

Risk Factors for Complicated Viridans Group Streptococcal Bacteremia in Adults: Impact of Oral Health

Kota Minami

Saga University Hospital

Takashi Matono

tmatono@cc.saga-u.ac.jp

Saga University Hospital

Yusuke Oka

Saga University Hospital

Toshiharu Urakami

Saga University Hospital

Yosuke Aoki

Saga University Hospital

Research Article

Keywords: Viridans group streptococci, invasive disease, complicated infection, Streptococcus anginosus group, odontogenic infection

Posted Date: June 29th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10045568/v1>

License:  This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Additional Declarations: Competing interest reported. TM has received speaker honoraria from Pfizer, Meiji Seika Pharma, GSK, MSD, Gilead Sciences, and Daiichi Sankyo. Other authors declare no conflicts of interest.

Version of Record: A version of this preprint was published at BMC Infectious Diseases on October 13th, 2026. See the published version at <https://doi.org/10.1186/s12879-026-14603-w>.

Abstract

Background

Viridans group streptococci can cause invasive disease. However, predictors of complicated infection in adult viridans group streptococcal bacteremia, including the role of oral sources, remain unclear.

Methods

This single-center retrospective cohort study was conducted at Saga University Hospital, Japan, among adults (≥ 18 years) with viridans group streptococci isolated from blood cultures between September 2014 and June 2025. Patients with polymicrobial bacteremia, cardiopulmonary arrest on arrival, contamination, or interhospital transfer were excluded. Complicated bacteremia was defined as infective endocarditis or metastatic pyogenic infection with isolation of viridans group streptococci from a sterile site other than blood. Multivariable logistic regression was used to identify independent risk factors.

Results

Among 164 patients, 54 (33%) developed complicated infection, most commonly infective endocarditis (41%), intra-abdominal abscess/peritonitis (22%), and deep neck infection (15%). Median age was similar between complicated and uncomplicated cases (68 vs. 70 years, $p = 0.81$). White blood cell counts ($p < 0.0001$) and C-reactive protein levels ($p = 0.0005$) were higher in complicated cases. In-hospital mortality rate was 0% in complicated cases and 5% in uncomplicated cases ($p = 0.18$). In multivariable analysis, independent predictors for complicated infection were community acquisition (adjusted odds ratio [aOR], 4.89; 95% confidence interval [CI], 2.31–10.8), odontogenic source (aOR, 3.07; 95%CI, 1.44–6.70), and *Streptococcus anginosus* group bacteremia (aOR, 2.87; 95%CI, 1.33–6.47).

Conclusions

One-third of adults with viridans group streptococcal bacteremia developed complicated infection. Community acquisition, odontogenic origin, and *S. anginosus* group etiology may help prioritize early imaging, dental evaluation, and timely source control.

Background

Viridans group streptococci are part of the normal flora of the oropharyngeal, urogenital, and gastrointestinal microbiota and are often referred to as oral streptococci[1]. Although generally considered commensal organisms, they comprise a heterogeneous group capable of causing invasive infections, including abscesses, bacteremia with neutropenic fever, and infective endocarditis[2, 3, 4, 5, 6]. In clinical practice, viridans group streptococci are recognized as important causative pathogens and

are included among the major criteria in the modified Duke criteria for the diagnosis of infective endocarditis[7]. In a Danish nationwide study, 7.3% of *Streptococcus* spp. bloodstream infections were accompanied by infective endocarditis [8].

In *Staphylococcus aureus* bacteremia, risk factors for invasive or complicated infection are well established, including persistent bacteremia, community acquisition, and presence of cardiac implantable devices[9], whereas comparatively little is known about predictors of complicated infection in viridans group streptococcal bacteremia. Previous studies have suggested species- and subspecies-level differences in clinical manifestations among viridans group streptococci. For example, the *Streptococcus mutans* group and *S. sanguinis* group are frequently detected in patients with infective endocarditis[10], whereas the *S. mitis* group is commonly observed in patients with febrile neutropenia[2, 3]. However, findings have varied across studies, and underlying clinical implications remain poorly understood. In addition, limited evidence is available regarding the causal link between oral health status as a potential portal of entry and development of severe invasive infections caused by viridans group streptococci.

