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CASE REPORT

A Case of Septicaemia Infection Induced by Vancomycin-Resistant *Streptococcus dysgalactiae subspecies equisimilis*

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Abstract: In recent years, reported infections caused by *Streptococcus dysgalactiae subspecies equisimilis* (SDSE)have notably increased. SDSE is a Group C or G β-hemolytic streptococcus, that frequently causes bloodstream infections and is highly susceptible to penicillins, cephalosporins and fluoroquinolones. In this study, we present the first reported case of vancomycin-resistant SDSE septicaemia, detailing its clinical diagnosis and treatment. A review of the relevant literature, noted that no other instances of vancomycin resistance in SDSE have been documented. It is hoped that this study will lead to further clinical consideration of vancomycin resistance.

Keywords: *S. dysgalactiae* subsp *equisimilis*, vancomycin resistance, septicaemia

Introduction

SDSE is an opportunistic pathogen that can cause diseases such as bloodstream infections, cellulitis, necrotizing fasciitis, pneumonia, meningitis, and endocarditis when host immunity is compromised. Bloodstream infections are the most frequently observed.[1] In recent years, the infection rate by SDSE has been increasing,[2] and reports of various disseminated diseases caused by this pathogen have also been steadily rising.[3] Several studies[4,5] have demonstrated that SDSE is sensitive to penicillins, cephalosporins, and fluoroquinolones but resistant to erythromycin, clindamycin, and tetracycline. Domestic and international research findings[6,7] largely support these observations, suggesting that penicillin and other beta-lactam antibiotics can be the first choice of treatment. Some reports have also recommended treatment with vancomycin in combination with piperacillin for nonpurulent severe infections. However, in patients with beta-lactam allergy, vancomycin is frequently employed as a reliable alternative, owing to its consistent in vitro activity against this organism.[8,9]

Vancomycin resistance in SDSE is exceptionally rare, with no previously documented cases. Its emergence would complicate empiric treatment for severe infections.

We report the first case of bloodstream infection caused by vancomycin-resistant SDSE, highlighting its clinical implications and the need for continued resistance surveillance.

Case Presentation

An 89-year-old patient was admitted to the Department of Respiratory Medicine of our hospital on September 14, 2024, presenting with a one-day history of cough and fever. The patient complained of chills and fever in the early morning on the day of admission with no apparent cause, and a maximum temperature of 39.5 °C, Symptoms included generalized pain and discomfort, accompanied by a productive cough with minimal white sputum, as well as transient episodes of

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chest tightness and shortness of breath. The patient had a history of type 2 diabetes mellitus and cavernous infarction for more than 10 years (currently taking oral selegiline metformin and hemosiderin). Upon admission: the body temperature was 38.4 °C, with coarse breath sounds in both lungs, erythema of the, left lower anterior tibial ski, increased skin temperature, and no obvious pain. Routine clinical blood tests revealed a serum amyloid A(SAA) concentration of 22.17 mg/L, a high-susceptibility C-reactive protein (hs-CRP) concentration of 2.70 mg/L, a white blood cell count of $18.43*10^9$/L, a hemoglobin concentration of 126.0 G/L, and a platelet count of $179*10^9$/L. Chest CT findings included: striated shadow in the upper lobe of the left lung, inflammatory? Partial solid nodules in the upper lobe of the right lung, small nodules in both lungs, scattered cord-like foci in both lower lungs, and calcification of the aorta and coronary wall. The initial diagnosis was community-acquired pneumonia.

Piperacillin (4 g, bid) combined with sulbactam (1 g, bid) was empirically administered to combat the infection. Ambroxol and dihydroxypropyl theophylline were used to reduce sputum and alleviate asthma, while physical cooling combined with an indomethacin bolus was used to control fever. The patient's temperature decreased, and the cough improved; however, the skin erythema persisted. On day 3 of admission bilateral blood cultures revealed SDSE, which was sensitive to penicillin, ceftriaxone, levofloxacin, and linezolid, but resistant to vancomycin, clindamycin, and erythromycin (Table 1, Microbiological culture and identification typically require approximately 3 days to yield results). A diagnosis of septicaemia was clear, and the current anti-infective regimen had proven effective, warranting continued maintenance therapy. By Day 10, the erythema of the left lower limb anterior tibial skin had improved, and the skin temperature had returned to normal. However, on 9.20, laboratory results showed elevated levels of SAA and hs-CRP, and a follow-up chest CT revealed a slight increase in exudate compared with the previous scan (Table 2 and Figure 1).

