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Characterization of bacterial isolates from patients with hidradenitis suppurativa

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Article

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Abstract

Hidradenitis suppurativa (HS) is a chronic, inflammatory, recurrent, and debilitating skin disease. In Japan, the skin flora of the affected areas of patients with HS has not been fully investigated. This study investigated bacterial species isolated from HS lesions and evaluated their antimicrobial susceptibility. A total of 47 strains isolated from 18 patients with HS at the Toranomon Hospital Dermatology Department were used. Bacterial species were identified using 16S rDNA gene analysis. As a result, coagulase-negative staphylococci were the most frequently isolated species, followed by several other bacteria, including *Streptococcus* spp., *Corynebacterium* spp., *Cutibacterium* spp., *Pseudomonas aeruginosa*, *Bacteroides fragilis*, *Peptoniphilus coxii*, and *Peptoniphilus harei/asaccharolyticus*. We evaluated the susceptibility of these isolates to various antimicrobial agents. All the strains were highly susceptible to fusidic acid and rifampicin. In contrast, *Staphylococcus* spp., *Enterococcus* spp., *Corynebacterium* spp., and *Streptococcus* spp. exhibited high rates of resistance to tetracyclines and macrolides. Detection of antimicrobial resistance genes in these strains revealed that 100% of *Corynebacterium* spp. carried *erm*(X), 60.0% of *Streptococcus* spp. carried *erm*(B), and 60.0% of coagulase-negative staphylococci strains carried *erm*(C). Additionally, 80.0% of *Streptococcus* spp. and 50.0% of *Enterococcus* spp. carried the *tet*(K). Our findings show that bacteria isolated from patients with HS have a high frequency of indigenous bacteria that are resistant to antimicrobial agents. These results highlight the importance of appropriate antimicrobial agents for the treatment of patients with HS.

Introduction

Hidradenitis suppurativa (HS) is a chronic, inflammatory, recurrent, and cutaneous follicular disease. This disease frequently results in the formation of subcutaneous abscesses and fistulas, often accompanied by secondary infections, which severely affect the patient's quality of life.[1] Despite its clinical significance, the prevalence of HS in Japan remains unknown owing to the lack of large-scale epidemiologic studies. In contrast, prevalence rates ranging from 0.1% to 4% have been reported in Europe and the United States.[2, 3, 4, 5] The disease predominantly affects males in Japan, whereas it is more common in women in Europe and the United States. The site of predilection differs between males and females. In men, lesions are commonly found in the perianal and gluteal regions, whereas in women, the axillary and vulvar regions are more commonly affected.[6, 7, 8]

However, HS has not yet been attributed to specific pathogenic microorganisms. Bacterial cultures from HS lesions are often negative, indicating that bacterial involvement is typically secondary rather than a primary feature of the disease.[1, 9] Previous studies have indicated that HS-affected skin harbors increased levels of anaerobic bacteria and opportunistic pathogens, including *Bacteroides* spp., *Corynebacterium* spp., *Porphyromonas* spp., *Peptoniphilus* spp., and *Prevotella spp*. compared with healthy skin, with a concomitant decrease in native skin commensals such as *Cutibacterium* spp.[1, 10, 11] Secondary infections in HS lesions are often polymicrobial, involving coagulase-negative staphylococci (CNS) and anaerobic bacteria.[1, 10, 11] Treatment of HS-associated secondary infections often involves the use of antibiotics with anti-inflammatory properties, including macrolides, lincosamides, and tetracyclines.[1, 10, 11] Rifampicin has shown efficacy by inhibiting suspended bacteria in HS tunnels and has anti-biofilm activity.[12, 13] However, the emergence of antimicrobial-resistant bacteria, especially to tetracyclines, macrolides, rifampicin, and clindamycin, is a major problem in the treatment of secondary infections in patients with HS.[14] Despite the problem of antimicrobial-resistant bacteria, there is a paucity of data on the cutaneous microbiota in HS-affected areas in Japan. Therefore, this study aimed to characterize the bacterial species isolated from patients with HS. Additionally, we performed antimicrobial susceptibility testing and analyzed antimicrobial resistance genes to elucidate the prevalence and characteristics of resistant bacterial strains in these patients.

