be evaluated with procedures such as the tuberculin skin test or interferon-gamma release assays (IGRA).[39–41] While a positive IGRA in a patient with a chronic wound might support the diagnosis, it is only an adjunct. Many patients with TB SSIs do have latent infection and, thus, test positive. In a case of cutaneous tuberculosis, the patient's T-SPOT test was markedly positive.[12] A negative IGRA may not rule out tuberculosis, particularly in immunocompromised persons.[42,43]

4.4. Imaging studies

While imaging is not diagnostic of TB, it aids in assessing the extent of infection. Plain X-rays or ultrasound of a chronic sinus can show underlying osteomyelitis or abscess cavities. In sternal TB SSI, a chest CT may reveal bony erosion of the sternum or cold abscesses in the mediastinum.[3] Magnetic resonance imaging is excellent for delineating sinus tracts and any musculoskeletal involvement. In spinal surgery TB infections, magnetic resonance imaging can help distinguish superficial vs deep infection and detect any concomitant spinal TB focus.

4.5. Diagnostic algorithm

In practice, when a TB SSI is suspected, the surgeon should obtain deep wound samples for AFB smear, culture, and TB PCR, and send a biopsy for histopathology.[4] Empiric broad-spectrum antibiotics should be avoided or minimized, as they can delay TB diagnosis by partially treating secondary infections and giving false reassurance. If suspicion is high, some experts advocate starting empirical antitubercular therapy (ATT) while awaiting confirmation[5]—especially if the patient's condition is deteriorating. Empiric therapy decision must weigh the likelihood of TB against differential diagnoses like atypical mycobacteria (nontuberculous mycobacteria).[44,45] Notably, atypical mycobacteria can also infect surgical wounds, usually earlier post-op and associated with water sources. Distinguishing them requires culture.[46] In general, a comprehensive workup that includes microbiology and pathology will yield the diagnosis in the majority of TB SSIs.

5. Intraoperative findings

When operating on a suspected or confirmed tuberculous SSI, surgeons encounter tissue characteristics that differ from typical pyogenic infections. Intraoperative findings often include pale granulation tissue, areas of caseous necrosis, and well-formed sinus tracts.[47,48] Upon incising the chronic wound or sinus, instead of frank pus, one may find thick, cheesy material (caseation) mixed with granulation tissue.[12] For example, a case report of cutaneous TB infection requiring debridement noted "yellow, fish-flesh-like necrotic tissue" beneath the ulcer surface, with copious caseous debris that had to be curetted out.[12] After thorough debridement, the wound bed typically appears red and bleeds, indicating viable tissue.

5.1. Granulomas and "cold abscesses"

In tuberculous soft-tissue abscesses, a classic finding is a so-called cold abscess. Intraoperatively, this may appear as a pocket of thick, yellow-white necrotic material with a fibrous rim, but without the high-pressure purulence seen in acute abscesses. The surrounding tissues are indurated and may have multiple interconnected cavities. Surgeons have described finding "rice grain" granulomas on the peritoneum or wound surfaces in abdominal TB cases. In port-site infections, small subcutaneous abscesses contiguous with the port tract have been noted, often with a sinus to the skin.

5.2. Sinus tracts

Intraoperatively, these sinus tracts can be extensive. In sternal TB infections after cardiac surgery, it is not uncommon to find multiple sinuses burrowing through the mediastinal fat or along suture lines. One case report described three fistulous tracts communicating with subcutaneous abscesses in a poststernotomy TB infection, which were all excised en bloc with the involved scar tissue.[3] The sinus walls typically are lined with granulation tissue. These tracts often extend to any retained foreign material (eg, nonabsorbed suture or mesh)—TB can preferentially involve such material.

5.3. Bone involvement

If the underlying bone is present near the surgical site, TB can cause osteomyelitis. In sternotomy infections, the sternal bone may be eroded and soft. In spinal instrumented surgery infections, vertebral bone is sometimes involved (though in the 2021 contaminated graft outbreak, significant bony involvement was not observed in most patients[7]). When TB involves bone, the term "caries" applies—the bone is crumbly with cavitations. If an implant is present, it may be loose due to bone destruction or chronic inflammation.

5.4. Vascularization and bleeding

Chronically infected TB tissue tends to be very vascular (except in the necrotic centers). Surgeons often note brisk bleeding from granulomatous tissue upon debridement. This is generally a good sign of viable tissue once debrided. Careful hemostasis is needed, but major bleeding is unusual unless a vessel is eroded.

5.5. Comparison with pyogenic infection

Unlike an acute staph infection where the surgeon might find intense pus and inflamed tissue that dissects easily, TB lesions cause a firmer, matted tissue plane. The tissues may feel more fibrotic and adherent due to the chronic inflammation. Lymph nodes near the wound might be enlarged, firm, and matted together.

