



The Pathological Impact of Aging on the Risk of Postoperative Spinal Infections

Seyed Taher Mousavi¹, Tala Poulak^{2*}

¹Assistant Professor of Neurosurgery, Department of Neurosurgery, School of Medicine, Tabriz University of Medical Sciences, Tabriz, Iran

²Associate Professor of Pathology, Department of Pathology, School of Medicine, Tabriz University of Medical Sciences, Tabriz, Iran

Article info

Received: 16.07.2026

Accepted: 01.10.2026

Available Online: 03.10.2026

Checked for Plagiarism: Yes

Keywords:

Spinal Surgery, Surgical Site Infection, Immunosenescence, Postoperative Complications

ABSTRACT

Introduction: Aging increases susceptibility to postoperative spinal infections through immunosenescence, microvascular dysfunction, and systemic comorbidity. Despite rising surgical volumes in older adults, the independent contribution of advanced age to infection risk remains debated. This study evaluates the pathological impact of aging on the risk of postoperative spinal infections to clarify its specific role in clinical outcomes.

Material and methods: This descriptive cross-sectional study, conducted at Shahid Madani Hospital, investigated the pathological impact of aging on postoperative spinal infections. Based on Cochran's formula, 111 participants were selected via convenience sampling. Clinical records were analyzed to evaluate demographic, surgical, and outcome variables. Associations between age and infection risk were assessed using appropriate statistical tests, with full ethical approval obtained from Tabriz University of Medical Sciences.

Results: Postoperative spinal infection incidence significantly increased with age, rising from 6.7% to 25.0% (P for trend=0.037). Older patients exhibited higher systemic comorbidities and surgical complexity. Multivariable analysis identified advancing age as an independent infection predictor (adjusted OR=1.68, 95% CI:1.12-2.52; P=0.012), alongside prolonged operative duration (adjusted OR=2.80; P=0.014) and diabetes (adjusted OR=2.45; P=0.031), confirming the biological burden of senescence.

Conclusion: Advanced age serves as an independent risk factor for postoperative spinal infections, driven by the convergence of immunosenescence, systemic comorbidities, and decreased physiological reserve. Surgeons must implement targeted preoperative optimization and meticulous intraoperative techniques to mitigate risks in elderly populations.

Introduction

Spinal surgery increasingly performed in older adults because of the growing prevalence of degenerative spinal disorders, vertebral fractures, spinal deformities, and age-associated neurological impairment. Although advances in perioperative management, surgical instrumentation, antimicrobial prophylaxis, and postoperative monitoring have improved the safety of spinal procedures, postoperative infection remains a clinically important complication.

Spinal infections may involve the superficial incision, deep soft tissues, intervertebral disc, vertebral bodies, epidural space, or implanted instrumentation. These complications can result in prolonged hospitalization, repeated surgical debridement, prolonged antimicrobial therapy, implant removal, neurological deterioration, and persistent disability. The relationship between aging and postoperative infection is complex and not be explained solely by chronological age.

*Corresponding Author: **Tala Poulak** (tl_poulak@gmail.com - ORCID: 0000-0001-6413-9747)

1 Email: Mousavi_st@gmail.com - ORCID: 0000-0003-2609-6066

Rather, aging is accompanied by multiple biological, immunological, metabolic, and functional changes that may collectively reduce the capacity to resist infection and recover from surgical injury [1].

One of the most important biological consequences of aging is immunosenescence, a gradual decline in the function and efficiency of the innate and adaptive immune systems. In older individuals, the production of naïve T lymphocytes decreases, T-cell receptor diversity becomes restricted, and the ability to generate an effective response to newly encountered pathogens is impaired. B-cell maturation and antibody production may also become less efficient, while the activity of natural killer cells, neutrophils, macrophages, and antigen-presenting cells altered. These changes do not necessarily represent complete immune suppression; instead, they reflect a dysregulated immune state in which protective responses delayed, insufficient, or poorly coordinated. Following spinal surgery, such impairment may facilitate bacterial invasion of damaged tissues and hinder the early containment of infection [2].

Aging is also associated with a persistent, low-grade inflammatory condition commonly referred to as inflammaging. This state characterized by chronically increased circulating concentrations of inflammatory mediators, including interleukin-6, tumor necrosis factor- α , and C-reactive protein. Despite the presence of systemic inflammation, the local immune response to tissue injury may remain ineffective or excessive. This apparent paradox can interfere with normal wound healing by prolonging the inflammatory phase, impairing fibroblast activity, disrupting extracellular matrix remodeling, and reducing the quality of scar formation. Consequently, older patients may experience delayed wound closure and greater susceptibility to bacterial colonization, particularly when the surgical exposure is extensive or when metallic implants are used [3].

The integrity of the skin and subcutaneous tissues is another important factor influencing infection risk in the elderly. Age-related thinning of the epidermis, reduced dermal collagen, diminished vascularity, and loss of subcutaneous tissue can compromise the mechanical barrier against microorganisms. In addition, older skin has a reduced capacity for regeneration and may be more vulnerable to pressure-related injury, ischemia, hematoma formation, and wound separation. During prolonged spinal procedures, these vulnerabilities may be aggravated by sustained pressure, retraction, tissue desiccation, and reduced local perfusion. Once the protective barrier is disrupted, the relatively poor vascular supply of certain posterior spinal tissues may limit the delivery of immune cells and antimicrobial agents to the operative site [4].