Results

Bacterial species and number of isolates from HS lesions

Bacteria were isolated from skin lesions, with *Staphylococcus* spp. being the most common (19 strains) (Table 1). This was followed by *Streptococcus* spp. (five strains), *Corynebacterium* spp. (four strains), *Enterococcus* spp. (four strains), and *Cutibacterium* spp. (three strains).

Table 1
Bacterial species and isolate counts from pyogenic hidradenitis suppurativa lesions

Species	No. of Strains	Species	No. of Strains
Staphylococcus		**Enterococcus**	
Coagulase positive		*E. avium*	2
S. aureus	4	*E. faecalis*	1
Coagulase negative		*E. gilvus/raffinosus*	1
S. epidermidis	7	**Others**	
S. lugdunensis	4	*Slackia exigua*	2
S. haemolyticus	2	*Peptoniphylus coxii*	1
S. hominis	1	*Peptoniphilus harei/asaccharolyticus*	1
S. caprae	1	*Finegoldia magna*	1
Streptococcus		*Bacteroides fragilis*	1
S. anginosus	2	*Porphyromonas somerae*	1
S. constellatus/intermedius	1	*Acidaminococcus intestini*	1
S. dysgalactiae	1	*Microbacterium paraoxydans*	1
S. agalactiae	1	*Pseudomonas aeruginosa*	1
Corynebacterium		*Escherichia coli*	1
C. striatum	2	*Proteus mirabilis*	1
C. amycolatum/ieikeium	1		
C. simulans	1		
Cutibacterium			
C. acnes	2		
C. avidum	1		

Major bacterial species by predilection sites

CNS was the most predominant bacterial group isolated from the upper body (33.3%; 9/27 strains), followed by *Corynebacterium* spp. (14.8%; 4/27 strains) (Fig. 1). In contrast, CNS was the predominant bacterial group in the lower body (29.4%; 5/17 strains), followed by *Enterococcus* spp. (23.5%; 4/17 strains).

Antimicrobial susceptibility

The antimicrobial susceptibilities of major bacterial species isolated from patients with HS were evaluated (Table 2). The amoxicillin-resistant strains were identified as *S. aureus* (75%; 3/4 strains), CNS (46.7%; 7/15 strains), and *Streptococcus* spp. (40%; 2/5 strains). Clindamycin-resistant strains were identified in CNS (40%; 6/15 strains), *Corynebacterium* spp. (100%; 4/4 strains), and *Streptococcus* spp. (60%; 3/5 strains). Gentamicin-resistant strains were detected in CNS (40%, 6/15 strains). Tetracycline-resistant or low-susceptibility strains were identified in CNS (46.7%; 7/15 strains), *Streptococcus* spp. (60%; 3/5 strains), and *S. aureus* (25%; 1/4 strains). Bacterial species isolated from the skin lesions showed high susceptibility to rifampicin and fusidic acid.

Table 2
Antimicrobial susceptibility of various bacterial species isolated from hidradenitis suppurativa lesions

