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Clinical and Microbiological Characteristics of *Eggerthella lenta* Bacteremia

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Eggerthella lenta* is an emerging pathogen that has been underrecognized due to historical difficulties with phenotypic identification. Until now, its pathogenicity, antimicrobial susceptibility profile, and optimal treatment have been poorly characterized. In this article, we report the largest cohort of patients with *E. lenta* bacteremia to date and describe in detail their clinical features, microbiologic characteristics, treatment, and outcomes. We identified 33 patients; the median age was 68 years, and there was no gender predominance. Twenty-seven patients (82%) had serious intra-abdominal pathology, often requiring a medical procedure. Of those who received antibiotics (28/33, 85%), the median duration of treatment was 21.5 days. Mortality from all causes was 6% at 7 days, 12% at 30 days, and 33% at 1 year. Of 26 isolates available for further testing, all were identified as *E. lenta* by both commercially available matrix-assisted laser desorption ionization–time of flight mass spectrometry (MALDI-TOF MS) systems, and none were found to harbor a *vanA* or *vanB* gene. Of 23 isolates which underwent susceptibility testing, all were susceptible to amoxicillin-clavulanate, cefoxitin, metronidazole, piperacillin-tazobactam, ertapenem, and meropenem, 91% were susceptible to clindamycin, 74% were susceptible to moxifloxacin, and 39% were susceptible to penicillin.

Eggerthella lenta is an anaerobic, nonsporulating, Gram-positive bacillus in the *Coriobacteriaceae* family that was first described in 1935 by Arnold Eggerth (1). It has since been characterized in more detail (2) and was called *Eubacterium lentum* until genetic analysis in 1999 placed it in its own distinct genus (3, 4). Two closely related species, *Paraeggerthella hongkongensis* and *Eggerthella sinensis*, have also been described recently (5–7). The complete genomic sequence was first published in 2009 (8, 9).

Due to historical difficulties with laboratory identification, there is a paucity of published data on the spectrum of disease that *E. lenta* causes and its optimal treatment. *E. lenta* is part of the normal human intestinal microbiome (10) and has been most commonly associated with infections from a gastrointestinal tract (GIT) source, which are often polymicrobial (11). Severe, disseminated disease has been described (12), and overall mortality is significant, ranging from 36% to 43% (13, 14).

E. lenta is being increasingly recognized in the modern clinical microbiology laboratory due to the widespread uptake of new technologies such as automated phenotypic identification systems and molecular sequencing techniques. More recently, it has been demonstrated that matrix-assisted laser desorption ionization–time of flight mass spectrometry (MALDI-TOF MS) is capable of rapidly identifying *E. lenta* (15–17).

The use of antimicrobial susceptibility testing (AST) for anaerobes remains restricted to specialized reference laboratories, so antimicrobial therapy for patients with infections due to anaerobic bacteria is often empirical. However, given that resistance among anaerobic organisms is increasing, susceptibility data are important to guide therapy (18–20). There are limited published data on the susceptibility profile of *E. lenta*, and breakpoints are extrapolated from those determined for other species. In 2001, Stinear et al. were developing a rapid PCR-based screening method for detection of fecal carriers of vancomycin-resistant enterococci (VRE) when they identified the *vanB* locus in *Eggerthella lenta*, hypothesizing that anaerobic bowel flora may represent the origin of VRE and demonstrating that *E. lenta* is capable of acquiring vancomycin resistance (21–23).

In this study, we have retrospectively reviewed 33 patients with *E. lenta* bacteremia, which represents the largest published series to date. We describe their clinical features, laboratory identification by both phenotypic and genotypic methods, and AST results.

