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Vacuum Sealing Drainage for Deep Surgical Site Infection Following Unilateral Biportal Endoscopic Spine Surgery: A Case Report and Literature Review

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Case Report

Keywords: unilateral biportal endoscopy, surgical site infection, vacuum sealing drainage, debridement, case report

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Abstract

Background:

Unilateral biportal endoscopy (UBE) features small incisions, relatively limited soft tissue injury and flexible visual field. However, postoperative deep surgical site infection may lead to persistent exudation, paravertebral or intraspinal infection, repeated debridement and prolonged hospitalization. Vacuum sealing drainage (VSD), a form of negative pressure wound therapy (NPWT), has been applied to infected wounds after complex spinal surgery. Nevertheless, dedicated reports on its use for deep infection after UBE remain scarce.

Case Summary:

An 80-year-old female patient underwent percutaneous unilateral biportal endoscopic lumbar discectomy at L4/5 for lumbar disc herniation with spinal stenosis. High fever occurred on postoperative day 6, with a maximum temperature of 41°C. Laboratory examinations showed procalcitonin (PCT) 0.45 ng/mL, C-reactive protein (CRP) 30.10 mg/L, serum amyloid A (SAA) > 1000.00 mg/L, interleukin-6 (IL-6) 14.30 pg/mL, and erythrocyte sedimentation rate (ESR) 23 mm/h. Contrast-enhanced lumbar MRI revealed marked edema in the left surgical access area at L4/5 and adjacent paravertebral soft tissues, with irregular fluid collection and abnormal enhancement, suggesting postoperative deep soft tissue infection. Enterobacter hormaechei was isolated from drainage fluid. After sufficient debridement and pathogen sampling, continuous VSD at − 125 mmHg was applied. The VSD dressing was replaced twice over 14 days, combined with cefoperazone-sulbactam sodium. After treatment, the incision healed well. Follow-up laboratory tests demonstrated ESR 19 mm/h, white blood cell count 5.13×10^9/L, and CRP 1.97 mg/L. Follow-up contrast-enhanced lumbar MRI showed marked regression of edema, fluid collection and abnormal enhancement at the L4/5 operative site and surrounding paravertebral soft tissues, indicating substantial improvement.

Conclusion:

This case suggests that for carefully evaluated deep surgical site infection after UBE, VSD can serve as an adjunct to adequate debridement, reliable pathogen sampling and targeted antimicrobial therapy, providing continuous drainage, dead-space management and wound-bed preparation. A single case cannot confirm that VSD is superior to other drainage or debridement modalities. Further case series and controlled studies are required to clarify its indications, optimal negative-pressure parameters and long-term outcomes.

1. Introduction

Unilateral biportal endoscopy (UBE) enables decompression, discectomy or fusion procedures via an observation portal and a working portal, and its application in degenerative lumbar spine disorders has gradually increased in recent years[1–3].UBE relies on continuous fluid irrigation and involves two separate incisions; its minimally invasive nature cannot completely eliminate the risk of surgical site infection (SSI). Large-scale studies on surgical complications have documented cases of infection following UBE. In 2025, another study specifically analyzed the causes and management of SSI after UBE for lumbar disc herniation. Two cases of deep SSI caused by Staphylococcus aureus after UBE published in 2026 further indicated that inadequate preoperative skin preparation, intraoperative extravasation of irrigation fluid, prolonged operative time and non-standard postoperative wound dressing changes may contribute to infection development[4–7].

The reported incidence of infection after unilateral biportal endoscopy (UBE) varies across published studies. One complication study including 4685 patients documented a small number of infectious events, whereas another French multicenter case series of 1566 patients undergoing lumbar UBE identified no surgical site infection (SSI) within a 90-day follow-up period [6, 7].Such discrepancies may be attributed to differences in case selection, surgical procedures, perioperative prophylactic measures, definitions of infection, and follow-up protocols. Once deep infection develops, timely delineation of the infection extension, acquisition of valid deep tissue specimens, thorough debridement of necrotic tissue, and administration of pathogen-directed antimicrobial therapy constitute the core components of the therapeutic algorithm.

Continuous negative pressure suction with VSD can serve as an adjuvant technique for the management of infected wounds after complex spinal surgery by continuously removing exudate, reducing local dead space, maintaining a relatively sealed wound environment, and promoting granulation tissue formation [8, 9].Previous systematic reviews, case series and comparative studies have demonstrated that VSD can be applied for deep surgical site infection following spinal surgery, including selected patients with retained implants. However, available evidence is predominantly derived from retrospective studies, with substantial heterogeneity in treatment protocols and negative

pressure parameters [10–15].Within the preliminary search scope of the present work, case reports focusing specifically on VSD therapy for deep SSI after UBE with complete documentation of the clinical timeline and technical details remain scarce. Herein, we present a case treated with VSD following limited debridement via the original UBE portals, and discuss the appropriate role, technical considerations and limitations of VSD in the management of UBE-related surgical site infection in light of current literature.

