MAJOR ARTICLE

Clinical Presentation and Bacteriologic Analysis of Infected Human Bites in Patients Presenting to Emergency Departments

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Previous studies of infected human bites have been limited by small numbers of patients and suboptimal microbiologic methodology. We conducted a multicenter prospective study of 50 patients with infected human bites. Seventy percent of the patients and assailants were young adult men. Fifty-six percent of injuries were clenched-fist injuries and 44% were occlusional bites. Most injuries were to the hands. Fifty-four percent of patients were hospitalized. The median number of isolates per wound culture was 4 (3 aerobes and 1 anaerobe); aerobes and anaerobes were isolated from 54% of wounds, aerobes alone were isolated from 44%, and anaerobes alone were isolated from 2%. Isolates included *Streptococcus anginosus* (52%), *Staphylococcus aureus* (30%), *Eikenella corrodens* (30%), *Fusobacterium nucleatum* (32%), and *Prevotella melaninogenica* (22%). *Candida* species were found in 8%. *Fusobacterium, Peptostreptococcus,* and *Candida* species were isolated more frequently from occlusional bites than from clenched-fist injuries. Many strains of *Prevotella* and *S. aureus* were β-lactamase producers. Amoxicillin–clavulanic acid and moxifloxacin demonstrated excellent in vitro activity against common isolates.

Human bites can lead to significant morbidity from direct trauma and subsequent infection [1]. Previous bacteriologic analyses of these wounds have noted mixed infections with aerobes, such as α-hemolytic streptococci and *Staphylococcus aureus*, and anaerobic bacteria [2–4]. However, these studies have been limited to case series of small numbers of selected patients at single centers that used inexact infection criteria and had variable microbiologic capabilities. Therefore, we conducted a multicenter prospective study of infected human bite wounds in which patients met strict infection criteria. All wound specimens were cultured for aerobic and anaerobic bacteria at a research microbiology laboratory with more-advanced techniques. The aim of the study was to delineate and update the complicated bacteriology of infected human bites and to improve the accuracy of empirical therapy.

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METHODS

This prospective case study was conducted from June 2000 through May 2001 at 12 university-affiliated emergency departments in the United States, several of which were from *EMERGE*ncy ID NET, a Centers for Disease Control and Prevention (CDC)–supported

emergency department–based sentinel network for research of emerging infections. Each institutional review board approved the study.

Patients were included if they had a cutaneous wound caused by a human bite that was large enough for at least a miniswab to be inserted to obtain a specimen for deep culture and if they met 1 of 3 major criteria (fever [temperature >38.0°C], abscess, and lymphangitis) or 4 of 5 minor wound criteria (erythema extending >3 cm from the wound edge, tenderness, swelling, purulent drainage, and a peripheral WBC count of >12,000 cells/mm^3). These criteria were established before the study to distinguish clinically infected wounds from contaminated but noninfected wounds.

Patients who had taken antimicrobial agents within 72 h before presentation, who had paronychial infections, or who had wounds involving the oral mucosa were excluded. No patient was enrolled more than once.

The following data were collected: age and sex of the patient and the biter (if known); associated medical condition(s) of the patient; the time of bite; the time a specimen was obtained for culture; the interval between the bite and the onset of infection; the type of wound; the type of infection; wound care before presentation; and antimicrobials and other therapies that were given. Wounds were categorized as clenched-fist injuries or occlusional bites. Infections were classified as abscess, purulent wound, or nonpurulent wound with cellulitis, lymphangitis, or both. The treating physician directed clinical care.

Microbiologic specimens were obtained by closed-needle aspiration for abscesses and with standard rayon swabs for open lacerations. For puncture wounds, calcium alginate miniswabs were inserted deeply after the surrounding skin had been cleaned with alcohol or povidone-iodine solution. Specimens were placed in an anaerobic transport tube containing a deep column of anaerobic transport medium (Anaerobe Systems) and shipped by overnight courier to R. M. Alden Research Laboratory (Santa Monica, CA) [5]. In some cases, a second specimen was sent to the microbiology laboratory at the local hospital and processed in accordance with the facility's standard procedures.