In the era of rapid and accurate microbial identification enabled by matrix-assisted laser desorption ionization–time of flight mass spectrometry (MALDI-TOF MS), we aimed to identify clinical and microbiological factors associated with complicated invasive infections in viridans group streptococcal bacteremia requiring antimicrobial therapy or invasive source control.

Methods

Study design and setting

This retrospective cohort study was conducted at Saga University Hospital (SUH) in Saga, Japan, a tertiary care teaching hospital with 602 inpatient beds. The annual number of blood culture specimens processed at SUH ranged from 11,643 to 13,970, recently. At SUH, infectious disease (ID) physicians routinely provide consultations for all patients with positive blood cultures, offering standardized assessment of infection source, antimicrobial management, and coordination of timely surgical or interventional source control when indicated. The clinical data used in this study were obtained from a laboratory database and by reviewing medical charts at SUH. This study was conducted according to the recommendations of the STROBE statement for observational studies.

Study population

Potentially eligible participants were patients aged ≥ 18 years who had viridans group streptococci isolated from at least one blood culture specimen obtained either in the outpatient setting or during hospitalization between September 2014 and June 2025. Exclusions included bacteremic episodes in which organisms other than viridans group streptococci were isolated in the same episode, patients who presented with cardiopulmonary arrest on arrival, cases deemed to be contamination by ID physicians,

and patients who were transferred to another hospital before diagnostic evaluation or outcome ascertainment. Multiple positive culture sets from a single admission were counted as one episode, and repeat positive cultures during the same admission period were considered part of the index episode.

Bacterial identification and taxonomy

Blood culture bottles were incubated in a BACTEC FX system (Becton, Dickinson, and Co. Japan, Tokyo, Japan) for up to 7 days. Bacterial isolates recovered from blood cultures were identified by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) using a MALDI Biotyper (Bruker Daltonics, Kanagawa, Japan). Viridans group streptococci were categorized into *Streptococcus mitis*, *S. sanguinis*, *S. anginosus*, *S. salivarius*, and *S. mutans* groups based on previous reports[11, 12].

Measurements and definitions

Extracted data included patient characteristics including age, sex, body mass index, comorbidities required to calculate the Charlson Comorbidity index[13], clinical presentation including acquisition setting, vital signs, presence of febrile neutropenia, adjudicated source of infection classified as odontogenic or non-odontogenic with other infectious foci described, intensive care unit admission, and in-hospital death. Laboratory values were collected at presentation, including leukocyte count and C-reactive protein levels measured closest to the time of the index positive blood culture. Findings related to infectious foci were reviewed, including ultrasonography, transthoracic and transesophageal echocardiography, computed tomography, magnetic resonance imaging, and operative or interventional radiology reports, when available.

Viridans group streptococcal bacteremia was defined as at least one positive blood culture for viridans group streptococci in the context of a compatible clinical syndrome for which clinicians provided antimicrobial therapy. Community-acquired infection was defined as a strain identified at the outpatient clinic or within 48 hours of admission. Febrile neutropenia was defined, in accordance with the Japanese Society of Medical Oncology clinical practice guidelines, as fever of at least 38.0°C once or at least 37.5°C sustained with an absolute neutrophil count less than 500 cells/ μ L or an anticipated decline to this level within 48 hours; this temperature threshold is lower than that of the Infectious Diseases Society of America definition (single oral temperature $\geq 38.3^\circ\text{C}$ or $\geq 38.0^\circ\text{C}$ sustained for ≥ 1 hour). An odontogenic source required a primary oral or dental focus determined by the attending physicians and/or ID physicians, with dental consultation when obtained. This included clinical evidence of dental infection such as dental abscess, periodontal infection, or periapical disease, with or without supportive imaging and no alternative source judged more likely. Complicated viridans group streptococcal bacteremia was defined as pyogenic infection in which viridans group streptococcal species was isolated from a sterile site (except for blood), such as deep-seated tissue; abscess; and cerebrospinal, intraocular, pleural, pericardial, or joint fluids, and infective endocarditis. Although echocardiography was

not performed in all patients, clearance of bacteremia was confirmed on follow-up blood cultures, and episodes without persistent bacteremia were classified as uncomplicated bacteremia.