Table 1 Results of Antimicrobial Susceptibility Testing

Streptococcus dysgalactiae subspecies equisimilis			
Antimicrobial Agents	**Minimum Inhibitory Concentration (μg/mL)**	**Interpretation of Susceptibility***	**Inhibitory Circle Diameter (mm)**
Penicillin	0.008	S	
Ceftriaxone		S	32
Clindamycin		R	12
Erythromycin		R	6
Levofloxacin		S	18
Linezolid		S	25
Vancomycin		R	6

Notes: Clarification of susceptibility testing methods: Matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) was employed for strain identification. Strains identified as *SDSE* resistant to vancomycin (score >2.0). Susceptibility was determined using a quantitative minimum inhibitory concentration test, combined with the Kirby-Bauer disk diffusion method. *Streptococcus pneumoniae* ATCC 49619 was used as the quality control strain for antimicrobial susceptibility testing of streptococci. *Interpretation was based on CLSI M100 (2023 edition): Performance Standards for Antimicrobial Susceptibility Testing standards along with WS/T 639—2018: Technical Requirement for Antimicrobial Susceptibility Testing.
Abbreviations: S, susceptible; R, resistant.

Table 2 Blood Test Inflammatory Indicator Chart

	2024.9.14	**2024.9.20**	**2024.9.26**
hs-CRP (MG/L)	2.70	28.78↑	6.28
SAA (MG/L)	22.17↑	283.62↑	2.37

Notes: The patient was tested three times during the admission period; the patient's hs-CRP and SAA levels were significantly elevated on 9.20, indicating that the infection was not under control. Tigecycline was then added to cover the antimicrobial spectrum, which returned to the normal range on 9.26. ↑Indicates exceeding the reference range (1–10 mg/L).

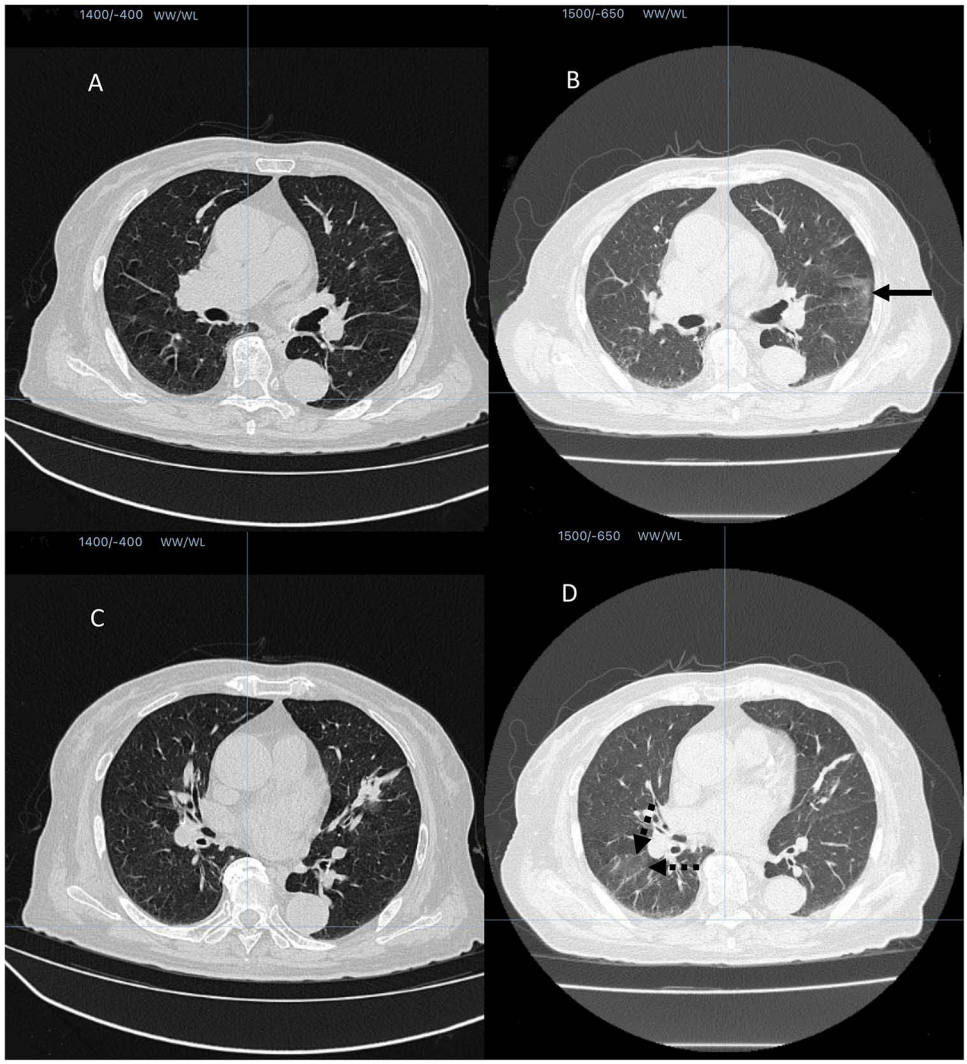

Figure 1 Chest CT scan results. A comparison of chest CT (**B** and **D**) on 9.23 with those upon admission (**A** and **C**) revealed that the upper lobe of the left lung (solid arrow) and the upper lobe of the right lung (dashed arrow) on 9.23 exhibited new pneumonia foci compared with the previous scans.

