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CASE REPORT

Neisseria sicca Bloodstream Infection in a Patient with Aortic Valve Replacement: Case Report and Genomic Analysis

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Abstract: *Neisseria sicca* is usually a commensal of the oropharynx but can occasionally cause invasive disease. Herein, we report a rare case of *N. sicca* bacteremia in a female with a 7-year history of aortic valve replacement (AVR), who presented with persistent fever but no respiratory symptoms or valvular vegetations. Blood cultures yielded *N. sicca*, identified by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) and confirmed via 16S rRNA sequencing. Appropriate antibiotic therapy with ticarcillin-clavulanate led to a significant clinical improvement. To gain insight into the pathogenicity and resistome of this strain, whole-genome sequencing of *N. sicca* junzhu_1 was performed using the Illumina NovaSeq platform. The resistome of the *N. sicca* strain consists of three antimicrobial resistance genes: *macB, macA*, and *mtrD*. Virulence gene analysis revealed that the pathogenicity of *N. sicca* was associated with *mntB, mntC, farA, farB, mtrD, mtrE, msrA*/*B* (*pilB), rfaF, rfaC, hpuB, pilT*, and *recN*. This case underscores the potential of *N. sicca* to act as an opportunistic pathogen in bloodstream infections (BSIs).

Keywords: *Neisseria sicca*, bloodstream infections, aortic valve replacement, whole-genome sequencing, case report

Introduction

Neisseria sicca (*N. sicca*) typically exists as a commensal bacterium on the mucosal surface of the human upper respiratory tract (URT), with an overall oropharyngeal carriage rate of approximately 9.4%.[1] However, in recent years, with an increasing number of clinical case reports on *N. sicca*, it has gradually exhibited its characteristics as an opportunistic pathogen rather than just a commensal bacterium on the mucosal surface. Similar to *N. meningitidis* and *N. gonorrhoeae*, its pathogenicity may involve a series of sequential steps, including adhesion to mucosal epithelial cells, invasion of host tissues, evasion of immune defenses, and induction of inflammatory damage.[2,3] This bacterium has been reported as a cause of various human infections, including endocarditis,[4,5] pneumonia,[6,7] sinusitis,[8] osteomyelitis,[9] meningitis,[10] conjunctivitis,[11] peritonitis,[12,13] and bloodstream infections (BSIs).[14,15]

BSIs are systemic inflammatory response syndromes caused by the invasion of pathogenic microorganisms such as bacteria or fungi into the bloodstream.[16] BSIs are associated with high mortality and increased healthcare costs.[16] These infections occur not only in immunocompromised individuals but also in healthy populations. Infectious endocarditis can serve as a nidus for ongoing BSIs because microorganisms colonizing the heart valves continuously disseminate into the bloodstream.[17] This may exacerbate BISs, leading to a vicious cycle of infection and inflammation. Based on the previous literature regarding infections caused by *N. sicca*, endocarditis constitutes the majority of cases.[18] Therefore, detection of N. *sicca* in the bloodstream warrants vigilance for potential endocardial diseases.

In a decade-long review of ~8,000 bacteremia cases, Feder et al identified only one *N. sicca*-positive culture that was ultimately deemed a contaminant.[19] While BSIs caused by *N. sicca* are uncommon, sporadic cases have been reported. Shaw et al first reported a case of *N. sicca* endocarditis in a 12-year-old patient.[20] Subsequent studies have also described

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true BSIs caused by this organism, though such occurrences remain uncommon.[5,18,21] These reports suggest *N. sicca* possesses greater pathogenic potential than is traditionally recognized.

Severe cardiac valve degeneration, including stenosis and regurgitation, often necessitates artificial valve replacement, yet this intervention carries a markedly increased risk for BSIs and endocarditis. Previous studies have reported BSI rates of ~8% after valve repair or replacement, of which 13–14.3% subsequently develop endocarditis.[22,23] Following aortic valve replacement (AVR), BSIs occur in approximately 10.1% of patients, of which nearly 40% progress to endocarditis.[24] The predominant causative organisms include gram-positive cocci—such as viridans group streptococci, β-hemolytic streptococci, coagulase-negative staphylococci (CoNS), methicillin-sensitive *Staphylococcus aureus* (MSSA), and *Enterococcus* spp.—and gram-negative bacilli, including *Escherichia coli, Klebsiella pneumoniae*, and *Pseudomonas aeruginos*a.[24] Recent reports have also documented rare cases of *Neisseria elongata* endocarditis in patients with AVR.[25,26]

In this study, we report a rare case of BSIs caused by the *N. sicca* strain junzhu_1 of unknown origin in a patient with a seven-year history of AVR. Whole-genome sequencing was performed to gain a deeper understanding of its pathogenicity and antimicrobial resistance. This case underscores the importance of recognizing *N. sicca* as an opportunistic pathogen in clinical settings.

Materials and Methods

Bacteria Isolation and Identification

Blood samples were inoculated into both aerobic and anaerobic blood culture bottles, and subsequently incubated in a BACT/ALERT 3D blood culture system (bioMérieux, France). Once a positive result was obtained, Gram staining was performed directly from the bottle, and the broth was plated onto 5% sheep blood agar. The agar plates were then incubated at 37°C for 18–24 hours. The bacterial isolate was identified by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF/MS) (bioMérieux, France) with 99.99% confidence. 16S rRNA-based taxonomic confirmation was performed by extracting and analyzing the corresponding genomic sequences from whole-genome assemblies using barrnap (https://github.com/tseemann/barrnap) with default parameters.