2. Case Presentation

2.1 Initial Admission

An 80-year-old female patient was admitted on March 8, 2026, presenting with a half-year history of low back pain combined with left lower extremity pain. She underwent thoracic vertebroplasty in 2023 and had no chronic medical conditions such as hypertension, diabetes mellitus or coronary heart disease. Admission physical examination revealed marked tenderness over the spinous processes and interspinous spaces of the lower lumbar spine. The muscle strength of left great toe dorsiflexion was grade 3, with diminished cutaneous sensation in the left lower extremity. The preoperative Visual Analogue Scale (VAS) score was 9, and the Oswestry Disability Index (ODI) was 82%. Preoperative imaging demonstrated L4/5 disc herniation predominantly at the left posterolateral (left paramedian) region, left lateral recess stenosis with compression of the left nerve root, degenerative lumbar spinal stenosis at L4/5, and multilevel degenerative changes of the lumbar spine (Figure 1). Integrating clinical symptoms, physical signs and imaging findings, the diagnoses were established as: 1. Lumbar disc herniation; 2. Lumbar spinal stenosis; 3. Severe osteoporosis. After exclusion of surgical contraindications and acquisition of informed consent, percutaneous unilateral biportal endoscopic lumbar discectomy with spinal canal decompression was performed at the L4/5 level under general anesthesia on March 10, 2026.

2.2 Infection Onset and Readmission

On postoperative day 6, the patient developed fever, chills, incision erythema, and low back pain. The peak body temperature reached 41°C. Wound examination revealed poor wound healing with drainage of pale red fluid. Repeat blood tests showed white blood cell (WBC) count 5.35×10^9/L, neutrophil percentage 59.0%, C-reactive protein (CRP) 30.10 mg/L, erythrocyte sedimentation rate (ESR) 23 mm/h, and procalcitonin (PCT) 0.45 ng/mL.

2.3 Diagnostic Evaluation

Contrast-enhanced MRI revealed marked edema in the left surgical access zone at L4/5 and adjacent paravertebral soft tissues, accompanied by irregular fluid collection and abnormal enhancement, consistent with postoperative deep soft tissue infection. Based on clinical manifestations, inflammatory markers and imaging findings, deep surgical site infection was initially suspected.

Drainage fluid was collected from the original endoscopic incision prior to antimicrobial adjustment. Culture yielded Enterobacter hormaechei. Antimicrobial susceptibility testing demonstrated susceptibility to cefoperazone/sulbactam, piperacillin/tazobactam, ceftazidime, cefepime, aztreonam, ertapenem, imipenem, amikacin, ciprofloxacin and trimethoprim-sulfamethoxazole, whereas resistance to ceftriaxone, gentamicin and tobramycin was observed.

2.4 Debridement and VSD Therapy

Given the presence of deep infection and persistent wound drainage in this patient, posterior lumbar debridement combined with vacuum sealing drainage was performed on March 23, 2026. Intraoperative findings revealed absent left lamina at L4/5, hyperplastic inflammatory granulomatous tissue within scar tissue, and a small amount of thin pale-red fluid outside the lamina. Specimens of pale-red fluid were collected for bacterial, fungal and other essential examinations. Inflammatory granulation tissue was thoroughly debrided, and the wound cavity was repeatedly irrigated with copious normal saline.

The VSD foam was trimmed according to the contour of the wound cavity and placed beneath the left fascia at L4/5, deep to the paraspinal muscles and within the deep cavity formed after laminar decompression. The foam fully covered the wound surface and obliterated local dead space, while direct compression on the thecal sac and nerve roots was avoided. After sealing the wound with semipermeable membrane, the foam was connected to a negative pressure system with continuous suction set at −125 mmHg.

Subsequently, VSD dressings were replaced twice on March 30 and April 7, 2026, with wound assessment performed at each dressing change. Once the patient achieved normalization of body temperature, erythrocyte sedimentation rate and CRP levels, without obvious necrotic tissue and with viable granulation tissue developing at the wound margins, secondary wound closure was conducted on April 14, 2026 (Figure 2).

2.5 Antimicrobial and Comprehensive Management

Empiric antimicrobial therapy was initiated with penicillin G sodium. According to culture and susceptibility results, the regimen was switched to piperacillin-tazobactam sodium on March 19, 2026, administered intravenously for 5 weeks, followed by oral antibiotic therapy. Discontinuation of antibiotics was guided by inflammatory markers including CRP and ESR. Nutritional support, analgesia, thromboprophylaxis and other supportive measures were provided concurrently.

2.6 Outcomes and Follow-up

The patient's body temperature returned to normal on March 26, 2026 after treatment. Laboratory tests before discharge revealed that CRP decreased from a peak value of 30.10 mg/L to 1.97 mg/L, and ESR declined from 23 mm/h to 19 mm/h (Figure 3). The patient was discharged on April 24, 2026, with a total hospital stay of 36 days. At discharge, the VAS score for back pain was 3 points, the VAS score for leg pain was 2 points, and the ODI was 20%.

The patient was followed up by telephone at 1 and 2 months after discharge. During these follow-ups, she reported a satisfactory general condition without fever, chills, wound erythema, dehiscence, or abnormal drainage. Her low back and lower-extremity symptoms continued to improve, and no infection-related rehospitalization or additional surgical intervention was required. At 3 months after discharge, the patient returned for further clinical and radiological evaluation. The surgical incision had healed well. Follow-up lumbar MRI demonstrated a marked reduction in abnormal signal changes at the L4/5 operative site and adjacent paravertebral soft tissues compared with the acute infectious phase, without obvious progressive fluid accumulation or definite abscess formation, indicating substantial improvement of the postoperative infectious changes(Figure 4). Taken together, the serial clinical follow-up and 3-month MRI findings suggested stable recovery, with no definite evidence of clinical or radiological recurrence of infection during the follow-up period.