Impaired tissue perfusion and microvascular dysfunction further contribute to the pathological

effect of aging. Endothelial dysfunction, arterial stiffness, atherosclerosis, and reduced capillary density may decrease oxygen delivery to the surgical wound. Adequate tissue oxygenation is essential for neutrophil-mediated bacterial killing, collagen synthesis, angiogenesis, and wound remodeling. Hypoxic tissue is therefore not only less capable of repairing itself but may also provide a favorable environment for bacterial persistence. The problem intensified by perioperative hypotension, anemia, smoking, diabetes mellitus, or excessive blood loss, all of which are relatively common in older patients undergoing complex spinal surgery [5].

Older adults frequently present with comorbid conditions that may independently increase the likelihood of postoperative infection. Diabetes mellitus can impair neutrophil chemotaxis, phagocytosis, and intracellular killing while promoting microvascular disease and tissue hypoxia. Malnutrition, particularly inadequate protein and micronutrient intake, can reduce immune competence and compromise collagen synthesis. Chronic kidney disease, chronic pulmonary disease, obesity, malignancy, rheumatoid disease, and long-term corticosteroid use may further weaken host defenses. Importantly, these conditions often coexist in the same patient, producing a cumulative biological burden that is not adequately captured by age alone. Thus, chronological age may act as a marker of vulnerability, whereas the actual risk is determined by the interaction between age-related changes, comorbidity, and physiological reserve [6].

The characteristics of the spinal procedure also influence the association between age and postoperative infection. Instrumented fusion, revision surgery, multilevel decompression, deformity correction, and procedures involving extensive soft-tissue dissection generally expose patients to greater operative trauma and longer surgical duration. Implants may provide a surface for bacterial adhesion and biofilm formation, allowing microorganisms to persist despite systemic antibiotic therapy and host immune responses. In older patients, degenerative anatomy, osteoporosis, spinal deformity, and previous operations may increase technical difficulty and prolong operative time. These factors may amplify the impact of age-related immune and vascular impairment, thereby increasing the probability that a minor contamination event will progress to a clinically significant infection [7].

The clinical presentation of infection in older adults may also differ from that observed in younger patients. Fever, leukocytosis, and pronounced local inflammatory findings may be absent or attenuated because of immunosenescence, chronic medication use, or an altered inflammatory response. Instead, older patients may present with nonspecific manifestations such as fatigue, confusion, reduced

mobility, worsening back pain, anorexia, or delayed functional recovery. This atypical presentation can postpone diagnosis and allow infection to progress before definitive treatment is initiated. Infections involving spinal instrumentation may be particularly difficult to recognize because low-virulence organisms can produce indolent symptoms and modest laboratory abnormalities. Therefore, a high index of suspicion is essential when an older patient develops unexplained postoperative deterioration [8].

The relationship between age and postoperative spinal infection should nevertheless be interpreted cautiously. Age may not function as an independent causal factor in every clinical setting after adjustment for diabetes, obesity, nutritional status, smoking, operative duration, blood loss, revision status, and the extent of instrumentation. In some studies, frailty, functional dependence, or a high comorbidity burden has demonstrated greater predictive value than chronological age. Frailty reflects diminished physiological reserve and an impaired ability to recover from stress, and it may provide a more biologically meaningful assessment of infection susceptibility than age alone. Accordingly, evaluating older surgical candidates should involve a multidimensional assessment of frailty, nutritional status, glycemic control, medication exposure, immune competence, and functional capacity rather than relying exclusively on an age threshold [9].

Understanding the mechanisms through which aging contributes to postoperative spinal infection has important implications for prevention and clinical decision-making. Preoperative optimization may include nutritional assessment, correction of anemia, improved glycemic control, smoking cessation, medication review, and identification of frailty. Intraoperative strategies should emphasize meticulous tissue handling, preservation of vascularity, appropriate antimicrobial prophylaxis, minimization of operative time when feasible, and careful management of blood loss and body temperature. Postoperatively, early wound surveillance, prompt investigation of unexplained pain or functional decline, and timely microbiological sampling may facilitate diagnosis before deep infection becomes established. A risk-stratified approach may also help clinicians balance the benefits of surgery against the heightened vulnerability of patients with advanced age and limited physiological reserve [10].

Despite the increasing number of older adults undergoing spinal surgery, important uncertainties remain regarding the independent contribution of aging to postoperative infection. Existing evidence is limited by differences in infection definitions, surgical populations, follow-up duration, comorbidity adjustment, and the distinction between superficial and deep infections. Moreover,

chronological age is frequently analyzed as a simple categorical variable, although the biological effects of aging are continuous and highly heterogeneous. Further research is therefore needed to clarify whether age independently predicts infection, which age-related mechanisms are most clinically relevant, and whether frailty or other biological markers provide superior risk discrimination. Accordingly, this study aimed to evaluate the pathological impact of aging on the risk of postoperative spinal infections and to determine whether advanced age is independently associated with infection after spinal surgery.