Antibiotic Susceptibility Assay

Antibiotic sensitivity testing was performed using the encompassed ten antibiotics: amoxicillin-clavulanate, ampicillin, tetracycline, cefotaxime, cefuroxime, rifampin, trimethoprim-sulfamethoxazole, chloramphenicol, cefaclor, and ofloxacin. MIC values were determined using ATB HAEMO CLSI (12) strips (bioMérieux, France) in accordance with the manufacturer's guidelines, and susceptibility was interpreted based on the breakpoints outlined in the US Clinical and Laboratory Standards Institute (CLSI) M100-S34 (2024) standards. Furthermore, the Kirby (K-B) disk diffusion method was employed to test for azithromycin and erythromycin.

Public Sequence Data

Whole-genome sequences of *N. sicca* were retrieved from GenBank using the search term "Neisseria sicca" with a genome length restriction of 1–10 Mb, yielding 28 available strains. Following a comprehensive evaluation of assembly metrics and annotation status, we selected 22 high-quality genomes for downstream comparative genomic analysis.

Whole Genome Sequencing and Bioinformatics Analysis

Total DNA of the *N. sicca* junzhu_1 isolate was extracted from pure cultures using a bacterial DNA extraction kit (Bacterial/Fungal DNA Extraction Kit [Magnetic Beads], China). A next-generation sequencing library was prepared using the NEBNext®Ultra™ DNA Library Prep Kit for Illumina (NEB, USA) (350 bp fragment library, paired-end) in accordance with the manufacturer's recommendations. The whole-genome sequence of *N. sicca* was obtained using the Illumina HiSeq 4000-PE150 platform (Illumina, Inc., USA), followed by assembly using Unicycler v. 0.5.0 (https://github.com/rrwick/Unicycler). The genome was annotated using the Prokaryotic Genome Annotation tool (Prokka v.1.14.6) (https:// github.com/ tseemann/prokka).

Antimicrobial Resistance and Virulence Analysis

We identified virulence and antibiotic resistance factors using the Virulence Factor Database (VFDB) (http://www.mgc.ac.cn/VFs/) and the Comprehensive Antibiotic Research Database (CARD) (https://card.mcmaster.ca/).

Phylogenetic Analysis

To characterize the evolutionary relationships among the 23 *N. sicca* isolates in a global context, we utilized CSI Phylogeny 1.4 for phylogenetic analyses (https://cge.food.dtu.dk/services/CSIPPhylogeny/). The phylogenetic tree was visualized and modified using iTOL (https://itol.embl.de).

Result

Case Presentation

A 73-year-old female with a 7-year history of AVR was admitted on November 8, 2023, with a one-day history of chills and high fever. Laboratory tests revealed a white blood cell (WBC) count of 17.1 × 10^9/L with neutrophilia (85.6%), markedly elevated C-reactive protein (CRP, 160.6 mg/L), and elevated procalcitonin (PCT, 1.53 ng/mL). Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and Mycoplasma pneumoniae tests were negative. Cytokine analysis showed significantly elevated interleukin-6 (IL-6, 566.10 pg/mL) and interleukin-10 (IL-10, 3888.73 pg/mL). Sputum cultures revealed normal respiratory flora, including *N. sicca* (1+). Blood culture yielded *N. sicca*, and chest computed tomography (CT) revealed an infectious lesion in the right lung (Figure 1A and D). The patient was diagnosed with pneumonia and bacteremia. The patient was administered ticarcillin-clavulanate (3.2 g q8h, IV). After one week of

