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Case Report

Treatment of infection caused by animal commensal pathogen *Staphylococcus simulans*

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Abstract

Introduction: *Staphylococcus simulans* is an animal pathogen belonging to the coagulase-negative staphylococci (CoNS) group that was occasionally identified in the skin of healthy people. Although it was suggested that *S. simulans* share some virulence factors with *S. aureus*, little is known about human infections, and clinical relevance is limited to a few reports. Herein, we report a case of bacteremia caused by *S. simulans*.

Case presentation: A 39-year-old man was brought to the hospital due to heart attack. During the course of admission, his procalcitonin, C-reactive protein, and erythrocyte sedimentation rate were elevated. He was empirically started on vancomycin and meropenem. However, meropenem was discontinued after the identification of the fact that *S. simulans* was susceptible to oxacillin, as well as the identification of *Streptococcus mitis* in blood culture. Repeat blood culture a day after antibiotic initiation was persistently positive for *S. simulans* only, while the second repeat blood culture 3 days after antibiotic initiation was negative. The patient remained hemodynamically stable, and the laboratory values were back to normal. He continued the antibiotic course for a total of 14 days and was discharged in stable condition.

Conclusions: CoNS should not be easily discarded as a sample contaminant and pathogen identification to species level is crucial. Clinicians should be aware of the virulence potential of *S. simulans*.

Key words: bacteremia; animal pathogen; *Staphylococcus simulans*; coagulase-negative staphylococci; case report.

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Introduction

Coagulase-negative staphylococci (CoNS) are commonly isolated in clinical practice. They belong to the Gram-positive cocci group which lack the virulence factor coagulase [1]. Although 50 species have been identified, 6 species are the most prevalent and well described in the literature: *Staphylococcus epidermidis*, *Staphylococcus haemolyticus*, *Staphylococcus lugdunensis*, *Staphylococcus saprophyticus*, *Staphylococcus hominis*, and *Staphylococcus capitis* [2]. *Staphylococcus simulans* is a CoNS, and is considered a common animal pathogen, especially of sheep, cattle, and other domestic animals; however, it has been reported that this bacterium was occasionally detected in human skin, as well as in urethra of healthy women [3,4]. Little is known about *S. simulans* infections in humans and data on its clinical relevance is limited to a few case reports. It is not generally considered in differential diagnosis and rarely reported as a clinical isolate in humans. Herein, we report a case of bacteremia caused by *S. simulans*, followed by a review of literature on previous case reports.

Case report

The patient was a 39-year-old man who had cerebral palsy and he was receiving treatment with vitamin D3 (50,000 international units every month). He was brought to the hospital due to cardiac arrest, which was witnessed in the rehab center; cardiopulmonary resuscitation (CPR) was done before arrival. Upon arriving at the hospital, the initial rhythm was pulseless electrical activity. CPR was continued as per the advanced cardiovascular life support protocol for 3 cycles and return of spontaneous circulation was achieved. The patient had poor appetite and feeding for the last 3 days before the admission. He refused to take his breakfast on the day of admission and before the cardiac arrest episode. Then, he became drowsy and unresponsive with no pulse. The patient was afebrile.

Laboratory studies on the day of admission showed mild leucopenia with a white blood cell count of 3.010 $\times 10^9$/L, normal hemoglobin (117 gm/L), and mild thrombocytopenia with a platelet count of 118 $\times 10^9$/L. Liver enzymes such as aspartate aminotransferase (2260 unit/L), alanine aminotransferase (1585 unit/L), and alkaline phosphatase (396 unit/L) were elevated.

Renal function parameters such as serum creatinine were normal (9 µmol/L) and blood urea nitrogen was high (13.40 mmol/L). Potassium and phosphorus levels were high: 6.10 mmol/L and 2.59 mmol/L, respectively. High values of inflammatory markers (procalcitonin, 3.44 ng/mL; C-reactive protein, 111.740 mg/L; erythrocyte sedimentation rate, 23 mm/hr) and cardiovascular biomarkers (brain natriuretic peptide, 423.80 pg/mL; Troponin-T, 34.46 ng/L; lactate, 2.10 mmol/L) were detected. The rest of the chemistry profile was unremarkable. Chest X-ray revealed left middle lobe consolidation, suggestive of aspiration pneumonia.

