

Risk factors, diagnosis, and management of postoperative spinal surgical site infections: a retrospective analysis of 36 cases

Postoperatif spinal cerrahi alan enfeksiyonlarında risk faktörleri, tanı ve tedavi yöntemleri: 36 vakanın retrospektif analizi

Serkan Civlan, Nevzat Doğukan Erbek, İlker Kiraz, Emrah Egemen, Mehmet Erdal Coşkun

Posted date:20.05.2025

Acceptance date:30.06.2025

Abstract

Purpose: Postoperative spinal surgical site infections (SSIs) significantly impact patient morbidity, healthcare costs, and recovery. This study aimed to identify risk factors and diagnostic indicators and discuss the treatment modalities for spinal SSI through retrospective analysis.

Materials and methods: Data from 36 patients with spinal SSI, operated between January 2014 and January 2018, were retrospectively evaluated. Patient-related and surgery-related risk factors, clinical presentations, laboratory findings, microbiological cultures, radiological imaging, and treatment outcomes were analysed.

Results: Among 2,596 spinal surgeries, 36 SSIs (1.39%) occurred. Common risk factors included hypertension, diabetes, coronary artery disease, immunosuppression, and smoking. Early-onset infections (<30 days; 72.2%) presented primarily with wound discharge; late-onset infections (>30 days; 27.8%) were characterised by persistent pain. CRP and neutrophil-to-lymphocyte ratio (NLR) were found to be important diagnostic markers. Culture positivity was higher in early-onset infections; *Escherichia coli* was the predominant pathogen. Deep infections required longer antibiotic therapy and hospital stay ($p<0.05$). Multidisciplinary management—including surgical debridement, irrigation, vacuum-assisted closure, and hyperbaric oxygen therapy—was essential. Despite aggressive treatment, five patients succumbed to complications.

Conclusion: Meticulous risk assessment, early diagnosis using NLR and CRP, culture-guided antibiotic therapy, and individualised multidisciplinary strategies remain crucial for managing spinal SSIs effectively.

Keywords: Spinal instrumentation, surgical site infection, spine surgery, neutrophil-to-lymphocyte ratio (NLR).

Civlan S, Dogukan Erbek N, Kiraz I, Egemen E, Coskun ME. Risk factors, diagnosis, and management of postoperative spinal surgical site infections: a retrospective analysis of 36 cases. Pam Med J 2025;18:771-782.

Öz

Amaç: Postoperatif spinal cerrahi alan enfeksiyonları (CAE), hasta morbiditesini, sağlık maliyetlerini ve iyileşmeyi ciddi oranda etkiler. Bu çalışmada spinal CAE'nin risk faktörleri, tanısal göstergeleri ve tedavi yöntemlerinin retrospektif olarak değerlendirilmesi amaçlandı.

Gereç ve yöntem: Ocak 2014 – Ocak 2018 tarihleri arasında spinal CAE gelişen 36 hasta retrospektif olarak incelendi. Hasta ve cerrahi kaynaklı risk faktörleri, klinik bulgular, laboratuvar değerleri, mikrobiyolojik kültürler, radyolojik görüntülemeler ve tedavi sonuçları analiz edildi.

Bulgular: Toplam 2.596 spinal cerrahi vakasında 36 CAE (%1,39) görüldü. En sık hipertansiyon, diyabet, koroner arter hastalığı, immünosupresyon ve sigara kullanımı risk faktörü olarak belirlendi. Erken dönemde gelişen enfeksiyonlar (<30 gün; %72,2) çoğunlukla yara akıntısıyla, geç dönemde gelişen enfeksiyonlar (>30 gün; %27,8) ise inatçı ağrı ile seyir gösterdi. Tanıda C-reaktif protein (CRP) ve nötrofil/lenfosit oranı (NLR) tanısal değeri yüksek bir bulgu olarak saptandı. Kültür pozitifliği erken enfeksiyonlarda daha yüksekti; en sık izole edilen patojen *Escherichia coli* idi. Derin enfeksiyonlu hastalarda antibiyotik tedavisi ve hastanede kalış süreleri daha uzundu ($p<0,05$). Cerrahi debridman, irrigasyon, vakum yardımcı kapama ve hiperbarik oksijen terapisi gibi multidisipliner yaklaşımlar gerekiyordu. Agresif tedaviye rağmen 5 hasta komplikasyonlara bağlı kaybedildi.

Sonuç: Detaylı risk değerlendirmesi, NLR ve CRP ile erken tanı, kültüre dayalı antibiyoterapi ve bireyselleştirilmiş multidisipliner yaklaşımlar spinal CAE yönetiminde kritik önem taşımaktadır.

Serkan Civlan, Asst. Prof. Pamukkale University Faculty of Medicine, Department of Neurosurgery, Denizli, Türkiye, e-mail: serkancivlan@hotmail.com (https://orcid.org/0000-0001-8915-8186) (Corresponding Author)

Nevzat Doğukan Erbek, M.D. Pamukkale University Faculty of Medicine, Department of Neurosurgery, Denizli, Türkiye, e-mail: drnevatdogukanerbek@gmail.com (https://orcid.org/0009-0001-1995-5611)

İlker Kiraz, Asst. Prof. Pamukkale University Faculty of Medicine, Department of Neurosurgery, Denizli, Türkiye, e-mail: ikiraz@pau.edu.tr (https://orcid.org/0000-0002-8393-9886)

Emrah Egemen, Assoc. Prof. Pamukkale University Faculty of Medicine, Department of Neurosurgery, Denizli, Türkiye, e-mail: eegemen@pau.edu.tr (https://orcid.org/0000-0003-4930-4577)

Mehmet Erdal Coşkun, Prof. Pamukkale University Faculty of Medicine, Department of Neurosurgery, Denizli, Türkiye, e-mail: ercoskun@yahoo.com (https://orcid.org/0000-0002-2816-0722)

Anahtar kelimeler: Spinal enstrümantasyon, cerrahi alan enfeksiyonu, omurga cerrahisi, nötrofil-lenfosit oranı (NLR).

Civlan S, Doğukan Erbek N, Kiraz İ, Egemen E, Coşkun ME. Postoperatif spinal cerrahi alan enfeksiyonlarında risk faktörleri, tanı ve tedavi yöntemleri: 36 vakanın retrospektif analizi. Pam Tıp Derg 2025;18:771-782.