Statistical analysis

Patients and microbiological characteristics were compared between complicated bacteremia and uncomplicated bacteremia using chi-square (χ^2) or Fisher's exact tests for nominal variables and Mann–Whitney U tests for continuous variables. To identify factors independently associated with complicated bacteremia, a multivariable logistic regression model was constructed with covariates selected for clinical plausibility and relevance. The model was adjusted for age ≥ 65 years and sex to account for potential bias in the analysis of complicated bacteremia. It was also adjusted for community acquisition, odontogenic infection, and *S. anginosus* group, as these have been reported as risk factors for complicated bacteremia and severe infections in previous reports[14, 15, 16]. A two-tailed p value < 0.05 was considered statistically significant, and the odds ratio (OR) and 95% confidence interval (95% CI) were calculated. All analyses were performed using JMP, version 18 (SAS Institute Japan Ltd., Tokyo, Japan).

Results

Baseline characteristics of patients and microorganisms

Of 249 potentially eligible patients hospitalized between September 2014 and June 2025, 85 were excluded, leaving 164 for analysis (Fig. 1). Among them, 54 patients (33%) met criteria for complicated bacteremia, while 110 patients (67%) were classified as having uncomplicated bacteremia. Patients with complicated bacteremia had a median age of 68 years (interquartile range [IQR], 57–78), compared with 70 years (IQR, 60–76) in uncomplicated cases ($p = 0.81$, Table 1). Charlson comorbidity index was lower in complicated cases than in uncomplicated cases (median 0.5 [IQR 0–2] vs. 2 [IQR 1–3], $p < 0.0001$). Hematological malignancy (0% vs. 28%, $p < 0.0001$) and febrile neutropenia (0% vs. 31%, $p < 0.0001$) were less frequently observed in complicated cases. Diabetes mellitus was noted in 15% of complicated cases and 27% of uncomplicated cases ($p = 0.076$). Inflammatory biomarkers were higher in complicated cases: total leukocytes (median $10.7 \times 10^3/\mu\text{L}$ vs. $6.7 \times 10^3/\mu\text{L}$, $p < 0.0001$) and C-reactive protein (98 mg/L vs. 43 mg/L, $p = 0.0005$). Community acquisition (59% vs. 25%, $p < 0.0001$) and odontogenic infection (54% vs. 29%, $p = 0.0022$) were significantly associated with complicated bacteremia. In-hospital mortality rate was 0% in complicated cases and 5% in uncomplicated cases ($p = 0.18$). Among the 54 patients with complicated bacteremia, the most frequent infectious foci were infective endocarditis (41%), followed by abdominal abscess or peritonitis (22%) and deep neck space infection (15%, Table 2). In the overall cohort ($n = 164$), the most common causative microorganism was *S. anginosus* group ($n = 73$, 45%), followed by *S. mitis* group ($n = 55$, 34%) and *S. sanguinis* group ($n = 23$, 14%). Species distribution differed between complicated and uncomplicated bacteremia (Table 1). *S.*

anginosus group was more frequently isolated in complicated bacteremia (56% vs. 39%, $p = 0.046$), whereas *S. mitis* group was less frequent in complicated bacteremia (22% vs. 39%, $p = 0.032$).