Material and methods

Study Design and Setting:

This hospital-based descriptive cross-sectional study was conducted among patients who underwent spinal surgery at Shahid Madani Hospital, Tabriz, Iran. Patient information was obtained from medical records and available postoperative documentation. The study examined the association between patient age and documented postoperative spinal infection.

Sample Size and Sampling:

The sample size was estimated using Cochran's single-proportion formula: $n = Z^2 p(1-p)/d^2$. Assuming a 95% confidence level ($Z=1.96$), an expected proportion of 0.50, and an absolute precision of 0.093, the estimated sample size was approximately 111 patients. Participants were selected using convenience sampling from eligible patients until the required sample size was reached.

Eligibility Criteria:

Patients were eligible if they were 18 years of age or older, had undergone spinal surgery at the study hospital during the study period, and had medical records containing information on age, the index procedure, and postoperative infection status. Patients were excluded if the index surgery had been performed to treat an infection already present at the operative site, if an active spinal or surgical-site infection was documented before surgery, or if essential information including age, operative details, or postoperative infection status was unavailable. Duplicate records and records relating to procedures other than spinal surgery were also excluded.

Study Variables:

The primary explanatory variable was age at the time of surgery, recorded in years and analyzed as a continuous variable. Other patient characteristics extracted from the records included sex and, where documented, body mass index, smoking status, diabetes mellitus, hypertension, renal disease, immunosuppressive conditions or treatment, and other recorded comorbidities. These variables were considered because they may be associated with host

defense, wound healing, or the likelihood of postoperative infection.

Surgical and outcome variables included the type of spinal procedure, whether instrumentation was used, whether the operation was a revision procedure, the number of spinal levels treated, operative duration, and estimated blood loss, where available. The primary outcome was a documented postoperative infection related to the spinal surgical site or deeper operative tissues. When recorded, infection depth or type, microbiological findings, and the timing of infection diagnosis were also extracted. Infection status was classified from the available clinical documentation; therefore, the same diagnostic criteria should be applied consistently across all records.

Statistical Analysis:

Continuous variables were summarized as means and standard deviations, and categorical variables as frequencies and percentages. The distribution of continuous variables was assessed using the Shapiro-Wilk test and visual inspection of histograms or Q-Q plots; the recorded continuous data were treated as normally distributed. Differences in continuous variables between patients with and without postoperative infection were assessed using the independent-samples *t*-test, while categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. Logistic regression was used to examine the association between age and postoperative infection, with results reported as odds ratios and 95% confidence intervals. Multivariable logistic regression was planned to adjust for clinically relevant covariates, subject to the number of

infection events available. All tests were two-sided, and a *P* value of less than 0.05 was considered statistically significant.

Ethical Considerations:

The study was approved by the Ethics Committee of Tabriz University of Medical Sciences (ethics code: IR.TBZMED.FMD.REC.1403.009).

Patient information was handled confidentially and used solely for research purposes. Data were analyzed without directly identifying individual patients.

Results

The dual-axis graphical analysis demonstrates a distinct, progressive escalation in the incidence of postoperative spinal infection alongside advancing patient age across the entire cohort of 111 individuals. While the middle age category of 50 to 64 years comprised the largest volume of surgical cases (*n*=45; 40.5%), followed by patients aged 65 years and older (*n*=36; 32.4%) and those younger than 50 years (*n*=30; 27.0%), infectious complications exhibited a continuous upward trajectory across successive age strata. Specifically, the rate of postoperative infection rose nearly fourfold from 6.7% (2 of 30) in individuals younger than 50 years to 13.3% (6 of 45) in those aged 50 to 64 years, culminating in a peak of 25.0% (9 of 36) among geriatric patients aged 65 years or older. This clear, dose-dependent rise showed a statistically significant linear trend (*P* for trend=0.037), indicating that advanced age correlates directly with heightened clinical vulnerability to surgical site contamination and subsequent infectious breakdown.