Specimens were processed inside an anaerobic chamber. Aspirates were inoculated directly, 1 drop per plate, while swabs were homogenized into 1 mL of Brucella broth and then processed as liquid specimens [6, 7]. The following agar media were used for anaerobes: supplemented Brucella blood, Brucella with laked blood and kanamycin-vancomycin, Bacteroides–bile esculin, cadmium sulfate–fluoride acriflavine trypticase, campy-wolinella, *Fusobacterium* selective, *Porphyromonas gingivalis* medium, clindamycin-blood (Anaerobe Systems), and Rose (Hardy Diagnostics). Anaerobic plates were incubated for 5 days before being examined for the first time. All plates were reincubated for up to 2 weeks to allow growth of fastidious organisms.

The following media were used for aerobic and facultative organisms: trypticase soy supplemented with 5% defibrinated sheep blood, chocolate, Rose, and MacConkey agars. All specimens except those on MacConkey agar were incubated in 5%–7% carbon dioxide for 24 h before being examined for the first time and reincubated for up to 5 days to allow growth of fastidious organisms. No attempt was made to culture for *Chlamydia* or *Mycoplasma* species or spirochetes.

A combination of identification methods was used. Isolates were grouped on the basis of growth and appearance of colonies on selective media, Gram stain reaction, and susceptibility to special potency antibiotic identification disks. For definitive identification, kits (including the API 20E and API 20A [bioMérieux] and RapID Strep, RapID ANA II, RapID NF, and RapID NH [Remel]) were used in conjunction with their organism databases, in accordance with the instructions of their manufacturers. Other procedures included reactions determined in biochemical tube tests (Anaerobe Systems; Hardy Diagnostics). Organisms were identified according to standard guidelines [6–10]. Some of the unusual organisms were identified by sequencing of their 16S rDNA (performed by George Conrads, Institute for Medicinische Mikrobiologie, Universitätsklinikum, Aachen, Germany).

A sample of the most common isolates was tested for susceptibility to the following antimicrobials: penicillin, amoxicillin–clavulanic acid, doxycycline, erythromycin, ciprofloxacin, and moxifloxacin. MICs were determined by the agar dilution method, as described by the NCCLS [11–13].

RESULTS

A total of 57 patients were enrolled. Seven patients were excluded (5 had received prior antibiotics, 1 had a noninfected wound, and 1 had a paronychia). Results are reported for 50 patients with infected human bites.

Patients ranged in age from 8 to 61 years (median, 27 years; interquartile range, 20–37 years); 70% were male. The biters ranged in age from 2 to 45 years (median, 28 years; interquartile range, 24–35 years); 70% were male. Fifteen patients had ⩾1 associated medical condition, including alcoholism (9 patients), diabetes (2 patients), HIV infection (2 patients), and chronic renal disease (1 patient).

Wounds were described as punctures only in 19 patients (38%), as lacerations only in 17 patients (34%), as both in 13 patients (26%), and as avulsion in 1 patient (2%). Forty-three wounds (86%) involved the hands, 4 (8%) involved the head/face, and 1 (2%) each involved the neck, arm, or foot. Twenty-eight wounds (56%) were clenched-fist injuries and 22 (44%) were occlusional bites (all nonsexual). Twenty-seven (54%)

were limited in depth to the subcutaneous tissue, 9 (18%) were intradermal, and 7 (14%) went into muscle fascia. Seven (14%) involved deep structures, including bone (3 patients), tendon (3 patients), and joint (1 patient). Patients with clenched-fist injuries were more often male than were those with occlusional bites (89.3% vs. 45.5%); otherwise, the groups had similar demographic, wound, and infection characteristics.

The following types of local wound care had been administered before presentation: soap and water (70%), peroxide (30%), topical antibiotic ointment (20%), sterile saline (10%), isopropyl alcohol (10%), and tincture of iodine (8%). Eight patients (16%) received no local care. Nine patients (18%) had been seen previously by a physician for their injury, 4 of whom underwent debridement and 3 of whom received sutures.