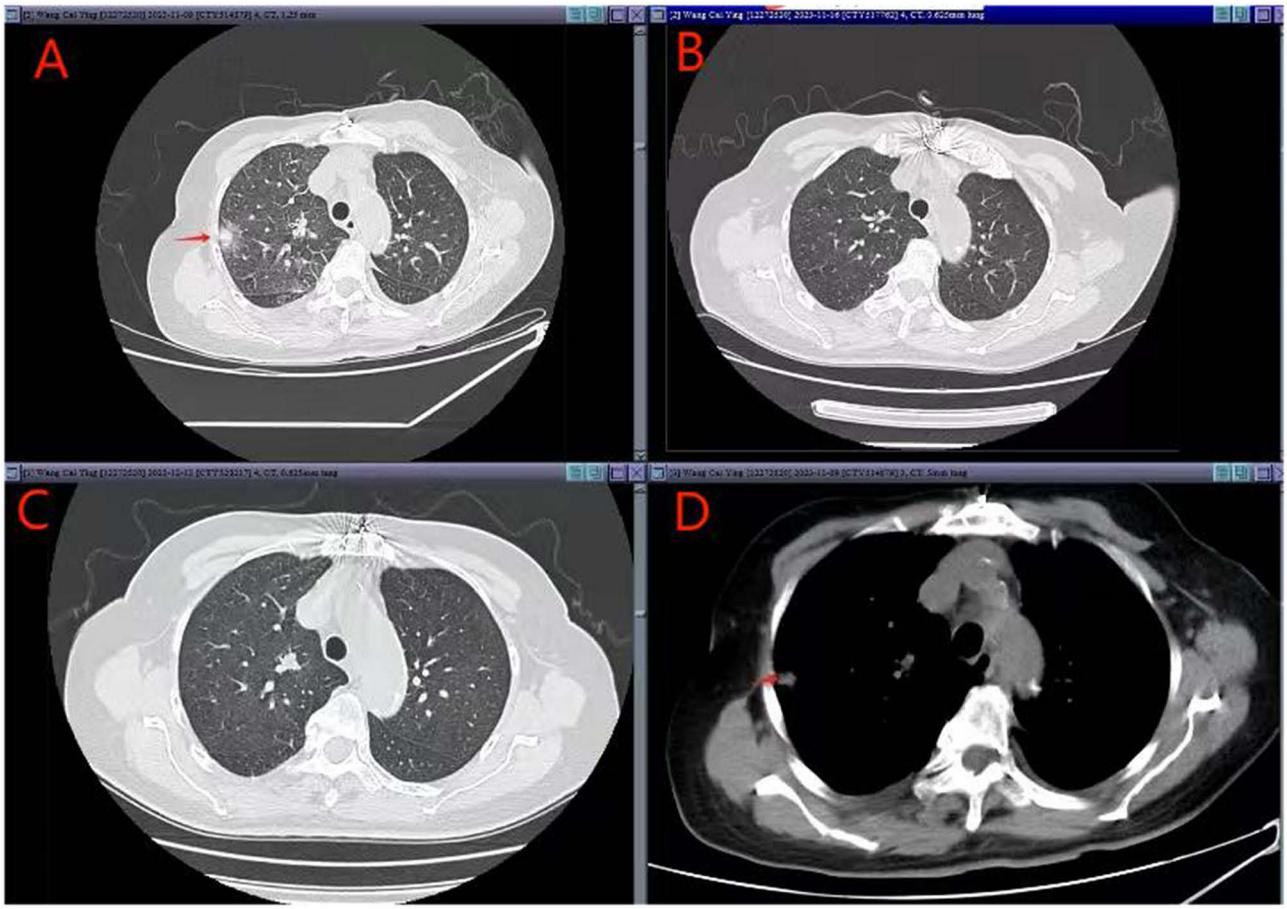

Figure 1 CT imaging changes between the two hospital admissions. (**A**) Local infectious lesion in upper lobe of right lung (red arrow); (**B**) Infectious lesion absorption in upper lobe of the right lung; (**C**) No infectious lesions in right upper lung lobe; (**D**) Plain transverse image of mediastinal window.

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treatment, the patient no longer had fever, cough, or sputum production. Follow-up chest CT indicated resolution of the infectious lesion in the right upper lung (Figure 1B). Oral amoxicillin-clavulanate therapy was continued for 4 days after discharge.

On December 11, 2023, the patient was readmitted to our hospital because of persistent high fever accompanied by chills for a day. Upon presentation, the patient had a body temperature of 39.1°C, a heart rate of 97 beats per minute, and a blood pressure of 113/53 mmHg. No symptoms of cough, sputum production, chest tightness, or shortness of breath were observed. Both lungs exhibited clear respiratory sounds, with no audible rales. The abdomen was soft and non-tender without rebound pain, and the liver and spleen were not palpable. The patient denied any symptoms, such as abdominal pain, diarrhea, urinary frequency, urgency, or dysuria. CT revealed multiple small nodules in both lungs with no inflammatory lesions (Figure 1C). Transthoracic echocardiography (TTE) revealed valvular dysfunction and aortic valve replacement with no vegetation (Figure 2).

Laboratory tests showed a WBC count of 13.5 $\times 10^9$/L, neutrophil ratio of 80.8%, hemoglobin of 109 g/L, platelet count of 129 $\times$ 10^9/L, CRP of 158.0 mg/L, troponin I of 0.034 ng/mL, brain natriuretic peptide (BNP) of 795 ng/L, and PCT of 8.10 ng/mL. Tests for SARS-CoV-2, influenza A virus, adenovirus, respiratory syncytial virus, and influenza B virus antigens were all negative. Prior to initiating empiric antimicrobial treatment with ticarcillin-clavulanate (3.2 g q8h IV) for eight days, two sets of aerobic and anaerobic blood cultures were performed. On the following day, sputum culture showed normal respiratory flora, and four culture bottles were positive for gram-negative Diplococcus

