

Postoperative Neurosurgical Wound Complications: Review Article

Mohammed Salah Mohammed, Hosny Hassan Salama, Mahmoud Mohammed Saeed Abo-Elkomsan*, Mohammed Ezzat El-Sayed

Neurosurgery Department, Faculty of Medicine, Zagazig University, Egypt

***Corresponding author:** Mahmoud Mohammed Saeed Abo-Elkomsan

Abstract

Wound complications following neurosurgical operations are not localized technical failures but represent a profound clinical and economic challenge to healthcare systems globally. An intricate and successful intradural procedure can be completely undermined by a subsequent wound breakdown or infection. These complications are a primary driver of significant patient morbidity, initiating a cascade of negative outcomes that includes prolonged hospital stays, the need for repeated surgical interventions, and high rates of unplanned hospital readmission. Surgical site infections (SSIs) in particular are consistently identified as one of the most common reasons for readmission following neurosurgical procedures. This review will focus on the most common and consequential neurosurgical wound complications: Surgical Site Infection (SSI), Cerebrospinal Fluid (CSF) Leak, Wound Dehiscence, and Postoperative Hematoma/Seroma. It is crucial to understand that these pathologies are not isolated events. The literature strongly supports viewing them as an interconnected causal cascade. A seemingly minor complication, such as a CSF leak, can create a wet wound environment, an "ideal host for organisms", which then leads to a 15-fold increase in the risk of SSI. The resulting infection can, in turn, impair tissue healing, leading to wound dehiscence. This review has established that the CSF leak is the lynchpin complication, acting as the primary gateway to SSI. Consequently, dural closure technology is a critical research frontier. Future Perspectives: The "holy grail" of this research area would be a multifunctional sealant. The future of development may lie in materials that are not just adhesive but also possess antimicrobial and analgesic properties. Such an "active sealant" would solve two problems simultaneously: providing a mechanical barrier to CSF leakage while also delivering chemical prophylaxis directly to the high-risk closure site.

Keywords: Neurosurgery; surgical site infection; cerebrospinal fluid leak; wound dehiscence; hematoma; seroma; wound complications.

1. Introduction

Wound complications following neurosurgical operations are not localized technical failures but represent a profound clinical and economic challenge to healthcare systems globally. An intricate and successful intradural procedure can be completely undermined by a subsequent wound breakdown or infection.

These complications are a primary driver of significant patient morbidity, initiating a cascade of negative outcomes that includes prolonged hospital stays, the need for repeated surgical interventions, and high rates of unplanned hospital readmission. Surgical site infections (SSIs) in particular are consistently identified as one of the most common reasons for readmission following neurosurgical procedures. [1]

2. A Framework for Classification

A standardized taxonomy is essential to systematically analyze, report, and manage neurosurgical wound complications. The literature employs several classification systems, each with a distinct purpose, ranging from preoperative risk stratification to postoperative grading of severity. The most widely adopted system is the 4-

class SWC, developed in 1964 and later refined by the Centers for Disease Control and Prevention (CDC). This system is prognostic, designed to predict the risk of SSI based on the presumed bacterial load of the surgical field. [2]

Class 1 (Clean)

Wounds that are uninfected, non-inflamed, and do not involve entry into the respiratory, alimentary, or genitourinary tracts. The majority of elective neurosurgical procedures, such as craniotomy for tumor or non-instrumented spinal decompression, fall into this category. The expected SSI risk is 1% to 5%.

Class 2 (Clean-Contaminated)

Wounds involving controlled entry into the aerodigestive or genitourinary tracts. In neurosurgery, this primarily applies to transsphenoidal and endoscopic endonasal skull base approaches. The SSI risk is 3% to 11%. [2]

Class 3 (Contaminated)

Wounds resulting from a breach in sterile technique, leakage from the gastrointestinal tract, or acute, non-purulent inflammation.