The entire treatment course for the patient over the two hospital admissions is shown in Figure 5.

Written informed consent was obtained from the patient for publication of this case report and all accompanying anonymized clinical and imaging data. All identifiable information has been removed to protect patient privacy.

2.7 Timeline of the Case

3. Literature Review Results

Although the existing literature concerning UBE-related infection remains limited, this field is no longer completely unexplored.Yang et al. [6]documented a small number of infectious cases in their complication analysis of 4685 patients undergoing UBE, and described managements including endoscopic debridement of infectious lesions and antimicrobial therapy.Zhang et al.[4]performed an analysis of risk factors and treatments for SSI after UBE for lumbar disc herniation in 2025. Hong et al. [5]reported two cases of deep SSI caused by Staphylococcus aureus following UBE in 2026. Both patients underwent repeat endoscopic irrigation and debridement combined with targeted antimicrobial therapy. That study focused on infectious risk factors and perioperative prevention rather than VSD. A French multicenter case series published by Sellier et al. [7]in the same period involving 1566 patients receiving lumbar UBE detected no SSI within 90 days, indicating considerable inter-center variability in infection incidence.

In contrast, a relatively large body of literature addresses VSD/NPWT for deep surgical site infection after spinal surgery. Early systematic reviews highlighted the promising potential of NPWT for spinal wounds; however, available evidence was mainly derived from low-quality studies, and its exact mechanisms and optimal protocols remained undetermined[10].Previous case series demonstrated that NPWT could be used in combination with repeated debridement, antimicrobial therapy and delayed wound closure[11–13].Wang et al. [14]compared NPWT with conventional debridement protocols in patients with deep SSI after posterior spinal surgery with instrumentation and reported differences regarding implant retention and infection recurrence. Nevertheless, their findings cannot be directly extrapolated to patients with UBE-associated infection. A single-arm meta-analysis published in 2025 supported the clinical application of NPWT for deep SSI after spinal surgery, yet the included studies were limited by non-randomized design, small sample sizes and substantial treatment heterogeneity [15].

Taken together, published case reports of UBE-related infection predominantly adopt repeat endoscopic debridement combined with antimicrobial therapy, while studies investigating VSD for spinal infection mainly focus on complex deep wounds after conventional open surgery or instrumented spinal fusion. Differing from those reports, the present case describes deep SSI after UBE successfully treated with VSD as a core adjuvant drainage strategy (Table 1).

Table 1
Comparison between representative published literature and the present case

Author/Year	Study type	Original surgery / Population	Infection profile	Primary treatment	Use of VSD/NPWT	Main outcomes	Relevance to the present case
Zhang et al., 2025[4]	Retrospective study	UBE for lumbar disc herniation	Analysis of causes and management of SSI	Open debridement and drainage combined with antibiotics	No	Analysis of risk factors and treatment	Key background literature regarding infection after UBE
Hong et al., 2026[5]	Case report (2 cases)	UBE L4/5 decompression / discectomy	Deep SSI caused by Staphylococcus aureus	Repeat endoscopic debridement plus targeted antimicrobial agents	No	Wound healing; optimization of preventive protocols	Case report most similar to the present UBE infection case
Yang et al., 2024[6]	Large-scale complication study	4685 patients undergoing UBE	A small number of infectious cases	Endoscopic debridement of infected lesions combined with antimicrobial therapy	Not specified	Outcomes of complication management	Demonstrates that UBE-related infection is rare yet clinically existent
Sellier et al., 2026[7]	Multicenter retrospective case series	1566 patients undergoing lumbar UBE	No SSI observed within 90 days	Not applicable	No	Absence of SSI	Reflects inter-center variability in incidence
Ousey et al., 2013[10]	Systematic review	Spinal wounds	Postoperative spinal wound complications	NPWT	Yes	Limited evidence; mechanisms and efficacy remain to be investigated	Provides theoretical and evidence background for NPWT
Kurra et al., 2020[13]	Retrospective study	Wounds associated with spinal SSI	Deep infectious wounds	NPWT applied at different tissue layers	Yes	Comparison of wound closure process	Supports the discussion regarding VSD technique
Wang et al., 2022[14]	Retrospective comparative study	Posterior instrumented lumbar surgery	Deep SSI	NPWT versus conventional debridement	Yes	Implant retention, infection recurrence and hospitalization parameters	Findings cannot be directly extrapolated to UBE, but allow for comparative analysis
Present case	Case report	Unilateral biportal endoscopic lumbar discectomy	Deep SSI caused by Enterobacter hormaechei	Debridement combined with VSD and antimicrobial therapy	Yes	Complete resolution of symptoms; cured and discharged	Highlights the unique UBE clinical scenario and VSD treatment strategy

4. Discussion

SSI following UBE may result from the combined effects of patient-related factors, surgical factors, and postoperative management. Risk factors for spinal SSI include diabetes mellitus, obesity, malnutrition, immunosuppression, prolonged operative time, and disruption of the perioperative sterile barrier [16, 17].UContinuous fluid irrigation, a distinctive feature of UBE, may lead to saturation of dressings or surgical drapes. Once the fluid barrier fails, skin colonizing bacteria can more easily invade deep wounds. The two infectious cases reported by Hong et al.[5]were both complicated by irrigation fluid leakage and soaked dressings as well as inadequate preoperative skin preparation; one case additionally involved prolonged surgery and unsupervised non-sterile dressing changes.