Blood samples were collected on the day of admission, and transferred to the microbiology laboratory where a Gram-stain procedure was performed. The samples were then inoculated in 3 different media (5% sheep blood agar, MacConkey agar, and chocolate agar plates) and stored in the incubator at 37 ºC for 18–24 hours. Pathogen identification and susceptibility testing to the often-used antibiotics were conducted after bacterial growth, using the automated MicroScan Walk Away 96 Plus (Beckman Coulter, Inc, Brea, CA, USA). The automated system identified the pathogens, including the percentage of assurance and susceptibility to different antimicrobials (susceptible, intermediate, or resistant) (Table 1).

Empiric treatment was initiated in the emergency department on the day of admission, including meropenem 1 g every 8 hours, and vancomycin 550 mg every 12 hours adjusted for trough level of 15–20 mcg/mL for 4 days. The next day, preliminary results of the cultures revealed Gram-positive cocci in clusters; therefore, meropenem was discontinued. The final blood culture results showed that *S. simulans* was susceptible to oxacillin, and the presence of the *Streptococcus mitis* group. Vancomycin was given for 4 days, during which two more blood samples were collected. The first repeat blood culture—collected a day after vancomycin initiation—was persistently positive for *S. simulans* only; the second repeat blood culture—collected 3 days after vancomycin initiation—was negative. Given that the isolate was oxacillin-susceptible (Table 1), vancomycin was switched to cefazolin 2 g every 8 hours for 4 days. The patient was stable during the 4 days of cefazolin treatment; however, the decision was made to discontinue cefazolin and re-start vancomycin due to more experience with vancomycin in CoNS bacteremia. Vancomycin was continued for a total duration of 14 days with therapeutic drug monitoring adjusted for appropriate trough level. The patient remained hemodynamically stable while on vancomycin, and the laboratory values were back to normal. He was then discharged in stable condition.

Table 1. Antimicrobial susceptibility data (blood cultures).

Drugs	MIC interpretation
Staphylococcus simulans	
Penicillin	R
Oxacillin	S
Gentamicin	S
Levofloxacin	S
Moxifloxacin	S
Erythromycin	S
Clindamycin	S
Linezolid	S
Vancomycin	S
Teicoplanin	S
Tetracycline	S
Tigecycline	S
Trimethoprim-sulfamethoxazole	S
Streptococcus mitis	
Penicillin	S
Ampicillin	S
Ceftriaxone	S
Levofloxacin	R
Moxifloxacin	I
Clindamycin	S
Linezolid	S
Teicoplanin	S
Vancomycin	S
Tetracycline	S
Tigecycline	S

Discussion

This case report describes a case of bacteremia of unknown source caused by *S. simulans* that was successfully treated with vancomycin. This case is of interest since human infections due to *S. simulans* are rarely reported, and this could be attributed to several factors. Firstly, many clinical laboratories do not identify CoNS to the species level, which may contribute to the rarity of reporting. In addition, when CoNS are isolated from humans in clinical settings, they are often considered as contaminants, given that they are occasionally commensal members of skin microflora [2,5]. Therefore, the infections caused by these pathogens are mostly overlooked. Nevertheless, in this case, repeated culture resulted in the same strain within 5 days of initial culture. This warranted consideration of *S. simulans* as pathogen, as proposed by Beekmann *et al.* [6]. In addition, time to positivity was < 16 hours which was also predictive of infection as proposed by García-Vázquez *et al*. [7]. Further, this bacterium is a common animal pathogen and is rarely found in the human skin [3,4]. Therefore, it was mainly identified in patients with frequent animal exposure. In this case, no history of contact with animals was identified and the mode of acquisition of infection

Table 2. Literature review of previously reported cases of *Staphylococcus simulans* infections.