Table 1

Patient and microbiological characteristics of complicated bacteremia and uncomplicated bacteremia caused by viridans streptococci (n = 164)

Age (years), median (IQR)	Complicated bacteremia (n = 54)	Uncomplicated bacteremia (n = 110)	p value
	68 (57–78)	70 (60–76)	0.81
Male gender, n (%)	40 (74)	68 (62)	0.12
Body mass index (kg/m ²), median (IQR)	22 (19–24)	21 (19–23)	0.75
Charlson comorbidity index, median (IQR)	0.5 (0–2)	2 (1–3)	< 0.0001
Comorbidity, n (%)			
Chronic hepatitis	7 (13)	23 (14)	0.22
Chronic kidney disease	6 (11)	16 (15)	0.54
Solid tumor	10 (19)	32 (29)	0.15
Hematological malignancy	0 (0)	31 (28)	< 0.0001
Diabetes mellitus	8 (15)	30 (27)	0.076
HIV infection	0 (0)	0 (0)	
quick SOFA score, median (range)	0 (0–2)	0 (0–3)	0.21
Total leukocytes (×10 ³ /μL), median (IQR)	10.7 (9.0–15.4)	6.7 (0.7–10.6)	< 0.0001
C-reactive protein (mg/L), median (IQR)	98 (50–169)	43 (14–128)	0.0005
Isolate group, n (%)			
<i>S. mutans</i>	0 (0)	1 (1)	0.48
<i>S. salivarius</i>	4 (7)	15 (14)	0.31
<i>S. anginosus</i>	30 (56)	43 (39)	0.046
<i>S. sanguinis</i>	9 (17)	14 (13)	0.49
<i>S. mitis</i>	12 (22)	43 (39)	0.032
HIV, human immunodeficiency virus; IQR, interquartile range; qSOFA, quick Sequential Organ Failure Assessment			

Age (years), median (IQR)	Complicated bacteremia (n = 54)	Uncomplicated bacteremia (n = 110)	p value
	68 (57–78)	70 (60–76)	0.81
Community acquired infections, n (%)	32 (59)	27 (25)	< 0.0001
Odontogenic infection, n (%)	29 (54)	32 (29)	0.0022
Febrile neutropenia condition, n (%)	0 (0)	34 (31)	< 0.0001
Intensive care unit admission, n (%)	6 (11)	18 (16)	0.37
In-hospital mortality, n (%)	0 (0)	6 (5)	0.18
HIV, human immunodeficiency virus; IQR, interquartile range; qSOFA, quick Sequential Organ Failure Assessment			

Table 2
Site of infections in patients with complicated bacteremia (n = 54)

Site of infection	n (%)
Infective endocarditis	22 (41)
Abdominal abscess/peritonitis	12 (22)
Deep neck space infection	8 (15)
Bone and joint infection	5 (9)
Empyema/lung abscess	4 (7)
Vascular graft infection	1 (2)
Meningitis	1 (2)
Perianal abscess	1 (2)

Risk factors for complicated bacteremia

Unadjusted logistic regression analysis showed that *S. anginosus* group bacteremia, community acquired infection, and odontogenic infection, were significantly associated complicated bacteremia (Table 3). After adjustment for age, sex, *S. anginosus* group infection, community acquisition, and odontogenic source (see Methods), multivariable logistic regression identified three independent risk factors for complicated bacteremia: community acquired infection (adjusted odds ratio [aOR], 4.89; 95%CI, 2.31–10.8; $p < 0.0001$); odontogenic source (aOR, 3.07; 95%CI, 1.44–6.70; $p = 0.0035$); and *S. anginosus* group bacteremia (aOR, 2.87; 95%CI, 1.33–6.47; $p = 0.0068$).