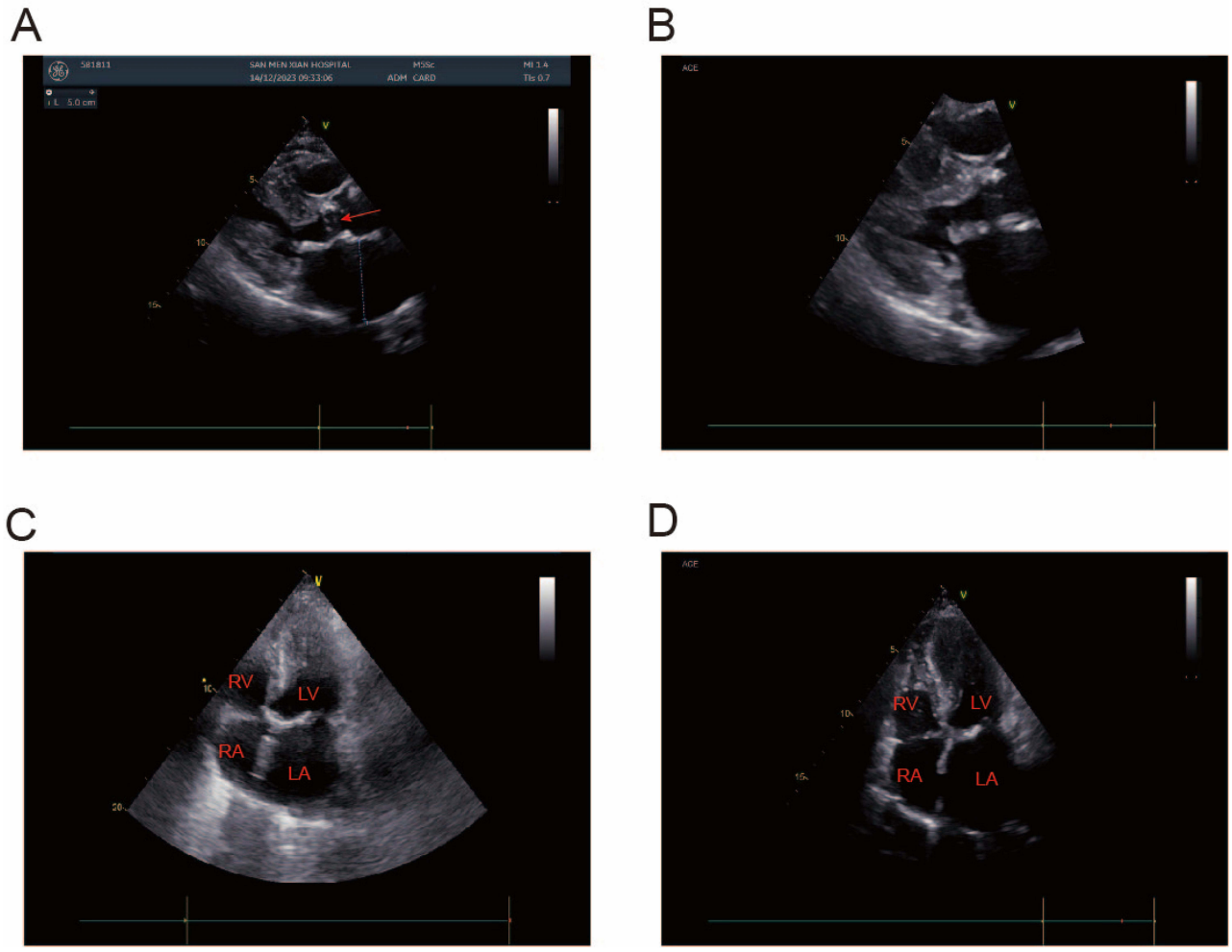

Figure 2 Echocardiogram showed no vegetation on the valves. (**A**) The artificial aortic valve image (red arrow); (**B**) The left ventricular long-axis section image; (**C**) Apical precordial four chamber view of the first admission; (**D**) Apical precordial four chamber view of the second admission.