Antimicrobial agent	*S. aureus* (n = 4)		CNS (n = 15)		*Corynebacterium* (n = 4)		*Streptococcus* (n = 5)		*C. acnes* (n = 2)		*Enterococcus* (n = 4)	
	MIC range	R%	MIC range	R%	MIC range	R%	MIC range	R%	MIC range	R%	MIC range	R%
Amoxicillin	0.13-2	75	≤ 0.06-4	46.7	≤ 0.13–0.5	N/A	≤ 0.06-≥128	40	≤ 0.06	0	0.25-8	N/A
Oxacillin	0.13-8	25	≤ 0.06-64	30.0	Not tested		Not tested		Not tested		Not tested	
Cefdinir	≤ 0.06–0.13	0	≤ 0.06-4	6.7	0.03–0.13	N/A	≤ 0.06-≥128	40	≤ 0.06	0	2-≥16	N/A
Levofloxacin	0.13-≥256	25	0.13-≥256	30.0	4–64	N/A	0.25-4	0	0.5-8	50	0.5–32	25
Nadifloxacin	≤ 0.06-4	N/A	≤ 0.06-32	N/A	0.5-4	N/A	0.25-4	N/A	0.5-4	N/A	0.25-8	N/A
Roxythromycin	0.06–0.25	0	≤ 0.06-≥256	73.3	16-≥256	N/A	0.13-≥128	60	≤ 0.06-≥256	50	≤ 0.13-≥256	N/A
Clarithromycin	≤ 0.06	0	≤ 0.06-≥256	66.7	4-≥256	N/A	≤ 0.06-≥128	60	≤ 0.06-≥256	50	≤ 0.13-≥256	N/A
Clindamycin	≤ 0.06	0	≤ 0.06-≥256	40.0	4-≥256	100	≤ 0.06-≥128	60	0.13-4	0	8-≥256	N/A
Gentamicin	0.13-8	0	≤ 0.06–128	40.0	≤ 0.13-2	N/A	1–32	N/A	4	N/A	0.25-32	N/A
Tetracycline	0.13–128	25	≤ 0.06–128	46.7	≤ 0.13-32	50	≤ 0.06-32	60	1–4	N/A	≤ 0.13-64	75
Doxycycline	≤ 0.06-16	25	≤ 0.06-16	40.0	≤ 0.13-4	N/A	≤ 0.06-16	60	0.13-2	0	≤ 0.13-8	0
Minocycline	≤ 0.06–0.5	0	≤ 0.06-32	26.7	≤ 0.13-4	N/A	≤ 0.06-32	60	0.13-2	0	≤ 0.13-8	0
Metronidazole	≥ 256	N/A	≥ 256	N/A	≥ 256	N/A	≥ 128	N/A	≥ 256	N/A	≥ 256	N/A
DDS	128-≥256	N/A	8-≥256	N/A	16-≥256	N/A	32-≥128	N/A	≥ 256	N/A	≥ 256	N/A

R%, resistance rate.

MIC range, range of MIC (μg/mL).

N/A, breakpoints were not defined by CLSI and EUCAST.

DDS, Diaminodiphenyl sulfone.

Antimicrobial agent	*S. aureus* (n = 4)		CNS (n = 15)		*Corynebacterium* (n = 4)		*Streptococcus* (n = 5)		*C. acnes* (n = 2)		*Enterococcus* (n = 4)	
	MIC range	R%	MIC range	R%	MIC range	R%	MIC range	R%	MIC range	R%	MIC range	R%
Fusidic acid	≤ 0.06	0	≤ 0.06	0	≤ 0.02–0.03	N/A	1-≥16	N/A	4–8	0	4–8	N/A
Rifampicin	≤ 0.06	0	≤ 0.06	0	≤ 0.02	0	≤ 0.01-≥16	40	≤ 0.06	N/A	0.125-≥32	25
R%, resistance rate.												
MIC range, range of MIC (μg/mL).												
N/A, breakpoints were not defined by CLSI and EUCAST.												
DDS, Diaminodiphenyl sulfone.												

Correlation between antimicrobial use and antimicrobial-resistant bacteria isolation

The association between prior antimicrobial use and the isolation of antimicrobial-resistant strains was analyzed using a questionnaire survey. Resistant strains were detected more frequently in patients with a history of clindamycin use (Fig. 2). No significant association was observed between prior tetracycline use and isolation of resistant strains.

Detection of antimicrobial resistance genes

The detection of macrolide resistance genes revealed that 100% of *Corynebacterium* spp. carried *erm*(X), 60.0% of *Streptococcus* spp. carried *erm*(B), 50.0% of *C. acnes* carried *erm*(X), 60.0% of CNS carried *erm*(C), and 25.0% of *Enterococcus* spp. carried *erm*(B) (Table 3).

Table 3
Detection of antimicrobial resistance genes in each bacterial species

Species	Resistance gene (%)						
	Macrolide				Tetracycline		Aminoglycoside
	erm(X)	*erm*(B)	*erm*(A)	*erm*(C)	*tet*(M)	*tet*(K)	*aacA-aphD*
S. aureus (n = 4)	-	-	-	-	75.0	50.0	25.0
CNS (n = 15)	-	-	6.7	60.0	100	46.7	40.0
Corynebacterium (n = 4)	100	-	-	-	-	-	-
Streptococcus (n = 5)	-	60.0	-	-	60.0	80.0	-
Enterococcus (n = 4)	-	25.0	-	-	50.0	50.0	-
C. acnes (n = 2)	50.0	-	-	-	-	-	-
(-), Not detected.							