MATERIALS AND METHODS

Case ascertainment and clinical data collection. This study was conducted at Monash Health, a large health care network encompassing 5 campuses and over 2,200 beds in Melbourne, Australia. Cases were identified by searching our Microbiology database for data from January 2000 to September 2013 for all positive blood cultures with *Eubacterium lentum* or *Eggerthella lenta*. Clinical data were collected from medical records. Cases were classified community onset if the positive blood culture was collected within 72 h of admission to the hospital and there was no identifiable prior health care contact, health care associated if the positive blood culture was collected within 72 h of admission and contact with the hospital system (e.g., dialysis, admissions, procedures, etc.) had occurred within 1 month after admission, and nosocomial if the positive blood culture was collected beyond 72 h

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TABLE 1 *E. lenta* bacteremia patient characteristics and clinical features[a]

Patient clinical characteristic	Values
Demographics	
Mean age ± SD (median, range)	62.4 ± 23.9 (68, 11–95)
Male sex	17 (52%)
Mean no. of days of stay ± SD (median, range)	23.6 ± 26.3 (11, 1–94)
Admission to intensive care, no. (%)	10 (30.3)
Admitting unit	
Medical, no. (%)	16 (48.5)
Surgical, no. (%)	13 (39.4)
Other, no. (%)	4 (12.1)
Onset	
Community, no. (%)	21 (63.6)
Health care associated, no. (%)	2 (6.1)
Nosocomial, no. (%)	10 (30.3)
Microbiological characteristics	
Anaerobic bottle only, no. (%)	33 (100)
Mean day blood culture flagged positive± SD (median, range)	2.85 ± 1.09 (3, 1–5)
Mean time to positivity (h) ± SD (median, range)	72.5 ± 25.2 (65.6, 40.1–122.6)
Polymicrobial bacteremia, no. (%)	13 (39.4)
Symptoms	
Fever, no. (%)	27 (81.8)
Abdominal pain, no. (%)	21 (63.6)
Vomiting, no. (%)	12 (36.4)
Diarrhea, no. (%)	8 (24.2)
Severity scores	
Mean simplified acute physiology score (SAPS II) ± SD (median, range)	27 ± 12.2 (25, 6–64)
Mean Pitt bacteremia score ± SD (median, range)	2.15 ± 1.9 (2, 0–8)
Medical comorbidities	
Diverticular disease, no. (%)	8 (24.2)
Colonic polyps, no. (%)	5 (15.2)
Gastrointestinal malignancy, no. (%)	4 (12.1)
Inflammatory bowel disease, no. (%)	1 (3)
Chronic liver disease, no. (%)	1 (3)
Gastrointestinal instrumentation or surgery within 30 days, no. (%)	5 (15.2)
Any chronic gastrointestinal pathology, no. (%)	19 (57.6)
Immunosuppression, no. (%)	5 (15.2)
Diabetes, no. (%)	9 (27.3)
Renal failure, no. (%)	12 (36.4), including 3 (9.1) dialysis
Nongastrointestinal malignancy, no. (%)	4 (12.1)
No significant comorbidities, no. (%)	5 (15.1)
Mean Charlson comorbidity score ± SD (median, range)	2.45 ± 2.25 (2, 0–7)
Likely source	
Hepatobiliary (liver, pancreas, gallbladder, biliary), no. (%)	3 (9.1)
Upper gastrointestinal tract, no. (%)	5 (15.2)
Colon, no. (%)	10 (30.3)
Bowel (unable to localize), no. (%)	5 (15.2)
Appendix, no. (%)	4 (12.1)
Skin or soft tissue, no. (%)	3 (9.1)
Other, no. (%)	3 (9.1)
Investigations	
Mean hemoglobin level (g/liter) ± SD (median, range) (RR, 119–160 g/liter)	112 ± 24 (112, 77–167)
Mean white cell count ($\times 10^9$/liter) ± SD (median, range) (RR, 4.0–11.0 $\times 10^9$/liter)	14.9 ± 8.7 (3.2–39)
Mean albumin level (g/liter) ± SD (median, range) (RR, 35–45 g/liter)	27 ± 6.8 (26, 14–39)
C-reactive protein level (mg/liter) ± SD (median, range) (RR, 0–5 mg/liter)	138.5 ± 107.1 (26, 14–39)
Abdominal CT, no. (%) (tested/abnormal)	17 (51.5)/13 (39.4)
Abdominal ultrasound, no. (%) (tested/abnormal)	9 (27.3)/6 (18.2)