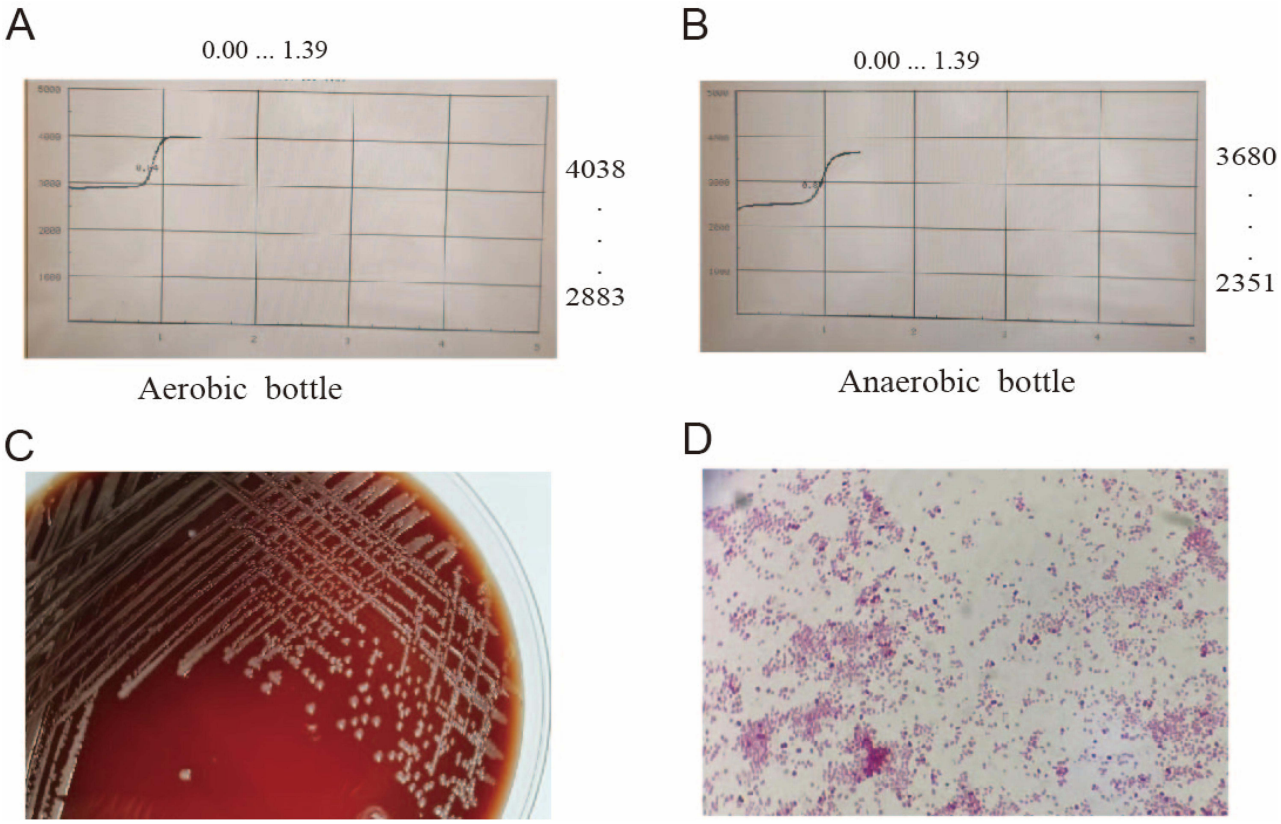

Figure 3 General overview of Neisseria sicca. (**A** and **B**) Growth curve of *N. sicca* under anaerobic (**A**) and microaerobic (**B**) conditions, respectively. (**C**) Columbia blood agar plate with growth of *N.sicca*. (**D**) Staining revealed Gram-negative diplococcus.

(Figure 3A, B and D). The organism grew on blood agar as irregular, elevated, smooth, dry, opaque, and yellowish colonies (Figure 3C). The isolated organism (*N. sicca* junzhu_1) was identified using MALDI-TOF MS and verified using 16S rRNA sequencing. Sepsis was diagnosed on the basis of the presence of this organism in the bloodstream. Antibiotic sensitivity testing indicated sensitivity to the current antibiotics, and the treatment was continued (Table 1).

After 8 days of treatment, the patient showed overall improvement with no fever or other symptoms. Follow-up blood culture results were negative. Pre-discharge blood tests showed a WBC count of 6.6×10^9/L with a neutrophil ratio of

Table 1 MIC Values (μg/Ml) of N. Sicca Junzhu_1

Antibiotics	MIC (μg/mL)	Sensitivity
Amoxicillin-clavulanate	<=4	S
Ampicillin	<=1	S
Tetracycline	<=2	S
Cefotaxime	<=2	S
Cefuroxime	<=4	S
Rifampin	<=1	S
Trimethoprim-sulfamethoxazole	<=0.5	S
Chloramphenicol	<=2	S
Cefaclor	<=8	S
Ofloxacin	<=2	S
Azithromycin	6 mm (Kirby-Bauer method)	R
Erythromycin	6 mm (Kirby-Bauer method)	R
β-lactamase	Negative	-

Abbreviations: S, Susceptible; R, Resistant.

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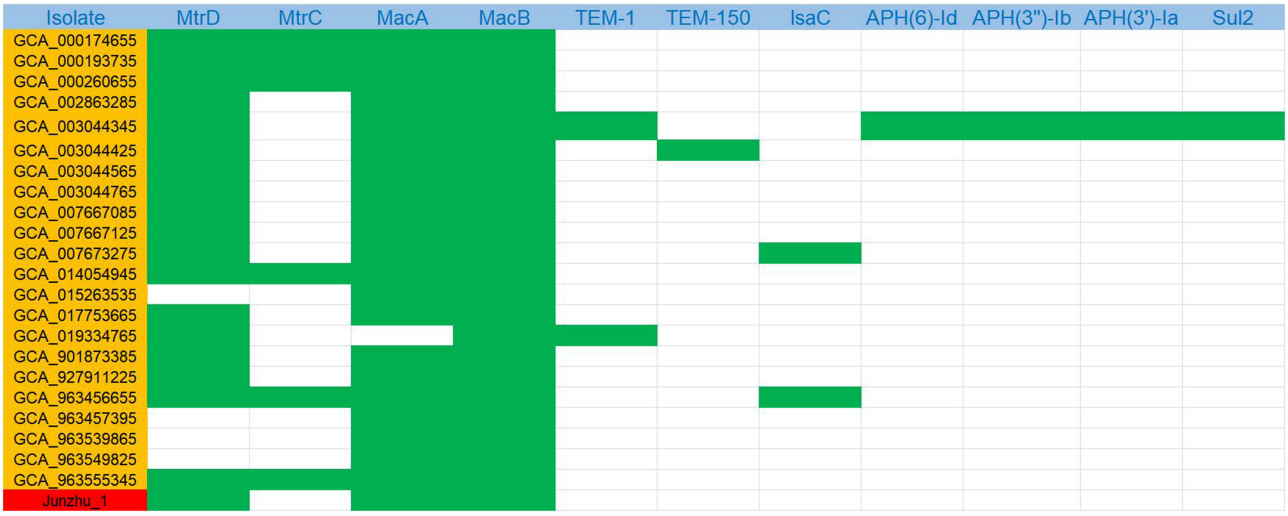

Figure 4 Distribution of antimicrobial resistance genes among 23 *N. sicca* isolates. Green rectangles indicate the presence of resistance genes in the corresponding isolates.

71.9%, CRP of 55.3 mg/L, and PCT of 0.99 ng/mL. The patient was discharged with instructions to continue oral amoxicillin-clavulanate for one week and to follow up regularly in the outpatient clinic. During the 18-month post-discharge follow-up period, the patient remained afebrile with no recurrence of cough or other infectious symptoms.