The detection of tetracycline resistance genes revealed that 80.0% of *Streptococcus* spp. carried *tet*(K), 50.0% of *Enterococcus* spp. carried *tet*(K), 50.0% of *S. aureus* carried *tet*(K), and 46.7% of CNS carried *tet*(K).

Discussion

In the treatment of patients with HS, the prevention and management of secondary skin lesion infections are crucial, and antimicrobial agents are often used.[14] However, in recent years, antimicrobial resistance has been reported in bacteria isolated from HS lesions, especially to therapeutic agents recommended in the Japanese Society of Dermatology's Guide to Clinical Practice.[14] It has been reported that normal skin bacteria such as *S. aureus*, CNS, *Streptococcus* spp., *Cutibacterium* spp., and *Corynebacterium* spp. and anaerobic bacteria such as *Bacteroides* spp., *Prevotella* spp., and *Peptostreptococcus* spp., and *Pseudomonas* spp. were frequently isolated from HS lesions, and opportunistic pathogens were also detected.[11] In this study, CNS was the most frequently isolated species, along with *Streptococcus* spp., *Corynebacterium* spp., *Cutibacterium* spp., *P. aeruginosa*, *Bacteroides fragilis*, *Peptoniphilus coxii*, and *Peptoniphilus harei/asaccharolyticus*. In contrast, *C. acnes* was isolated less frequently in this study. In HS lesions, the amount of *C. acnes*, a normal skin commensal, has been reported to decrease in association with disease severity.[11] Therefore, the patients in this study had a severe form of HS, suggesting a decrease in normal skin commensal bacteria and an increase in opportunistic pathogens in the skin lesions.

The antimicrobial susceptibility of the bacterial isolates was measured. All tested bacterial species showed high susceptibility to fusidic acid and rifampicin. However, tetracyclines, clindamycin and macrolides showed high resistance rates in *Staphylococcus* spp., *Enterococcus* spp., *Corynebacterium* spp., and *Streptococcus* spp. Resistance to clindamycin and macrolides tends to be more common than that to tetracyclines. It has been reported that the clearance rates of macrolides, such as erythromycin and azithromycin, from the skin are slower than those from the plasma, resulting in prolonged drug retention in the skin.[26] Thus, macrolides may result in prolonged bacterial exposure at sub-minimum inhibitory concentrations on the skin. Additionally, clindamycin concentrations in synovial fluid and bone have been reported to reach 60–80% of concurrent serum levels, and it is therefore inferred that similar concentrations may be achieved in the skin. [27] In contrast, tetracyclines such as doxycycline are highly lipophilic and exhibit widespread tissue distribution, with greater accumulation observed in the skin compared to plasma.[28] Furthermore, tetracyclines generally have a longer half-life than macrolides such as clarithromycin and clindamycin, allowing for the maintenance of effective drug levels over extended periods. Additionally, Japanese clinical practice guidelines recommend tetracyclines, clindamycin and macrolides, which are frequently prescribed. Thus, the emergence of macrolide-resistant bacteria may be promoted by the repeated exposure of skin flora to low concentrations of clindamycin and macrolides. Moreover, macrolide resistance is known to exhibit cross-resistance with clindamycin, suggesting that inappropriate use of both drugs may further accelerate the development of antimicrobial resistance.

Antimicrobial resistance genes have been detected in various bacteria isolated from patients with HS. Macrolide and clindamycin resistance was mainly associated with *erm*(C) in CNS, *erm*(X) in *Corynebacterium* spp., and *erm*(B) in *Enterococcus* spp. and *Streptococcus* spp. Additionally, *erm*(X) was highly prevalent in *C. acnes*. Therefore, the acquisition of these resistance genes can be attributed to the development of resistance to various antimicrobial agents. *tet*(M) was frequently detected in tetracycline-susceptible and low-resistant strains, but there was no correlation with the minimum inhibitory concentration. However, *tet*(M) has been reported to induce minocycline resistance.[25] When using tetracyclines, care should be taken with *tet*(M) harboring strains. Additionally, *tet*(M), *tet*(O), and *erm*(B) are known to be involved in horizontal gene transfer between strains of bacteria, such as *Streptococcus agalactiae*, which are capable of transformation.[29] Therefore, antimicrobial resistance genes detected in bacterial strains isolated from skin lesions in this study may have been horizontally transferred between strains in the skin lesions.