5.6. Foreign materials

If synthetic material (mesh, prosthesis) is involved, TB can produce a biofilm-like colonization, though it is more sparse than bacterial biofilm.[49,50] Mesh infected with TB might be encased in granulomas. In one case of TB involving a hernia mesh, the mesh had to be completely removed as it was enveloped in infected tissue.[46] Implants like joint prostheses, if not removed initially, often show evidence of chronic inflammation with granulomas at reoperation.

5.7. Diagnostic frozen section

Intraoperative frozen pathology (if available) can be very helpful. A quick frozen section of debrided tissue may show granulomas, alerting the surgical team to likely TB so they can obtain appropriate cultures if not already done.

6. Treatment strategies

Management of TB SSIs requires a combination of adequate surgical intervention and prolonged antitubercular chemotherapy. The goals are to eradicate *M. tuberculosis* from the site, remove any necrotic tissue or infected foreign material, and allow the wound to heal. Treatment must be coordinated with infectious disease specialists for optimal outcomes.

6.1. Antitubercular therapy

A standard regimen for drug-susceptible TB is implemented, which typically comprises a four-drug induction phase (2 months of isoniazid, rifampicin, pyrazinamide, and ethambutol) and a continuation phase (typically isoniazid and rifampicin) for a minimum of 6 months.[4] However, for TB SSIs, especially with deep or extensive involvement, many experts recommend extended durations (9–12 months) of therapy.[7,11] There are several reasons: tuberculous wounds often have areas of poor drug penetration (caseous material, possibly biofilm on implants), and they behave similarly to osteoarticular TB, which historically is treated for longer durations. In a series of six patients with spinal TB SSI from allografts, all were treated with a full 12 months of therapy (2HRZE + 10HR) and achieved complete wound healing.[7] Likewise, a multicenter review of 15 TB prosthetic joint infections reported a median of 12 months of ATT, after which all surviving patients recovered without recurrence.[16] Shorter courses (6 months) might suffice for very superficial cutaneous TB infections once fully debrided, but evidence suggests that 6-month regimens have a higher relapse risk in skeletal or deep soft-tissue TB.[16] Therefore, the conservative approach is to treat TB SSIs for at least 9 months, and 12 months if there is any doubt or extensive disease.

Regimens must be adjusted if drug resistance is present. If the isolate is rifampin-resistant or multidrug-resistant (MDR-TB), specialized regimens with second-line drugs (fluoroquinolones, injectables, newer oral agents such as bedaquiline) are needed, often for 18+ months.[51] Rapid drug susceptibility testing from culture or molecular tests should guide drug choices early.

6.2. Surgical management

Surgical intervention complements ATT in TB SSIs through thorough debridement of necrotic tissue, abscess drainage, and sinus tract excision. A single comprehensive debridement with effective ATT often achieves healing, as demonstrated in cardiac surgery cases where sternal wound debridement with fistulae excision led to successful flap coverage.[3] Similarly, four of six spine TB SSI patients required surgical debridement alongside TB medications, with all wounds healing completely.[7]

Infected implants should typically be removed unless structurally essential. Some prosthetic joint TB cases were managed with one or two-stage replacements during ATT.[16] Interestingly, during the bone graft outbreak, most spinal hardware was retained while infection resolved through medications and debridement.[7] Postdebridement wound management includes open packing, negative pressure wound therapy (NPWT), or closure. NPWT proves particularly effective by enhancing vascularity while ATT works—evidenced by an abdominal TB wound case achieving >95% closure after NPWT and skin grafting.[21] Complex cases, especially chest wall or neck TB sinuses, may require flap closure with well-vascularized tissue, as seen in a sternal TB infection successfully treated with pectoralis muscle flap.[3]

6.3. Monitoring and adjustment

Patients on treatment need regular monitoring for both wound healing and drug-related issues. Wound examinations should show gradual improvement—decreased discharge, filling of cavities with healthy granulation, and contracting of wound size. If there is no improvement after 2–3 months of appropriate therapy, one must reassess for potential issues: drug resistance, nonadherence to medications, or an alternative diagnosis. At times, repeat debridement may be necessary if residual necrotic material remains. In the cesarean wound TB case, the patient's ulcer initially healed with ATT but then recurred at month 4, prompting another debridement and instillation of local isoniazid powder, after which it finally closed by 6 months.[8]

6.4. Adjunct therapies

There is some experience with topical or local TB therapy. In the above case, topical isoniazid and streptomycin were applied to the wound bed in addition to systemic drugs, seemingly helping cure the persistent ulcer.[8] Such local therapy has historical precedent in skeletal TB and can be considered in refractory cases, though robust evidence is lacking. Another adjunct is the use of corticosteroids in TB wounds—generally not indicated unless there is severe concomitant inflammation causing issues. In most TB SSIs, steroids are avoided due to concern of impairing healing further. Nutritional support is an often under-appreciated adjunct: correcting anemia, hypoalbuminemia, and vitamin D deficiency can help the immune system fight TB and assist wound repair.[15]