Introduction

Postoperative spinal surgical site infections (SSI) are serious complications causing substantial morbidity, prolonged hospitalisation, increased healthcare costs, and disability risk. The rising prevalence correlates with ageing populations, surgical complexity, prolonged operative times, frequent use of instrumentation, and higher comorbidities [1-3]. Predisposing patient-related factors include diabetes mellitus, obesity, immunosuppression, malnutrition, smoking, and chronic illnesses [4, 5]. Surgically, prolonged procedures, extensive dissection, significant blood loss, revision surgeries, and instrumentation elevate infection risks [2, 4, 6].

Infections present either early (<30 days) with wound discharge, erythema, and pain or late (>30 days), featuring persistent deep pain, delayed healing, or sinus tract formation [7]. Diagnosis integrates clinical evaluation, elevated C-reactive protein (CRP), erythrocyte sedimentation rate (ESR) levels, neutrophil-to-lymphocyte ratio (NLR), and magnetic resonance imaging (MRI) [8-10]. While pathogen identification through microbiological cultures guides the treatment, culture-negative results are common and often necessitate empirical therapy [11, 12].

Management involves multidisciplinary strategies: superficial infections benefit from antibiotics and wound care; deep infections require surgical debridement, prolonged antimicrobials, surgical implant management, and adjunct therapies like vacuum-assisted closure or hyperbaric oxygen [13-17]. Prevention through strict aseptic protocols, antibiotic prophylaxis, and meticulous surgical techniques remains paramount. This retrospective study aimed to analyse 36 patients regarding risk factors, diagnostic indicators, and treatment strategies and may contribute to the limited body of literature evaluating the NLR as a potential early diagnostic marker in spinal surgical site infections.

Materials and methods

This retrospective study analysed 36 patients diagnosed with spinal SSI between January 2014 and January 2018. Patients with a minimum follow-up duration of 12 months were included in the study. Detailed medical histories were obtained, and risk factors were evaluated. Patient-related risk factors included age, gender, obesity, smoking, diabetes mellitus, hypertension, coronary artery disease, chronic obstructive pulmonary disease, rheumatoid arthritis, ankylosing spondylitis, chronic renal failure, osteoporosis, steroid use, and immunosuppression. Surgery-related risk factors included the duration of surgery, whether the surgery was a revision procedure, the number of surgical sessions, intraoperative or postoperative blood transfusion, and utilising instrumentation. The presence of these risk factors was quantified. Postoperative symptoms and findings suggestive of infection—onset time, pain, fever, and wound discharge—were recorded.

All patients' laboratory parameters—white blood cell (WBC) count, ESR, CRP, neutrophil count, lymphocyte count, and the NLR—were evaluated before the initial operation, at the time of infection onset, at discharge, and during outpatient follow-up. These parameters were then compared between early-onset (<30 days) and late-onset (>30 days) infections. All cultures (blood, puncture, and surgical tissue specimens) were examined via Gram staining and aerobic, anaerobic, fungal, and acid-fast bacillus (AFB) microbiological assessments. Based on culture results, the causative pathogens were analysed separately for early-onset and late-onset infections.

Contrast-enhanced MRI scans were reviewed to categorise infections as paraspinal/superficial soft tissue or deep tissue infections (e.g., osteomyelitis, discitis, epidural abscess). Based on the patient's symptoms, laboratory findings, and radiological evaluations, debridement,

pressurised irrigation, closed irrigation-drainage system, instrumentation removal, flap coverage in patients whose surgical wounds could not be primarily closed, vacuum-assisted closure (VAC) therapy, hyperbaric oxygen therapy, and ultrasound-guided abscess drainage were applied in various combinations, depending on clinical necessity.

Written informed consent was obtained from all patients. The study was conducted according to the ethical standards of the Declaration of Helsinki. Approval was granted by the Non-Interventional Clinical Research Ethics Committee of Pamukkale University (approval date: 28.05.2024 and approval number: 10). The intravenous (IV) and subsequent oral treatment durations were analysed and compared between patients with deep tissue infection and those with only superficial soft tissue infection.

Statistical analyses

All statistical analyses were performed using SPSS software (version 22.0, Chicago, USA). Continuous variables were expressed as mean \pm standard deviation (SD), while categorical variables were presented as frequencies and percentages. No sample size calculation was performed before the study to determine the number of patients to be included; instead, all patients from the specified groups over the four years were included. For group comparisons, non-parametric tests (Mann–Whitney U and Kruskal–Wallis H) were applied to continuous data, while categorical variables were analysed using the Pearson chi-square test. A two-tailed p -value <0.05 was considered statistically significant.

Results

Of the 2,596 patients who underwent spinal surgery, 36 cases of spinal SSI were identified, corresponding to an incidence rate of 1.39%. The minimum follow-up duration was 12 months, with a mean follow-up of 29.7 ± 10.8 months. Among these 36 patients, 19 (52.8%) were female and 17 (47.2%) were male. The

mean body mass index (BMI) was 27.89 ± 4.82 kg/m², and the mean age was 57.50 ± 15.58 years. Hypertension was present in 12 patients (33.3%), coronary artery disease in 10 (27.8%), and diabetes mellitus in 9 (25%); additionally, 9 patients (25%) were smokers and 10 (27.8%) were immunosuppressed. Twenty-three patients (63.8%) had two or more risk factors, whereas 9 (25%) had no identifiable ones. From the perspective of perioperative risk factors, patients were divided into three groups:

Group A (n=10): Procedures without instrumentation/minimally invasive surgeries.

Group B (n=20): Decompressive procedures/stabilisation surgeries (with or without instrumentation) for degenerative or traumatic etiologies.

Group C (n=6): Tumour excision/corpectomy.

Table 1 below summarises perioperative risk factors across the three groups, including the number of operated levels, session details, revision rates, instrumentation usage, blood transfusion requirements, dural tear incidences, and mean operative times. The overall figures across all groups include 16 revision procedures (44%), 13 cases requiring blood transfusion (36%), and six instances of dural tears (16.7%). Increasing surgical complexity is associated with longer operative times, higher transfusion rates, and greater incidence of dural injuries.