A survey of antimicrobial use history was conducted to analyze the association between a history of antimicrobial use and the emergence of antimicrobial-resistant bacteria. No statistically significant difference was found between a history of clindamycin and tetracycline use and the incidence of antimicrobial-resistant bacteria. However, macrolide-resistant strains were more frequently isolated from patients with a history of clindamycin use, indicating a correlation.

A limitation of this questionnaire survey was that patients with no documented history of antimicrobial use or unclear information about the antimicrobials used were classified as having no history of antimicrobial use. The group classified as having no history of antimicrobial use might have included patients with such a history. Owing to the limited sample size in this study, a more comprehensive analysis is needed to gain a clearer understanding of the relationship between the history of antimicrobial use in patients with HS and the isolation rate of resistant bacteria.

These results suggest that antimicrobial-resistant indigenous bacteria are highly prevalent in HS lesions. However, these bacteria remained susceptible to rifampicin and fusidic acid, suggesting that these agents are the antimicrobials of choice for preventing secondary infections. Ongoing surveillance is essential to identify effective antimicrobial agents for the treatment of secondary bacterial infections, and the prevalence of antimicrobial-resistant bacteria must be closely monitored. Additionally, Japanese clinical guidelines for the treatment of HS recommend biological agents for patients with severe disease. However, to reduce the risk of the emergence of antimicrobial-resistant bacteria, biological agents, rather than antimicrobial agents, should also be considered for patients with mild-to-moderate disease.

Materials and Methods

Bacterial strains

The study protocol was approved by the Ethics Committees of the Tokyo University of Pharmacy and Life Sciences (20 – 06) and Toranomon Hospital (2016). Forty-seven bacterial strains were isolated from the affected areas of 15 patients with HS who visited the Dermatology Department of Toranomon Hospital in 2021. These bacterial strains were collected from the head, posterior neck, upper back, neck, axilla, buttocks, thighs, perineum, pubis, and groin using Transystem™ Cary-Blair Agar Gel Medium (COPAN). *Cutibacterium acnes* ATCC 6919 and ATCC11828 and *S. aureus* JCM2874 were used as control strains for the antimicrobial susceptibility analysis.

Culture conditions and identification of bacterial species

Sample swabs were cultured on blood agar medium under both aerobic and anaerobic conditions at 37°C for 24–48 h. The blood agar medium was prepared by supplementing brain heart infusion broth (Becton Dickinson) with 1.5% Bacto™ agar (Becton Dickinson) and 5% defibrinated horse blood (Japan Bio Serum). Gram staining was performed using Favor G "Nissui" (Nissui). *Staphylococcus* spp. were identified using PCR amplification of the *nuc* gene.[15] Species identification was performed using the Basic Local Alignment Search Tool (BLAST) and 16S rRNA gene sequences determined by Sanger sequencing.[16]

Antimicrobial susceptibility testing

Antimicrobial susceptibility testing for *Staphylococcus* spp., *Enterococcus* spp., *Corynebacterium* spp., and *Streptococcus* spp. was performed by the agar or broth dilution method according to the Clinical and Laboratory Standards Institute (CLSI).[17] The antimicrobial susceptibility of *C. acnes* was determined using the agar dilution method based on our previous method and the CLSI for anaerobic bacteria.[18, 19] The following antimicrobial agents were tested: amoxicillin (FUJIFILM Wako Pure Chemical), cefdinir (Tokyo Chemical Industry), levofloxacin hydrochloride (Tokyo Chemical Industry), nadifloxacin (Tokyo Chemical Industry), roxithromycin (Tokyo Chemical Industry), clarithromycin (Tokyo Chemical Industry), clindamycin hydrochloride (Tokyo Chemical Industry), gentamicin sulfate (FUJIFILM Wako Pure Chemical), tetracycline hydrochloride (FUJIFILM Wako Pure Chemical), doxycycline hyclate (Tokyo Chemical Industry), minocycline hydrochloride (FUJIFILM Wako Pure Chemical), metronidazole (FUJIFILM Wako Pure Chemical), 4,4'-diaminodiphenyl sulfone (FUJIFILM Wako Pure Chemical), and fusidic acid (Tokyo Chemical industry). Oxacillin sodium salt monohydrate (Tokyo Chemical Industry) was used only for *Staphylococcus* spp. Resistance breakpoints for antimicrobial agents were determined according to CLSI for *C. acnes*, *Staphylococcus* spp., *Enterococcus* spp., and *Streptococcus* spp., and EUCAST for *Corynebacterium* spp.[19, 20, 21] For antimicrobial agents without breakpoints, these were defined by our laboratory (Table S1).