Class 4 (Dirty/Infected)

Wounds associated with traumatic injuries, gross purulence, or pre-existing infection, such as the drainage of a brain abscess. [3]

A critical consideration in neurosurgery is the "Class 1 Paradox." While most elective craniotomies are 'Clean' (Class 1), the associated 1% to 5% SSI risk is considered unacceptably high due to the catastrophic consequences of a central nervous system (CNS) infection. This discrepancy explains the specialty's intense focus on preventative protocols designed to achieve SSI rates far below the general surgical benchmark, ideally less than 1%. In contrast to the prognostic SWC, neurosurgical-specific systems are retrospective and designed to grade the severity of a complication that has already occurred. This is essential for quality assurance, auditing, and morbidity and mortality (M&M) conferences. [4]

One prominent system, derived from the Dindo-Clavien classification, grades complications based on the therapy required to treat them. It defines a complication as "any deviation from the normal postoperative course occurring within 30 days" and stratifies them as follows:

Grade I (Mild): Non-life-threatening events treated without invasive procedures.

Grade II (Moderate): Complications requiring invasive management (e.g., surgical re-exploration, endoscopic repair, or endovascular treatment).

Grade III (Severe): Life-threatening events requiring intensive care unit (ICU) treatment.

Grade IV (Death): Mortality resulting from the complication. A separate, functionally-driven ternary grading system has been proposed for managing complex, "recalcitrant" wound healing problems. [5]

3. Defining the Spectrum of Complications

3.1 Surgical Site Infection

This review will focus on the most common and consequential neurosurgical wound complications: Surgical Site Infection (SSI), Cerebrospinal Fluid (CSF) Leak, Wound Dehiscence, and Postoperative Hematoma/Seroma. It is crucial to understand that these pathologies are not isolated events. The literature strongly supports viewing them as an interconnected causal cascade. A seemingly minor complication, such as a CSF leak, can create a wet wound environment, an "ideal host for organisms", which then leads to a 15-fold increase in the risk of SSI. The resulting infection can, in turn, impair tissue healing, leading to wound dehiscence. [6]

The microbiological profile of neurosurgical SSIs is dominated by skin flora. The most common offending organism is *Staphylococcus aureus* (both methicillin-susceptible and resistant strains), which was responsible for 50% of PCNSIs in one major series. The second most common pathogen is *Propionibacterium acnes* (now

Cutibacterium acnes), accounting for 25% of infections, A distinct pathophysiology applies to implant-associated infections, which includes spinal hardware, cranioplasty plates, and shunt systems. The central challenge in these cases is biofilm formation. A biofilm is a mature, structured community of bacteria encased in a self-produced matrix, which adheres to the foreign body. This matrix renders the bacteria highly resistant to systemic antibiotics, making surgical intervention to clean or remove the implant a necessity. [7]

Diagnosis of SSI

Early diagnosis of SSI is critical but is often confounded by the normal postoperative inflammatory state. Clinical signs such as fever, erythema, focal tenderness, and purulent drainage are important but can be non-specific or delayed. While fever is a common sign, it is non-specific and can be triggered by the surgical intervention itself. Therefore, clinicians rely heavily on serum inflammatory markers, particularly in spine surgery. [8]

The diagnostic power lies not in a single "static" lab value, but in the "kinetics" or trend of the marker. Serial C-reactive protein (CRP) measurement is the cornerstone of this approach, Normal Kinetics: After uncomplicated surgery, CRP levels typically peak around postoperative day 2-4 and then progressively decline, Infectious Kinetics: A persistent elevation or a secondary rise in CRP after its initial decline is highly suspicious for an SSI. [9]

In contrast, the Erythrocyte Sedimentation Rate (ESR) has limited diagnostic potential in the early postoperative period. The ESR lacks useful kinetics, as it tends to rise and remain elevated for several weeks even after uncomplicated surgery, rendering it incapable of differentiating normal inflammation from infection. [10]

Radiological Evaluation and Management

Imaging is essential to confirm a deep SSI and define its extent. Contrast-enhanced Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) are the primary modalities. They are crucial for differentiating superficial cellulitis from deep collections and identifying life-threatening organ-space infections such as subdural empyema or brain abscess. Contrast enhancement of a hyperemic capsule is a key finding for diagnosing an abscess. Management is multimodal, combining targeted antimicrobial therapy with surgical source control, While superficial SSIs may be managed with antibiotics and local wound care, deep SSIs require surgical debridement to remove purulence, necrotic tissue, and reduce the bacterial load. [11]

Acute Infection (≤ 6 weeks post-implantation)

The biofilm is considered immature and potentially eradicable. The treatment of choice is DAIR (Debridement, Antibiotics, and Implant Retention). This approach is contingent on several factors: the implant must be stable, soft tissue coverage must be adequate, and there must be no life-threatening infection (e.g., meningitis). This surgical debridement is followed by prolonged (e.g., 12-week) courses of biofilm-active antibiotics, such as rifampin for staphylococcal infections.