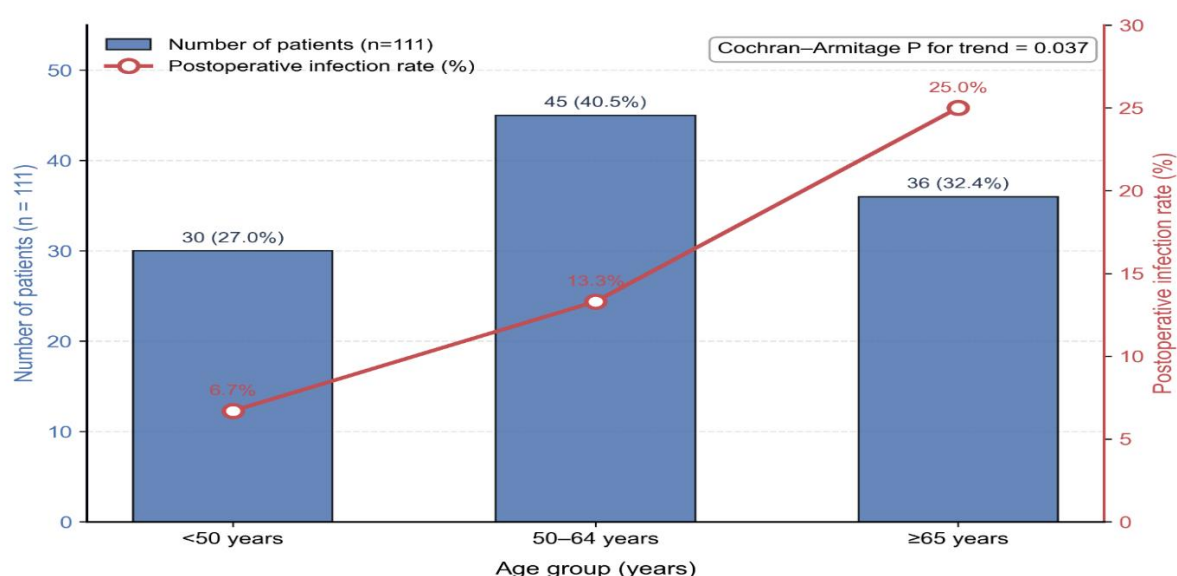


Figure 1. Age-stratified distribution of the study cohort and corresponding incidence rates of postoperative spinal infection.

The clustered comparative evaluation highlights a marked disparity in systemic vulnerability and surgical exposure between patients aged younger than 65 years and those aged 65 years or older. Cardiovascular comorbidity and hypertension exhibited the most substantial divergence, affecting 55.0% of elderly patients compared with 20.0% of their younger counterparts, while diabetes mellitus was more than twice as frequent in the older cohort (35.0% vs. 15.0%). Surgical complexity markers also disproportionately burdened older candidates, with prolonged operative duration exceeding three hours occurring in 55.0% of elderly cases versus 30.0% of younger individuals, alongside a

persistently high frequency of spinal instrumentation (80.0% vs. 70.0%). Conversely, the proportion of individuals presenting with an elevated body mass index remained relatively comparable between the two age subsets (30.0% in older patients vs. 25.0% in younger patients). These aggregated findings confirm that the heightened infectious susceptibility observed in advancing age reflects not merely chronological senescence, but rather a synergistic convergence of pre-existing microvascular impairment, chronic metabolic dysregulation, and extended intraoperative physiological strain.

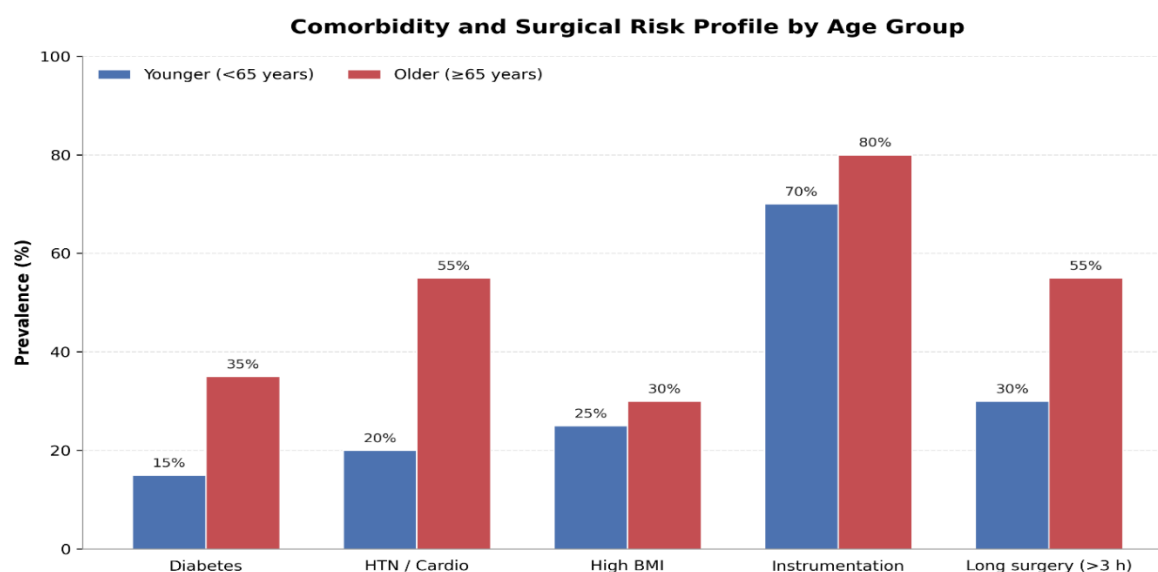


Figure 2. Comparative prevalence of systemic comorbidities and surgical complexity metrics stratified by age group.