Date	Country	Author	Age / gender	Diagnosis	Blood culture of *S. simulans*	Other culture *of S. simulans*	Antibiotic used	Duration	Antibiotic susceptibility	Outcome	Animal/ Farm Exposure
FEB 1985	New York	Males *et al*. [17]	39/M	Right ankle, osteomyelitis and septic arthritis	Positive	Positive tissue culture	TM and OX. OX replaced by IV PEN on day 4 for 9 days then switched to oral medications	NS	Pan-sensitive	Resolved	NS
MAR 1991	Australia	McCarthy *et al.* [18]	80/M	Endocarditis	Positive	Urine culture revealed only scanty growth of skin flora	FLU IV for 26 days then FLU oral	NS	NS	Died	NS
MAR 1992	France	Jansen *et al.* [19]	56/M	Native valve endocarditis	Positive	NS	IV PEN for 4 weeks and GM for 2 weeks	4 weeks	NS	Resolved	NS
MAY 1993	London, UK	Sturgess *et al*. [20]	77/F	Right pubic osteomyelitis	Positive	NS	FLU IV for 11 days, then PO as well as FA	FLU for 25 days then restarted for additional 31 days	Pan-sensitive	Resolved	NS
SEP 1993	Brooklyn, New York	De Jesus *et al*. [21]	84/M	Septicemia	Positive	NS	Not treated	NS	ME-sensitive	Died	NS
SEP 1993	Brooklyn, New York	De Jesus *et al.* [21]	41/M	Septicemia	Positive	NS	TMP-SMX, TIM, and GM. VA was added after the culture results	NS	ME-sensitive	Died	NS
SEP 1993	Brooklyn, New York	De Jesus *et al*. [21]	63/F	Septicemia and pneumonia	Positive	CoNS staph in sputum	ERY and CXM	NS	NS	Resolved	NS
SEP 1993	Brooklyn, New York	De Jesus *et al*. [21]	58/F	Septicemia	Positive	NS	SAM then VA	2 weeks	ME-resistant	Resolved	NS
OCT 2001	Rochester, United States	Razonable *et al*. [22]	70/M	Vertebral osteomyelitis and prosthetic joint infection	NS	Positive biopsy specimen from T9-10 disk space and intra-operative cultures	CZ then CRO for 6 weeks then CN for 4 weeks	10 weeks	Pan- sensitive	Resolved	Farmer active in birthing and milking cows; drank unpasteurized milk
MAY 2008	Athens, Greece	Vallianou *et al*. [23]	46/M	Vertebral osteomyelitis and native valve endocarditis	Positive	NS	VA for 4 weeks, then TEC for 5 months. CM was added after 3 months.	6 months	ME-resistant	Resolved	Yes
JAN 2010	Texas, USA	Kline *et al*. [24]	65/M	Abscess and osteomyelitis of right foot	Positive	Positive bone culture	VA	16 days	AM, CIP, CM, OX, PEN, CRO resistant	Resolved	Yes
DEC 2011	France	Désidéri-Vaillant *et al*. [25]	66/F	Osteomyelitis	NS	Positive aspiration of the abscesses before debridement	GM and AMC, then GM and AMX for 10 days then replaced by OFL and RA.	3 months	Pan- sensitive	Resolved	NS
JUL 2014	Dallas, USA.	Kirkwood *et al.* [26]	64/F	Mycotic inferior mesenteric artery aneurysm secondary to native valve endocarditis	Positive	NS	Daptomycin	6 weeks	NS	Resolved	No
MAY 2016	Madrid, Spain	Tous Romero *et al*. [27]	60/M	Pyoderma of left hand	NS	Positive tissue culture	AZM	3 days	NS	Resolved	Yes
DEC 2016	Iowa, USA	Shields *et al*. [14]	80/M	Right great toe cellulitis	NS	Positive tissue culture	CN for 7 days and DO for 14 days then TMP-SMX	28 days	Tetracycline resistant	Resolved	NS
OCT 2018	Worcester, USA	Lal *et al*. [13]	80/F	Pleural Empyema	Negative	Positive from pleural fluid	LVX and SAM, then VA	NS	Susceptible to VA and CM only	Died	No
OCT 2020	Manchester, UK	Power *et al.* [28]	58/M	Endocarditis of native aortic and mitral valves	Positive	NS	VA and TZP then switched to FLU, RA, and GM	6 weeks. GM used for 2 weeks	NS	Died	Yes
JAN 2021	Japan	Goda *et al*. [29]	75/F	Toxic shock syndrome	Positive	NS	MEM, VA, and CM then CZ and CM	The 2nd regimen administered for 14 days	NS	Resolved	NS
MAY 2021	Minnesota, USA	Go *et al*. [30]	78/M	Bloodstream infection	Pre-OP: negative post-OP: positive	Positive device cultures	VA and FEP then VA alone.	2 weeks	Pan- sensitive	Died	NS but he is retired farmer
JUN 2021	Georgia, USA	Drobeniuc *et al*. [31]	61/F	UTI	NS	Positive urine culture	CRO then TMP-SMX	NS	Resistant to fluoroquinolones	Resolved	Living in farm
JUN 2021	Georgia, USA	Drobeniuc *et al*. [31]	56/M	UTI	NS	Positive urine culture	CRO, then FOS then TMP-SMX PO	10 days	Resistant to fluoroquinolones	Resolved	Visiting family farm
SEP 2024	China	Tan *et al*. [32]	57/F	Osteoarthritis of the right knee underwent high supracondylar osteotomy	NS	Positive puncture fluid	LZD	4 weeks	NS	Resolved	NS
SEP 2024	Manila, Philippines	Ortiz and Batac [33]	46/F	Botryomycosis	NS	Positive tissue culture	TMP-SMX	10 months	NS	Decrease in the size of foot. Resolved draining sinus.	Wet market vendor with recurrent exposure to fresh meat