Early-onset (<30 days) infections were seen in 26 patients (72.2%), and late-onset (>30 days) infections were seen in 10 patients (27.8%). The mean onset time in early-onset infections was 14.38 ± 11.07 days, compared to 102.5 ± 99.81 days in late-onset infections. The mean onset time in all patients was 38.86 ± 65.20 days. Presenting symptoms included discharge (70%), pain (50%), and fever (24%). Among those with early-onset infections, 85% reported wound discharge, followed by pain (38%) and fever (26%). In late-onset infections, 80% presented with pain and 30% with discharge; none of these late-onset cases had a fever.

Table 1. Perioperative risk factors in patients with iatrogenic spinal infections

Variables	Group A (n=10)	Group B (n=20)	Group C (n=6)	p	Total (n=36)
Instrumentation (Yes/No)	0/10	18/2	5/1	$X^2=24.586$ $p=0.001^{**}$	23 (64%) / 13 (36%)
Levels Operated (Mean±SD)	1.70±0.67	4.70±2.03	4.33±1.21	H=17.219 $p=0.001^*$ (Group A)	3.81±2.08
Sessions (1/2/3)	10/0/0	18/2/0	2/3/1	$X^2=14.480$ $p=0.006^{**}$	30/5/1
Revisions (2/3/4)	2/1/0	4/3/2	3/1/0	$X^2=2.153$ $p=0.708^{**}$	9/5/2
Blood Units Transfused (0/1/2/3/>3 units)	8/0/2/0/0	14/1/1/1/3	1/1/0/1/3	$X^2=14.517$ $p=0.069^{**}$	23/2/3/2/6
Mean Operative Time (min)	153.75±23.87	290±87.49	415±170.15	H=11.245 $p=0.004^*$ (Group A)	276±137.22
Dural Tear (Yes/No)	0/10 (100%)	5/15 (25%/75%)	1/5 (16%/84%)	$X^2=3.000$ $p=0.223^{**}$	6/30 (16%/84%)

H=Kruskal wallis H test, χ^2 =Pearson chi-square test

WBC levels were not significantly elevated at any time point. CRP levels were elevated in all cases. CRP levels were already elevated preoperatively in early-onset infections compared to late-onset infections. CRP levels decreased significantly by discharge and at follow-up. ESR levels were elevated in all early-onset infections, though CRP levels declined more rapidly and noticeably than ESR during the treatment process. The neutrophil/lymphocyte ratio was considerably higher, particularly in early-onset infections. The NLR in early-onset infections reached a markedly supranormal mean of 7.74 ± 5.13 . It also returned to its reference range sooner than the surgery-related rise in CRP and was within normal limits at both discharge and end-of-therapy follow-up for all patients. Pre- and post-treatment laboratory results are summarised in Table 2.

Blood cultures, puncture cultures, and surgical tissue cultures were compared between early-onset and late-onset infection groups. Blood cultures were taken in 12 (33.3%)

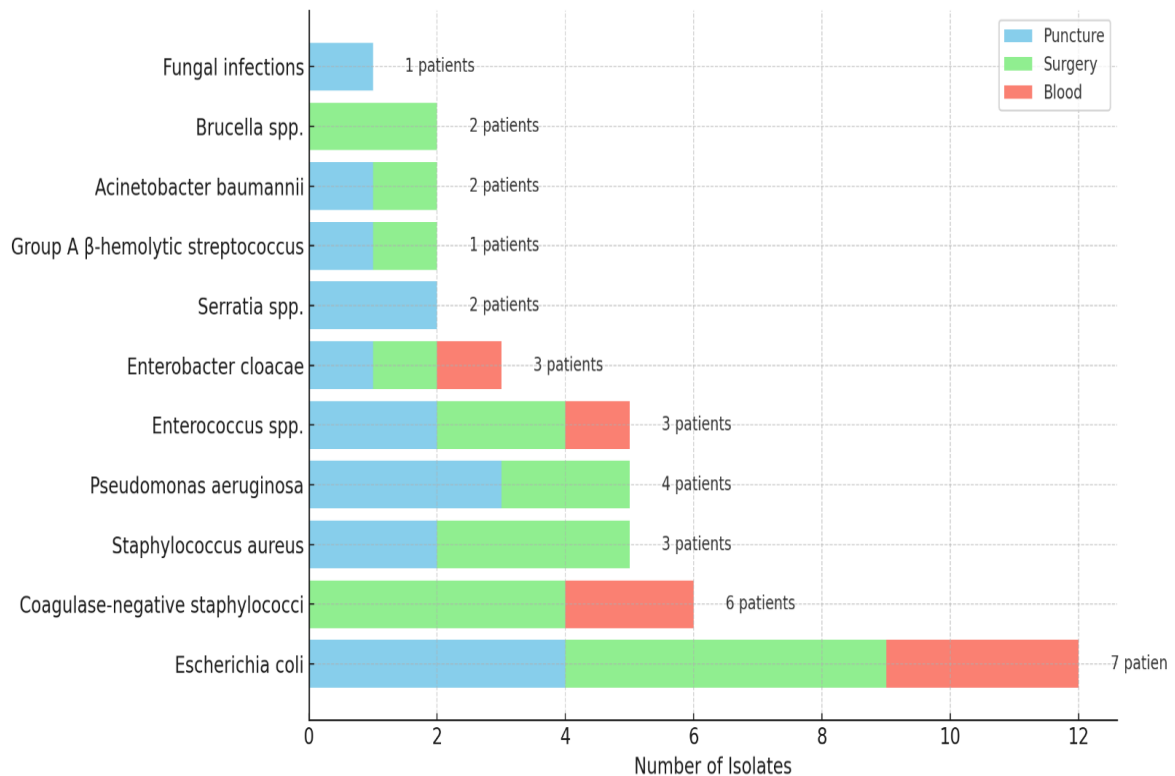
patients, 10 with early-onset infections and 2 with late-onset infections; overall, 50% of these cultures were positive. No growth was observed in blood cultures of the late-onset group, whereas 60% were positive in the early-onset group. Among 20 patients with puncture cultures, 70% demonstrated growth (72% in early-onset infections). Surgical tissue cultures obtained from 36 patients showed a 50% positivity rate. When examined per patient, the overall positivity of at least one culture type was 69.4%.