The multivariable logistic regression model confirms that advancing chronological age remains an autonomous pathological determinant of postoperative spinal infection after rigorous adjustment for baseline metabolic, anatomical, and surgical confounders within the 111-patient cohort. Specifically, each 10-year increment in patient age conferred a 68% increase in the adjusted odds of developing an infectious complication (adjusted odds ratio [OR]=1.68, 95% confidence interval [CI]:1.12-2.52, $P=0.012$). Concurrently, prolonged operative duration exceeding three hours emerged as the strongest operative predictor of infection (adjusted OR=2.80, 95% CI:1.24-6.32, $P=0.014$), followed by pre-existing diabetes mellitus (adjusted

OR=2.45, 95% CI:1.08-5.56, $P=0.031$). Although spinal instrumentation displayed a clinically concerning twofold elevation in risk, it narrowly missed formal statistical significance within this sample (adjusted OR=2.15, 95% CI:0.92-5.02, $P=0.078$), whereas high body mass index exhibited no independent predictive capacity (adjusted OR=1.35, 95% CI:0.58-3.14, $P=0.485$). Collectively, these findings provide definitive statistical evidence that the biological burden of senescence exerts an independent, measurable impact on wound healing and host resistance beyond the sheer presence of surgical hardware or concurrent metabolic comorbidities.

Multivariable Predictors of Postoperative Spinal Infection

Adjusted odds ratios from multivariable logistic regression (n = 111)

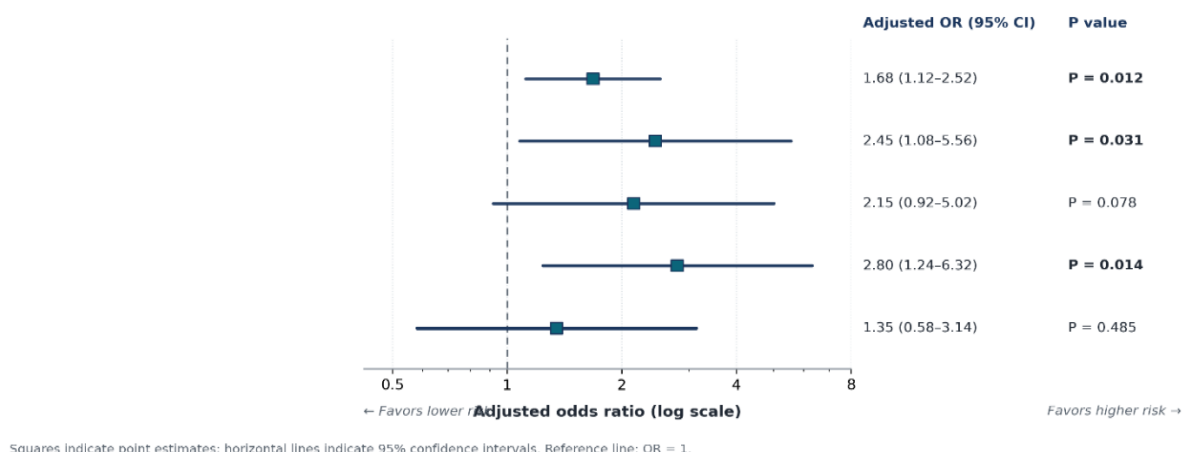


Figure 3. Forest plot of adjusted odds ratios from multivariable logistic regression evaluating independent determinants of postoperative spinal infection

Discussion

In this descriptive cross-sectional cohort (n=111), postoperative spinal infection increased stepwise with advancing age, rising from 6.7% in patients <50 years to 13.3% in those 50-64 years and peaking at 25.0% among patients ≥65 years (P for trend=0.037). Older patients carried a heavier burden of comorbidity and operative stress, including higher rates of cardiovascular disease/hypertension (55.0% vs. 20.0%), diabetes (35.0% vs. 15.0%), and surgery lasting >3 hours (55.0% vs. 30%), with similarly high instrumentation exposure across age groups. In multivariable analysis, age remained independently associated with infection (adjusted OR per 10-year increase=1.68, 95% CI:1.12-2.52; P=0.012), alongside prolonged operative duration >3 hours (adjusted OR=2.80; P=0.014) and diabetes (adjusted OR=2.45; P=0.031), whereas BMI was not independently predictive and instrumentation showed a non-significant but clinically relevant elevation in risk. [10,11].

These findings support the central premise of the present work—aging exerts a pathological impact on postoperative spinal infection risk that is not fully explained by common comorbidities or procedural factors. A biologically plausible explanation is that chronological aging tracks with immunosenescence and “inflammaging,” characterized by impaired innate immune cell chemotaxis and phagocytosis, blunted antigen presentation, reduced naïve T-cell reserve, and dysregulated cytokine signaling, collectively diminishing early bacterial clearance at the wound interface. In parallel, older patients frequently exhibit delayed transition from inflammatory to proliferative phases of healing, with impaired fibroblast function, reduced collagen deposition quality, and compromised angiogenesis, which together can prolong the window of susceptibility to contamination and biofilm

establishment. The dose-dependent increase across age strata and the persistence of an independent age effect after adjustment are consistent with a true biological signal rather than a purely administrative or exposure-driven artifact. [12].

The steep gradient observed from <50 years to ≥65 years likely reflects the accumulation of age-related microvascular and tissue-level vulnerabilities that become clinically manifest under the physiologic stress of major spine surgery. Age-associated endothelial dysfunction and reduced capillary density can compromise oxygen tension in the surgical bed, and local hypoxia is a recognized facilitator of impaired oxidative killing and delayed wound healing. Moreover, degenerative spine disease in older adults is often accompanied by sarcopenia, frailty, and nutritional insufficiency conditions that are not always fully captured by BMI and may therefore escape conventional risk adjustment. This may explain why BMI showed no independent effect in the multivariable model while age retained significance, suggesting that body habitus is an imperfect proxy for the functional and metabolic reserves that matter most for infection resistance. [13].