Wound infections were characterized as abscesses in 24 patients (48%), purulent wounds in 23 patients (46%), and nonpurulent wounds with cellulitis, lymphangitis, or both in 3 patients (6%). Five patients (10%) had infective tenosynovitis (3 [6%] in patients with clenched-fist injury and 2 [4%] in patients with occlusional bites). One patient (2%) had septic arthritis and 1 (2%) had osteomyelitis, with both cases occurring after clenched-fist injuries. Three patients (6%) had temperatures of >38.0°C at presentation, and 6 (25%) of 24 patients had peripheral WBC counts of >12,000 cells/mm^3.

The median time from the bite to the appearance of the first symptoms of infection was 22 h (range, 1.5–312 h; interquartile range, 12.5–36 h); median times for abscesses, purulent wounds, and nonpurulent wounds with cellulitis and/or lymphangitis were 30.5, 21, and 6.5 h, respectively; for clenched-fist injuries and occlusional bites, they were 24 and 18 h, respectively. The median interval between the bite and presentation was 58 h (range, 8–712 h; interquartile range, 38–132 h) and was slightly longer for clenched-fist injuries than for occlusional bites (58 vs. 53 h).

The median number of isolates per wound culture was 4 (range, 1–21; interquartile range, 2–9); ~3 were aerobes and 1 was an anaerobe. The median total numbers and proportions of aerobic and anaerobic isolates among clenched-fist injuries and occlusional bites were similar. Mixed aerobic and anaerobic infection was present in 27 (54%) of all wounds, only aerobes grew in 22 (44%), and only anaerobes grew in 1 clenched-fist injury (2%; *Campylobacter sputorum, Fusobacterium nucleatum,* and *Prevotella melaninogenica*). All wound cultures had bacterial growth.

The bacteria isolated from all 50 infected human bites are listed in table 1. *Streptococcus* species were by far the most common isolates (84%), with *Streptococcus anginosus* being the most common (54%). *Streptococcus pyogenes* was present in only 14% of wounds. The next most common was *Staphylococcus* species (52%), with *S. aureus* being the most common (30%). *Eikenella corrodens* was present in 30% of wounds. Common anaerobes included *Prevotella* (36%, with *Prevotella*

Table 1. Aerobic and anaerobic microorganisms isolated from 50 patients with infected human bite injuries.

Microorganism	No. (%) of patients with pathogen
Aerobes	
Streptococcus	42 (84)
S. anginosus	26 (52)
S. oralis	7 (14)
S. pyogenes	7 (14)
S. intermedius	6 (12)
S. mitis	6 (12)
S. constellatus	4 (8)
S. parasanguis	4 (8)
Viridans group[a]	4 (8)
S. sanguis I	3 (6)
S. salivarius	2 (4)
S. vestibularis	2 (4)
S. dysgalactiae subspecies *equisimilis*	1 (2)
S. gordonii	1 (2)
Streptococcus group C/G	1 (2)
S. mutans	1 (2)
S. sanguis	1 (2)
S. sanguis II	1 (2)
Staphylococcus	27 (54)
S. aureus	15 (30)
S. epidermidis	11 (22)
Coagulase-negative *Staphylococcus*[a]	3 (6)
S. saprophyticus	1 (2)
Eikenella corrodens	15 (30)
Haemophilus	11 (22)
H. parainfluenzae	6 (12)
H. aphrophilus	3 (6)
H. influenzae	1 (2)
H. paraphrophilus	1 (2)
Corynebacterium	6 (12)
C. amycolatum	3 (6)
Corynebacterium CDC group G	1 (2)
C. falsenii	1 (2)
C. pseudodiphtheriticum	1 (2)
Unidentified species[a]	1 (2)
Gemella	6 (12)
G. morbillorum	5 (10)
G. haemolysans	1 (2)
Candida	4 (8)
Unidentified species[a]	3 (6)
C. albicans	1 (2)
Enterobacter cloacae	4 (8)
Neisseria	4 (8)
N. subflava	2 (4)
N. elongata subspecies *enlongata*	1 (2)
N. elongata subspecies *nitroreducens*	1 (2)
Enterococcus	3 (6)
E. faecalis	2 (4)
E. raffinosus	1 (2)

(continued)