General Genomic Features of N. sicca Junzhu_1

The genome of *N. sicca* junzhu_1 comprises 2,420,190 bp and exhibits a G+C content of 51.21%. The strain was predicted to have 68 contigs, 2129 coding sequences (CDSs), and 61 RNA genes [58 tRNAs, one 5S rRNA, one 16S rRNA, and one 23S rRNA]. The genome sequence of *N. sicca* junzhu_1 has been deposited in NCBI GenBank under the accession number SRR29061210.

The Antimicrobial Resistance of N. sicca Junzhu_1

N. sicca junzhu_1 is susceptible to a variety of antimicrobial agents including amoxicillin-clavulanate, ampicillin, tetracycline, cefotaxime, cefuroxime, rifampin, trimethoprim-sulfamethoxazole, chloramphenicol, cefaclor, and ofloxacin. However, the strain was resistant to azithromycin and erythromycin (Table 1). Consistent with these findings, the resistome of *N. sicca* junzhu_1 comprised three antimicrobial resistance genes: *macB, macA*, and *mtrD* (Figure 4).

Virulence Genes in N. sicca Junzhu_1

Virulence gene analysis showed that the pathogenicity of *N. sicca* junzhu-1 was related to the MntABC pathway (*mntB* and *mntC*), efflux pump system (*farA, farB, mtrD*, and *mtrE*), oxidative damage (*msrA*/*B* [*pilB*]), lipooligosaccharide synthesis pathway (*rfaF* and *rfaC*), membrane receptor (*hpuB*), motor protein (*pilT*), catalase (*KatA*), and DNA repair (*recN*), which may be related to the poor prognosis of infection.

Phylogenetic Analysis of 23 N. sicca Strains

To elucidate the phylogenetic relationships between *Neisseria* strains from different regions and our strain, junzhu_1, 22 genomic datasets from the NCBI database (Table S1), and the sequence of junzhu_1 were used to construct a phylogenetic tree. These isolates were primarily distributed in the USA (17, accounting for 73.91%), and the majority of hosts from which they were isolated were humans (n=19, 82.61%). The isolates were obtained from various samples, including oral cavity, nasopharynx, blood, urine, and duodenal aspirates. Phylogenetic analysis revealed that the two strains most closely related to the isolated strain junzhu_1 were GCA_007673275 and GCA_017753665. Strain GCA_017753665 was recovered from a blood sample in Zhejiang, China, and differed by 3585 single nucleotide polymorphisms (SNPs) in the same region (Figure 5).