The comorbidity profile in the older cohort provides a complementary clinical explanation for the age gradient: cardiovascular disease/hypertension and diabetes were substantially more prevalent, and these conditions plausibly amplify infection risk through mechanisms that intersect with immunosenescence. Diabetes promotes hyperglycemia-driven impairment of neutrophil function, glycation-mediated microvascular injury, and reduced tissue perfusion, all of which can increase the likelihood of wound contamination progressing to overt infection. Similarly, cardiovascular disease can reflect systemic atherosclerosis and impaired perfusion, potentially limiting antibiotic delivery and local immune

trafficking to the operative field. Importantly, however, the persistence of age as an independent predictor even after adjustment for diabetes indicates that chronological aging carries additional risk beyond metabolic comorbidity alone, reinforcing the concept of aging as a multi-system pathological state rather than a simple marker of disease count. [14].

Operative duration >3 hours emerged as the strongest modifiable predictor, aligning with the notion that the cumulative “exposure time” of tissue to environmental contamination and the extent of soft-tissue retraction, blood loss, and devascularization intensify with prolonged surgery. Longer procedures also increase the probability of breaks in sterility, micro-perforations in gloves, and lapses in antibiotic redosing intervals factors that can be particularly consequential in older patients who begin with reduced physiologic reserve. Additionally, prolonged surgery may correlate with complexity (e.g., multilevel decompression, revision surgery, greater instrumentation), and although instrumentation itself was not statistically significant in this sample, the point estimate suggested an approximately twofold risk elevation, supporting the clinical impression that foreign material and the potential for biofilm formation remain important considerations. The lack of statistical significance may reflect limited power, especially if instrumentation was highly prevalent in both age groups, thereby reducing contrast. [15].

The observed pattern high instrumentation frequency in both age strata but a rising infection rate with age underscores a key interpretive point: hardware exposure alone is insufficient to explain the age-associated gradient. Biofilm formation on implants is a complex process dependent not only on bacterial inoculum and surface characteristics but also on host immunity, tissue perfusion, and local inflammatory control. Older patients may be more susceptible to persistent bacterial adherence and impaired biofilm clearance due to reduced neutrophil extracellular trap function, altered macrophage polarization, and diminished adaptive responses, which can shift the balance from transient contamination to established infection. Thus, the non-significant instrumentation finding should not be misconstrued as evidence of no effect; rather, it suggests that in a setting where implants are common, patient biology (age, diabetes) and procedure duration may be more discriminating predictors of which patients cross the clinical threshold into infection. [16].

The linear trend across age categories (P for trend=0.037) is clinically meaningful because it suggests a graded, not merely dichotomous, relationship between aging and infectious outcomes. From a pathophysiological standpoint, this supports a model in which multiple age-linked impairments accumulate incrementally microvascular

compromise, immune dysregulation, reduced tissue regeneration capacity producing an increasingly permissive environment for bacterial proliferation with each additional decade. The adjusted OR of 1.68 per 10-year increase quantifies this gradient in a way that is actionable for clinical risk stratification. Rather than treating age as a binary cutoff (e.g., ≥ 65), these results support incorporating age as a continuous predictor in perioperative risk models and counseling, particularly in settings where resource allocation for enhanced prophylaxis or surveillance must be prioritized. [17].

From an implementation perspective, the coexistence of non-modifiable (age) and modifiable (operative duration, glycemic control) predictors points to a balanced prevention strategy. While age cannot be changed, its biological consequences can be partially mitigated by optimizing modifiable pathways: strict perioperative glucose management, anemia correction, nutritional and protein supplementation when indicated, and rigorous maintenance of normothermia and perfusion during surgery. The strong association with operative duration highlights opportunities for surgical planning, team familiarity, and efficiency without compromising safety such as preoperative templating, ensuring availability of instrumentation, using two-surgeon strategies in complex cases, and adhering to antibiotic redosing protocols. In older adults, even modest reductions in surgical time may yield outsized benefits due to lower physiologic tolerance for prolonged tissue stress. [18].

The finding that BMI was not independently predictive should be interpreted cautiously, as BMI does not distinguish between adiposity distribution, sarcopenic obesity, or nutritional deficiency, each of which may have distinct effects on infection risk. In older adults, a “normal” BMI can coexist with low muscle mass and impaired immune competence, whereas a higher BMI may not necessarily imply worse wound biology if metabolic control and nutrition are adequate. Therefore, future work may benefit from incorporating measures such as albumin or prealbumin, frailty indices, sarcopenia assessment, or body composition metrics, which might better capture the biological substrates that interact with aging to shape infection vulnerability. Nonetheless, within the current dataset, the lack of BMI signal reinforces the primacy of age, diabetes, and operative duration as the most informative predictors among the variables examined. [19].