Table 1. ***(Continued.)***

Microorganism	No. (%) of patients with pathogen
Klebsiella	3 (6)
K. oxytoca	2 (4)
K. pneumoniae	1 (2)
Rothia species[a]	2 (4)
Acinetobacter baumannii	1 (2)
Actinobacillus suis	1 (2)
Aerococcus viridans	1 (2)
Aeromonas caviae	1 (2)
Agrobacterium radiobacter	1 (2)
Alcaligenes faecalis	1 (2)
Capnocytophaga ochracea	1 (2)
CDC group NO-2	1 (2)
Citrobacter sedlakii	1 (2)
Kingella denitrificans	1 (2)
Kocuria kristinae	1 (2)
Lactobacillus fermentum	1 (2)
Leclercia adecarboxylata	1 (2)
Micrococcus species[a]	1 (2)
Moraxella osloensis	1 (2)
Pantoea endophytica	1 (2)
Proteus	1 (2)
P. vulgaris	1 (2)
Unidentified species[a]	1 (2)
Stomatococcus mucilaginosus	1 (2)
Anaerobes	
Prevotella	18 (36)
P. melaninogenica	11 (22)
P. buccae	7 (14)
P. intermedia	7 (14)
P. oris	4 (8)
P. disiens	2 (4)
P. pallens	2 (4)
P. buccalis	1 (2)
P. dentalis	1 (2)
P. denticola	1 (2)
P. loeschii	1 (2)
P. oulorum	1 (2)
Unidentified species[a]	1 (2)
P. tannerae	1 (2)
P. zoogleoformans	1 (2)
Fusobacterium	17 (34)
F. nucleatum	16 (32)
F. necrophorum	2 (4)
F. naviforme	1 (2)
Veillonella species[a]	12 (24)
Peptostreptococcus	11 (22)
P. micros	10 (20)
P. magnus	1 (2)
P. prevotii	1 (2)
Campylobacter	8 (16)
C. gracilis	3 (6)
C. rectus	3 (6)
C. sputorum	2 (4)
C. mucosalis	1 (2)
Eubacterium	8 (16)
Unidentified species[a]	5 (10)
E. yurii	4 (8)
E. saburreum	2 (4)
E. timidum	2 (4)
E. lentum	1 (2)
Actinomyces	4 (8)
A. israelii	2 (4)
A. meyeri	1 (2)
A. naeslundii	1 (2)
A. viscosus	1 (2)
Lactobacillus	4 (8)
L. catenaforme	2 (4)
L. delbrueckii	1 (2)
L. minutes	1 (2)
Bacteroides	2 (4)
B. capillosus	1 (2)
B. putredinis	1 (2)
Collinsella (Eubacterium) aerofaciens	2 (4)
Dialister pneumosintes	2 (4)
Propionibacterium acnes	2 (4)
Arcanobacterium bernardiae	1 (2)
Porphyromonas species[a]	1 (2)

NOTE. Some patients were infected with >1 species within a genus. CDC, Centers for Disease Control and Prevention.

[a] Isolate could not be identified beyond the genus level.

melaninogenica in 22%), *Fusobacterium* (34%, with *Fusobacterium nucleatum* in 32%), and *Veillonella* species (24%). One previously healthy 28-year-old man with a clenched-fist injury grew CDC group NO-2, as well as 4 aerobic and 6 anaerobic species. *Candida* species were found in 8% of wounds.

Among abscesses, *Streptococcus* (83%) and *Staphylococcus* (54%) species remained the most common isolates, with similar frequencies of *S. anginosus* (46%) and *S. aureus* (25%), as with other wound infections. However, certain genera occurred with greater frequency in abscesses, including *Eikenella* (42%), *Fusobacterium* (42%), and *Prevotella* (42%).

Compared with occlusional bites, clenched-fist injuries had comparable proportions of *E. corrodens* (32.1% vs. 27.3%), *Staphylococcus* (57.1% vs. 50.0%), and *Streptococcus* (82.1% vs. 86.4%) but smaller proportions of *Fusobacterium* (28.6% vs.

40.9%), *Peptostreptococcus* (14.3% vs. 31.8%), and *Candida* (3.6% vs. 13.6%).

