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CASE REPORT

Corynebacterium striatum is Not Just a Contaminant: Experience with Antibiotic Treatment of Spondylodiscitis in an Immunocompetent Adult

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Background: *Corynebacterium striatum* is a commensal skin agent rarely described as a cause of infective spondylodiscitis. In this study, we report the first case of an infected patient who was successfully treated with conservative measures.

Case Presentation: A 54-year-old immunocompetent patient presented with progressive low back pain that had persisted for 1 month. Magnetic resonance imaging revealed abnormal signals in the L4–L5 vertebrae, indicating lumbar spine infection. Laboratory investigations revealed elevation of the serum C-reactive protein level and erythrocyte sedimentation rate. Blood and disc biopsy tissue cultures produced cream-colored round raised colonies on blood agar plates, which were identified as *C. striatum* using matrix-assisted laser desorption ionization time-of-flight mass spectrometry and 16S rRNA sequencing. Based on the antibiotic sensitivity test results, vancomycin and linezolid were sequentially administered to treat *C. striatum* infection; however, this strategy proved ineffective after 12 days. Despite delayed symptomatic treatment, the patient was successfully treated with a 2-week course of linezolid based on the use of amikacin to control other pathogens.

Conclusion: *C. striatum* can cause discitis in patients without any medical or surgical complications. The infection was successfully treated with anti-infective agents, providing empirical information on spinal infections.

Keywords: *C. striatum*, spondylodiscitis, conservative treatment, spinal infections

Introduction

Corynebacterium striatum is an aerobic and facultative anaerobic gram-positive bacterium that is part of normal human skin and mucous membranes.[1] In many countries, *C. striatum* is considered a potential MDR pathogen usually associated with surgery or bloodstream infections, causing bacteremia, artificial knee infections, and acute or chronic spondylitis.[2–5] While usually associated with postoperative infections, *C. striatum* may also emerge secondary to hematogenous spread.[6] Notably, spontaneous spondylodiscitis caused by *C. striatum* remains exceptionally rare, with only one documented case in medical literature.[7] This infectious process involves inflammatory destruction of intervertebral discs, adjacent vertebrae, and paraspinal structures, potentially leading to acute sepsis, neurological compromise from epidural abscess formation, vertebral collapse, and multiorgan dysfunction.[4,8] Although post-traumatic and postoperative cases have increased in frequency, approximately 20% of spondylodiscitis cases occur spontaneously without identifiable predisposing factors.[9,10] The diagnostic challenge posed by nonspecific clinical presentation frequently results in delayed treatment, contributing to mortality rates approaching 27%.[8] Therapeutic management of *C. striatum* infections presents significant clinical challenges due to limited evidence guiding antimicrobial selection and dosing strategies.[11] Current

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guidelines emphasize the importance of integrating pharmacokinetic/pharmacodynamic (PK/PD) principles with in vitro susceptibility data to ensure adequate tissue penetration at infection sites.[12] Glycopeptides (vancomycin, teicoplanin) and oxazolidinones (linezolid) demonstrate reliable in vitro activity and clinical efficacy, often requiring combination therapy with aminoglycosides like gentamicin for synergistic effects.[13,14] Amikacin, a broad-spectrum aminoglycoside primarily indicated for Gram-negative infections, shows particular utility in managing severe systemic infections when combined with anti-Gram-positive agents.[15] We present a novel case of spontaneous *C. striatum* spondylodiscitis in an immunocompetent patient without identifiable risk factors, successfully managed through combination therapy with linezolid and amikacin.

Case Description

A 54-year-old man presented to our department with a month-long history of low back pain that had intensified over the past 20 days and had radiated to the lower hip in the last 2 days. He had no history of trauma, fever, weight loss, surgery, or cancer. The only positive findings on physical examination were pain in flexion and extension of the lumbar spine, tenderness and percussion of the spinous process at L4–L5, and radiation to the left buttock, including hypoesthesia of the skin of the left buttock and the lateral thigh. Laboratory tests revealed a serum C-reactive protein (CRP) concentration of 16.47 mg/L, erythrocyte sedimentation rate (ESR) of 35 mm/h, white blood cell count of 14.6 × 109/L, blood urea nitrogen (BUN) level of 6.90 mmol/L, and serum creatinine (CRE) level of 75 μmol/L. Magnetic resonance imaging (MRI) indicated a narrow left lateral recess at L5–S1 and changes in bone marrow signals at L4–L5 (Figure 1A).