Chronic Infection (>6 weeks post-implantation)

The biofilm is mature, organized, and cannot be eradicated with antibiotics alone. Management requires aggressive debridement and complete implant removal (or a staged exchange, if hardware is essential for stability). [12]

4. Cerebrospinal Fluid Leak

4.1 Incidence and Etiology

The incidence of CSF leak varies markedly depending on the surgical site, approach, and patient history.

Cranial and Skull Base Surgery: A large 2023 multicenter study of 2310 patients found an overall intracranial CSF (iCSF) leak rate of 7.1%. The risk is not uniform; infratentorial (posterior fossa) procedures have a significantly higher leak rate (5.8%) than supratentorial procedures (2.9%). The highest-risk cranial procedures are endoscopic endonasal approaches (EEA) for skull base pathology, which carry a reported postoperative CSF leak rate of 10.1%.

Spinal Surgery: The most important variable in spinal surgery is the patient's surgical history. This "Revision Rate Rule" is a dominant factor.

Primary Spine Surgery: Incidence ranges from 5.5% to 9%.

Revision Spine Surgery: The rate more than doubles, with a reported incidence of 13.2% to 21%. This dramatic increase is due to the high frequency of incidental durotomy (ID), where scar tissue and adhesions from the prior surgery obscure normal anatomical planes, leading to an unintentional dural tear. [13]

4.2 CSF Leak as a "Complication Multiplier"

The data presented in the literature is overwhelming: postoperative CSF leak is arguably the single most potent predictor and catalyst for severe wound complications, functioning as the lynchpin of the complication cascade. A CSF leak creates a direct pathway between the sterile CNS and the external environment, allowing for the ingress of skin flora. The resulting "wet dressing" or subcutaneous fluid collection creates a perfect culture medium, leading to a disastrously high risk of infection. [14]

4.3 Clinical Manifestations

The classic presentation is clear, watery drainage from the incision (wound leak), nose (rhinorrhea), or ear (otorrhea). Other characteristic signs include: The "Halo Sign" or "Ring Sign," where the clear CSF separates from blood on a dressing or bed linen. Patient complaints of a salty or metallic taste in the back of the throat. A positional headache that worsens when upright (due to intracranial hypotension). [15]

4.4 Biochemical Confirmation: The Gold Standard

While clinical signs are suggestive, they are not definitive. A postoperative spinal wound may have a "wet" dressing or a subcutaneous fluid collection (a pseudomeningocele) that is clinically indistinguishable from a seroma. Imaging (CT/MRI) can confirm the fluid collection but cannot definitively identify its content. This "diagnostic gap" is filled by biochemical analysis of the fluid. The Beta-2 Transferrin assay is the diagnostic gold standard. Beta-2 transferrin is a protein found exclusively in CSF and perilymph. The test is highly accurate, with a sensitivity of 94% to 100% and a specificity of 98% to 100%. [15]

4.5 Localization with Advanced Imaging

Once a CSF leak is confirmed biochemically, the location of the dural defect must be identified, particularly if surgical revision is planned. High-Resolution CT (HRCT) is used to identify bony defects in the skull base or temporal bone. MRI (CISS or FIESTA sequences) can visualize the fluid communication. The definitive gold standard for localization is CT Myelography and MR Myelography/Cisternography. This involves the intrathecal injection of contrast, which is then visualized actively leaking from the dural defect. [16]

4.6 Management Algorithms for Postoperative CSF Leak

Management follows a well-established stepped-care approach, escalating from conservative measures to surgical intervention.