Some cultures yielded multiple pathogens. Table 3 summarizes the corresponding microbiological findings. The pathogen distribution reflects individual isolates rather than patient counts. Overall, the most frequently identified pathogens were *Escherichia coli* (12 isolates), coagulase-negative staphylococci (6 isolates), *Staphylococcus aureus* (5 isolates), *Pseudomonas aeruginosa* (5 isolates), and *Enterococcus spp.* (5 isolates).

Table 2. Pre- and post-treatment laboratory results

Variables (Reference Range)	Timepoint	Early (n=26)	Late (n=10)	z	p	Overall (n=36)
WBC (4–10 K/uL)	Pre-op	8.02±2.85	9.21±2.68	1.14	0.283	8.21±2.81
	Infection Onset	10.56±4.08	8.24±2.17	1.41	0.166	9.92±3.77
	Discharge	7.21±2.97	6.16±1.67	0.84	0.419	6.93±2.70
	Follow-up	5.96±1.34	7.27±1.82	1.78	0.075	6.51±1.66
CRP (<0.5 mg/dL)	Pre-op	1.42±1.31	0.25±0.21	2.00	0.045	1.22±1.27
	Infection Onset	9.56±8.12	6.19±5.76	1.09	0.274	8.62±7.61
	Discharge	2.84±2.06	1.64±1.08	1.56	0.120	2.47±1.88
	Follow-up	0.81±1.14	0.39±0.24	0.12	0.901	0.64±0.89
ESR (<20 mm/h)	Pre-op	-	-	-	-	-
	Infection Onset	83.76±32.45	71.14±28.38	0.98	0.325	80.08±31.26
	Discharge	71.63±33.87	62.75±22.56	0.80	0.426	68.67±30.35
	Follow-up	24.75±25.19	31.71±18.84	0.69	0.487	28.00±21.96
NLR	Pre-op	2.61±1.15	3.16±2.52	0.24	0.811	2.66±1.24
	Infection Onset	7.74±5.13	3.05±1.00	3.67	0.001	6.44±4.86
	Discharge	3.93±3.98	1.71±0.43	2.19	0.029	3.35±3.55
	Follow-up	2.88±2.05	1.84±0.31	1.36	0.172	2.50±1.71

z: Mann Whitney U test

Table 3. Pathogen distribution by isolate and patient counts


MRI was performed in 29 patients, revealing superficial soft tissue infection in 28/29 (96%), osteomyelitis in 10/29 (34%), epidural abscess in 7/29 (24%), and psoas abscess in 4/29 (14%). Treatment modalities were combined according to clinical and radiological findings. All patients underwent debridement of necrotic tissues. At the end of surgery, a closed irrigation-drainage system was placed in 20 patients (56%). Instrumentation was removed in 3 patients with suspected screw loosening. Myocutaneous flap coverage was necessary for three patients whose surgical wounds could not be primarily closed. VAC therapy was applied in 3 patients with extensive dead space in the surgical area. The incorporation of hyperbaric oxygen therapy was limited to two patients who demonstrated persistent deep infection despite adequate debridement and culture-directed antibiotics, chosen on a case-by-case basis, particularly when tissue hypoxia was suspected. Four patients with imaging-confirmed psoas abscesses underwent ultrasound-guided catheter drainage.

Among patients with deep tissue infection, the mean duration of IV antibiotic therapy was 38.50 ± 26.93 days, postoperative oral antibiotic therapy 97.78 ± 42.65 days, and hospital stay 67.10 ± 61.82 days. In patients with only superficial soft tissue infection, IV antibiotic therapy lasted 17.84 ± 8.12 days, postoperative oral antibiotic therapy 17.31 ± 16.59 days, and hospital stay 28.41 ± 10.49 days. Comparisons between the deep infection group and the superficial infection group revealed statistically significant differences in IV therapy duration ($p=0.004$), postoperative oral antibiotic duration ($p=0.0001$), and length of hospital stay ($p=0.040$).

The mean duration of the index surgery leading to infection was 253.15 ± 181.02 minutes in patients with only superficial soft tissue involvement, compared to 120 ± 141.42 minutes in those with deep infection. This difference was statistically significant ($p=0.040$).

Discussion

Spinal surgery is frequently performed for a wide range of conditions, most commonly lumbar disc herniation, as well as spinal stenosis, deformity, trauma, and spinal infections

[18]. Spinal SSIs remain among the most serious complications following spinal surgery, significantly increasing patient morbidity, healthcare costs and the length of hospital stay. The reported incidence ranges from 0.7% to 16%, depending on patient comorbidities, surgical complexity and perioperative protocols [1-3]. The heterogeneity in incidence is mainly attributable to the diversity of patient-related and surgery-related risk factors.

When preoperative (patient-related) risk factors are examined, the decreased vascularity and immune response of adipose tissue in obese patients leads to fat necrosis, providing a favourable milieu for bacterial proliferation and increasing the risk of infection after surgery [19]. The literature identifies body mass index (BMI) as a risk factor for iatrogenic spinal infections. Abdallah et al. [20] reported that every 5 kg m² increase in BMI was associated with a 13% increase in the risk of spinal SSI.

Lin et al. [21] demonstrated that in long-term smokers, nicotine and other toxic substances attack the fatty acids in cell membranes, producing lipid peroxidation products that directly or indirectly delay tissue healing and increase infection risk. Although there is no consensus in the literature that smoking increases SSI risk, Schimmel et al. [22] reported that the risk of deep infection doubled in smokers [23, 24]. Subsequent studies have supported this relationship.

In patients with diabetes mellitus, hyperglycaemia exerts inhibitory effects on the vascular and immune systems, impairing wound healing in surgical tissues and elevating infection risk [25]. Approximately 17% of diabetic patients develop postoperative complications, two-thirds of which are infectious [15]. Moreover, a preoperative serum glucose level >125 mg/dL (>6.9 mmol/L) or a postoperative level >200 mg/dL has been shown to increase the risk of spinal SSI even in patients without diabetes mellitus [5]. Cardiovascular disease, hypertension and renal failure also compromise soft-tissue viability and increase infection risk [25]. Other preoperative risk factors identified in the literature include steroid use, alcohol consumption, immunosuppression and advanced age.