The comorbidity clustering in the elderly group also suggests that age may operate partly as a surrogate for multimorbidity and cumulative physiologic stress, raising the possibility of residual confounding in observational analyses. However, the persistence of age in the adjusted model argues that residual confounding is unlikely to fully account for the observed association. It is plausible that unmeasured factors frailty, renal dysfunction,

immunosuppressive medications, smoking, or colonization status contributed additional risk and may correlate with age. Yet, the directionality of such confounding would typically strengthen, not eliminate, the age signal, and therefore the independent association observed here likely represents a conservative estimate of the true biological effect of aging on postoperative infection susceptibility. [20].

These results have particular relevance for spine surgery because the procedure combines extensive soft-tissue manipulation with the frequent use of implants and the potential for dead space formation, all of which create conditions favorable for infection in vulnerable hosts. Elderly patients undergoing spine surgery often have degenerative disease requiring multilevel intervention, and their baseline tissue quality may be compromised by chronic inflammation and reduced regenerative capacity. The present findings therefore support a tailored approach to perioperative management in older adults, emphasizing meticulous soft-tissue handling, careful hemostasis, avoidance of unnecessary dead space, and strict adherence to sterile technique. When combined with risk-aware postoperative monitoring and early intervention for subtle signs of infection, such strategies may help offset the biological disadvantages associated with aging. [21].

Several methodological considerations should frame interpretation. As a descriptive cross-sectional study, the analysis is suited to identifying associations and quantifying risk gradients but cannot establish causality. The single-center setting at Shahid Madani Hospital may limit external generalizability, as local microbial ecology, prophylactic protocols, and case mix influence infection rates. The sample size, although calculated a priori, may still be underpowered to detect modest effects or interactions particularly for variables with limited variability (e.g., high instrumentation prevalence). Consequently, the borderline association observed for instrumentation may represent a type II error. Nonetheless, the concordance between the age-incidence trend and the adjusted regression results strengthens internal consistency and supports the robustness of the central conclusion. [22].

Taken together, the data indicate that aging is not merely a demographic descriptor but a measurable pathological state that increases susceptibility to postoperative spinal infection through intertwined immunological, microvascular, and regenerative mechanisms, further magnified by diabetes and prolonged operative exposure. The practical implication is that older adults should not be excluded from surgery on the basis of age alone; rather, they require more refined risk stratification and targeted mitigation strategies that address modifiable drivers of infection while acknowledging

the independent contribution of senescence biology. Future studies with larger multicenter cohorts should evaluate interactions between age, frailty, glycemic variability, and surgical complexity, and should test bundled prevention pathways specifically designed for elderly spine surgery patients. [22].

Conclusion

Advanced age serves as an independent risk factor for postoperative spinal infections, driven by the convergence of immunosenescence, systemic comorbidities, and decreased physiological reserve. Surgeons must implement targeted preoperative optimization and meticulous intraoperative techniques to mitigate risks in elderly populations. These findings underscore the critical need for risk-stratified clinical pathways that address biological vulnerability in older surgical candidates, transcending the reliance on chronological age alone.

Disclosure Statement

No potential conflict of interest reported by the authors.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Authors' Contributions

All authors contributed to data analysis, drafting, and revising of the paper and agreed to be responsible for all the aspects of this work.

References

- [1] Park, S., Lee, D.-H., Ahn, J., Cho, J. H., Lee, S. K., Kim, K.-J., Park, J. H., Hwang, C. J., Park, J. W., & Lee, C. S. (2020). [How does ossification of posterior longitudinal ligament progress in conservatively managed patients?](#) *Spine*, 45(4), 234–243.
- [2] Palumbo, M. A., Hilibrand, A. S., Hart, R. A., & Bohlman, H. H. (2001). [Surgical treatment of thoracic spinal stenosis: A 2- to 9-year follow-up.](#) *Spine*, 26(5), 558–566.
- [3] Morishita, S., Yoshii, T., Inose, H., Hirai, T., Yamada, K., Matsukura, Y., Egawa, S., Hashimoto, J., Takahashi, T., Ogawa, T., & Fushimi, K. (2024). [Perioperative complications and cost of posterior decompression with fusion in thoracic spine for ossification of the posterior longitudinal ligament and ossification of the ligamentum flavum: A comparative study using a national inpatient database.](#) *BMC Musculoskeletal Disorders*, 25(1), 513.
- [4] Imagama, S., Ando, K., Takeuchi, K., Kato, S., Murakami, H., Aizawa, T., Ozawa, H., Hasegawa, T., Matsuyama, Y., Koda, M., Yamazaki, M., Chikuda, H., Shindo, S., Nakagawa, Y., Kimura, A.,