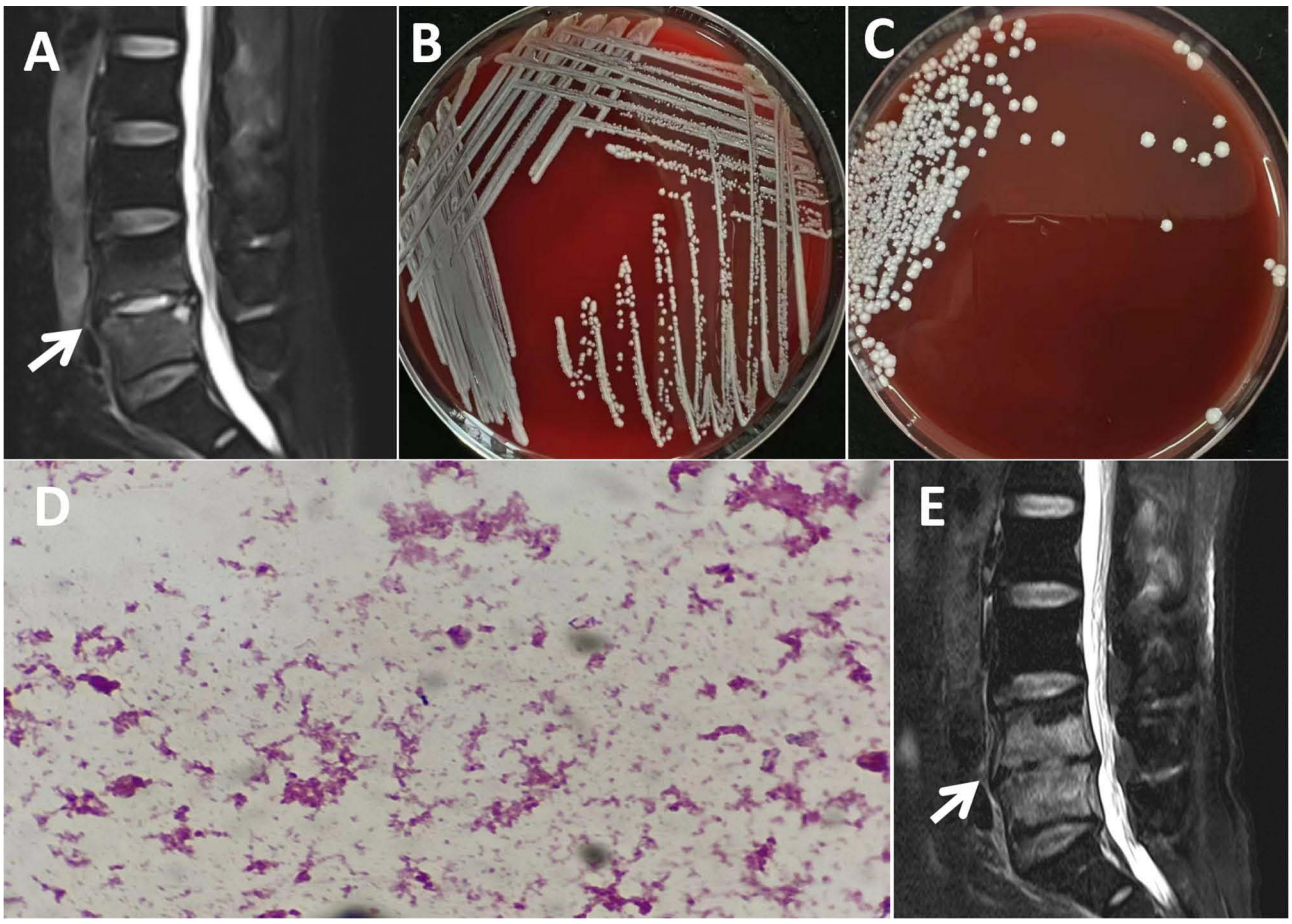

Figure 1 Representative results of MRI and sample culture. (**A**) T2-WI-FS image for diagnosis before antibiotic treatment (white arrow).(**B**) After 2 days of incubation, tiny, nonhemolytic, and milky white colonies were observed on the Columbia blood agar plate of blood samples. (**C**) After 2 days of incubation, tiny, nonhemolytic, and milky white colonies were observed on the Columbia blood agar plate of tissue samples. (**D**) Morphology of the tissue smear. (**E**) MRI reassessment 68 days after presentation (white arrow).