(Continued on following page)

TABLE 1 (Continued)

Patient clinical characteristic	Values
Treatment	
No directed treatment, no. (%)	5 (15.2%)
Antibiotic therapy, no. (%)	28 (84.8%)
Mean duration of antibiotics if treated (days) ± SD (median, range)	36.7 ± 56.2 (21.5, 2–301)
Surgical procedure, no. (%)	12 (36.4)
Radiological procedure, no. (%)	3 (9.1)
Endoscopy performed (diagnostic and/or therapeutic), no. (%)	11 (33.3)
Outcome	
7-day mortality, no. (%)	2 (6.1)
30-day mortality, no. (%)	4 (12.1)
1-yr mortality, no. (%)	11 (33.3)
Readmission	
Within 3 mos, no. (%)	11 (33.3)
Within 12 mos, no. (%)	16 (48.5)

[a] $n = 33$ patients. CT, computed tomography; RR, reference range; SD, standard deviation.

after admission. Measures of illness severity (simplified acute physiology score [SAPS II] [24] and Pitt bacteremia score [25]) as well as comorbidity (Charlson index) (26, 27) were calculated. The study was approved by our Human Research Ethics Committee.

Blood cultures. *E. lenta* was isolated from BacT/Alert anaerobic blood culture bottles using a BacT/Alert three-dimensional (3D) automated microbial detection system (bioMérieux, Marcy l'Etoile, France). The number of hours (or days if hour data were not available) to detection of growth was recorded for each case.

Isolate identification. Available isolates were retrieved from storage and purity plated on Oxoid Anaero plates (Thermo Fisher Scientific, Basingstoke, United Kingdom). After 48 h of incubation, they were identified via multiple methods. Phenotypic identification was performed based on Gram staining, colony morphology, and Vitek-2 ANC card (bioMérieux, Marcy, l'Etoile, France) results. MALDI-TOF identification was performed using both available commercial systems, Bruker MS (Bruker Daltonics, Bremen, Germany) and Vitek MS (bioMérieux, Marcy l'Etoile, France) (formerly known as Axima@SARAMIS; Shimadzu). For isolates that could not be retrieved from storage, original phenotypic identification was performed with a RapID-ANA II system (Innovative Diagnostic Systems, Inc., Atlanta, GA).

Bruker MS analysis was performed as previously described (28, 29). Each isolate was anaerobically grown on a chocolate agar plate for 72 h. The extended direct transfer method was used whereby a single colony was touched and applied as a thin film directly onto a spot on a MALDI target plate, overlaid with 1 μl of 70% formic acid, and allowed to dry at room temperature. A 1-μl volume of HCCA matrix solution (α-cyano-4-hydroxy-cinnamic acid–50% acetonitrile–47.5% water–2.5% trifluoroacetic acid) (Sigma-Aldrich) was added to each spot and allowed to dry. *Escherichia coli* (strain MB11464_1) was used as a calibration standard. Spectrometric measurements were performed using a Microflex LT mass spectrometer (Bruker Daltonik) and analyzed using MALDI Biotyper software (version 3.1). Per manufacturer instructions, a log score value of ≥2.0 met the criteria for species identification. A score of 1.700 to 1.999 allowed correct identification to the level of the genus, while a score of <1.7 was interpreted as showing no identity.