When all 36 patients with postoperative SSI were assessed for preoperative risk factors, hypertension (n=12) was most prevalent, followed by coronary artery disease (n=10), immunosuppression (n=10), smoking (n=9) and diabetes mellitus (n=9). Additionally, eight patients were immunosuppressed, and seven had rheumatoid arthritis. Fourteen patients had three or more risk factors, whereas nine had none. Overall, our findings were concordant with the published literature. The mean BMI of patients was calculated as 27.89; no significant association was found between BMI and SSI risk. This result stems from the limited sample size and the narrow distribution of BMI values among patients.

Procedure type is a significant determinant among perioperative (surgery-related) risk factors. In a study of 108,419 cases published in 2011, instrumented procedures were associated with a 28% higher SSI risk. For fusion surgeries, purely anterior approaches carried the lowest risk. The same study linked minimally invasive techniques to lower SSI rates, whereas revision cases exhibited a 65% higher infection rate than primary surgeries. At the spinal level, thoracic procedures had the highest and cervical the lowest SSI risk [26].

Prolonged operative times can induce tissue ischaemia and hypoxia, increasing the risk of infection. Several studies have reported that lengthy procedures are associated with higher postoperative infection rates in spinal surgery. Generally, a duration of ≥ 2 h is significantly associated with increased SSI risk, and each additional hour beyond two hours further elevates this risk [27-29].

The literature also indicates that excessive intraoperative blood loss increases postoperative SSI risk. Olsen et al. [5] reported significantly greater mean blood loss in patients with SSI compared to those without (275 ml vs. 150 ml). Another study found that blood loss >1000 mL during spinal surgery increased SSI risk [2].

In our study, the mean operative time was 273.0 ± 127.0 minutes; notably, the mean duration in all three patient groups exceeded 2.5 hours and was even longer in complex cases, supporting the reported association between prolonged surgery and SSI. Forty-four per cent

of the procedures were revision surgeries, 36% of patients required blood transfusions, and 64% of cases involved multilevel interventions. These findings are consistent with perioperative risk factors previously reported in the literature. We believe minimising operative duration, reducing intraoperative blood loss, and avoiding unnecessary multilevel surgeries through appropriate surgical indications may reduce the risk of spinal SSIs.

Fang et al. [4] reported that spinal SSIs most often occur within the first three months after surgery. SSIs may be categorised as early (≤ 30 days) and late (> 30 days) according to time of onset [12, 30]. Early infections usually manifest with severe pain, wound drainage, erythema, swelling and mild fever. Late infections are more insidious and more complicated to diagnose, typically presenting with back pain or tenderness on deep palpation. In our series, early infections (≤ 30 days) emerged at a mean of 14.38 ± 11.07 days, late infections (> 30 days) at 102.5 ± 99.81 days, and the overall mean onset time was 38.86 ± 65.20 days, in agreement with the literature.

The most used laboratory tests for diagnosing and monitoring SSIs are WBC count, CRP and ESR. Because WBC elevation occurs in fewer than 50% of cases, its sensitivity is inferior to other markers [31]. In our study, WBC values were only slightly above the upper limit of normal ($10.56 \pm 4.08 \times 10^3/\mu\text{L}$) in early infections and remained within normal limits at other time points, corroborating its limited utility.

Although CRP and ESR are sensitive indicators, both rise physiologically after surgery. CRP peaks on postoperative days 2-3 and usually returns to baseline by days 5-21; a second CRP peak raises suspicion of infection [10]. ESR peaks on postoperative days 3-5 and may remain elevated for 3-6 weeks. Because CRP normalises earlier, it is more sensitive than ESR for SSI detection [32, 33]. Khan et al. [9] observed that CRP declined rapidly in response to intravenous antibiotics, whereas ESR normalised more slowly, underscoring CRP's superiority for monitoring treatment response. In our series, mean CRP levels were significantly elevated in early (9.56 ± 8.12 mg/dL) and late (6.19 ± 5.76 mg/dL) infections and normalised after therapy; ESR declined but less markedly. Thus, our data support CRP's greater

utility. Notably, preoperative CRP was already elevated (1.42 ± 1.31 mg/dL) in early-infection patients, suggesting that high preoperative CRP may predict SSI, consistent with recent literature [34-36].

Following bacterial invasion, neutrophils rise while lymphocytes fall. Takahashi et al. [37] reported that lymphocyte counts dropped after surgery in infection-free patients but began to rise after day 4, returning to normal within three weeks. In contrast, lymphocyte proportions fell below 10% in infected patients until postoperative day 11. The NLR has been widely studied in inflammatory and infectious diseases over the past decade [38, 39]. To our knowledge, Shen et al. [40] conducted the first study on NLR in spinal SSIs in 2019, demonstrating that NLR values >5.19 on postoperative day 4 and >3.85 on day 7 were associated with SSI after instrumented spinal surgery. Inose et al. [41] subsequently showed that NLR >4.91 on postoperative days 3-4 and >3.21 on days 6-7 predicted SSI after spinal decompression. Later studies confirmed that early postoperative NLR elevation correlates with SSI risk and that combining NLR with CRP or lymphocyte percentage improves predictive accuracy [42, 43]. In our cohort, the mean NLR in early infections was markedly elevated (7.74 ± 5.13) and returned to normal at discharge and after treatment, reinforcing NLR's diagnostic and monitoring value. Because the NLR normalised sooner than the surgery-related rise in CRP, we believe it enhances diagnostic sensitivity by distinguishing early-onset infections from the physiological postoperative inflammatory response.

Although blood cultures and aspiration biopsies aid diagnosis, the gold standard is intraoperative tissue culture [31, 44]. Jiménez Mejías et al. [45] found positive blood cultures in 55% of spinal infection cases. Another study reported that, although wound cultures were positive in all 16 patients, blood cultures were positive in only six [46]. Culture negativity rates vary from 16% to 80% across series; however, extending incubation can yield positive cultures in up to 90% of cases [11, 12, 47]. Early spinal SSIs (≤ 30 days) are most commonly caused by *Staphylococcus aureus*, including methicillin-sensitive and methicillin-resistant strains, reflecting direct inoculation of skin flora.