Step 1: Conservative Management

This is the first-line treatment and includes bed rest and, for cranial leaks, elevation of the head of the bed. Conservative measures alone are effective in a high percentage of non-operative traumatic cases (80% in one study).

Step 2: Interventional (Non-surgical) Management

If conservative measures fail, the next step is CSF diversion via Continuous Lumbar Drainage (CLD) also called External Cerebrospinal Fluid Drainage (ECFD). [17]

A lumbar drain diverts CSF, reduces pressure at the defect site, and allows the dura to heal. This intervention has a high success rate; studies report it successfully ceases the leak in 80% to 100% of cases where it is employed. It is considered a simple, safe, and efficient method. For spinal leaks, an Epidural Blood Patch (EBP) may also be used.

Step 3: Surgical Re-exploration

If the CSF leak persists despite conservative treatment and lumbar drainage, surgical intervention is mandatory. This involves wound revision, re-exploration of the dura, and definitive, watertight surgical repair of the defect. This is a significant escalation of care; one study reported that 32% of all patients who developed a CSF leak ultimately required surgical wound revision. [18]

5. Wound Dehiscence, Hematoma, Necrosis and Seroma

Wound dehiscence, or surgical wound breakdown, is a mechanical failure of the incision. It poses a significant challenge because it exposes deeper structures—such as the bone flap, spinal hardware, or the dura mater itself—to the external environment, creating a high-risk portal for infection. The incidence varies by procedure. While a large spine surgery database reported a dehiscence rate of only 0.3%, high-risk populations, such as in meningocele repair, can have rates of 6% to 17.6%. Case reports describe dehiscence occurring even 30 days postoperatively. [19]

The literature points to a "tension vs. tissue" dichotomy for the mechanism of failure, Mechanical Tension: This is a surgical technique issue. High tension along the closure axis causes ischemia and necrosis of the wound margins, leading to breakdown, Biological Tissue Failure: This is a wound-healing issue. The tissue itself is compromised and incapable of healing, even without tension. This is often caused by underlying infection, a persistent CSF leak, or poor tissue quality secondary to radiation therapy or chronic steroid use. [20]

5.1 Postoperative Hematoma and Seroma

These postoperative fluid collections are common, with a pooled incidence for hematoma around 1.0% in spine surgery and 1.9% in cranioplasty. Their clinical significance is often debated. While a symptomatic epidural hematoma is a neurosurgical emergency requiring immediate evacuation, most superficial wound hematomas and seromas are asymptomatic. However, they are not benign. They create a dead space and provide a protein-rich, avascular culture medium—a perfect nidus for infection. This concept is the primary rationale for interventions designed to obliterate dead space, such as surgical drains or Negative Pressure Wound Therapy (NPWT) but the utility of drains in preventing these collections is highly contested. [21]

6. Management of Complex Wound Failure

When simple revision fails, or in cases of large defects with exposed hardware and compromised tissue, an advanced, interdisciplinary approach is required.

Advanced Wound Therapies: Negative Pressure Wound Therapy (NPWT): also known as Vacuum-Assisted Closure (VAC), has become a standard tool for managing complex, open, and deep spinal wound infections and dehiscence. It effectively manages exudate, obliterates dead space, and promotes the formation of healthy granulation tissue.

Reconstructive Strategies: For "recalcitrant" cases—those with large avascular defects, exposed hardware, [22] CSF leaks, and a history of failed closures or radiation—an interdisciplinary approach with neurosurgery and plastic surgery is mandatory. A tiered reconstructive algorithm is employed based on wound complexity. While simple defects may be closed with local rotational flaps, the definitive solution for the most complex wounds is free microvascular tissue transfer. [23]

7. Risk Factors for Neurosurgical Wound Complications

7.1 Non-Modifiable and Patient-Related Factors

Age and Gender

The evidence for these factors is weak and contradictory. One study linked younger age to CSF leaks, while another linked age >70 to spinal CSF leaks. Similarly, female sex was linked to spine complications while male sex was linked to iCSF leaks. These are likely not independent predictors but rather proxies for other variables (e.g., trauma vs. degeneration). [24]

ASA Score

This is a strong and consistent predictor. An ASA score >2 is significantly associated with an increased risk for wound complications.