Table 3
Logistic regression analysis of variables in the prediction of complicated bacteremia

Variables	OR (95% CI)	Adjusted OR (95%CI)	p value
Aged ≥ 65 years	0.93 (0.48–1.83)	0.73 (0.33–1.61)	0.44
Male gender	1.76 (0.87–3.71)	1.01 (0.44–2.33)	0.98
Diabetes mellitus	0.46 (0.19–1.05)		
<i>S. anginosus</i> group bacteremia	1.95 (1.01–3.79)	2.87 (1.33–6.47)	0.0068
Community acquired infections	4.47 (2.25–9.09)	4.89 (2.31–10.8)	< 0.0001
Odontogenic infection	2.83 (1.45–5.60)	3.07 (1.44–6.70)	0.0035
OR, odds ratio; CI, confidence interval			

Discussion

In this cohort of adults with viridans group streptococcal bacteremia, approximately one-third of patients developed complicated infection. Community acquisition, odontogenic source, and *S. anginosus* group bacteremia were each independently associated with complicated infection. Notably, the presence of these features in patients with viridans group streptococcal bacteremia may serve as practical clinical cues to prompt evaluation for infective endocarditis and invasive infection.

The present study yielded three key findings. First, *S. anginosus* group bacteremia may be a potential risk factor for complicated infection among patients with viridans group streptococcal bacteremia. Although viridans group streptococci are generally regarded as organisms with relatively low virulence, 33% of patients in our cohort developed complicated infection, most commonly infective endocarditis and intra-abdominal infections. This prevalence is consistent with a previous report in which infective endocarditis was observed in 24.6% of patients with viridans group streptococcal bacteremia[4]. Among viridans group streptococci, *S. anginosus* group—particularly *S. intermedius* and *S. constellatus*—has been reported to have a tendency toward abscess formation.[5, 6]. Beyond these observations, our study provides more robust evidence by showing, through multivariable analysis, that bacteremia caused by *S. anginosus* group was independently associated with complicated infection. In contrast, several studies have shown that *S. mutans* group and *S. sanguinis* group are more prevalent than *S. anginosus* group in patients with infective endocarditis[10]. This discrepancy may reflect differences in outcome definitions, as our study included pyogenic infections and other metastatic infections in addition to infective endocarditis. Metastatic pyogenic infections, as well as infective endocarditis, are clinically important because they require surgical drainage and prolonged antimicrobial therapy. Given the well-recognized propensity of *S. anginosus* group for abscess formation[5, 6], our findings may offer practical insights for clinical decision-making.

Second, odontogenic source was independently associated with complicated infection, emphasizing the potential role of early dental assessment and preventive interventions. Poor oral hygiene can generally lead to invasive infections, including infective endocarditis, brain abscess, lung abscess, osteomyelitis, and prosthetic joint infection[16]. Previous studies shown that approximately 8% of infective endocarditis cases were associated with periodontal or dental diseases in the absence of a dental procedure[17]. Therefore, accumulating evidence suggests that bacteremia from odontogenic infections correlates with the severity of oral inflammation, underscoring the importance of good oral health in the prevention of infective endocarditis[18]. However, among patients with viridans group streptococcal bacteremia, several studies have failed to show a significant association between odontogenic infections and infective endocarditis or pyogenic infections[19]. In addition, odontogenic infection is not included in the HANDOC score for predicting infective endocarditis in patients with non- β -hemolytic streptococcal bacteremia[14]. Taken together, our findings highlight the need for further studies to clarify the potential causal relationship between odontogenic infections and metastatic pyogenic infections in patients with viridans group streptococcal bacteremia.

Third, community-acquired infection was associated with an increased risk of complicated infection. In *Staphylococcus aureus* bacteremia, community acquisition has consistently been linked to a higher risk of complicated bacteremia[9, 20], possibly reflecting prolonged bacteremia prior to treatment initiation and a greater propensity for metastatic infection. Similarly, community acquisition has been reported as a risk factor for infective endocarditis in viridans group streptococcal bacteremia, consistent with our findings[14]. Conversely, in hospital-acquired cases, early detection and prompt initiation of treatment may help prevent the development of complicated infections. In our cohort, hematological malignancy and febrile neutropenia were more frequently observed in patients with uncomplicated bacteremia, most of whom had hospital acquisition, which may partly explain our findings. Interestingly, consistent with our findings, a previous study showed that the presence of malignancy, particularly hematological malignancy, is independently associated with a reduced risk of infective endocarditis in patients with viridans group streptococcal bacteremia[4]. Thus, our observations may provide additional insight into the clinical impact of community acquisition and underlying hematological malignancy in this population.