Vitek MS (bioMérieux) analysis was also performed as previously described (28, 30). Single colonies from a 72-h brain heart infusion agar plate were directly smeared onto wells of the target plate; 0.5 μl of ready-to-use 40% formic acid (bioMérieux, Marcy l'Etoile, France) was added to each sample and allowed to air dry at room temperature. A 1-μl volume of ready-to-use HCCA matrix solution (bioMérieux, Marcy l'Etoile, France) was added to each spot and allowed to dry. *E. coli* strain ATCC 8739 was used as a calibration standard. To generate protein mass fingerprints, the FlexiMass-DS target was loaded onto an adapter, prior to insertion into a Vitek MS instrument. Raw spectra were generated with Launchpad v. 2.8 software (Shimadzu-Biotech), and protein mass spectrum fingerprints were then directed to SARAMIS (AnagnosTec GmbH) for analysis. Isolates were identified by comparison with spectra in the reference MS database (containing 3,178 SuperSpectrum [SuperS] entries). Identification at the genus or species level was considered reliable when the score was above the 75% confidence level when matched to the SuperS data, a consensus spectrum for that genus or species compiled from reference spectra of that organism in the database. A result was also acceptable if a "comparison" result, indicating a good match to one or more reference spectra but not to the SuperS data, was obtained.

Bacterial DNA was extracted, amplified, and sequenced using a MicroSEQ 16S rRNA bacterial identification kit (PerkinElmer Applied Biosystems Inc., Foster City, CA, USA). The resulting 16S rRNA gene sequences were then analyzed with MicroSeq 500 software (version 2.2.1). If the sequences were ≥99.0% compatible with the database, the identity was acceptable to the level of the species.

AST. Antimicrobial susceptibility testing (AST) was performed for *E. lenta* clinical isolates and *E. lenta* strain ATCC 43055 (31) using the reference agar dilution procedure (Wadsworth method) and Brucella agar supplemented with 5 μg/ml hemin plus 1 μg/ml vitamin K plus 5% (vol/vol) laked sheep blood, with a final volume of 20 ml/plate (18 ml agar plus 2 ml antibiotic). The "direct colony suspension" method (32, 33) was used. Plates were incubated in an anaerobic chamber for 48 h in an atmosphere of 4% to 7% CO_2. An inoculum of 10^5 CFU/spot was used. Antibiotic powders were supplied by Sigma-Aldrich (penicillin, cefoxitin, ceftriaxone, vancomycin, piperacillin-tazobactam, metronidazole, clindamycin), GlaxoSmithKline (amoxicillin-clavulanate), Merck (ertapenem), Pfizer (tazobactam, tigecycline), Bayer (moxifloxacin), and AstraZeneca (meropenem).

Testing for teicoplanin was performed using the Etest method (bioMérieux) (lot number 1002276240) according to the manufacturer's recommended guidelines (34).

Interpretative criteria. MICs were compared to standard Clinical and Laboratory Standards Institute (CLSI) and European Committee on Antimicrobial Susceptibility Testing (EUCAST) breakpoints (available only for Gram-positive anaerobes) and are reported as susceptible, intermediate, or resistant.

***van* gene PCR.** After incubation for 48 h, the Anaero (Oxoid) plates were swabbed with a dry sterile cotton swab and the organisms were placed into sterile saline solution. Bacteria were pelleted at 10,000 × *g* for 10 min, and the supernatant was discarded. DNA was extracted

TABLE 2 Individual patient diagnoses and treatment information[a]