On the third day of hospitalization, the patient underwent an L4/5 disc biopsy. The excised tissue was sent for pathological and microbiological analyses. Immediate postoperative cefazolin sodium (2.0 g, every 8 h) was administered for usual spondylodiscitis. By day 6, *C. striatum* was detected in the bacterial cultures of blood samples. Two days of incubation led to the growth of tiny, non-hemolytic, milky white colonies on Columbia blood agar plates from four blood sample cultures (Figure 1B). *C. striatum* was identified in bacterial cultures of disc biopsy tissues by day 7. After 2 days of incubation, small, non-hemolytic, milky-white colonies were observed on Columbia blood agar plates (Figure 1C). Gram staining of the tissue smear identified *C. striatum* (Figure 1D).

The identification of the two colonies from blood and tissues biopsies as *C. striatum* was achieved through matrix-assisted laser desorption ionization time-of-flight mass spectrometry (MALDI-TOF MS) and confirmed by 16S rRNA sequencing (primers: 27F, TTGGAGAGTTTGATCCTGGCTC; 1492R, TTGGAGAGTTTGATCCTGGCTC) and phylogenetic tree analysis (Figure 2). The GenBank accession numbers are OR653924 (isolated from tissue biopsies samples) and OR653923 (isolated from blood samples). Thus, vancomycin (1.0 g, every 12 h) was added to the anti-infective regimen.

After 12 days of vancomycin treatment, the CRP level and ESR remained higher than normal, indicating a lack of efficacy. Therefore, the patient received linezolid (0.6 g, every 12 h instead of vancomycin for 8 days. Despite this management, the CRP level and ESR were not decreased, but BUN (7.73 mmol/L) and CRE levels (88.4 μmol/L) were within their normal ranges. An antibiotic sensitivity test (AST) was performed using a commercial kit (broth dilution method, Zhongaisheng, Hebei, China), and the results revealed that the strains isolated from blood and tissue cultures were sensitive to gentamicin, cefepime, cefotaxime, meropenem, ciprofloxacin, tetracycline, doxycycline, daptomycin, rifampicin, vancomycin, and linezolid but resistant to erythromycin and clindamycin (Table 1).

Because of uncontrolled infection and AST results, other pathogens were suspected. Therefore, amikacin (0.6 g/day) was added to the anti-infective regimen. Treatment with linezolid (0.6 g, every 12 h) plus amikacin (0.6 g/day) was initiated instead of linezolid alone on the 27th day of hospitalization. Hematological data and CRP levels returned to normal by day 41, indicating the infection was controlled. The patient was discharged with a 2-week oral linezolid