Enterobacterales, such as *Escherichia coli* and *Enterobacter cloacae*, are also commonly observed, particularly in lumbar and sacral surgeries. Late infections (>3 months, typically after instrumented fusion) are dominated by low-virulence, biofilm-forming organisms, principally coagulase-negative staphylococci and *Cutibacterium acnes* [8, 47, 48].

In our series, positive blood cultures were obtained in 50% of patients, aspiration cultures in 70%, and intraoperative cultures in 50%. The lower rate of intraoperative growth than aspiration growth is attributed to the antibiotics administered between diagnosis and surgery. When early and late infections were compared, aspiration and blood culture positivity were higher in early cases, whereas intraoperative positivity was identical (50%) in both groups. Overall, culture positivity was 76.92% in early and 60% in late infections. The most commonly isolated pathogens were *Escherichia coli* (12 isolates), coagulase-negative staphylococci (6 isolates), *Staphylococcus aureus* (5 isolates), *Pseudomonas aeruginosa* (5 isolates), and *Enterococcus spp.* (5 isolates), while other organisms were detected less frequently. Notably, and in contrast to most published series where gram-positive organisms are more predominant, *E. coli* emerged as the leading pathogen in our cohort based on both isolate and patient counts.

Early and aggressive management is essential for the successful treatment of spinal SSIs [14]. While superficial infections may respond to antibiotic therapy alone, persistent cases often require adjunctive drainage. In contrast, deep infections frequently necessitate surgical intervention, including thorough debridement, irrigation, and closed drainage systems. Empirical broad-spectrum antibiotics should be initiated after obtaining tissue samples and adjusted based on culture results [14]. The optimal duration of antibiotic therapy typically involves 4–6 weeks of intravenous treatment followed by tailored oral regimens.

The management of instrumentation-associated infections remains controversial. Several studies suggest retaining implants in early-onset infections to prevent instability due to lack of solid fusion, whereas late-onset infections, complicated by biofilm formation, often necessitate hardware removal [7, 10].

Adjunctive techniques, including closed suction irrigation systems, VAC therapy, myocutaneous flap coverage, and hyperbaric oxygen therapy, have been reported as effective strategies for managing complex spinal SSIs [3, 15, 18, 49, 50]. These approaches are particularly beneficial in cases with extensive soft tissue defects, persistent infections, or when primary wound closure is not feasible. Several studies have demonstrated that VAC therapy and flap reconstruction can enhance wound healing, while closed irrigation systems aid in reducing bacterial load [15, 49]. Hyperbaric oxygen therapy has also been shown to improve infection control, potentially avoiding the need for implant removal in selected cases [16].

In our series of 36 patients, comprehensive surgical management—including staged debridement, pressurised irrigation, and appropriate use of drainage systems—proved effective. Closed irrigation systems were applied in 20 patients, VAC therapy in 3 cases, and myocutaneous flaps in another three patients with significant tissue loss. Instrumentation was removed in 3 of 23 patients due to mechanical loosening. Hyperbaric oxygen therapy was employed in 2 patients. Despite aggressive management, five patients succumbed to sepsis and multiorgan failure.

Patients with deep infections required significantly longer intravenous (38.5 ± 26.9 days) and oral antibiotic therapy (97.8 ± 42.6 days) than those with superficial involvement. Interestingly, prolonged operative time was associated with superficial soft tissue infections, likely due to ischemic effects from prolonged retraction. This finding underscores the importance of intermittent release of retractors during extended procedures to preserve tissue perfusion and minimise infection risk.

Overall, our multidisciplinary, tailored approach was consistent with existing literature and highlights the necessity of individualised treatment strategies based on infection depth, timing, and implant status to optimise clinical outcomes in postoperative spinal infections.

This study is subject to several significant limitations. Most notably, its retrospective design inherently restricts the ability to establish

causality and increases susceptibility to selection and information biases. Additionally, the absence of a control group prevents comparative analysis against non-infected or differently managed cohorts, limiting the generalizability of observed associations and treatment outcomes. The relatively small sample size and single-centre nature of the investigation may also reduce statistical power and external validity.

In conclusion, spinal SSIs significantly impact surgical success, functional recovery, and healthcare costs. Our analysis highlights that meticulous risk assessment—from preoperative optimisation to intraoperative vigilance—is critical for prevention. The neutrophil-to-lymphocyte ratio has emerged as a sensitive marker for detecting early infections and should be routinely monitored. *Escherichia coli* emerged as the most commonly identified pathogen in our cohort. Rapid initiation of culture-guided antimicrobial therapy, complemented by timely surgical intervention, remains essential once infection is suspected. Ultimately, systematic risk management, early diagnostic recognition, and coordinated multidisciplinary care represent fundamental strategies for reducing the incidence and severity of spinal SSIs.

Funding: None.

Authors' contributions to the article

S.C. constructed the main idea and hypothesis of the study and developed the concept and design. S.C. primarily attended surgical procedures, collected the data, performed analysis and interpretation, conducted the literature review, and wrote the original thesis. N.D.E., E.E., and I.K. reviewed the most recent literature. S.C. and N.D.E. wrote the manuscript based on the original thesis and incorporated recent literature. I.K. and E.E. critically reviewed and corrected the manuscript. E.C. supervised the study, attended surgical procedures, and performed the final critical revision. In addition, all authors discussed the entire study and approved the final version.

Conflict of interest: No conflict of interest was declared by the authors.