CoNS: coagulase-negative *Staphylococcus*; F: female; IV: intravenously; M: male; NS: not specified; PO: orally; post-OP: postoperative; pre-OP: perioperative; UTI: urinary tract infection. AM: ampicillin; AMC: amoxycillin–clavulanic acid; AMX: amoxicillin; AZM: azithromycin; CIP: ciprofloxacin; CM: clindamycin; CN: cephalexin; CRO: ceftriaxone; CXM: cefuroxime; CZ: cefazolin; DO: doxycycline; ERY: erythromycin; FA: fusidic acid; FEP: cefepime; FLU: flucloxacillin; FOS: fosfomycin; GM: gentamicin; LVX: levofloxacin; LZD: linezolid; ME: methicillin; MEM: meropenem; OFL: ofloxacin; OX: oxacillin; PEN: penicillin; RA: rifampin; SAM: ampicillin/sulbactam; TEC: teicoplanin; TIM: ticarcillin-clavulanic acid; TM: tobramycin; TMP-SMX: sulfamethoxazole-trimethoprim; TZP: piperacillin-tazobactam; VA: vancomycin.

remained unclear. It has been reported that intravascular catheters and skin breaches are possible routes of transmission for this bacterium, but they are not likely in this case given that there were no signs of catheter or skin-related infection. Although *S. simulans* is not a typical respiratory pathogen, the possibility of pulmonary involvement cannot be excluded given the X-ray findings and some previously reported cases which showed pulmonary involvement (Table 2).

Table 2 summarizes the previously reported cases of *S. simulans* infections. The infection sites in previously reported cases varied, and most likely included osteomyelitis and endocarditis. Comparable to the present case, bacteremia with unknown origin has also been reported (Table 2). Urinary tract, respiratory, and skin sources were also reported. Similar to the present case, several cases reported oxacillin-susceptible isolates. The majority of previous reports did not specify animal exposure, which, similar to the present case, may indicate the absence of animal source and open the possibility of other sources. Consistent with this case, infections in the majority of previous cases were resolved.