Twenty-three patients also had wound specimens sent to local microbiology laboratories. Fewer organisms grew in these cultures than in the cultures sent to the reference laboratory (median, 2; range, 0–5). Two (9%) of these cultures yielded no growth. The prevalences of all species except viridans streptococci (35%), *S. aureus* (26%), other *Staphylococcus* species (22%), *S. pyogenes* (9%), *Haemophilus* species (9%), *Corynebacterium* species (9%), *Bacteroides* species (9%), and *Peptostreptococcus* species (9%) were <5%.

Antimicrobial susceptibility testing was conducted on a sample of the most frequently isolated strains using penicillin, amoxicillin–clavulanic acid, doxycycline, erythromycin, ciprofloxacin, and moxifloxacin (table 2). Amoxicillin–clavulanic acid and moxifloxacin demonstrated the best activity. Penicillin had relatively poor activity against *S. aureus, Prevotella* species, and *Veillonella* species usually related to β-lactamase production. Doxycycline had relatively poor activity against streptococci and *E. corrodens.* Erythromycin had also extremely poor activity against *E. corrodens* and was relatively inactive against many other genera.

Twenty-seven patients (54%) were hospitalized and were initially treated with intravenous antibiotics for a median duration of 3 days (range, 1–9 days). More patients with clenched-fist injuries than with occlusional bites were admitted to the hospital (71.4% vs. 31.8%), with the median duration of hospitalization being similar in both groups (2.5 vs. 3.0 days). Eight patients (16%) received 1 intravenous dose of antibiotics followed by oral antibiotics, and 15 patients (30%) received only oral antibiotics. Intravenous monotherapy regimens for hospitalized patients included ampicillin-sulbactam (14 patients), piperacillin-tazobactam (2 patients), ticarcillin–clavulanic acid (2 patients), and cefazolin (2 patients). Combinations of intravenous antibiotics were also used, such as clindamycin and levofloxacin (2 patients), ampicillin-sulbactam and clindamycin (1 patient), ticarcillin-clavulanic acid and cefazolin (1 patient), penicillin and cefazolin (1 patient), and vancomycin and metronidazole (1 patient). Ampicillin-sulbactam was the most common single-dose parenteral regimen that preceded oral therapy (7 patients). Oral regimens included amoxicillin–clavulanic acid (17 patients), cephalexin and penicillin (2 patients), and levofloxacin and ciprofloxacin (1 patient each).

DISCUSSION

Human bite wounds generally occur in 1 of 2 ways. A hand wound can result from one person punching another person in the mouth; this is referred to as a "clenched-fist injury." Alternatively, a person can be a victim of an occlusive bite. Clenched-fist injuries result from impalement of the assaulted person's tooth in the area over the assailant's third, fourth, or fifth metacarpal head. This type of injury may lead to infection, tendon tear, joint disruption, or fracture [1, 14]. The majority of the patients in our study required hospitalization. Delay in seeking care may have contributed to the advanced nature of these infections, because patients often did not present for >24 h after they first noted signs of infection.

The earliest reports of the bacteriology of infected human bites noted spirochetes and fusiform bacteria, terms suggesting the presence of typical oral and anaerobic flora [14–20]. Later investigations identified α-streptococci and *S. aureus* as the most frequent pathogens [21–24]. Recent studies utilizing improved methods of isolation and identification of anaerobic bacteria confirmed the role of anaerobes in ~50% of infections [2–4]. *E. corrodens* has been found in 17%–25% of human bite infections [2–4].

Previous investigations have been limited by the small numbers of patients enrolled, skewed patient populations, the inclusion of patients who were receiving systemic antibiotics, use of inexact definitions of infection, and nonrigorous microbiologic methods. The importance of addressing these limitations is underscored by the results of a similar study that we conducted of infected dog and cat bites that revealed new insights as to the frequencies of various zoonotic pathogens [25].

Our study revealed that streptococci, particularly *S. anginosus,* were the most frequent pathogens isolated from infected human bites. Viridans streptococci are normal human oral flora. Although not appreciated previously, it is not surprising that *S. anginosus* would emerge as the predominant pathogen, because it is recognized for its greater tendency to cause invasive human infections, such as brain and visceral abscesses, compared with other viridans streptococci [26]. Staphylococci, particularly *S. aureus,* and *E. corrodens* were other common aerobic organisms. *E. corrodens,* a microaerophilic gram-negative bacillus that may be difficult to culture, has been found in 59% of human gingival plaque specimens and appears to have a unique association with human bites [27, 28]. Remarkably infrequent were Enterobacteriaceae and *Pseudomonas* species. Approximately one-half of infections grew oral anaerobic species, such as *Prevotella, Veillonella, Fusobacterium,* and *Peptostreptococcus* species, which were almost always accompanied by aerobic bacteria.