Timely and adequate surgical debridement remains the cornerstone of management for deep postoperative spinal infection, which involves resection of devitalized and necrotic tissue, acquisition of deep tissue specimens intraoperatively for etiological testing, and administration of appropriate antimicrobial therapy based on culture and susceptibility results[18–20].VSD cannot serve as a substitute for debridement or antimicrobial treatment. Its potential benefits include continuous removal of exudate, reduction of local fluid collection, management of irregular dead space, minimization of secondary contamination induced by frequent open dressing changes, and optimization of wound conditions to facilitate delayed closure [10–15].For narrow, elongated deep tracts or paraspinal dead space formed after UBE, VSD achieves more extensive contact drainage than conventional drainage tubes. In patients with spinal implants, clinicians should clarify the therapeutic goal: either control of acute infection with retention of stable implants whenever feasible, or implant removal indicated by loosening, delayed infection or biofilm formation. The decision to retain implants relies on comprehensive evaluation of infection duration, spinal stability, fusion status, pathogens and the accessibility of debridement, and should not be determined solely based on VSD application[21].

For VSD application on spinal wounds, inconsistencies exist across previous studies regarding the depth of foam placement, foam proximity to the dura and neural structures, negative pressure magnitude, continuous versus intermittent mode, dressing change intervals, and criteria for wound closure. Several publications address special scenarios with exposed or incised dura. Therefore, a fixed negative pressure value cannot be generalized as a standard protocol for all patients with UBE-associated infection. In cases with dural exposure, the use of protective barrier materials should be documented, and clinicians should stay alert to risks including cerebrospinal fluid leakage, hemorrhage, pain, retained foam fragments and adjacent tissue injury[22, 23].

Both patients with deep Staphylococcus aureus SSI after UBE described by Hong et al. [5]underwent repeat endoscopic debridement and targeted antimicrobial therapy without VSD. The large-scale complication analysis by Yang et al. [6]also emphasized timely debridement of infectious lesions and full-course antimicrobial treatment. Compared with those reports, the present case featured a deep infectious cavity that could not be adequately drained by conventional tubes and persistent wound drainage. Management was performed through the original UBE incision without immediate conversion to a large open wound. Current evidence recognizes deep SSI after UBE as a severe complication requiring early identification and active intervention. For patients presenting with progressively worsening postoperative pain, persistent wound drainage, fever or markedly elevated CRP, contrast-enhanced MRI should be adopted to evaluate infection extension, and deep tissue specimens should be obtained whenever possible prior to antimicrobial adjustment. VSD can act as an adjuvant tool for selected complex wounds, yet it must not delay debridement or mask uncontrolled infection within the intervertebral space, epidural space or around implants. Treatment endpoints encompass not only superficial wound closure, but also resolution of systemic symptoms, steady decline of inflammatory markers, radiological improvement of deep infection, and absence of recurrence over an adequate follow-up period.

5. Limitations

This report has several limitations. First, being a single case, it cannot assess the comparative efficacy of VSD against conventional drainage, open wound dressing, or repeat endoscopic debridement. Second, infection control was achieved through the combined effects of debridement, antimicrobial therapy, VSD, and the patient's intrinsic recovery; hence, the treatment outcome cannot be fully attributed to VSD alone. Third, empirical antibiotics were administered before bacterial culture sampling. Fourth, this narrative literature review is restricted by the limited number of published studies on UBE-associated infection. Further multicenter case series or prospective studies are needed to define the indications, technical protocols, and long-term safety of VSD.

6 Conclusion

Deep surgical site infection after UBE is relatively uncommon. However, delayed management may lead to infection spread, persistent non-healing wounds, and secondary extended surgery. In the present case, after adequate debridement, vacuum sealing drainage (VSD), and targeted anti-infective therapy, local infectious manifestations were controlled, the incision healed, relevant clinical symptoms improved, and the patient was discharged upon achieving clinical cure criteria. This case suggests that for selected patients with deep infection following UBE, VSD can serve as an adjuvant measure combined with thorough debridement and systematic anti-infective treatment. It facilitates continuous drainage, reduces local dead space, and optimizes wound conditions.

Declarations

Conflict of Interest: The authors declare that they have no conflict of interest.

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Availability of data and material (data transparency): All data generated or analyzed during this study are included in this article.

Code availability (software application or custom code): Not applicable

Consent for publication: Written consent to publish this article was obtained from study participants. Proof of consent to publish from study participants can be requested at any time.

Informed consent: Informed consent was obtained from individual participant included in the study.