The present study has several limitations. First, because this was a single-center study conducted at a tertiary care hospital, referral bias and limited generalizability should be considered. However, it is noteworthy that the exclusion of polymicrobial infections enabled a clearer evaluation of species- and subspecies-level diversity. Second, transthoracic echocardiography was not systematically performed in all patients, which may have led to misclassification of complicated and uncomplicated bacteremia. Nevertheless, all cases were co-managed by ID physicians who conducted systematic assessments, supporting the overall reliability of the clinical classification in this study. Third, identification of odontogenic sources relied on clinical assessment and may have been subject to misclassification and detection bias. However, standardized evaluations by ID physicians and appropriate referrals to oral surgery services likely improved the accuracy of odontogenic source identification. Finally, the modest sample size restricted the number of covariates included in the multivariable analysis to preserve model

stability, leaving the possibility of residual confounding. In particular, hematological malignancy was absent in the complicated bacteremia group, limiting inference regarding its association with complicated infection and highlighting the need for validation in larger cohorts.

Conclusion

In this study, complicated infection occurred frequently among adult patients with viridans group streptococcal bacteremia. Community acquisition, odontogenic infection, and *S. anginosus* group bacteremia emerged as independent predictors of complicated disease, providing practical clinical cues for early diagnostic evaluation and management. Recognition of these factors may facilitate timely identification of infective endocarditis and pyogenic infection and support more targeted clinical decision-making. Furthermore, these findings underscore the potential importance of proactive multidisciplinary management and preventive oral care strategies in reducing the risk of severe infection.

Abbreviations

aOR

Adjusted odds ratio

CI

Confidence interval

ICMJE

International Committee of Medical Journal Editors

ID

Infectious disease

IQR

Interquartile range

MALDI-TOF MS

Matrix-assisted laser desorption/ionization time-of-flight mass spectrometry

OR

Odds ratio

STROBE

Strengthening the Reporting of Observational Studies in Epidemiology

SUH

Saga University Hospital

Declarations

Ethics approval and consent to participate

This study was approved by the Institutional Review Board of Saga University Hospital (approval number 2024-09-07). Because of the retrospective design using deidentified data, the requirement for written

informed consent was waived by the Institutional Review Board, and all patient information was anonymized to the extent possible. All procedures were conducted in accordance with the ethical standards of the Declaration of Helsinki and institutional policies for patient privacy and data security.

Consent for publication

Not applicable.

Competing Interests

TM has received speaker honoraria from Pfizer, Meiji Seika Pharma, GSK, MSD, Gilead Sciences, and Daiichi Sankyo. Other authors declare no conflicts of interest.

Funding

No funding was received for this study.

Author Contribution

KM: Data curation, Investigation, Visualization, Writing – original draft.

TM: Conceptualization, Formal analysis, Project administration, Supervision, Visualization, Writing – original draft.

YO: Writing – review & editing.

TU: Writing – review & editing.

YA: Writing – review & editing.

All authors read and approved the final version of the manuscript, and all authors meet the ICMJE criteria for authorship.

Acknowledgement

We thank the physicians, nurses, and clinical and technical staff at the Saga University Hospital for their excellent work.

Availability of data and materials

The deidentified data underlying this article are not publicly available because of institutional privacy restrictions and the conditions of ethics approval. The data underlying this article will be shared on reasonable request to the corresponding author.