Based on the clinical pharmacist's recommendation, the antimicrobial spectrum was expanded by combining other antimicrobial drug and then adding tigecycline (50 mg, q12h) to combat the infection. On Day 13, the pathogenetic examination results remained negative; however, the patient's vital signs were stable, and his condition had stabilized. He was subsequently discharged with oral antimicrobial therapy, with plans for a repeat chest CT one month later.

Discussion and Conclusion

Given that this isolate was obtained from blood cultures during a febrile episode with systemic inflammatory response, it is confirmed as the causative pathogen of the bloodstream infection.

Although the isolate exhibited well-characterized resistance to vancomycin, the underlying mechanism remains speculative due to the absence of molecular or genetic analyses; however, plausible hypotheses can be formulated based on the existing literature. Vancomycin-resistant *Enterococcus faecium* (VRE) typically harbors the *VanA* and *VanB* genes, which mediate the synthesis of modified cell wall precursors, such as D-Ala-D-Lac, thereby reducing the binding affinity of vancomycin.[10] Similarly, vancomycin-resistant *Staphylococcus aureus* (VRSA) carries the *VanA* gene, which is encoded by the Tn1546 transposon and acquired through plasmid exchange with enterococci.[11] Type *VanA* is resistant to both vancomycin and teicoplanin, whereas type *VanB* is resistant to vancomycin only and sensitive to ticoranin.[12] It is

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well documented that, compared with point mutations, SDSE undergoes genetic recombination at a significantly higher rate, facilitating genetic diversification.[13] Based on these findings, it is hypothesized that the SDSE strain in this case may have acquired a vancomycin resistance gene, possibly resembling the *VanB* gene, through genetic recombination. Such recombination events could enable the strain to produce modified cell wall precursors, conferring resistance to vancomycin. The retrospective nature of this case report, which precluded the availability of an isolate for advanced molecular analysis (such as genomic sequencing), must be considered a limitation. Consequently, our discussion on the potential resistance mechanisms is extrapolated from the phenotypic antibiogram patterns and prior published studies.

Although direct experimental evidence is lacking, the clinical context provides indirect support for this hypothesis. The suboptimal efficacy of the initial regimen combining piperacillin with sulbactam could be attributed to the patient's compromised immune function and potential coinfection with other pathogens. The observed vancomycin resistance highlights the need for vigilant monitoring and individualized treatment strategies, particularly in immunocompromised patients.

Given the absence of prior reports on vancomycin-resistant SDSE, this case underscores the potential emergence of new drug-resistant strains and highlights the critical need for further research. Future studies should focus on the following:

1. Conducting genome sequencing and transcriptomic analyses to identify resistance-related genetic alterations in SDSE.
2. Investigating the molecular mechanisms underlying vancomycin resistance in SDSE through protein-level studies and functional assays.
3. Expanding clinical surveillance to identify similar cases and establish epidemiological trends.

By pursuing these directions, future research can deepen insights into the molecular biology of SDSE, identify actionable targets for therapeutic intervention, and develop robust strategies to prevent the spread of vancomycin-resistant strains. Additionally, the emergence of vancomycin resistance may be associated with its increasing use in recent years. The 2024 National Institute of Hospital Administration (NHC) Annual Report highlighted that vancomycin consumption increased from 623,000 defined daily doses (DDDs) in 2014 to 1,046,600 DDDs in 2023.

In conclusion, we report the first case of bloodstream infection caused by vancomycin-resistant *Streptococcus dysgalactiae subspecies equisimilis*. Therefore, our case serves as a critical alert to clinicians that vancomycin may not be a reliable empiric option for severe SDSE infections, particularly in regions where this pathogen is prevalent or when prior antibiotic exposure is suspected. While the current understanding of mechanisms underlying resistance remains speculative, this may indicate this case provides a valuable foundation for future investigations on the molecular biology and clinical management of vancomycin-resistant SDSE. So this report underscores the urgent need for enhanced surveillance of vancomycin resistance in SDSE. Further studies are warranted to investigate the molecular mechanisms and potential transmission of resistance genes in this species.

Ethics Approval and Consent to Participate

This case report was approved by the Institutional Review Board (or Ethics Committee) of Shidong Hospital Affiliated to University of Shanghai for Science and Technology, Yangpu District, Shanghai, China. Written informed consent for publication was obtained from the patient's legal guardian.

Consent for Publication

Given that the 89-year-old patient has communication difficulties due to a long-term history of lacunar infarction, consent for publication was obtained from her legal guardian (daughter).

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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

All authors report no potential conflicts of interest in this work.

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