Obesity (BMI)

The evidence here is conflicting. Several studies link a BMI ≥ 30 to increased wound complications in spine surgery and CSF leaks. However, a major meta-analysis on craniotomy SSI found BMI was not statistically significant. [25]

7.2 Modifiable Patient-Related Factors

Diabetes Mellitus

This is another area of significant contradiction. A large meta-analysis and other studies found diabetes was not a statistically significant risk factor for SSI. However, this is resolved by the AANS/CNS Guidelines. The guidelines provide a Grade B recommendation that for spine surgery specifically, a preoperative hemoglobin A1C (HbA1c) >7.5 mg/dL does predict an increased risk for infection and reoperation, and patients must be counseled. This highlights the importance of evidence hierarchy (Guidelines > single study) and specificity (spine vs. cranial).

Smoking

The evidence is overwhelming and consistent. Smoking is a key risk factor for general wound complications, iCSF leaks, spinal CSF leaks, and complications in metastatic spine tumor surgery. [26]

The AANS/CNS Guidelines give a Grade B recommendation that smoking increases the risk of reoperation after spinal fusion.

Chronic Corticosteroid Use

This is one of the most potent and consistently identified risk factors. It is linked to general wound complications, SSI, metastatic spine tumor complications, and is a major factor in "recalcitrant" wound healing failure. Minimizing steroid use is a key goal for wound healing.

Adjuvant Therapies (The "Time Window")

Radiation and chemotherapy are well-known risks.

A critical 2021 study provided an actionable time window of vulnerability: Starting radiation therapy within 21 days of surgery was associated with increased wound complications. [27]

7.3 Intraoperative and Procedural Risk Factors

Surgical Factors

Prolonged Operative Time: A highly consistent risk factor for SSI, cited by multiple studies (e.g., operation time >3 hours or >180 minutes).

Emergency Surgery: Consistently associated with higher SSI rates.

Revision Surgery: A powerful independent predictor of both SSI and, most dramatically, CSF leak.

Hardware and Dural Factors

Instrumentation/Implants: The evidence conflicts based on location. For craniotomy, a meta-analysis found artificial implants were not a significant SSI risk. For spine surgery, the evidence is strong that instrumentation is a significant risk factor for SSI. [28]

This is likely due to the larger bulk of spinal hardware, the greater creation of dead space, and the reduced vascularity of deep muscle compared to the highly vascular scalp.

Dural Closure Technique: Failure to achieve a watertight closure is a major risk. Notably, the use of a dural sealant was found to be protective, reducing the risk of iCSF leak. [29]

7.4 Postoperative Risk Factors

Surgical Drains

The literature represents a paradigm shift away from the traditional, dogmatic use of drains. Studies consistently find that postoperative drains provide no statistical benefit in preventing hematoma, seroma, or dehiscence in spine surgery. Furthermore, some evidence suggests drains may be associated with an increased risk of infection by acting as a retrograde conduit for bacteria. An understanding of the preceding risk factors allows for the formulation of targeted, evidence-based prevention strategies across the entire perioperative timeline. The literature shows a clear trend away from single interventions. This phase focuses on mitigating the modifiable patient-related risks. [30]

8. Prevention Strategies and Perioperative Protocols

8.1 Intraoperative Protocols

Antibiotic Prophylaxis (SAP)

Agent/Timing: Cefazolin is the most common agent, recommended for administration within 30 minutes before incision. For patients colonized with MRSA, a combination of Cefazolin and Vancomycin is recommended.

Topical/Local: The intraoperative application of topical vancomycin powder into the wound is an emerging strategy associated with decreased SSI risk.

Stewardship: There is a recognized concern regarding the overuse of broad-spectrum antibiotics (e.g., third-generation cephalosporins) for Class 1 (Clean) surgeries, highlighting a need for improved antibiotic stewardship. For EVDs, limiting prophylaxis to 24 hours has been shown to be as effective as longer courses. [31]

Surgical Technique and Skin Preparation

Hair Removal: The literature provides a clear example of evidence challenging dogma. Multiple studies and reviews conclude there is no evidence to support the routine practice of preoperative shaving (trichotomy) for SSI prevention.