We did not define breakpoints for nadifloxacin and ozenoxacin, which are topical agents, or gentamicin, which is ineffective against anaerobes.

Detection of antimicrobial resistance genes

The macrolide-clindamycin resistance genes *erm*(X), *erm*(50), *erm*(A), *erm*(B), *erm*(C), *mph*(C), and *msr*(A/B); the aminoglycoside resistance genes *aacA-aphD*; and the tetracycline resistance genes *tet*(M) and *tet*(K) were detected using PCR.[19, 22–25]

Prior antimicrobial use

We examined the association between antimicrobial use and the isolation of antimicrobial-resistant bacteria by analysing data on oral tetracycline and clindamycin use obtained from a questionnaire survey of 15 patients. Patients with no documented history of antimicrobial use or with unclear information about administration were classified as having no history of antimicrobial use.

Statistical analysis

Statistical significance was analyzed using Fisher's exact test and Student's *t*-test ($p < 0.05$).

Declarations

Ethical approval

All methods were performed in accordance with relevant guidelines and regulations. This study was approved by the Ethics Committees of Tokyo University of Pharmacy and Life Sciences (approval no. 20 – 6) and Toranomon Hospital (approval no. 2016). Written informed consent was obtained from all patients prior to sample collection.

Conflict of interest statement

Nobukazu Hayashi received lecture and research fees from Abbvie Inc.

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Author Contribution

Shoji Seyama, Yuji Miyazaki, Yu Sakaki, Nobukazu Hayashi and Hidemasa Nakaminami contributed to the study conceptualization and methodology. Data collections were performed by Shoji Seyama, Yuji Miyazaki, Koyo Yoshihara, and Yu Sasaki. Formal analysis and visualization were performed by Shoji Seyama, Yuji Miyazaki, Koyo Yoshihara and Hidemasa Nakaminami. The first writing – original draft was written by Shoji Seyama and Yuji Miyazaki. The writing – review & editing was performed by Shoji Seyama, Nobukazu Hayashi, and Hidemasa Nakaminami. All authors read and approved the final manuscript.

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Data Availability

All data generated or analyzed during this study are included here and are available upon reasonable request to the corresponding author.