References

1. Pull ter Gunne AF, Mohamed AS, Skolasky RL, van Laarhoven CJ, Cohen DB. The presentation, incidence, etiology, and treatment of surgical site infections after spinal surgery. *Spine (Phila Pa 1976)*. 2010;35(13):1323-1328. doi:10.1097/BRS.0b013e3181bcde61
2. Wimmer C, Gluch H, Franzreb M, Ogon M. Predisposing factors for infection in spine surgery: a survey of 850 spinal procedures. *J Spinal Disord*. 1998;11(2):124-128.
3. Singh K, Heller JG. Postoperative spinal infections. *Contemporary Spine Surgery*. 2005;6(9):61-68. doi:10.1097/01075922-200509000-00001
4. Fang A, Hu SS, Endres N, Bradford DS. Risk factors for infection after spinal surgery. *Spine (Phila Pa 1976)*. 2005;30(12):1460-1465. doi:10.1097/01.brs.0000166532.58227.4f
5. Olsen MA, Nepple JJ, Riew KD, et al. Risk factors for surgical site infection following orthopaedic spinal operations. *J Bone Joint Surg Am*. 2008;90(1):62-69. doi:10.2106/JBJS.F.01515
6. Simchen E, Stein H, Sacks TG, Shapiro M, Michel J. Multivariate analysis of determinants of postoperative wound infection in orthopaedic patients. *J Hosp Infect*. 1984;5(2):137-146. doi:10.1016/0195-6701(84)90117-8
7. Sierra Hoffman M, Jinadatha C, Carpenter JL, Rahm M. Postoperative instrumented spine infections: a retrospective review. *South Med J*. 2010;103(1):25-30. doi:10.1097/SMJ.0b013e3181c4e00b
8. Stambough JL, Beringer D. Postoperative wound infections complicating adult spine surgery. *J Spinal Disord*. 1992;5(3):277-285. doi:10.1097/00002517-199209000-00005
9. Khan MH, Smith PN, Rao N, Donaldson WF. Serum C-reactive protein levels correlate with clinical response in patients treated with antibiotics for wound infections after spinal surgery. *Spine J*. 2006;6(3):311-315. doi:10.1016/j.spinee.2005.07.006
10. Mok JM, Guillaume TJ, Talu U, et al. Clinical outcome of deep wound infection after instrumented posterior spinal fusion: a matched cohort analysis. *Spine (Phila Pa 1976)*. 2009;34(6):578-583. doi:10.1097/BRS.0b013e31819a827c
11. Muschik M, Lück W, Schlenszka D. Implant removal for late-developing infection after instrumented posterior spinal fusion for scoliosis: reinstrumentation reduces loss of correction. A retrospective analysis of 45 cases. *Eur Spine J*. 2004;13(7):645-651. doi:10.1007/s00586-004-0694-4
12. Kowalski TJ, Berbari EF, Huddleston PM, Steckelberg JM, Mandrekar JN, Osmon DR. The management and outcome of spinal implant infections: contemporary retrospective cohort study. *Clin Infect Dis*. 2007;44(7):913-920. doi:10.1086/512194
13. Hegde V, Meredith DS, Kepler CK, Huang RC. Management of postoperative spinal infections. *World J Orthop*. 2012;3(11):182-189. doi:10.5312/wjo.v3.i11.182
14. Finn M, Schmidt M. Postoperative infections of the spine. Youmans Neurological Surgery Sixth Edition Philadelphia, PA: Saunders. 2011;570:e577.
15. Pappalardo G, Schneider S, Kotsias A, Jeyaraman M, Schäfer L, Migliorini F. Negative pressure wound therapy in the management of postoperative spinal wound infections: a systematic review. *Eur J Orthop Surg Traumatol*. 2024;34(5):2303-2313. doi:10.1007/s00590-024-03983-x
16. Larsson A, Engström M, Uusijärvi J, Kihlström L, Lind F, Mathiesen T. Hyperbaric oxygen treatment of postoperative neurosurgical infections. *Neurosurgery*. 2002;50(2):287-296.
17. Bavinzski G, Schoeggel A, Trattinig S, et al. Microsurgical management of postoperative disc space infection. *Neurosurg Rev*. 2003;26(2):102-107. doi:10.1007/s10143-002-0241-x
18. Keskin E, Açıkgöz B, Kalaycı M, et al. Lomber disk cerrahisinde insizyon büyüklüğünün ameliyat sonrası paraspinal adale iyileşmesine etkisi. *Med J West Black Sea*. 2020;4(2):71-77. doi:10.29058/mjwbs.2020.2.5
19. Abbey DM, Turner DM, Warson JS, Wirt TC, Scalley RD. Treatment of postoperative wound infections following spinal fusion with instrumentation. *J Spinal Disord*. 1995;8(4):278-283. doi:10.1097/00002517-199508040-00003
20. Abdallah DY, Jadaan MM, McCabe JP. Body mass index and risk of surgical site infection following spine surgery: a meta-analysis. *Eur Spine J*. 2013;22(12):2800-2809. doi:10.1007/s00586-013-2890-6
21. Lin AH, Liu MH, Ko HB, Perng DW, Lee TS, Kou YR. Inflammatory effects of menthol vs non-menthol cigarette smoke extract on human lung epithelial cells: a double-hit on TRPM8 by reactive oxygen species and menthol. *Front Physiol*. 2017;8:263. doi:10.3389/fphys.2017.00263
22. Schimmel JJ, Horsting PP, de Kleuver M, Wonders G, van Limbeek J. Risk factors for deep surgical site infections after spinal fusion. *Eur Spine J*. 2010;19(10):1711-1719. doi:10.1007/s00586-010-1421-y
23. Meng F, Cao J, Meng X. Risk factors for surgical site infections following spinal surgery. *J Clin Neurosci*. 2015;22(12):1862-1866. doi:10.1016/j.jocn.2015.03.065