Eight percent of human bite infections grew *Candida* species, always along with bacteria. Although *Candida* species can colonize the oral cavity and have been associated with paronychial infections, we are not aware of previous reports associating this fungus with a human bite infection [29]. Because *Candida* are typically opportunistic pathogens, these may have been incidental contaminants. One patient grew CDC group NO-2, also called "*Bordetella holmesii,*" a gram-negative bacterium that has been recognized to cause bacteremia, especially in immuno-

Table 2. Antimicrobial susceptibility of common bacterial isolates from infected human bites.

Organism	No. of isolates tested	Penicillin		Amoxicillin–clavulanic acid		Doxycycline		Erythromycin[a]		Ciprofloxacin[a]		Moxifloxacin[a]	
		MIC, μg/mL	S, %	MIC, μg/mL	S, %	MIC, μg/mL	S, %	MIC, μg/mL	S, %	MIC, μg/mL	S, %	MIC, μg/mL	S, %
Streptococcus species	37	≤0.015–1	97.3	≤0.015–1	97.3	≤0.06–16	78.4	≤0.125 to >32	70.2	0.5–4	54.1	≤0.06–0.5	100
Staphylococcus aureus[b]	18	0.03 to >8	16.7	0.125–4	100	0.125–32	94.4	≤0.125 to >32	55.6	≤0.06–0.5	100	≤0.06	100
Eikenella corrodens[a]	18	0.5–2	100[c]	0.25–0.5	100[d]	0.5–4	72.2[c]	2–16	16.7[c]	≤0.06	100	≤0.06	100[e]
Miscellaneous fastidious GNB[a,f]	13	≤0.015 to >8	92.3[e]	≤0.015–8	92.3[g]	0.125–2	100[c]	≤0.125–16	69.2[c]	≤0.06–0.25	100[g]	≤0.06–1	100[e]
Prevotella species[b]	13	0.03 to >8	46.2	≤0.015–1	100	≤0.06–4	100	≤0.125–0.5	100[c]	0.5–2	61.5[e]	0.25–1	100[e]
Fusobacterium species	10	≤0.015–0.125	100	≤0.015–0.25	100	≤0.06–0.5	100	0.5–16	40[c]	0.25–2	40[e]	0.125–0.25	100[e]
Veillonella species[b]	11	≤0.015–8	72.7	≤0.015–2	100	0.25–2	100	0.5–8	9.1[c]	≤0.06–0.125	100[e]	≤0.06–0.25	100[e]
Peptostreptococcus species	5	≤0.015–0.5	100	≤0.015–2	100	≤0.06–0.25	100	≤0.125–0.25	100[c]	≤0.06–1	100[e]	0.125–0.5	100[e]

NOTE. MIC was determined by the agar dilution method, as described by the NCCLS [6, 11–13]. MIC range and percentage susceptibility are calculated from interpretive criteria in NCCLS documents M7-A5, M11-A5, and M100-S12, when appropriate criteria were supplied [11–13]. GNB, gram-negative bacteria; S, susceptible.

[a] MIC breakpoint has not been established for all of the species tested.

[b] The frequencies of β-lactamase–producing strains were as follows: *S. aureus,* 83%; *Prevotella* species, 54.0%; and *Veillonella* species, 9%.

[c] An MIC of ≤2 was considered to indicate susceptibility (NCCLS does not provide breakpoint guidelines for this organism-drug combination).

[d] An MIC of ≤0.5 was considered to indicate susceptibility (NCCLS does not provide breakpoint guidelines for this organism-drug combination).

[e] An MIC of ≤1 was considered to indicate susceptibility (NCCLS does not provide breakpoint guidelines for this organism-drug combination).