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Author Contributions Yanan Wang (YW) contributed to the conceptualization and design of the study, collection and organization of clinical data, literature review, preparation of figures and tables, and drafting of the original manuscript. Hao Xing (HX) contributed to clinical data verification, interpretation of the treatment course, and critical revision of the manuscript. Huan Liu (HL) contributed to literature retrieval, data organization, and manuscript review and editing. Zhengqi Chang (ZC) contributed to the conceptualization of the study, clinical supervision, interpretation of the findings, critical revision of the manuscript, and overall project supervision. All authors read and approved the final version of the manuscript.

Ethics approval and consent to participate: This study has been approved by the 960th hospital of PLA. Each author certifies that all investigations were conducted in accordance with ethical principles. The participant involved in the study gave their informed consent and signed an informed consent form.

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Figures

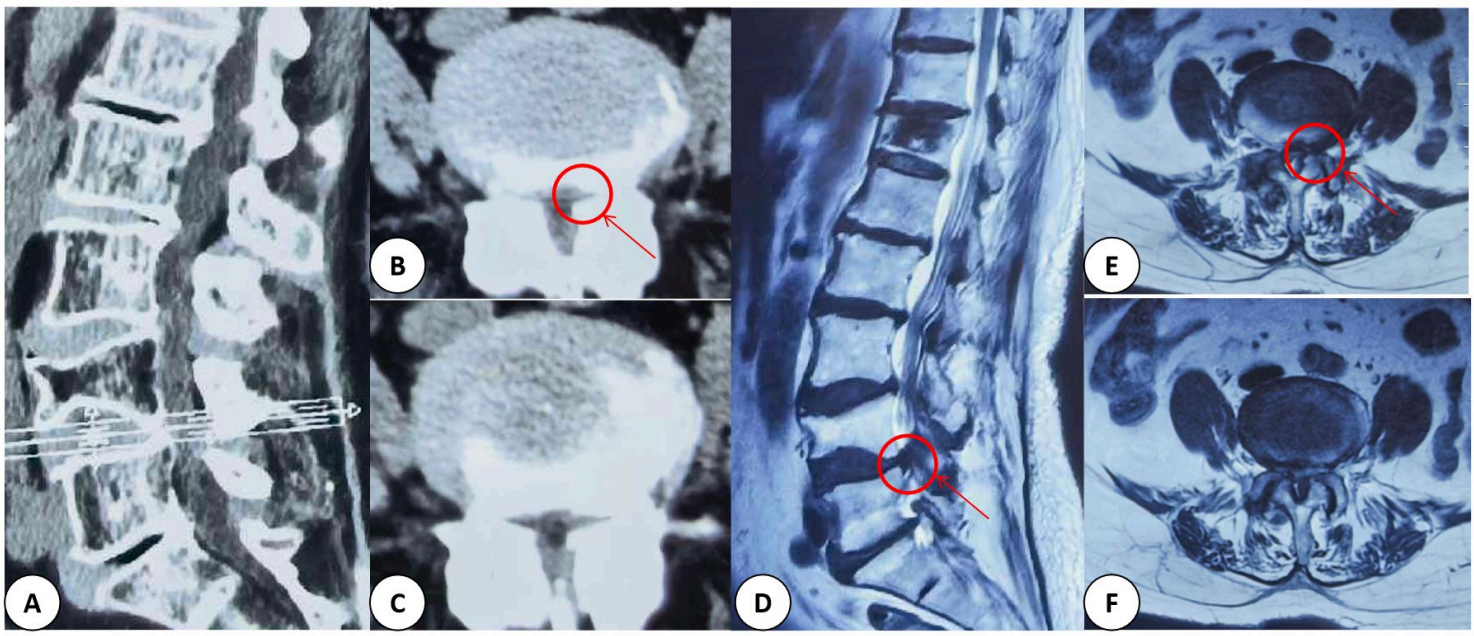


Figure 1

A–C show posterior protrusion of the L4/5 intervertebral disc into the spinal canal, predominantly on the left posterolateral side, resulting in stenosis of the spinal canal and left lateral recess, as well as degenerative hypertrophy of bilateral facet joints. Figures 1D–F demonstrate decreased signal intensity of the L4/5 intervertebral disc with left posterolateral disc herniation compressing the anterior

edge of the thecal sac and the course of the left nerve root, accompanied by marked left lateral recess stenosis. Degenerative changes of bilateral facet joints, thickened ligamentum flavum and L4/5 spinal stenosis are also observed.