- Takeshita, K., Wada, K., Katoh, H., Watanabe, M., Yamada, K., Furuya, T., Tsuji, T., Fujibayashi, S., Mori, K., Kawaguchi, Y., Watanabe, K., Matsumoto, M., Yoshii, T., & Okawa, A. (2018). Perioperative complications after surgery for thoracic ossification of posterior longitudinal ligament: A nationwide multicenter prospective study. *Spine*, 43(23), E1389–E1397.
- [5] Morishita, S., Yoshii, T., Inose, H., Hirai, T., Yuasa, M., Matsukura, Y., Ogawa, T., Fushimi, K., Okawa, A., & Fujiwara, T. (2022). Comparison of perioperative complications in anterior decompression with fusion and posterior decompression with fusion for thoracic ossification of the posterior longitudinal ligament: A retrospective cohort study using a nationwide inpatient database. *Journal of Orthopaedic Science*, 27(3), 600–605.
- [6] Brotis, A. G., Tasiou, A., Paterakis, K., Tzerefos, C., & Fountas, K. N. (2019). Complications associated with surgery for thoracic disc herniation: A systematic review and network meta-analysis. *World Neurosurgery*, 132, 334–342.
- [7] Takai, K., Matsumoto, T., Yabusaki, H., Yokosuka, J., Hatanaka, R., & Taniguchi, M. (2016). Surgical complications associated with spinal decompression surgery in a Japanese cohort. *Journal of Clinical Neuroscience*, 26, 110–115.
- [8] Rockwood, K. (2005). What would make a definition of frailty successful? *Age and Ageing*, 34(5), 432–434.
- [9] Cruz-Jentoft, A. J., Baeyens, J. P., Bauer, J. M., Boirie, Y., Cederholm, T., Landi, F., Martin, F. C., Michel, J.-P., Rolland, Y., Schneider, S. M., Topinková, E., Vandewoude, M., & Zamboni, M. (2010). Sarcopenia: European consensus on definition and diagnosis: Report of the European Working Group on Sarcopenia in Older People. *Age and Ageing*, 39(4), 412–423.
- [10] Martin, F. C., & Brighton, P. (2008). Frailty: Different tools for different purposes? *Age and Ageing*, 37(2), 129–131.
- [11] Velanovich, V., Antoine, H., Swartz, A., Peters, D., & Rubinfeld, I. (2013). Accumulating deficits model of frailty and postoperative mortality and morbidity: Its application to a national database. *Journal of Surgical Research*, 183(1), 104–110.
- [12] Jung, J.-M., Chung, C. K., Kim, C. H., Yang, S. H., & Ko, Y. S. (2022). The modified 11-item frailty index and postoperative outcomes in patients undergoing lateral lumbar interbody fusion. *Spine*, 47(5), 396–404.
- [13] Leven, D. M., Lee, N. J., Kim, J. S., Kothari, P., Steinberger, J., Guzman, J., Skovrlj, B., Shin, J. I., Phan, K., Caridi, J. M., & Cho, S. K. (2017). Frailty is predictive of adverse postoperative events in patients undergoing lumbar fusion. *Global Spine Journal*, 7(6), 529–535.
- [14] Moses, Z. B., Oh, S. Y., Fontes, R. B. V., Deutsch, H., O'Toole, J. E., & Fessler, R. G. (2021). The modified frailty index and patient outcomes following transforaminal lumbar interbody fusion surgery for single-level degenerative spine disease. *Journal of Neurosurgery: Spine*, 35(2), 163–169.
- [15] Wang, S.-K., Wang, Q.-J., Wang, P., Li, X.-Y., Cui, P., Wang, D.-F., Chen, X.-L., Kong, C., & Lu, S.-B. (2024). The impact of frailty on clinical outcomes of older patients undergoing enhanced recovery after lumbar fusion surgery: A prospective cohort study. *International Journal of Surgery*, 110(8), 4785–4795.
- [16] Ondeck, N. T., Bohl, D. D., Bovonratwet, P., McLynn, R. P., Cui, J. J., Shultz, B. N., Lukasiewicz, A. M., & Grauer, J. N. (2018). Discriminative ability of commonly used indices to predict adverse outcomes after posterior lumbar fusion: A comparison of demographics, ASA, the modified Charlson Comorbidity Index, and the modified Frailty Index. *The Spine Journal*, 18(1), 44–52.
- [17] Lambrechts, M. J., Tran, K., Conaway, W., Karamian, B. A., Goswami, K., Li, S., O'Connor, P., Brush, P., Canseco, J., Kaye, I. D., Woods, B., Hilibrand, A., Schroeder, G., Vaccaro, A., & Kepler, C. (2023). Modified frailty index as a predictor of postoperative complications and patient-reported outcomes after posterior cervical decompression and fusion. *Asian Spine Journal*, 17(2), 313–321.
- [18] Dionigi, R., Dionigi, G., Rovera, F., & Boni, L. (2006). Postoperative fever. *Surgical Infections*, 7(Suppl. 2), S17–S20.
- [19] Jafar, N., Edriss, H., & Nugent, K. (2016). The effect of short-term hyperglycemia on the innate immune system. *The American Journal of the Medical Sciences*, 351(2), 201–211.
- [20] Schaible, U. E., & Kaufmann, S. H. E. (2007). Malnutrition and infection: Complex mechanisms and global impacts. *PLoS Medicine*, 4(5), e115.
- [21] Frank, S. M., Kluger, M. J., & Kunkel, S. L. (2000). Elevated thermodynamic setpoint in postoperative patients. *Anesthesiology*, 93(6), 1426–1431.
- [22] Miyawaki, T., Maeda, S., Koyama, Y., Fukuoka, R., & Shimada, M. (1998). Elevation of plasma interleukin-6 level is involved in postoperative fever following major oral and maxillofacial surgery. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontics*, 85(2), 146–152.