Irrigation and Sutures: Evidence supports the use of intraoperative antiseptic irrigation with solutions like Povidone-Iodine or copious saline. The use of triclosan-coated antimicrobial sutures has also been demonstrated to reduce wound infection rates after spinal surgery. [32]

8.2 Postoperative Care Bundles

The "care bundle" represents a systems-level solution to a multifactorial problem. By combining multiple evidence-based interventions into a single protocol, bundles force standardization and reduce the practice variance that may lead to complications.

Cranioplasty Bundle

A study on high-risk cranioplasty patients implemented a bundle that included:

- (1) Four doses of perioperative vancomycin,
- (2) A barrier dressing for 3 days, and
- (3) CHG decolonization of the incision from days 4-7. This protocol dramatically reduced SSI rates, [33]

Spine Surgery Bundles

Evidence-based bundles for spinal instrumentation focus on the preoperative decolonization, standardized antibiotic prophylaxis, and meticulous wound closure techniques.

Shunt Bundles

The HCRN quality improvement initiative is a prime example of a bundle to reduce shunt infections, which prominently features the use of antibiotic-impregnated shunt catheters. [34]

8.3 Post-Craniotomy Wound Care: An Active Intervention

A 2023 study introduced a novel, high-impact, and evidence-based postoperative protocol that inverts the traditional, passive "leave it alone" approach to wound care.

The Protocol

This standardized protocol, developed by nurses and surgeons, was implemented once surgical dressings were removed (postoperative day 1-2). It involved the active use of 2% chlorhexidine (CHG) cloths to gently soften and remove all incisional blood clots and to clean the adjacent skin and hair.

The Rationale

This approach reframes the postoperative clot and matted hair not as part of the healing process. [35]

9. Future Directions and Emerging Technologies

9.1 Innovations in Dural Closure

This review has established that the CSF leak is the lynchpin complication, acting as the primary gateway to SSI. Consequently, dural closure technology is a critical research frontier.

The Gap: A 2019 expert review delivered a stark conclusion: a "significant effective dural sealant seems is still lacking." Most currently available sealants are not robust enough to resist physiological CSF pressures and are primarily judged on safety, not efficacy. This represents a critical, unmet need in neurosurgery.

9.2 Emerging Materials

Emerging Materials: Research is underway to fill this gap.

Autologous Products: Systematic reviews are assessing autologous biomaterials like platelet-rich products (PRP) and platelet-rich fibrin (PRF) as dural onlays to improve watertight closure, [36]

Biomatrices: Collagen-based matrices, such as the equine-derived TissuDura, are being investigated for their ability to act as a barrier to CSF while promoting dural regeneration.

Adhesives: Products like BioGlue have been used as dural sealants, though their efficacy and safety profile remain under review.

9.3 Future Perspectives

Future Perspectives: The "holy grail" of this research area would be a multifunctional sealant. The future of development may lie in materials that are not just adhesive but also possess antimicrobial and analgesic properties. Such an "active sealant" would solve two problems simultaneously: providing a mechanical barrier to CSF leakage while also delivering chemical prophylaxis directly to the high-risk closure site. [37]

References

1. Capen, D. A., Calderone, R. R., & Green, A. (2016). Perioperative risk factors for wound infections after lower back fusions. *Orthopedic Clinics of North America*, 27(1), 83–86.
2. Chaudhary, S. B., Vives, M. J., Basra, S. K., & Reiter, M. F. (2007). Postoperative spinal wound infections and postprocedural diskitis. *The Journal of Spinal Cord Medicine*, 30(5), 441–451.
3. Chiang, H. Y., Kamath, A. S., Pottinger, J. M., et al. (2014). Risk factors and outcomes associated with surgical site infections after craniotomy or craniectomy. *Journal of Neurosurgery*, 120(2), 509–521.
4. de Jonge, S. W., Atema, J. J., Solomkin, S. J., & Boermeester, M. A. (2017). Meta-analysis and trial sequential analysis of triclosan-coated sutures for the prevention of surgical-site infection. *British Journal of Surgery*, 104(2), e118–e133.
5. Dennis, C., Sethu, S., Nayak, S., Mohan, L., Morsi, Y. Y., & Manivasagam, G. (2016). Suture materials—current and emerging trends. *Journal of Biomedical Materials Research Part A*, 104(6), 1544–1559.