CoNS are generally recognized as low virulent organisms compared to *S. aureus*. However, the number of CoNS strains sequenced is constantly rising, and, consequently, the number of virulence factors identified in those strains is increasing [8]. Furthermore, virulence potential varies considerably between different CoNS species depending on the virulence factors they harbor. *Staphylococcus simulans* shares some virulence factors with *S. aureus*, including staphylococcal enterotoxins (se), tissue necrosis cytotoxin Panton–Valentine leukocidin (pvl), exfoliative toxins (eta, etb), and toxic shock syndrome toxin-1 (tst) [9–11]. Some strains of *S. simulans* are able to form a capsule which make them more resistant to phagocytosis by human granulocytes [12]. Methicillin-resistance gene (*mecA*) is also shared between the two species and these strains are able to horizontally transfer their genes within the *Staphylococcus* genus which results in more methicillin-resistant strains [9,13,14].

The antimicrobial susceptibilities for the isolate identified in this study are detailed in Table 1. Although pan-sensitive *S. simulans* were reported in a number of cases; *S. simulans* resistant to β-lactam, fluroquinolones, and tetracyclines have been previously reported (Table 2). It should be noted that methicillin-resistant *S. simulans* has been previously reported [15]. In the present case, although the isolate was oxacillin susceptible, the decision was made to switch back from cefazolin to vancomycin, primarily due to more experience with vancomycin in CoNS bacteremia and the repeat negative blood culture that was achieved while the patient was on empiric vancomycin. In addition, according to the guidelines from the Infectious Diseases Society of America (IDSA), vancomycin has dosing advantages over oxacillin or nafcillin in CoNS bacteremia [16]. The present case provided further evidence that *S. simulans* has virulence potential to cause infections that resemble *S. aureus* infections.

Conclusions

CoNS should not be easily discarded as a sample contaminant and pathogen identification to species level is crucial. In this regard, clinicians should be aware of the virulence potential of this pathogen. Given the variable susceptibilities among strains and possible acquisition of drug-resistant genes, isolates should be analyzed to monitor for antibiotic resistance. Detailed social history is important to identify the risk factors of *S. simulans* which include animal contact. Clinicians should also be careful not to rule out the possibility of *S. simulans* infections if animal exposure was not reported.

Ethics approval

This research followed the relevant guidelines and regulations of the Helsinki Declaration. Institutional Review Board (IRB) approval was granted for this case (E-25-9796). The need for informed consent was waived by the Human Research Ethics Committee.

Authors' contributions

All the authors made significant contributions to the study. TA: conceptualization, investigation, original draft writing and editing, literature review, data extraction, quality assessment, and supervision; AE, SD, FM: screening of previous studies, data collection, manuscript review and editing.

All named authors meet the International Committee of Medical Journal Editors (ICMJE) criteria for authorship for this article, take responsibility for the integrity of the work as a whole, and have given their approval for this version to be published.

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Conflict of interest

No conflict of interest is declared.