References

1. Doern CD, Burnham CAD. It's not easy being green: the viridans group streptococci, with a focus on pediatric clinical manifestations. *J Clin Microbiol*. 2010;48(11):3829–35. 10.1128/JCM.01563-10.
2. Nielsen MJ, Claxton S, Pizer B, et al. Viridans group streptococcal infections in children after chemotherapy or stem cell transplantation: a 10-year review from a tertiary pediatric hospital. *Med (Baltim)*. 2016;95(9):e2952. 10.1097/MD.0000000000002952.
3. Marron A, Carratalà J, González-Barca E, Fernández-Sevilla A, Alcaide F, Gudiol F. Serious complications of bacteremia caused by viridans streptococci in neutropenic patients with cancer. *Clin Infect Dis*. 2000;31(5):1126–30. 10.1086/317460.
4. Bae J, Park JH, Lee M, et al. Clinicomicrobiological risk factors for infective endocarditis in viridans group streptococci bacteraemia. *J Antimicrob Chemother*. 2024;79(9):2327–33. 10.1093/jac/dkae231.
5. Jiang S, Li M, Fu T, Shan F, Jiang L, Shao Z. Clinical characteristics of infections caused by *Streptococcus anginosus* group. *Sci Rep*. 2020;10(1):9032. 10.1038/s41598-020-65977-z.
6. Suzuki H, Hase R, Otsuka Y, Hosokawa N. Bloodstream infections caused by *Streptococcus anginosus* group bacteria: a retrospective analysis of 78 cases at a Japanese tertiary hospital. *J Infect Chemother*. 2016;22(7):456–60. 10.1016/j.jiac.2016.03.017.
7. Delgado V, Ajmone Marsan N, de Waha S, et al. 2023 ESC Guidelines for the management of endocarditis. *Eur Heart J*. 2023;44(39):3948–4042. 10.1093/eurheartj/ehad193.
8. Østergaard L, Bruun NE, Voldstedlund M, et al. Prevalence of infective endocarditis in patients with positive blood cultures: a Danish nationwide study. *Eur Heart J*. 2019;40(39):3237–44. 10.1093/eurheartj/ehz327.
9. Tong SYC, Fowler VG, Skalla L, Holland TL. Management of *Staphylococcus aureus* bacteremia: a review. *JAMA*. 2025;334(9):798–808. 10.1001/jama.2025.4288.
10. Deas G, Lee TC, Colston J, et al. Streptococcal endocarditis: a meta-analysis of species dependant risk. *EClinicalMedicine*. 2025;87:103425. 10.1016/j.eclinm.2025.103425.
11. Facklam R. What happened to the streptococci: overview of taxonomic and nomenclature changes. *Clin Microbiol Rev*. 2002;15(4):613–30. 10.1128/CMR.15.4.613-630.2002.
12. Abranches J, Zeng L, Kajfasz JK, et al. Biology of oral streptococci. *Microbiol Spectr*. 2018;6(5). 10.1128/microbiolspec.GPP3-0042-2018.
13. Charlson ME, Pompei P, Ales KL, MacKenzie CR. A new method of classifying prognostic comorbidity in longitudinal studies: development and validation. *J Chronic Dis*. 1987;40(5):373–83. 10.1016/0021-9681(87)90171-8.

14. Sunnerhagen T, Törnell A, Vikbrant M, Nilson B, Rasmussen M. HANDOC: a handy score to determine the need for echocardiography in non-β-hemolytic streptococcal bacteremia. Clin Infect Dis. 2018;66(5):693–8. 10.1093/cid/cix880.
15. Isa K, Matsuo T, Onishi K, Mori N. Predicting bacteremia caused by *Streptococcus anginosus* group: a new scoring method. J Infect Chemother. 2022;28(12):1628–31. 10.1016/j.jiac.2022.08.018.
16. Li X, Kolltveit KM, Tronstad L, Olsen I. Systemic diseases caused by oral infection. Clin Microbiol Rev. 2000;13(4):547–58. 10.1128/CMR.13.4.547.
17. Drangsholt MT. A new causal model of dental diseases associated with endocarditis. Ann Periodontol. 1998;3(1):184–96. 10.1902/annals.1998.3.1.184.
18. Bumm CV, Folwaczny M. Infective endocarditis and oral health—a narrative review. Cardiovasc Diagn Ther. 2021;11(6):1403–15. 10.21037/cdt-20-908.
19. Thoresen T, Jordal S, Lie SA, Wünsche F, Jacobsen MR, Lund B. Infective endocarditis: association between origin of causing bacteria and findings during oral infection screening. BMC Oral Health. 2022;22(1):491. 10.1186/s12903-022-02509-3.
20. Fowler VG, Olsen MK, Corey GR, et al. Clinical identifiers of complicated *Staphylococcus aureus* bacteremia. Arch Intern Med. 2003;163(17):2066–72. 10.1001/archinte.163.17.2066.