6.5. Outcomes with treatment

The prognosis for TB SSIs is generally favorable if appropriate therapy is given. Case series report high cure rates: in the six-patient spinal graft TB series, all patients were infection-free and had healed wounds at 1-year follow-up (excluding one who died of unrelated illness).[7] None of those patients developed TB recurrence or pulmonary TB during that period.[7] In the prosthetic joint TB review, all treated patients (with the exception of one who did not receive therapy in time) recovered without ongoing infection.[16] These outcomes underscore that TB SSIs, while protracted, respond well to the combination of debridement and ATT.

On the other hand, delays in diagnosis or treatment can lead to complications: persistent sinus tracts, extension of infection to deeper structures, or systemic spread. Mortality directly from TB SSI is rare but documented in severe cases—for example, an elderly cardiac surgery patient with unrecognized sternal TB wound infection succumbed, the diagnosis only made postmortem.[3] In the recent U.S. bone graft outbreak, two patients died with TB disease before the outbreak was recognized.[6] Such tragedies highlight the need for vigilance and timely therapy.

6.6. Special scenarios

If TB SSI occurs in an MDR TB strain, management becomes much more challenging. There are case reports of extensively drug-resistant TB causing wound infections (for instance, TB of a breast abscess in an MDR-TB patient[52]). In those, surgery to remove infected tissue plus an individualized MDR-TB regimen for 18–24 months is necessary. Another scenario is coinfection with atypical mycobacteria or fungi. Each pathogen must be treated—as in the case of mixed *Kodamaea* fungal and TB infection, where antifungals were given alongside ATT.[5]

To summarize treatment, all patients with TB SSIs should receive full-course TB chemotherapy, and most will benefit from at least one surgical debridement to remove infected tissue. Implants should be removed if feasible. With this combined approach, the majority of patients achieve resolution of infection and wound healing.[7,11]

7. Outcomes and prognosis

With proper treatment, tuberculous SSIs generally have favorable outcomes, though results depend on diagnosis timing and patient condition.

7.1. Healing and function

Successful treatment closes chronic sinuses and restores tissue integrity. Superficial infections typically heal within 2–3 months, while deeper ones may take 6+ months.[8] Cesarean TB wounds can achieve complete healing after 6 months of ATT,[8] and sternal wounds typically close and stabilize by 1-year follow-up.[7] Though scarring can be significant, normal healing resumes once TB is cured. Orthopedic TB SSI patients showed no reactivation after 12-month therapy.[7]

7.2. Recurrence

Posttreatment recurrence is rare in immunocompetent patients with drug-susceptible TB. Spinal TB SSI patients showed no recurrence during 12-month follow-up.[7] In one case noted earlier, incomplete initial healing led to a relapse of the ulcer which was then cured after extending therapy and adding local treatment.[8] In MDR-TB, relapse rates are higher and close monitoring is needed for several years.

7.3. Morbidity

TB SSIs cause morbidity through extended wound care and additional surgeries. Patients endure months of dressing changes and the psychological burden of chronic wounds. An abdominal TB wound required 46 days from NPWT application to graft closure[21]—significantly longer than typical SSIs. Spine or joint infections may cause residual stiffness.

7.4. Mortality

Direct death from tuberculous SSIs is rare with modern chemotherapy. Fatalities typically result from late diagnosis or severe comorbidities, as with the 74-year-old cardiac patient whose TB sternal infection was diagnosed posthumously.[3] In a 2023 bone graft TB outbreak, two early patients died before diagnosis, but no deaths occurred after treatment began.[6] A review of 155 prosthetic joint TB cases found nearly all infections curable.[16]

7.5. Quality of life

Curing TB SSIs significantly improves patient well-being, transitioning from daily wound care to normal activities. Unlike

pulmonary TB, properly treated TB SSI patients rarely require prolonged isolation as the covered wound poses minimal transmission risk.[8]

Finally, an important outcome is that successfully treated TB SSIs rarely require prolonged isolation or public health measures once therapy is started. Unlike pulmonary TB, a TB SSI patient is typically not infectious via airborne route. There is minimal risk of transmission from an appropriately covered wound.

8. Prevention of tuberculosis surgical site infections

Preventing tuberculosis involvement in surgical sites relies on both broader TB control measures and specific surgical practices. While TB SSIs are uncommon, the consequences and outbreak potential (as seen with contaminated grafts) make prevention efforts important, especially in regions with higher TB prevalence or in surgeries involving transplanted material.