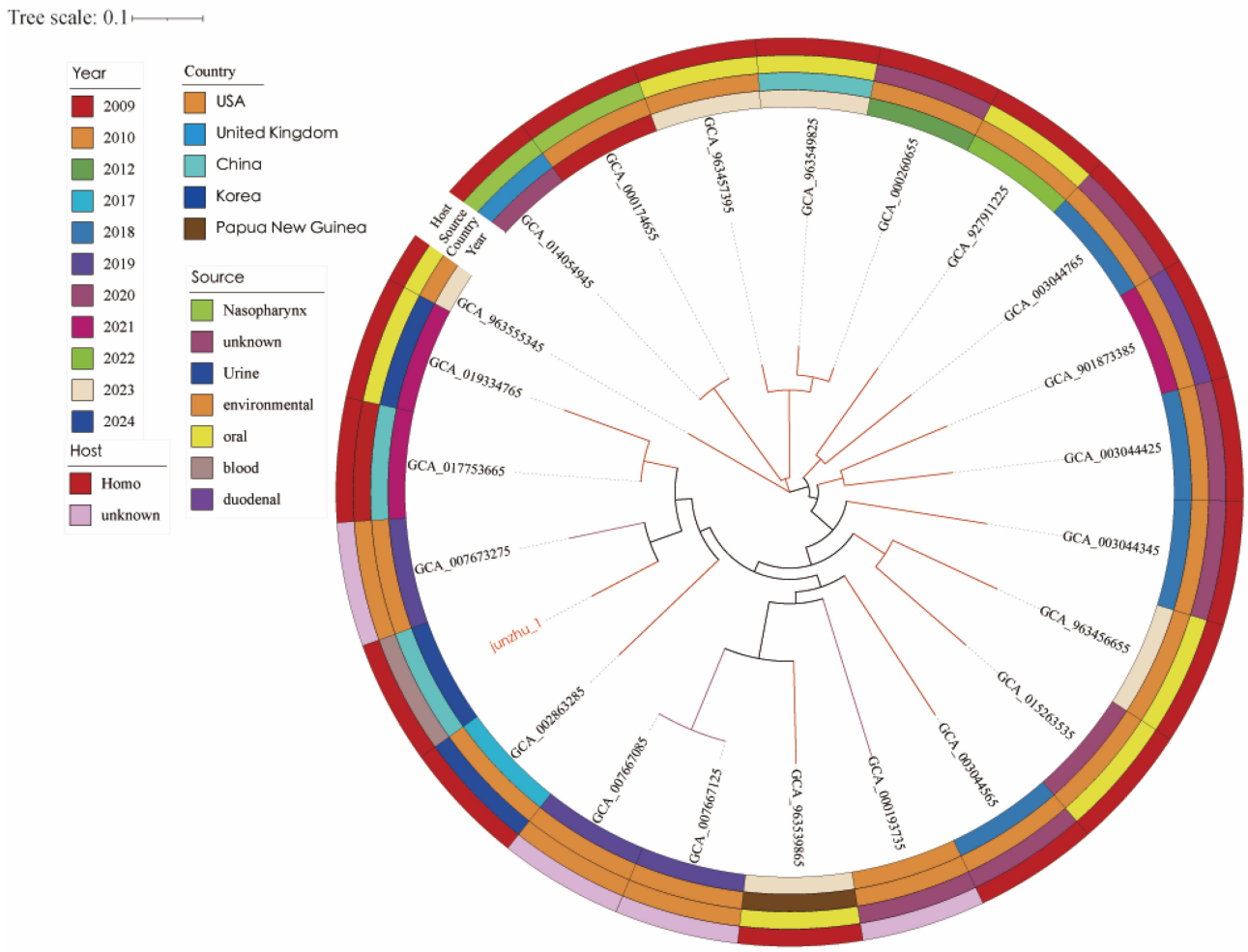

Figure 5 Recombination-filtered core genome phylogeny for a total of 23 *Neisseria sicca* isolates worldwide deposited in the NCBI GenBank database. The isolation date, host, source and country are represented by squares of different colors. The isolate junzhu_I recovered in this study is highlighted in red font.

Discussion

This case report presents a patient with a history of AVR who was confirmed to be infected with *N. sicca*, the origin of the infection remains elusive.

Upon admission, the patient presented with a localized inflammatory lesion in the right lung lobe, as revealed by chest CT. Blood cultures were positive for *N. sicca*, whereas sputum cultures revealed the presence of normal upper respiratory tract flora, including *N. sicca*. Unfortunately, bronchoalveolar lavage fluid was not obtained from the bacterial culture. While the exact cause of the lesion is uncertain, *N. sicca* infection is a possible contributor, as it has been reported to cause pneumonia in prior cases.[6,7] Following antibiotic treatment, the pulmonary lesion resolved completely and the patient's temperature normalized. However, the patient was readmitted one month later with fever, and subsequent blood cultures again detected *N. sicca*, CT imaging showed no pulmonary or other lesions. Comparative echocardiography from both admissions showed no evidence of vegetation on the cardiac valves.

Diagnosing prosthetic valve endocarditis is challenging and requires multiple imaging techniques beyond standard microbiological analysis.[27] The complexity arises from device-related infection characteristics, biofilm formation, and limitations in imaging interpretation.[27,28] Considering the possibility of infective endocarditis, a negative TTE result does not definitively exclude the diagnosis, as vegetations may be too small, located in atypical positions, or obscured by suboptimal image quality. Therefore, transesophageal echocardiography (TEE) should be considered, alongside other diagnostic modalities such as cardiac CT or PET/CT, to confirm or rule out occult infective endocarditis.[29]

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To our knowledge, only two cases of BSIs caused by *N. sicca* following AVR have been reported. Cheng et al[30] described a male patient with a history of AVR who presented with 10-day fever. Although TTE was negative for vegetation, TEE revealed vegetation on the aortic valve. The isolated *N. sicca* was found to be sensitive to meropenem, trimethoprim-sulfamethoxazole, chloramphenicol, and minocycline, but resistant or insensitive to penicillin, azithromycin, ciprofloxacin, and cefuroxime. After an 8-week treatment regimen, the patient's blood cultures were negative, and the follow-up TEE showed no evidence of vegetation. Locke et al[31] reported the case of a female patient with a history of AVR and pacemaker implantation who presented with recurrent fever. Although TTE was negative, TEE revealed two vegetations in the lead of the right atrial pacemaker. The patient subsequently underwent complete pacemaker and lead extraction, received initial intravenous ertapenem, and was later transitioned to ceftriaxone. The patient fully recovered and was discharged after completing a 6-week course of intravenous cefuroxime at home.

We retrieved 22 *N. sicca* sequences from GenBank and analyzed them together with the strain junzhu_1 from this study. Most strains were of human origin (n = 19), followed by environmental sources (n = 3), with one strain of unknown origin, suggesting that *N. sicca* can inhabit both human hosts and environmental niches. Within humans, it primarily colonizes the upper respiratory tract but has also been isolated from blood, the gastrointestinal tract, and urine. Notably, two bloodstream-derived strains were identified, including junzhu_1 and NS20201025 (GCA_017753665.1). Phylogenetic analysis revealed that NS20201025, one of the closest relatives of junzhu_1, was isolated from Zhejiang—the same geographic region as our strain. This strain has been reported to cause native-valve endocarditis complicated by multiple embolic cerebral infarctions in a patient with underlying heart disease. Collectively, these findings suggest that the strain junzhu_1 may have pathogenic potential for infective endocarditis.

In the present study, we performed whole-genome sequencing of the isolated *N. sicca* junzhu-1 strain to further study its pathogenicity and resistance. *N.sicca* junzhu-1 harbors virulence factors, including fatty acid efflux system genes (*MtrCDE* and *FarAB-MtrE*), Type IV pili (*PilT*), lipooligosaccharides (involving *rfaC* and *rfaF* genes), and reactive oxygen species (ROS) defense mechanisms (involving *Recn, KatA, MntABC*, and *MsrA*/*B*). These virulence genes are also present in the genomes of pathogenic *N. gonorrhoeae* and *N. meningitidis* and may contribute to poor infection prognosis.