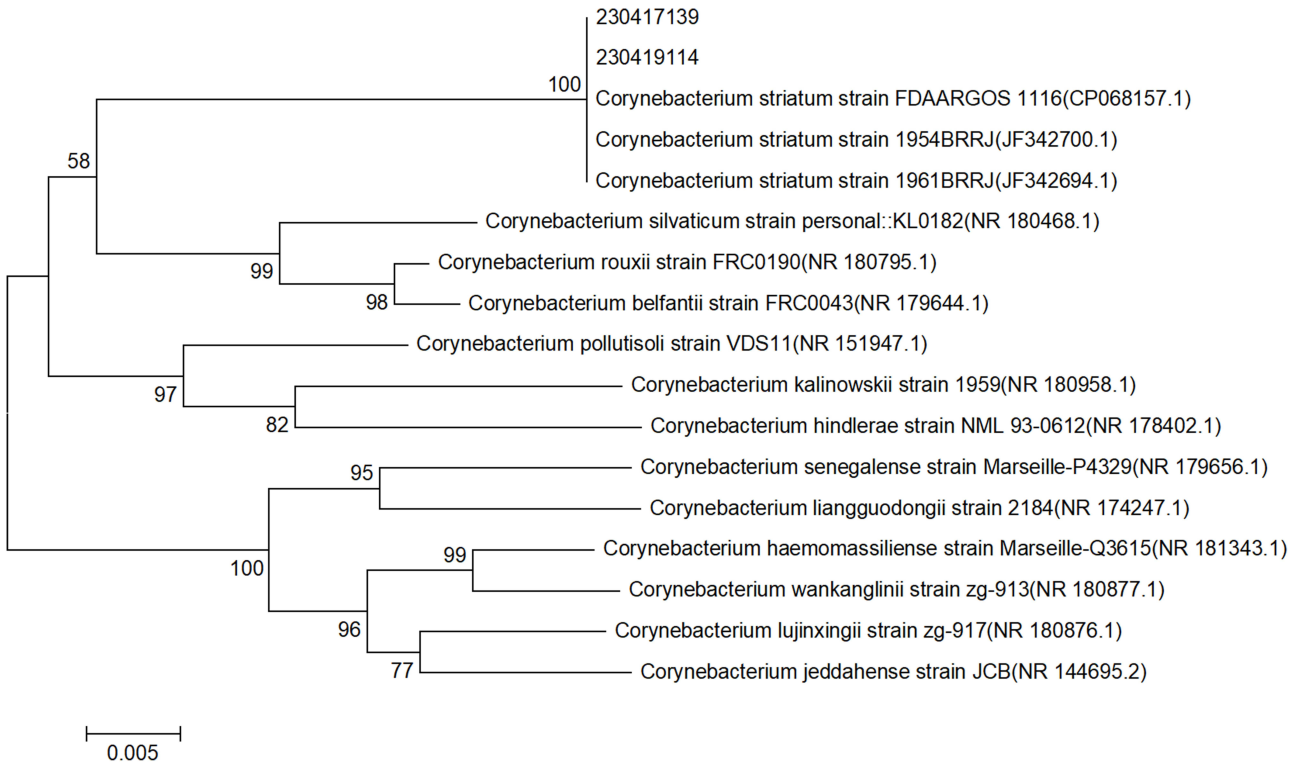

Figure 2 Phylogenetic analyses of *C. striatum* isolated from tissues (230419114) and blood samples (230417139).

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Table 1 Antimicrobial Susceptibility Profiles of *C. striatum*

Antimicrobial Agents	Blood Sample MIC (mg/L)*	Tissue Sample MIC (mg/L)*	Clinical Breakpoint (mg/L)#		
			S	I	R
Penicillin	0.5	0.5	≤0.12	0.25–2	≥4
Gentamicin	<0.25	<0.25	≤4	8	≥16
Cefepime	1	1	≤1	2	≥4
Cefotaxime	1	1	≤1	2	≥4
Ceftriaxone	2	2	≤1	2	≥4
Meropenem	<0.125	<0.125	≤0.25	0.5	≥1
Erythromycin	>8	>8	≤0.5	1	≥2
Chloramphenicol	>16	4	≤0.5	1–2	≥4
Ciprofloxacin	1	<0.5	≤1	2	≥4
Tetracycline	2	<1	≤4	8	≥16
Doxycycline	<1	<1	≤4	8	≥16
Daptomycin	0.25	0.25	≤1	-	-
Rifampicin	<0.5	<0.5	≤1	2	≥4
Linezolid	0.5	0.5	≤2	-	-
Vancomycin	1	0.5	≤2	-	-
Trimethoprim-sulfamethoxazole	1	1	≤2/38	-	≥4/76

Notes: *Obtained by broth dilution method based on breakpoints available in CLSI guidelines M45. # Susceptibility was interpreted based on breakpoints available in CLSI guidelines M45.
Abbreviations: MIC, minimum inhibitory concentration; S, susceptible; I, intermediate; R, resistance.

regimen. At the last follow-up after 68 days (June 24, 2023), MRI revealed no clinical recurrence (Figure 1E). Details on diagnosis and treatment are presented in Figure 3.

Discussion and Conclusions

Spontaneous spondylitis caused by *C. striatum* is rarely reported, and it usually observed in immunocompromised individuals, especially those with diabetes, immunosuppression, and end-stage renal failure, and in older patients.[7] It is widely agreed that the occurrence of spinal infections, such as idiopathic discitis, is increasing.[16] Based on the current case, it is further believed that colonizers that were initially considered harmless contaminants can cause human

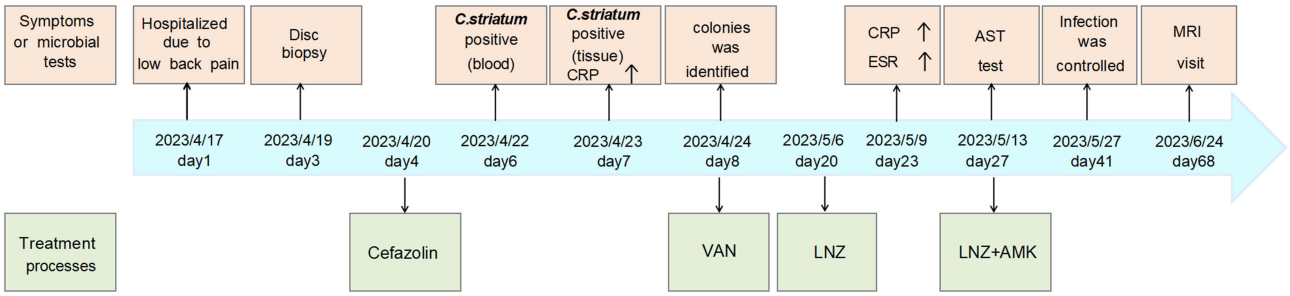

Figure 3 Timeline of the patient's diagnosis and treatment.
Abbreviations: VAN, vancomycin; LNZ, linezolid; AMK, amikacin; CFZ, cefazolin.

infection. More crucially, this case emphasizes the necessity to view *C. striatum* as a pathogen in certain medical contexts.