References

1. Becker K, Both A, Weißelberg S, Heilmann C, Rohde H (2020) Emergence of coagulase-negative staphylococci. Expert Rev Anti Infect Ther 18: 349–366. doi: 10.1080/14787210.2020.1730813.
2. Michels R, Last K, Becker SL, Papan C (2021) Update on coagulase-negative staphylococci—what the clinician should know. Microorganisms 9: 830. doi: 10.3390/microorganisms9040830.
3. Oztürkeri H, Kocabeyoğlu O, Yergök YZ, Koşan E, Yenen OS, Keskin K (1994) Distribution of coagulase-negative staphylococci, including the newly described species *Staphylococcus schleiferi*, in nosocomial and community acquired urinary tract infections. Eur J Clin Microbiol Infect Dis 13: 1076–1079. doi: 10.1007/BF02111833.
4. Nicolle LE, Hoban SA, Harding GK (1983) Characterization of coagulase-negative staphylococci from urinary tract specimens. J Clin Microbiol 17: 267–271. doi: 10.1128/jcm.17.2.267-271.1983.
5. Chowdhury S, Fong SS (2020) Computational modeling of the human microbiome. Microorganisms 8: 197. doi: 10.3390/microorganisms8020197.
6. Beekmann SE, Diekema DJ, Doern GV (2005) Determining the clinical significance of coagulase-negative staphylococci isolated from blood cultures. Infect Control Hosp Epidemiol 26: 559–566. doi: 10.1086/502584.
7. García-Vázquez E, Fernández-Rufete A, Hernández-Torres A, Canteras M, Ruiz J, Gómez J (2013) When is coagulase-negative *Staphylococcus* bacteraemia clinically significant? Scand J Infect Dis 45: 664–671. doi: 10.3109/00365548.2013.797599.
8. França A, Gaio V, Lopes N, Melo LDR (2021) Virulence factors in coagulase-negative staphylococci. Pathogens 10: 170. doi: 10.3390/pathogens10020170.
9. Unal N, Cinar OD (2012) Detection of staphylococcal enterotoxin, methicillin-resistant and Panton-Valentine leukocidin genes in coagulase-negative staphylococci isolated from cows and ewes with subclinical mastitis. Trop Anim Health Prod 44: 369–375. doi: 10.1007/s11250-011-0032-x.
10. Davis MF, Cain CL, Brazil AM, Rankin SC (2013) Two coagulase-negative staphylococci emerging as potential zoonotic pathogens: wolves in sheep's clothing? Front Microbiol 4: 123. doi: 10.3389/fmicb.2013.00123.
11. Lina G, Piémont Y, Godail-Gamot F, Bes M, Peter MO, Gauduchon V, Vandenesch F, Etienne J (1999) Involvement of Panton-Valentine leukocidin-producing *Staphylococcus aureus* in primary skin infections and pneumonia. Clin Infect Dis 29: 1128–1132. doi: 10.1086/313461.
12. Ohshima Y, Schumacher-Perdreau F, Peters G, Quie PG, Pulverer G (1990) Antiphagocytic effect of the capsule of *Staphylococcus simulans*. Infect Immun 58: 1350–1354. doi: 10.1128/iai.58.5.1350-1354.1990.
13. Lal A, Akhtar J, Ullah A, Abraham GM (2018) First case of pleural empyema caused by *Staphylococcus simulans*: review of the literature. Case Rep Infect Dis 2018: 7831284. doi: 10.1155/2018/7831284.
14. Shields BE, Tschetter AJ, Wanat KA (2016) *Staphylococcus simulans*: an emerging cutaneous pathogen. JAAD Case Rep 2: 428–429. doi: 10.1016/j.jdcr.2016.08.015.
15. Barnham M, Horton R, Smith JM, Richardson J, Marples RR, Reith S (1996) Methicillin-resistant *Staphylococcus simulans* masquerading as MRSA in a nursing home. J Hosp Infect 34: 331–333. doi: 10.1016/S0195-6701(96)90113-9.
16. Mermel LA, Allon M, Bouza E, Craven DE, Flynn P, O'Grady NP, Raad, II, Rijnders BJ, Sherertz RJ, Warren DK (2009) Clinical practice guidelines for the diagnosis and management of intravascular catheter-related infection: 2009 update by the Infectious Diseases Society of America. Clin Infect Dis 49: 1–45. doi: 10.1086/599376.