24. Kong L, Liu Z, Meng F, Shen Y. Smoking and Risk of Surgical Site Infection after Spinal Surgery: A Systematic Review and Meta-Analysis. *Surg Infect (Larchmt)*. 2017;18(2):206-214. doi:10.1089/sur.2016.209
25. Deng H, Chan AK, Ammanuel S, et al. Risk factors for deep surgical site infection following thoracolumbar spinal surgery. *J Neurosurg Spine*. 2019;32(2):292-301. doi:10.3171/2019.8.SPINE19479
26. Smith JS, Shaffrey CI, Sansur CA, et al. Rates of infection after spine surgery based on 108,419 procedures: a report from the Scoliosis Research Society Morbidity and Mortality Committee. *Spine (Phila Pa 1976)*. 2011;36(7):556-563. doi:10.1097/BRS.0b013e3181eadd41
27. Valentini LG, Casali C, Chatenoud L, Chiaffarino F, Uberti Foppa C, Broggi G. Surgical site infections after elective neurosurgery: a survey of 1747 patients. *Neurosurgery*. 2008;62(1):88-96. doi:10.1227/01.NEU.0000311065.95496.C5
28. Zhang L, Li EN. Risk factors for surgical site infection following lumbar spinal surgery: a meta-analysis. *Ther Clin Risk Manag*. 2018;14:2161-2169. Published 2018 Oct 31. doi:10.2147/TCRM.S181477
29. Kim BD, Hsu WK, De Oliveira GS Jr, Saha S, Kim JY. Operative duration as an independent risk factor for postoperative complications in single-level lumbar fusion: an analysis of 4588 surgical cases. *Spine (Phila Pa 1976)*. 2014;39(6):510-520. doi:10.1097/BRS.0000000000000163
30. Köder K, Hardt S, Gellert MS, et al. Outcome of spinal implant-associated infections treated with or without biofilm-active antibiotics: results from a 10-year cohort study. *Infection*. 2020;48(4):559-568. doi:10.1007/s15010-020-01435-2
31. Dowdell J, Brochin R, Kim J, et al. Postoperative Spine Infection: Diagnosis and Management. *Global Spine J*. 2018;8(4 Suppl):37S-43S. doi:10.1177/2192568217745512
32. Kunakornsawat S, Tungsiripat R, Putthiwara D, et al. Postoperative Kinetics of C-Reactive Protein and Erythrocyte Sediment Rate in One-, Two-, and Multilevel Posterior Spinal Decompressions and Instrumentations. *Global Spine J*. 2017;7(5):448-451. doi:10.1177/2192568217699389
33. Takahashi J, Ebara S, Kamimura M, et al. Early-phase enhanced inflammatory reaction after spinal instrumentation surgery. *Spine (Phila Pa 1976)*. 2001;26(15):1698-1704. doi:10.1097/00007632-200108010-00014
34. Zhang Y, Zhong G, Fan K, He J, Sun Y, Li L. Preoperative C-reactive Protein and Other Inflammatory Biomarkers as Predictors of Postoperative Complications in Colorectal Tumor Patients. *Altern Ther Health Med*. 2024;30(8):152-157.
35. Inoue D, Kabata T, Kajino Y, et al. Do elevated preoperative serum inflammatory markers influence surgical site or periprosthetic joint infections following primary total hip arthroplasty?. *J Orthop Sci*. doi:10.1016/j.jos.2024.11.003
36. Tang S, Gong W, Han X, Han S, Zhang H, Lian Z. Predictive value of the preoperative C-reactive protein-to-albumin ratio for surgical site infection after percutaneous kyphoplasty: a single-center retrospective study. *Front Cell Infect Microbiol*. 2025;15:1565468. doi:10.3389/fcimb.2025.1565468
37. Takahashi J, Shono Y, Hirabayashi H, et al. Usefulness of white blood cell differential for early diagnosis of surgical wound infection following spinal instrumentation surgery. *Spine (Phila Pa 1976)*. 2006;31(9):1020-1025. doi:10.1097/01.brs.0000214895.67956.60
38. de Jager CP, Wever PC, Gemen EF, et al. The neutrophil-lymphocyte count ratio in patients with community-acquired pneumonia. *PLoS One*. 2012;7(10):e46561. doi:10.1371/journal.pone.0046561
39. Narci A, Tuncer AA, Cetinkursun S. Diagnostic importance of neutrophil/lymphocyte ratio in childhood appendicitis. *Med J Kocatepe*. 2009;10:5-7.
40. Shen CJ, Miao T, Wang ZF, et al. Predictive value of post-operative neutrophil/lymphocyte count ratio for surgical site infection in patients following posterior lumbar spinal surgery. *Int Immunopharmacol*. 2019;74:105705. doi:10.1016/j.intimp.2019.105705
41. Inose H, Kobayashi Y, Yuasa M, Hirai T, Yoshii T, Okawa A. Postoperative lymphocyte percentage and neutrophil-lymphocyte ratio are useful markers for the early prediction of surgical site infection in spinal decompression surgery. *J Orthop Surg (Hong Kong)*. 2020;28(2):2309499020918402. doi:10.1177/2309499020918402
42. Salimi M, Mosalamiaghili S, Mafhoumi A, Riaz M. The neutrophil-to-lymphocyte ratio (NLR) levels predicting the surgical site infection in spinal surgery: a systematic review. *J Spine Surg*. 2025;11(1):135-147. doi:10.21037/jss-24-106
43. Peng Z, Jia Y, Li J, Wang G. Diagnostic Value of Neutrophil-Lymphocyte Ratio in Predicting Post-Operative Infection after Orthopedic Surgery: A Systematic Review and Meta-Analysis. *Surg Infect (Larchmt)*. 2024;25(7):527-537. doi:10.1089/sur.2024.002
44. Lazennec JY, Fourniols E, Lenoir T, et al. Infections in the operated spine: update on risk management and therapeutic strategies. *Orthop Traumatol Surg Res*. 2011;97(6 Suppl):S107-S116. doi:10.1016/j.otsr.2011.07.002
45. Jiménez Mejías ME, de Dios Colmenero J, Sánchez-Lora FJ, et al. Postoperative spondylodiskitis: etiology, clinical findings, prognosis, and comparison with nonoperative pyogenic spondylodiskitis. *Clin Infect Dis*. 1999;29(2):339-345. doi:10.1086/520212

46. Dobran M, Marini A, Gladi M, et al. Deep spinal infection in instrumented spinal surgery: diagnostic factors and therapy. *G Chir.* 2017;38(3):124-129. doi:10.11138/gchir/2017.38.3.124
47. Clark CE, Shufflebarger HL. Late-developing infection in instrumented idiopathic scoliosis. *Spine (Phila Pa 1976).* 1999;24(18):1909-1912. doi:10.1097/00007632-199909150-00008
48. Alfin DJ, Shilong DJ, Bot GM, Dengunu Salun W. Surgical site infection rate in spine surgery, incidence, and risk factors: a ten-year retrospective cohort review in a developing neurosurgical centre. *BMC Surg.* 2025;25(1):127. doi:10.1186/s12893-025-02846-4
49. Levi AD, Dickman CA, Sonntag VK. Management of postoperative infections after spinal instrumentation. *J Neurosurg.* 1997;86(6):975-980. doi:10.3171/jns.1997.86.6.0975
50. Mitra A, Mitra A, Harlin S. Treatment of massive thoracolumbar wounds and vertebral osteomyelitis following scoliosis surgery. *Plast Reconstr Surg.* 2004;113(1):206-213. doi:10.1097/01.PRS.0000097440.15013.5C