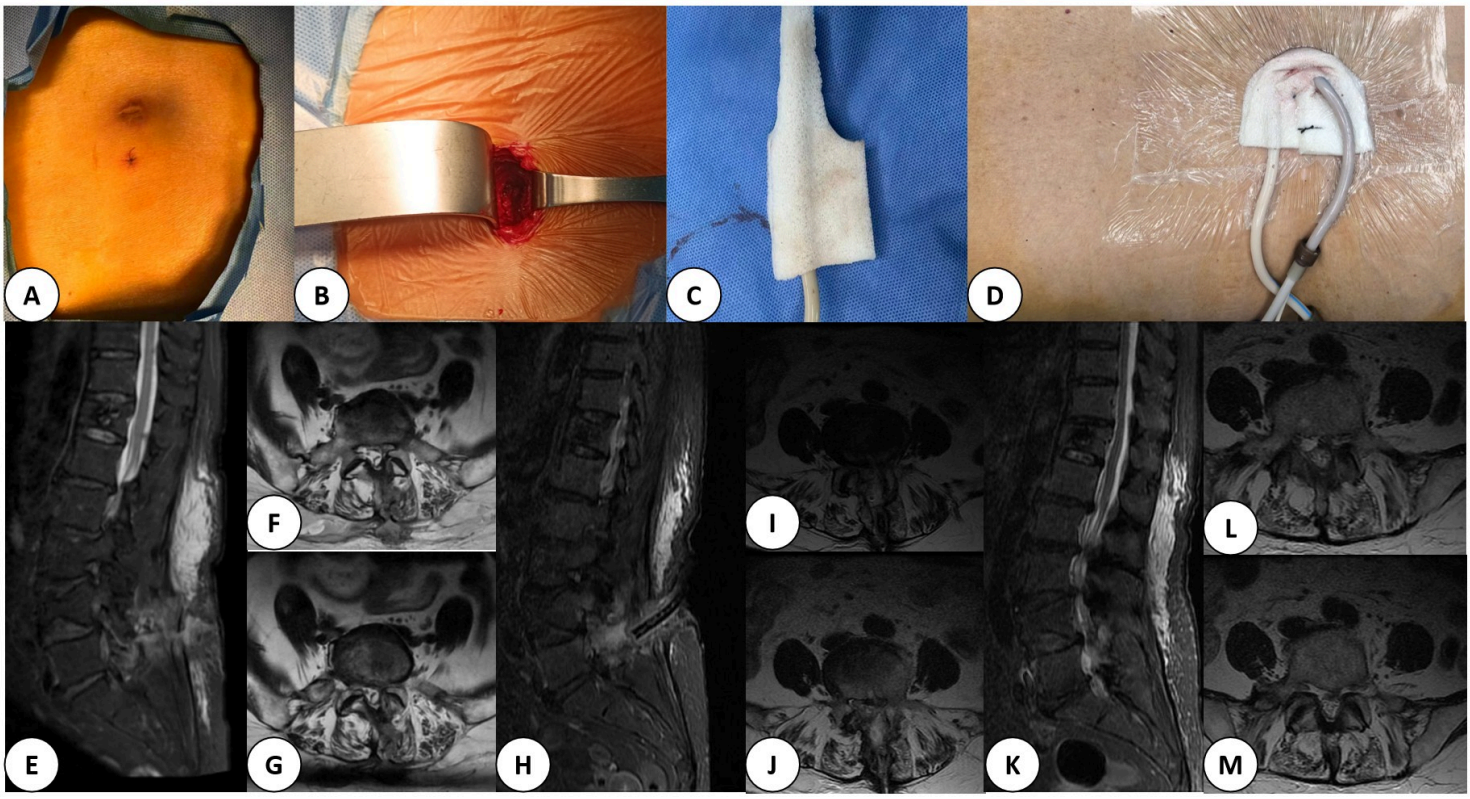


Figure 2

(A) Appearance of the infected surgical wound before debridement; (B) Intraoperative debridement through the original surgical incision; (C) Trimmed VSD foam suitable for insertion via the original incision; (D) Postoperative wound appearance. (E–G) Magnetic resonance images obtained at readmission due to infection; (H–J) Magnetic resonance images after debridement combined with VSD negative-pressure therapy; (K–M) Follow-up magnetic resonance images acquired before discharge upon completion of treatment.