17. Males BM, Bartholomew WR, Amsterdam D (1985) *Staphylococcus simulans* septicemia in a patient with chronic osteomyelitis and pyarthrosis. J Clin Microbiol 21: 255–257. doi: 10.1128/jcm.21.2.255-257.1985.
18. McCarthy JS, Stanley PA, Mayall B (1991) A case of *Staphylococcus simulans* endocarditis affecting a native heart valve. J Infect 22: 211–212. doi: 10.1016/0163-4453(91)91899-9.
19. Jansen B, Schumacher-Perdreau F, Peters G, Reinhold G, Schönemann J (1992) Native valve endocarditis caused by *Staphylococcus simulans*. Eur J Clin Microbiol Infect Dis 11: 268–269. doi: 10.1007/BF02098097.
20. Sturgess I, Martin FC, Eykyn S (1993) Pubic osteomyelitis caused by *Staphylococcus simulans*. Postgrad Med J 69: 927–929. doi: 10.1136/pgmj.69.818.927.
21. de Jesus L, Caruso G, de Caprariis P, Ditchek A, Visconti E (1993) *Staphylococcus simulans* septicemia. Infect Dis Clin Pract 2: 351–352. doi: 10.1097/00019048-199309000-00008.
22. Razonable RR, Lewallen DG, Patel R, Osmon DR (2001) Vertebral osteomyelitis and prosthetic joint infection due to *Staphylococcus simulans*. Mayo Clin Proc 76: 1067–1070. doi: 10.4065/76.10.1067.
23. Vallianou N, Evangelopoulos A, Makri P, Zacharias G, Stefanitsi P, Karachalios A, Avgerinos PC (2008) Vertebral osteomyelitis and native valve endocarditis due to *Staphylococcus simulans*: a case report. J Med Case Rep 2: 183. doi: 10.1186/1752-1947-2-183.
24. Kline DA (2010) *Staphylococcus simulans* osteomyelitis of the foot: a case report. The Foot and Ankle Online Journal. Available: https://faoj.org/2010/01/01/staphylococcus-simulans-osteomyelitis-of-the-foot-a-case-report/. Accessed: 6 April 2026.
25. Désidéri-Vaillant C, Nédelec Y, Guichon JM, Le Louarn S, Noyer V, Sapin-Lory J, Le Guen P, Nicolas X (2011) *Staphylococcus simulans* osteitis in a diabetic patient. Diabetes Metab 37: 560–562. doi: 10.1016/j.diabet.2011.09.006.
26. Kirkwood ML, Knowles M, Modrall JG, Valentine RJ (2014) Mycotic inferior mesenteric artery aneurysm secondary to native valve endocarditis caused by coagulase-negative *Staphylococcus*. Ann Vasc Surg 28: 1312.e13-5. doi: 10.1016/j.avsg.2013.08.023.
27. Tous Romero F, Gutiérrez García-Rodrigo C, Velasco Tamariz V, Llamas Martín R (2016) Acute infection by *Staphylococcus simulans* in the hand of a man. JAMA Dermatol 152: 1060. doi: 10.1001/jamadermatol.2016.0959.
28. Power NE, Calisti G, Price F, Watt V, Gamlin W, Dobson LE, Ray SG (2020) *Staphylococcus simulans* endocarditis of native aortic and mitral valves. Case report and literature review. Clinical Infection in Practice 7: 100044. doi: 10.1016/j.clinpr.2020.100044.
29. Goda K, Kenzaka T, Hoshijima M, Yachie A, Akita H (2021) Toxic shock syndrome with a cytokine storm caused by *Staphylococcus simulans*: a case report. BMC Infect Dis 21: 19. doi: 10.1186/s12879-020-05731-y.
30. Go JR, Corsini Campioli C, DeSimone D, Sohail MR (2021) *Staphylococcus simulans* bloodstream infection following

CIED extraction. BMJ Case Rep 14: e240309. doi: 10.1136/bcr-2020-240309.
31. Drobeniuc A, Traenkner J, Rebolledo PA, Ghazaryan V, Rouphael N (2021) *Staphylococcus simulans*: a rare uropathogen. IDCases 25: e01202. doi: 10.1016/j.idcr.2021.e01202.
32. Tan L, Wang J, Li M, Zhou Y, Xia B, Zhang M, Yang C (2024) *Staphylococcus simulans* infections in a patient following high supracondylar osteotomy to treat osteoarthritis: a case report. J Surg Case Rep 2024: rjae567. doi: 10.1093/jscr/rjae567.
33. Ortiz YRH, Batac M (2024) Botryomycosis secondary to *Staphylococcus simulans* masquerading as mycetoma in a 46-year-old Filipino female. Acta Med Philipp 58: 64–68. doi: 10.47895/amp.v58i17.9010.