Case	Age (yrs)/ sex	Diagnosis	Treatment details			Outcome
			Procedure	Antibiotic[b]	Duration (days)	
1	57/F	Viral gastroenteritis	None	Metronidazole	10	Alive at 12 mos
2	75/F	Metastatic gastric cancer	Distal gastrectomy and Roux-en-y reconstruction	Trimethoprim	16	Death at 9 mos
3	44/F	Chronic cholelithiasis	Cholecystectomy	None	0	Alive at 12 mos
4	68/F	Urinary tract sepsis	Sigmoidoscopy	Amoxicillin and metronidazole	12	Alive at 12 mos
5	85/F	Sepsis of unknown source	None	Ceftriaxone	8	Alive at 12 mos
6	87/M	Diabetic foot infection	None	Ticarcillin-clavulanate	12	Death on day 6
7	17/F	Fecal peritonitis secondary to anastomotic leak	Laparotomy, washout, end-ileostomy	Meropenem	71	Alive at 12 mos
8	68/M	Perforated gangrenous appendicitis	Open appendectomy	Ceftriaxone and metronidazole	8	Alive at 12 mos
9	57/F	Perforated sigmoid diverticulum	Pelvic washout + Hartmann's	Amoxicillin-clavulanate	48	Alive at 12 mos
10	81/M	Diverticulitis	Colonoscopy	Ciprofloxacin and metronidazole	19	Death at 8 mos
11	81/M	Prosthetic hip joint infection	None	Ticarcillin-clavulanate	2	Death on day 17
12	12/M	Gastroenteritis	None	None	0	Alive at 12 mos
13	45/F	Diverticulitis	Colonoscopy	Cephalexin and metronidazole	21	Alive at 12 mos
14	94/M	Ischial osteomyelitis	Debridement	Cotrimoxazole and ciprofloxacin	301	Alive at 12 mos
15	81/M	Acute appendicitis	Laparoscopic appendectomy	Metronidazole	3	Alive at 12 mos
16	37/M	Diverticular abscess with colovesical fistula	Gastroscopy	Meropenem	32	Death on day 24
17	11/M	Portal vein thrombophlebitis	None	Clindamycin and ciprofloxacin	43	Alive at 12 mos
18	95/F	Myocardial infarction	Gastroscopy	Ceftriaxone and azithromycin	4	Death on day 4
19	29/F	Labor	None	None	0	Alive at 12 mos
20	77/M	Colitis	Colonoscopy	Amoxicillin-clavulanate	42	Alive at 12 mos
21	70/M	L1–L2 discitis, osteomyelitis, paravertebral abscess, psoas abscess, and meningitis[c]	CT-guided drainage	Meropenem and linezolid	90	Alive at 12 mos
22	47/M	Perforated appendix	Laparoscopic appendectomy	Amoxicillin-clavulanate	18	Alive at 12 mos
23	78/F	Viral gastroenteritis	None	None	0	Alive at 12 mos
24	66/M	Stroke	Colonoscopy	Ceftriaxone and metronidazole	44	Alive at 12 mos
25	87/F	Exacerbation of COPD	None	Amoxicillin and doxycycline	6	Death at 11 mos
26	88/F	Colonic arteriovenous malformations, diverticular disease	Colonoscopy	Amoxicillin-clavulanate	7	Death at 7 mos
27	66/M	Infected femoral artery puncture site, ischemic gut	Debridement and femoral arterial repair; gastroscopy, colonoscopy, and push enteroscopy	Meropenem	43	Death at 3 mos
28	72/M	Biliary sepsis, pancreatic cancer	Percutaneous transhepatic cholangiogram	Amoxicillin-clavulanate and metronidazole	46	Death at 4 mos
29	30/M	Gastroenteritis	None	None	0	Alive at 12 mos
30	87/M	Sigmoid diverticulitis; *Clostridium difficile* colitis	None	Metronidazole	23	Death at 3 mos

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from pellets of bacteria using a PureLink DNA miniprep kit with the Gram-positive modification protocol (Invitrogen). DNA was eluted using 200 μl of 10 mM Tris (pH 8.5). DNA concentrations were determined by Qubit assay. Real-time PCR for *vanA* and *vanB* was performed using a Roche LightCycler FastStart DNA Master HybProbe kit as described by Palladino et al. (35). Control isolates were strain MLG043 (*E. lenta* containing *vanB* [23]) and clinical isolates of *Enterococcus faecium* containing *vanA* and *E. faecium* containing *vanB* (22).