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Figures

Fig. 1

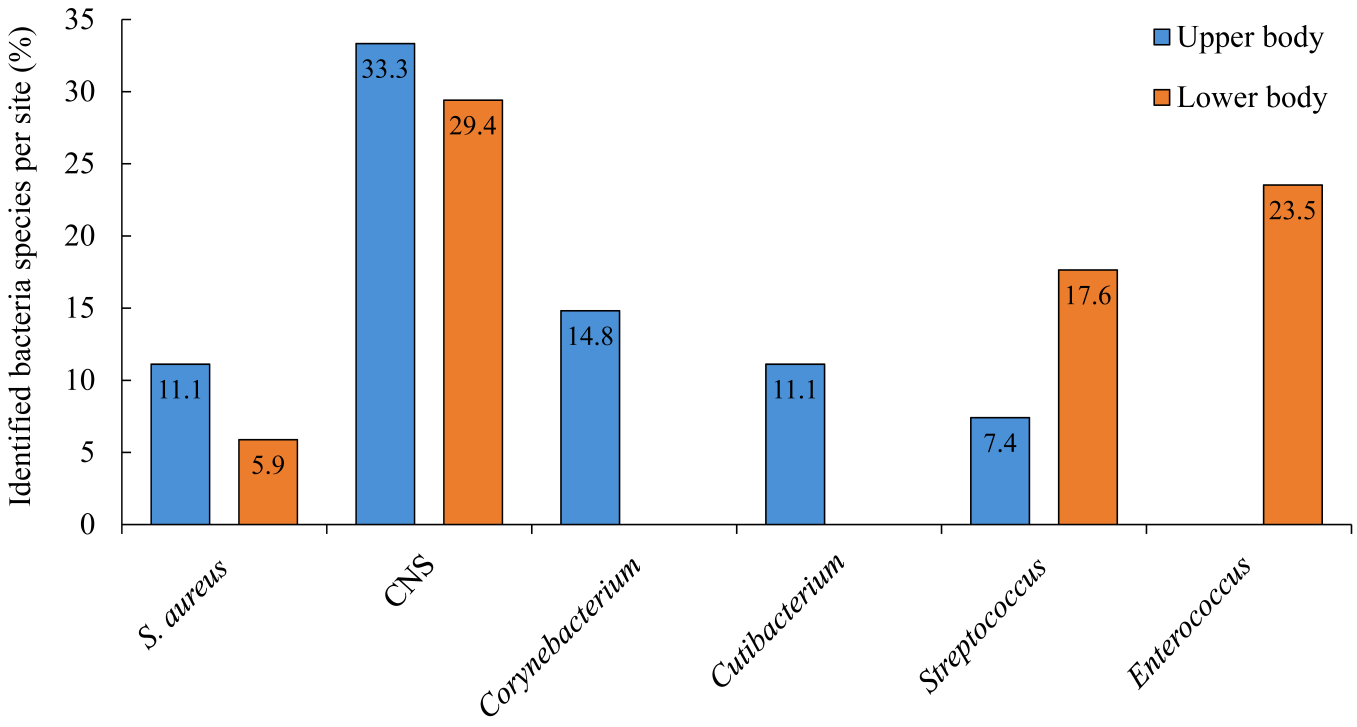


Figure 1

Major bacterial species identified by site of predilection.

Blue bars represent the percentage of bacterial species isolated from the upper body, and orange bars represent those isolated from the lower body.

Fig. 2

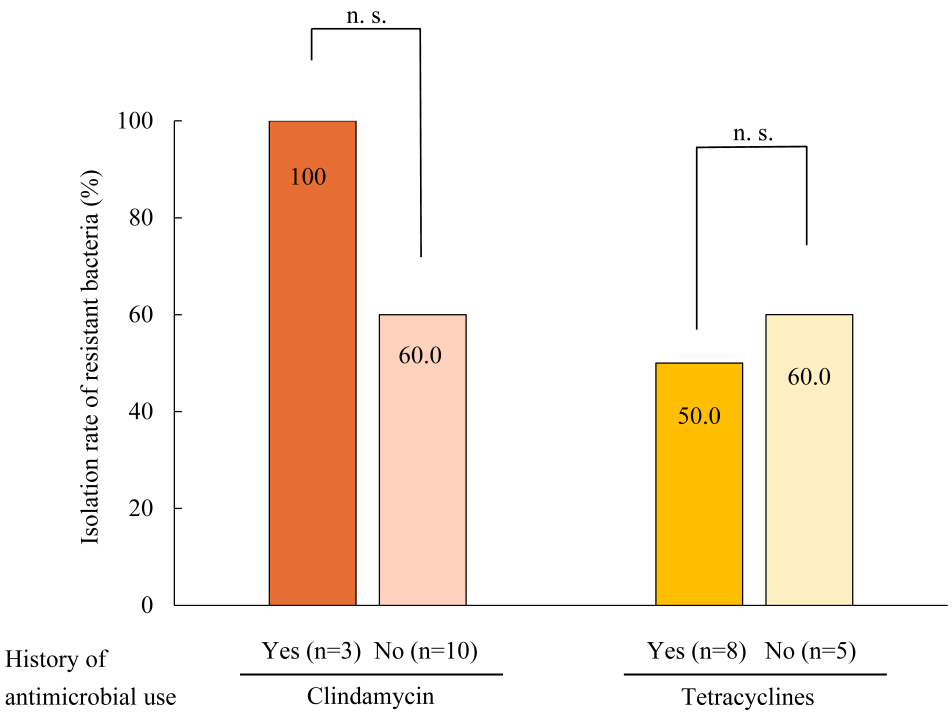


Figure 2

Association between history of antimicrobial use history and the isolation of antimicrobial-resistant bacteria.

The orange and green bars represent the history of macrolide and tetracycline use, respectively. Dark and light bars represent patients with and without history of antimicrobial use, respectively.

n.s. indicates no significant difference.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- TableS120251218.docx