The virulence of *C. striatum* species should not be underestimated, particularly in patients with normal immune function. The patient in this case had a *C. striatum* infection that resulted in lower back pain and higher CRP and ESR levels. However, the patient had no fever that is consistent with the literature description: fever is less common in cases of clinical suspicion of discitis, accounting for 48% to 63% of cases.[17] Thus, identification of the pathogen should be considered in patients with normal immune function who develop unexplained spinal pain, particularly in those with elevated laboratory inflammation markers. Not only in cases of fever, but also in clinically bland cases and in patients without fever.

Studies revealed that 16S rRNA sequencing and MALDI-TOF MS are highly reliable for identifying pathogens.[18] In our case, the colonies were identified as *C. striatum*, with scores of 2.300 (blood) and 2.245 (tissue) after 2 days of incubation on blood agar. Similarly, 16S rRNA sequencing confirmed species-level identity (100% homology) between blood and tissue samples, with identical sequences observed for both specimen types. For swift detection of this pathogen, we advise employing MALDI-TOF MS once colonies are isolated. Due to its convenience, accuracy, and low cost of reagents and consumables, it is also being cost-effectively applied in various areas of clinical microbiology.[19]

As Streifel et al recommended, antibiotic therapy can be considered a reasonable treatment option.[20] Vancomycin and linezolid are among the anti-infective agents that have been successfully employed to treat *C. striatum* infections.[21] The *C. striatum* isolates in our study were susceptible to gentamicin, vancomycin, and linezolid but intrinsically resistant to some antibiotics, such as erythromycin, clindamycin, and co-trimoxazole. The antibiotic susceptibility profile is mostly akin to that reported by Alibi et al.[22] Given that each isolate's antibiotic susceptibility profile varied in an unpredictable manner, an appropriate AST should be conducted to determine which antimicrobial agents, including macrolides, lincosamides, fluoroquinolones, beta-lactams, and rifampicin, are effective for treating *C. striatum* infections before beginning antibiotic therapy. For our patient, despite microbiologically guided therapy and administration of vancomycin-linezolid monotherapy-a regimen associated with clinical cure-elevated inflammatory markers suggested suboptimal infection control. This discrepancy highlights multifactorial challenges in managing spinal infections, including comorbidities or immunometabolic variations, pathogen virulence dynamics, and antibiotic pharmacokinetic limitations. Notably, experimental models reveal diminishing bone penetration of vancomycin during progressive osteomyelitis, potentially explaining attenuated therapeutic responses in deep-seated infections.[23] Other than that the rising prevalence of MDR pathogens further complicates treatment strategies. Current data indicate high resistance rates among Gram-negative organisms to first-line agents like cefazolin and ciprofloxacin,[24] necessitating broad-spectrum coverage in polymicrobial or high-risk scenarios. Amikacin was added to the anti-infective regimen to control other pathogens. But,Combination therapies remain contentious, with heterogeneous evidence supporting β-lactam/glycopeptide synergism.[25]

Overall, this case is interesting because it is extremely rare for a severe non-staphylococcal organism to invade the spine of a healthy individual. *C. striatum* can cause severe invasive infections of various tissues, and these infections can also occur in immunocompetent hosts. According to the existing literature and susceptibility trends, linezolid is the most suitable first-line treatment.

Abbreviations

MDR, multidrug-resistant pathogen; CRP, C-reactive protein; ESR, erythrocyte sedimentation; AST, An antibiotic sensitivity test; MALDI-TOF MS, matrix-assisted laser desorption ionization time-of-flight mass spectrometry; PK, pharmacokinetic; PD, pharmacodynamic; BUN, blood urea nitrogen; CRE, serum creatinine; MRI, Magnetic resonance imaging.

Data Sharing Statement

The original contributions of this study are included in the article. Further inquiries can be directed to the corresponding author.

Ethics Approval

This study, involving a human participant, was reviewed and approved by the Ethics Committee of Shengli Oilfield Central Hospital (NO. YXLL 202515301). Written informed consent for experiment and publication was obtained from the patient. The consent form is available for review by the editor if needed.

Consent for Publication

No potentially identifiable human images or data are presented in this study.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no other conflicts of interest in this work.

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