MtrCDE and FarAB-MtrE efflux pumps enhance resistance against host-derived long-chain fatty acids by relying on the outer membrane protein MtrE for export. The expression of these pumps may be differentially regulated by the transcriptional regulatory protein MtrR.[32] Type IV pili (TFP) are crucial for bacterial attachment to host cells, with PilT proteins mediating retraction and intimate attachment.[33] Lipooligosaccharides (LOS) are key virulence factors that are involved in immune evasion, tissue attachment, and host cell invasion. Its biosynthesis involves branched oligosaccharide production linked to lipid A via two 3-deoxy-D-manno-2-octulosonic acid (KDO) molecules, with rfaC and rfaF playing essential roles.[34] ROS play crucial roles in bacterial physiology and stress responses. Both *N. meningitidis* and *N. gonorrhoeae* possess ROS defense mechanisms essential for survival. These include RecN (a putative zinc metalloprotease), KatA (catalase), MntABC (an ABC-type Mn transporter), and MsrA/B (methionine sulfoxide reductases), which collectively contribute to ROS detoxification.[35] Płaczkiewicz et al[36] demonstrated that both *N. gonorrhoeae* and N. *sicca* induce the secretion of pro-inflammatory cytokines, including IL-6 and TNF-α, as well as chemokines CXCL8 and CCL20, in infected epithelial cells. A study[37] found that commensal species of *Neisseria*, specifically *N. sicca* and *N. lactamica*, can cause toxic damage to cultured human endothelial cells. Considering the virulence factors of *N. sicca* and its ability to elicit inflammatory responses, a potential link exists between its pathogenicity and its ability to cause BSIs.

Recent research suggests that commensal *Neisseria* species serve as reservoirs for antibiotic resistance genes, particularly those conferring resistance to azithromycin and erythromycin, which can be horizontally transferred to pathogenic *Neisseria* species.[38] Consistent with this, *N. sicca* junzhu_1 harbored resistance determinants-including *macB, macA*, and *mtrD*-that are associated with resistance to both azithromycin and erythromycin. *N. lactamica* harbors mutated *gyrA* and *penA*, leading to resistance to quinolones and penicillin, respectively.[39] One study found that the blaTEM gene, responsible for β-lactamase production, was present in 93.9% of *Neisseria* spp. isolates, all of which showed resistance to penicillin.[40] Genomic analysis of the 23 *N. sicca* isolates revealed resistance patterns, all of which harbored multiple macrolide resistance genes (including *mtrC, mtrD, marA, marB*, and *lsaC*), whereas specific isolates carried additional β-

lactamase genes (TEM-1 in strains GCA_003044345 and GCA_019334765; TEM-50 in GCA_963456655) (Figure 4). Fortunately, most blaTEM-positive commensal *Neisseria* spp. are susceptible to cephalosporins.[40] However, the commensal species, *N. cinerea* and *N. elongata*, demonstrated resistance to ceftriaxone.[38]

To the best of our knowledge, published reports indicates that *N. sicca* is generally susceptible to third-generation cephalosporins, β-lactamase inhibitors, penicillins, and quinolones. However, no CLSI criteria are currently available for this organism, and breakpoints for *Neisseria meningitidis* were therefore applied as a reference in susceptibility testing. In patients with AVR who develop BSIs concomitant with infective endocarditis, treatment poses additional challenges due to the potential formation of bacterial biofilms on prosthetic materials, which can reduce antibiotic efficacy and increase the risk of persistent infection.[41] Therefore, combination therapy—including aminoglycosides or rifampin—may be considered to enhance antimicrobial activity and target biofilm-associated bacteria,[42,43] with the final regimen tailored according to local resistance patterns and individual susceptibility results.

This case report had several limitations. First, the definitive diagnosis of endocarditis in our patient remains elusive, as we lacked TEE and other advanced imaging modalities such as cardiac CT or PET/CT, relying solely on TTE. Second, in the absence of CLSI susceptibility breakpoints for *N. sicca*, we used ATB HAEMO CLSI (12) strips, referring to the susceptibility breakpoints for *Haemophilus influenzae*. Third, although the virulence genes of *N. sicca* have been identified through whole-genome sequencing, their expression levels have not yet been verified using qPCR. Furthermore, we did not perform in vivo animal model experiments to validate virulence.

Conclusions

In summary, this case study provides the first genomic insight into the virulence and resistance characteristics of *N. sicca* implicated in BSIs in patients with AVR. These findings provide valuable information for future research on the pathogenicity and antibiotic resistance mechanisms of this microorganism in clinical contexts. Although *N. sicca* is generally susceptible to β-lactams, determining its sensitivity is highly advisable for guiding appropriate therapies. *N. sicca* commonly resides as a commensal bacterium on the mucosal surface of the human URT. However, its potential pathogenicity, particularly in BSIs arising from underlying heart valve disease, warrants further investigation.

Data Sharing Statement

Data supporting the findings of this study are openly available at https://trace.ncbi.nlm.nih.gov/Traces/index.html?view=run_browser&acc=SRR29061210&display=metadata reference number SRR29061210.

Ethics Approval and Consent to Participate

Whole genome sequencing, along with a case report, was approved by the Ethics Committee of Sanmen People's Hospital in Taizhou, China.

Patient Consent for Publication

The patient provided written informed consent for the publication of case details and accompanying images.

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Disclosure

The authors declare that they have no competing interests in this work.

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