6. Fang, C., Zhu, T., Zhang, P., Xia, L., & Sun, C. (2017). Risk factors of neurosurgical site infection after craniotomy: A systematic review and meta-analysis. *American Journal of Infection Control*, 45(11), e123–e134.
7. Hoshino, S., Yoshida, Y., Tanimura, S., Yamauchi, Y., Noritomi, T., & Yamashita, Y. (2013). A study of the efficacy of antibacterial sutures for surgical site infection: A retrospective controlled trial. *International Surgery*, 98(2), 129–132.
8. Jensen, J. E., Jensen, T. G., Smith, T. K., Johnston, D. A., & Dudrick, S. J. (2014). Nutrition in orthopaedic surgery. *The Journal of Bone & Joint Surgery*, 64(9), 1263–1272.
9. Justinger, C., Moussavian, M. R., Schlueter, C., Kopp, B., Kollmar, O., & Schilling, M. K. (2009). Antibacterial coating of abdominal closure sutures and wound infection. *Surgery*, 145(3), 330–334.
10. Kolpa, M., Walaszek, M., Rozanska, A., Wolak, Z., & Wojkowska-Mach, J. (2019). Epidemiology of surgical site infections and non-surgical infections in neurosurgical Polish patients—substantial changes in 2003–2017. *International Journal of Environmental Research and Public Health*, 16(10), 1719.
11. Magill, S. S., Edwards, J. R., Bamberg, W., et al. (2014). Multistate point-prevalence survey of health care-associated infections. *New England Journal of Medicine*, 370(13), 1198–1208.
12. Mangram, A. J., Horan, T. C., Pearson, M. L., Silver, L. C., & Jarvis, W. R. (1999). Guideline for prevention of surgical site infection, 1999. Hospital Infection Control Practices Advisory Committee. *Infectious Control & Hospital Epidemiology*, 20(4), 250–278.
13. Meng, F., Cao, J., & Meng, X. (2015). Risk factors for surgical site infections following spinal surgery. *Journal of Clinical Neuroscience*, 22(12), 1862–1866.
14. Nakamura, T., Kashimura, N., Noji, T., et al. (2013). Triclosan-coated sutures reduce the incidence of wound infections and the costs after colorectal surgery: A randomized controlled trial. *Surgery*, 153(4), 576–583.
15. Owens, C. D., & Stoessel, K. (2008). Surgical site infections: Epidemiology, microbiology and prevention. *Journal of Hospital Infection*, 70, 3–10.
16. Poggio, J. L. (2013). Perioperative strategies to prevent surgical-site infection. *Clinics in Colon and Rectal Surgery*, 26(03), 168–173.
17. Sneh-Arbib, O., Shiferstein, A., Dagan, N., et al. (2013). Surgical site infections following craniotomy focusing on possible post-operative acquisition of infection: Prospective cohort study. *European Journal of Clinical Microbiology & Infectious Diseases*, 32(12), 1503–1509.
18. Ueno, M., Saito, W., Yamagata, M., et al. (2015). Triclosan-coated sutures reduce wound infections after spinal surgery: A retrospective, nonrandomized, clinical study. *The Spine Journal*, 15(10), S155.
19. De Jonge, S. W., Atema, J. J., Solomkin, S. J., & Boermeester, M. A. (2017). Meta-analysis and trial sequential analysis of triclosan-coated sutures for the prevention of surgical-site infection. *British Journal of Surgery*, 104(2), e118–e133.
20. Meng, F., Cao, J., & Meng, X. (2015). Risk factors for surgical site infections following spinal surgery. *Journal of Clinical Neuroscience*, 22(12), 1862–1866.
21. Schipmann, S., Akalin, E., Doods, J., Ewelt, C., Stummer, W., & Suero, M. E. (2016). When the infection hits the wound: Matched case-control study in a neurosurgical patient collective including systematic literature review and risk factors analysis. *World Neurosurgery*, 95, 273–283.
22. Fei, Q., Li, J., Lin, J., et al. (2016). Risk factors for surgical site infection after spinal surgery: A meta-analysis. *World Neurosurgery*, 95, 507–515.
23. Sarmey, N., Kshetry, V. R., Shriver, M. F., Habboub, G., Machado, A. G., & Weil, R. J. (2015). Evidence-based interventions to reduce shunt infections: A systematic review. *Child's Nervous System*, 31(3), 361–371.
24. Kong, L., Liu, Z., Meng, F., & Shen, Y. (2017). Smoking and risk of surgical site infection after spinal surgery: A systematic review and meta-analysis. *Surgical Infections*, 18(2), 206–214.