Figures

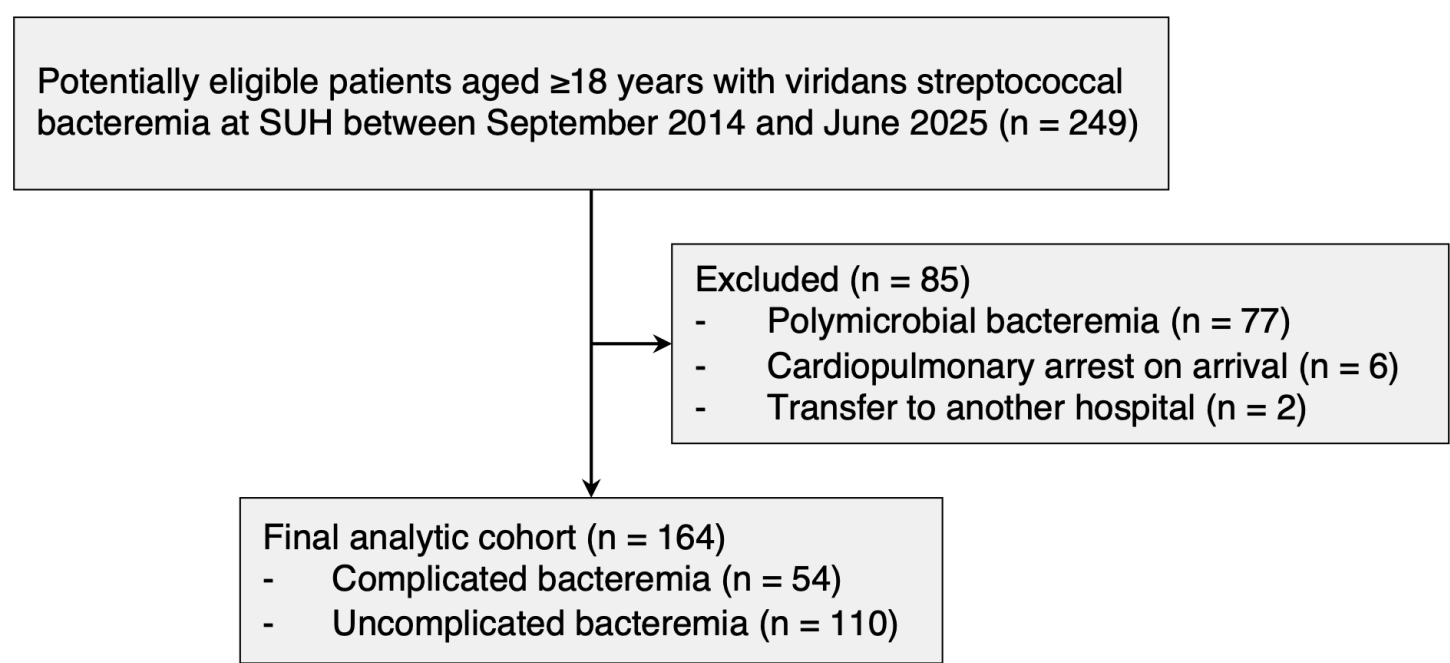


Figure 1

Flow diagram of patient selection

Flow diagram showing the selection of adults with viridans group streptococcal bacteremia, including exclusions and the final analytic cohort.