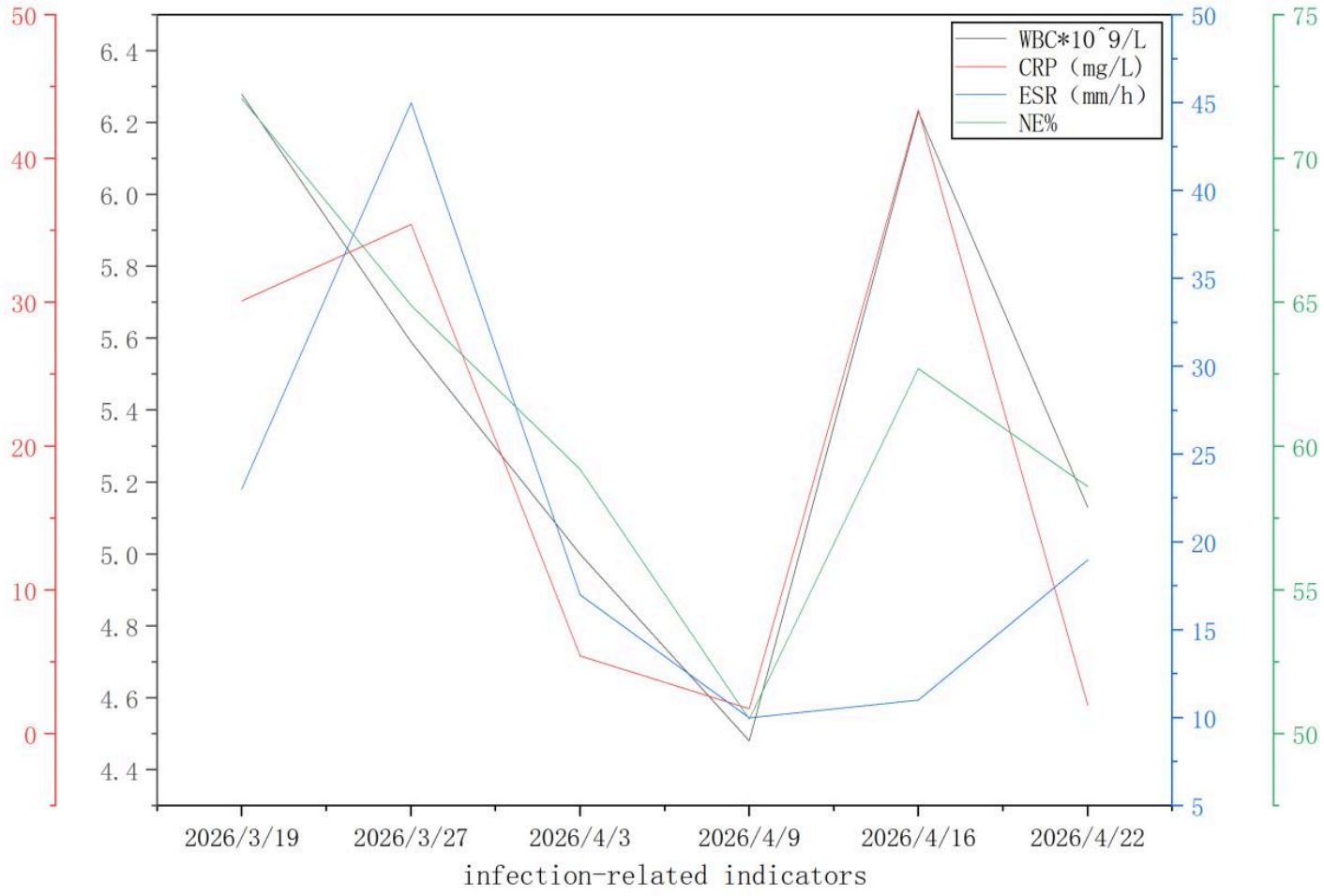


Figure 3

The line graph illustrates the changing trends of infectious biomarkers in the patient from admission to discharge.