TABLE 2 (Continued)

Case	Age (yrs)/ sex	Diagnosis	Treatment details: Procedure	Treatment details: Antibiotic[b]	Treatment details: Duration (days)	Outcome
31	45/F	Left thigh abscess	Debridement and washout of abscess	Amoxicillin-clavulanate	23	Alive at 12 mos
32	65/F	Non-small-cell lung cancer	Right upper/middle lobectomy; sigmoidoscopy	Piperacillin-tazobactam	54	Alive at 12 mos
33	57/F	Enterocutaneous fistula	Laparotomy, mesh removal, fistula repair, cholecystectomy	Piperacillin-tazobactam	22	Alive at 12 mos

[a] F, female; M, male; COPD, chronic obstructive pulmonary disease; CT, computed tomography.
[b] As most patients received multiple antibiotics, those displayed are the ones used for the longest time.
[c] Previously described case report (12).

Statistical analysis. Categorical data are presented as total numbers and percentages. Continuous data are expressed as means and standard deviations with medians and ranges as required.

RESULTS

Clinical data. The clinical characteristics of 33 patients with *E. lenta* bacteremia are summarized in Tables 1 and 2. The median age was 68 years (mean, 62.4 ± 23.9; range, 11 to 95), and 17/33 (52%) were male. The median length of stay was 11 days (24 ± 26; 1 to 94), and 10/33 (30%) patients were admitted to the intensive care unit (ICU). The majority (64%) of patients had community onset disease, and 39% had polymicrobial bacteremia, with a variety of enteric organisms isolated. Blood cultures flagged positive after a median of 3 days of incubation (2.85 ± 1.09; 1 to 5). Time to positivity could be determined for 30 patients; the median time was 65.5 h (72.5 ± 25.2; 40.1 to 122.6). The most common presenting symptoms were fever (82%) and abdominal pain (64%). A total of 82% of patients had the GIT identified as the likely source. The median SAPS II score was 25 (27 ± 12.2; 6 to 64), the median Charlson comorbidity score was 2 (2.45 ± 2.25; 0 to 7), and the Pitt bacteremia score was 2 (2.15 ± 1.9; 0 to 8). A total of 67% of patients had an abnormal white cell count (WCC; $<4 \times 10^9$/liter or $>11 \times 10^9$/liter) at the time of bacteremia, and the C-reactive protein (CRP) level was elevated in all 31 patients in whom it was measured.

Abdominal imaging was performed in more than half of the total number of patients, and in three-fourths, an abnormality was found. Treatment was dependent on the underlying pathology, with 21/33 patients undergoing at least one procedure. Twelve patients underwent surgery, 3 had radiologically guided procedures (e.g., drainage of abscess), and 11 proceeded to diagnostic and/or therapeutic endoscopy. Antibiotic treatment was variable. Five patients received no antibiotic therapy, and all were alive at 1 year. These were generally patients with self-limiting illnesses who had clinically improved by the time their blood cultures had flagged positive, so their treating clinicians had decided that antibiotic therapy was not indicated. Of those treated with antibiotics, the median duration of treatment was 21.5 days (36.7 ± 56.2; 2 to 301). A wide range of antibiotics were used, most often metronidazole-containing regimens (8 patients) or amoxicillin-clavulanate (6 patients). A 1-year follow-up was available for all patients. Mortality from all causes was 6% at 7 days, 12% at 30 days, and 33% at 1 year.

Isolate identification. A total of 28 isolates were available for comprehensive testing. All were confirmed as *E. lenta* using a Vitek-2 ANC card and 16S rRNA gene sequencing. Five older isolates originally identified by Rapid-ANA were not viable. Twenty-six isolates were available for testing in the Bruker MS system, which accurately identified all 26 to the genus level (log score, >1.7), including 19/26 as *E. lenta* (>2.0). Twenty-three isolates were tested in the bioMérieux MALDI system; all were identified as *E. lenta* with a 99.9% confidence value.