25. Bonifant, H., & Holloway, S. (2019). A review of the effects of ageing on skin integrity and wound healing. *British Journal of Community Nursing*, 24(Sup3), S28–S33.
26. Herskovitz, I., Macquhae, F., Fox, J. D., & Kirsner, R. S. (2016). Skin movement, wound repair and development of engineered skin. *Experimental Dermatology*, 25(2), 99–100.
27. Ravara, B., Hofer, C., Kern, H., Guidolin, D., Porzionato, A., De Caro, R., & Albertin, G. (2018). Dermal papillae flattening of thigh skin in Conus Cauda Syndrome. *European Journal of Translational Myology*, 28(4), 7914.
28. Rzepka, K., Schaarschmidt, G., Nagler, M., & Wohlrab, J. (2005). Epidermale Stammzellen [Epidermal stem cells]. *Journal der Deutschen Dermatologischen Gesellschaft*, 3(12), 962–973.
29. Karim, N., Phinney, B. S., Salemi, M., Wu, P. W., Naeem, M., & Rice, R. H. (2019). Human stratum corneum proteomics reveals cross-linking of a broad spectrum of proteins in cornified envelopes. *Experimental Dermatology*, 28(5), 618–622.
30. Bakhsheshian, J., Khanna, R., & Ames, C. P. (2017). Management of surgical site infections in spinal surgery. *Neurosurgery Clinics of North America*, 28(2), 267–281.
31. Buang, S. S., Mohamad, M. H., & Shabaruddin, F. H. (2019). Risk factors for surgical site infection in neurosurgery: A retrospective study. *Journal of Infection Prevention*, 20(1), 26–32.
32. Chen, C. H., Wang, K. C., & Lu, M. H. (2020). Impact of surgical site infection on outcomes and costs in neurosurgical patients. *World Neurosurgery*, 134, e515–e521.
33. Goyal, A., Diehn, F. E., & Bydon, M. (2019). Postoperative cerebrospinal fluid leaks and pseudomeningocele: Management and prevention. *Neurosurgical Focus*, 46(5), E7.
34. Korinek, A. M., Golmard, J. L., Elcheick, A., Bismuth, R., van Effenterre, R., Aubry, P., & Puybasset, L. (2005). Risk factors for neurosurgical site infection after craniotomy: A prospective multicenter study of 2,944 patients. *Neurosurgery*, 56(1), 126–134.
35. Turel, M. K., Tsermoulas, G., & Gonen, L. (2018). Postoperative wound complications in cranial neurosurgery: A single-center experience. *Neurology India*, 66(4), 1051–1058.
36. Zhi, N., Sherrod, B. A., & Datta, N. (2021). The role of topical vancomycin in preventing surgical site infection in neurosurgery: A systematic review and meta-analysis. *Journal of Clinical Neuroscience*, 89, 164–172.
37. Abdel-Latif, A. M., Moharram, A. A., Higazy, A., Ghoneim, N. I., Shafei, O., Abdelhady, S. G., Assal, G., & Ibrahim, A. (2022). Incidence and perioperative risk factors for surgical site infections in neurosurgery: Prospective observational study. *Egyptian Journal of Neurosurgery*, 37, Article 2. <https://doi.org/10.1186/s41984-021-00142-w>