8.1. Preoperative screening and management

Identifying patients with active or latent TB before surgery can allow interventions to reduce SSI risk. All patients must have a comprehensive medical history assessment; individuals with a history of tuberculosis or exposure to it may require a chest X-ray or IGRA test during the preoperative evaluation. If a patient is found to have active TB infection elsewhere, elective surgery should be postponed until TB treatment has rendered them noninfectious and in better health. In urgent cases, appropriate airborne isolation and perioperative ATT should be instituted. LTBI, if detected, could be treated with isoniazid or rifamycin prophylaxis in certain high-risk surgical patients.[53,54] There are no formal guidelines to universally treat LTBI before surgery, but a case-by-case approach is sensible to lower the risk of postoperative reactivation.

8.2. Infection control and sterilization

Strict adherence to standard infection control practices will also prevent unusual pathogens such as TB from entering surgical wounds. One documented cause of port-site TB infections was inadequate sterilization of laparoscopic instruments with 2% glutaraldehyde. Modern autoclaving at appropriate temperature/time will kill *M. tuberculosis*. If chemical disinfectants are used, they must be of sufficient concentration and exposure time. Single-use instruments or disposable trocars could be considered in settings where TB SSI outbreaks have occurred. Additionally, surgical technique that minimizes tissue devitalization reduces any infection risk.

8.3. Allograft and tissue donor screening

A major prevention lesson emerged from the recent bone graft-associated outbreaks. Tissue banks and hospitals must implement rigorous screening for TB in donors of bone or other tissues that retain viable cells. After the 2021 outbreak of TB from a bone allograft product involving 113 patients, it became clear that improved donor testing was needed.[6] The involved tissue bank started nucleic acid testing on bone grafts for TB,[6] but PCR may miss low levels of bacteria. Thus, adding culture-based testing of donor tissue is recommended for high-risk tissue grafts.[6] For surgeons, it is prudent to use only grafts from reputable sources following these guidelines. If an autograft is an option, it inherently avoids donor-derived TB.

8.4. Surveillance and early reporting

Healthcare systems should have surveillance for SSIs. While routine surveillance is aimed at common bacteria rates, it can also pick up unusual patterns—for instance, if multiple patients develop culture-negative chronic wounds after similar procedures, this should trigger an investigation. The 2021 TB outbreak was identified when a cluster of spinal surgery patients at one hospital all developed TB SSIs; recognizing the link led to a nationwide alert. Early notification of public health authorities can prevent more cases. In that outbreak, once recognized, they identified 36 recipients of the contaminated product and prevented its use in 53 more patients, likely avoiding additional TB cases.[6]

8.5. Environmental controls and patient education

Though TB SSIs are not airborne in transmission, OR ventilation and airborne precautions should be used for known TB patients. These measures primarily protect staff, but also indirectly protect the patient's wound from aerosolized TB droplets. In regions where patients might apply home remedies that are not sterile, education can help—for instance, there have been examples of inoculation TB from patients applying unpasteurized animal products to wounds, resulting in *Mycobacterium bovis* infection.[55]; such practices should be discouraged.

8.6. Addressing risk factors

Mitigating patient risk factors pre- and postoperatively is part of prevention. Good glycemic control in diabetics, optimization of nutrition, and minimizing steroid use around the time of surgery can all reduce the chance of an opportunistic TB infection. For example, if a rheumatoid patient on high-dose steroids is to have elective surgery, working with their rheumatologist to taper steroids and ensure they do not have latent TB (with prophylaxis if they do) could prevent a TB SSI in that immunosuppressed host.

9. Conclusion

Tuberculous SSIs, though rare, require vigilance among surgeons in all settings as *M. tuberculosis* can manifest as persistent "non-healing" wounds. Their unique pathophysiology features granulomatous inflammation with delayed onset, chronic discharge, and resistance to conventional antibiotics. Early diagnosis through biopsy and mycobacterial cultures, triggered by clinical suspicion, is crucial for proper management, which combines thorough surgical debridement with extended antitubercular chemotherapy.

Successful outcomes depend on multidisciplinary collaboration between surgeons, infectious disease specialists, and pathologists, with most patients achieving complete wound healing when promptly treated. Prevention strategies include proper sterilization, careful allograft screening, and risk-based patient screening, while case reporting helps identify patterns. Recent investigations have enhanced understanding of this uncommon complication, ensuring tuberculosis remains on the differential in surgical settings and helping restore patient health while preventing transmission.

Author contributions

Cen-Hung Lin: Writing - Original Draft, Writing - Review & Editing; Pi-Chieh Lin: Resources; Ching-Hua Hsieh: Conceptualization, Supervision. All authors have read and agreed to the final version of the manuscript.

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All data generated or analyzed during this study are included in this published article.

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