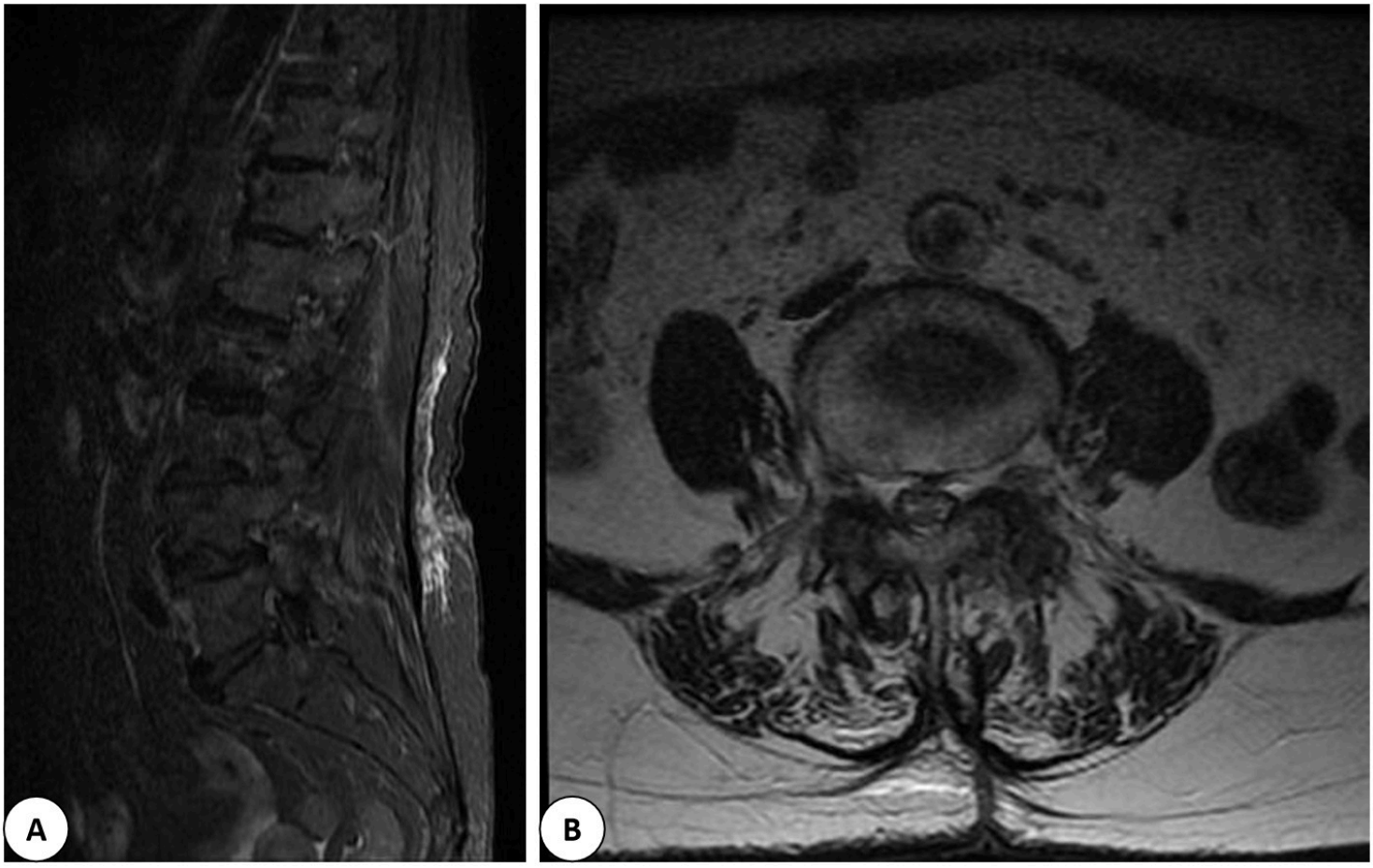


Figure 4

the 3-month follow-up lumbar MRI demonstrated markedly reduced abnormal signal changes at the L4/5 operative site and adjacent paravertebral soft tissues, with no obvious fluid collection or definite abscess formation.

The entire treatment course for the patient over the two hospital admissions is shown in Figure 5.

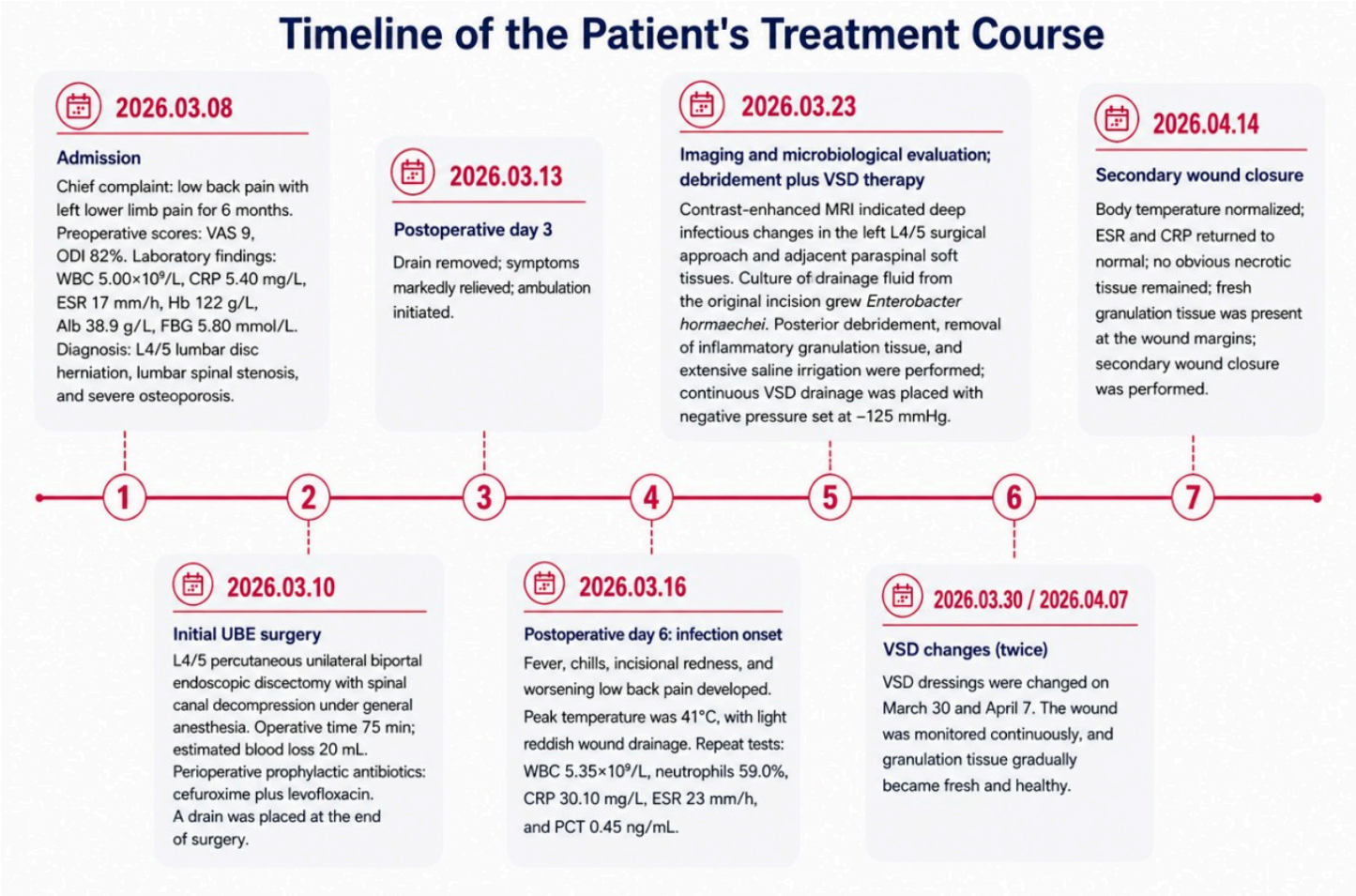


Figure 5

Timeline of the Case

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