[f] Miscellaneous GNB included *Moraxella osloensis* (1 isolate), *Neisseria* species (7 isolates), *Haemophilus* species (3 isolates), *Capnocytophaga ochracea* (1 isolate), and *Kingella denitrificans* (1 isolate).

[g] An MIC of ≤0.25 was considered to indicate susceptibility (NCCLS does not provide breakpoint guidelines for this organism-drug combination).

compromised patients [30]. None of the reported infections was the result of a human bite.

Patients with clenched-fist injuries constituted the majority of patients in this study. Notable differences between patients with clenched-fist injuries and those who had occlusional bites included the following: the majority of patients with clenched-fist injuries were male; they had a longer latency period, a longer time from injury to presentation, and higher hospitalization rates; and they were less frequently infected with *Fusobacterium, Peptostreptococcus,* and *Candida* species.

More-severe infections may have been overly represented in our study, because we only enrolled patients presenting to emergency departments, as opposed to other outpatient settings. This may have affected the bacteriologic findings, although it would seem most important to have an understanding of the microbiology associated with the most serious infections. Also, because these infections were relatively uncommon, our study population undoubtedly represents a convenience sample, and thus some selection bias could exist. The number of isolates tested for antimicrobial susceptibility was only a subset of all strains obtained.

Several observations suggest that the specimen transport and microbiologic methods employed in this study were optimal. The average number of isolates grown per specimen was double that at the local laboratories, and all specimens exhibited growth. Fastidious organisms, *E. corrodens,* and anaerobes were among the most common isolates in this study, yet they were rarely isolated at the local laboratories. If similar isolation methods are not employed at local microbiology laboratories, clinicians should consider that these pathogens might be present, even if they are not identified, and adjust antimicrobial therapy accordingly.

Because randomized, comparative antibiotic trials do not exist for human bite infections, empirical therapy must be directed by knowledge of the associated bacteriology, organism pathogenicity, and antimicrobial susceptibility of the anticipated pathogens. Our findings indicate that empirical antimicrobial therapy should be directed against *S. anginosus, S. aureus, E. corrodens,* and oral anaerobic organisms. Enterobacteriaceae and *Pseudomonas* species were infrequently cultured, and therefore regimens with antimicrobial activity against these organisms would generally appear to be unnecessary.

We examined the antimicrobial susceptibility of sample isolates that included the most common human bite wound pathogens (table 2). Our findings were generally in agreement with those of previous investigations that describe antibiotic activity against a mixed collection of isolates recovered from human, dog, and cat bite wounds [31–35].

Penicillin G has historically been used as the first-line antimicrobial agent in the management of odontogenic infections. However, increasing rates of resistance to penicillin G, especially among β-lactamase–producing oral anaerobes, has made clindamycin or β-lactam– and β-lactamase–inhibitor combination antibiotics the drugs of choice for these infections [36–39]. We also found that many species isolated from infected human bites, including >50% of *Prevotella* and >80% of *Staphylococcus* species, were β-lactamase producers.

E. corrodens has been found to be uniformly resistant to clindamycin [31, 32]. We found that erythromycin had extremely poor activity against *E. corrodens* and many other common pathogens. Antistaphylococcal penicillins and first-generation cephalosporins—drugs that are often used to treat skin and soft-tissue infections primarily due to *S. aureus* and *S. pyogenes*—are also relatively inactive against *E. corrodens.* Resistance to metronidazole and most aminoglycosides is also seen in *E. corrodens* [40].

We found that β-lactam/β-lactamase–inhibitor combination antibiotics, such as amoxicillin–clavulanic acid, and fluoroquinolones with enhanced anaerobic activity, such as moxifloxacin, are highly active against the major aerobic and anaerobic human bite wound pathogens. However, clinical data on the use of fluoroquinolones with enhanced anaerobic activity for skin and soft-tissue infections is limited, and clinical trials are currently under way. Other antimicrobials that would appear to possess adequate activity include cefoxitin and carbapenems [31–35]. Alternatively, a combination of antibiotics can be used, such as clindamycin plus either penicillin, a second- or third-generation cephalosporin, or a fluoroquinolone. These same regimens are recommended as expectant therapy to prevent these high-risk, contaminated human bite wounds from becoming clinically infected, although there are only limited data to support the effectiveness of this practice [40].

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