Antimicrobial susceptibility testing (AST). Table 3 shows the AST results for 23 *E. lenta* isolates. Applying CLSI interpretative criteria (32), all isolates were susceptible to amoxicillin-clavulanate, cefoxitin, metronidazole, ertapenem, piperacillin-tazobactam, and meropenem. Susceptibility to clindamycin was 91%, to moxifloxacin was 74%, and to penicillin was 39%; all isolates were resistant to ceftriaxone. No CLSI breakpoints are available for tigecycline, vancomycin, or teicoplanin; however, all isolates had an MIC of ≤1 mg/liter. Using EUCAST breakpoints, interpretations were similar, except for penicillin and piperacillin-tazobactam, to which no isolates were susceptible (36).

***van* gene PCR.** A total of 23 *E. lenta* isolates were tested; all were negative for *vanA* and *vanB* genes. All positive controls tested positive.

DISCUSSION

Our report represents the largest, most comprehensive study of patients with *E. lenta* bacteremia described in the literature to date. Our findings suggest that *E. lenta* bacteremia is associated with a spectrum of disease, ranging from asymptomatic bacteremia (generally seen in the setting of a transient gastrointestinal illness) to part-polymicrobial bacteremia from an intra-abdominal source (e.g., perforated viscus) to severe monomicrobial disseminated disease. A significant proportion of our cohort had serious GIT pathology, often requiring surgical or radiological intervention and ICU admission to achieve cure. This suggests that when identified in blood cultures, *E. lenta* should not necessarily be dismissed as a contaminant but rather should represent a trigger for more-detailed evaluation of the patient and appropriate abdominal diagnostic imaging.

Published reports of *E. lenta* bacteremia are outlined in Table 4. Our cohort has some differences from previous case series. Venugopal et al. (14) provided detailed clinical information on 25 cases but no microbiological data. They reported a higher proportion of health care onset bacteremias (50%). Only 44% of their patients had an abdominal disease source, and 40% had a skin or soft-tissue disease source, most often infected decubitus ulcers, associated with high (44%) rates of hospitalized, bedridden patients.

TABLE 3 Antimicrobial susceptibility testing results for 23 isolates plus control strain[a]

Patient isolate	MIC, mg/liter (interpretive criteria) or [no. (%) susceptible]												
	PEN	FOX[b]	CRO	AMC	TZP	CLI	MOX[b]	MET	ERT	MEM	TGC	VAN[c]	TEC
ATCC 43055	0.5 (S/I)	8 (S)	>128 (R)	1 (S)	16 (S/I)	0.25 (S)	0.25 (S)	2 (S)	1 (S)	0.5 (S)	0.5	1 (S)	0.12
3	1 (I/R)	8 (S)	>128 (R)	1 (S)	16 (S/I)	1 (S)	0.5 (S)	2 (S)	1 (S)	0.5 (S)	0.5	1 (S)	0.12
6	1 (I/R)	8 (S)	>128 (R)	1 (S)	16 (S/I)	0.25 (S)	4 (I)	2 (S)	1 (S)	0.5 (S)	0.5	1 (S)	0.12
8	0.5 (S/I)	4 (S)	128 (R)	1 (S)	16 (S/I)	0.25 (S)	4 (I)	2 (S)	1 (S)	0.25 (S)	0.5	1 (S)	0.12
9	1 (I/R)	8 (S)	128 (R)	1 (S)	16 (S/I)	0.5 (S)	8 (R)	2 (S)	1 (S)	0.5 (S)	0.5	1 (S)	0.12
10	0.5 (S/I)	8 (S)	128 (R)	1 (S)	16 (S/I)	0.25 (S)	4 (I)	2 (S)	1 (S)	0.25 (S)	1	1 (S)	0.12
11	1 (I/R)	16 (S)	>128 (R)	1 (S)	32 (S/R)	0.5 (S)	0.25 (S)	2 (S)	1 (S)	0.5 (S)	0.5	1 (S)	0.12
12	0.5 (S/I)	8 (S)	>128 (R)	1 (S)	16 (S/I)	0.25 (S)	0.5 (S)	2 (S)	1 (S)	0.5 (S)	0.5	1 (S)	0.12
13	1 (I/R)	8 (S)	>128 (R)	1 (S)	16 (S/I)	0.25 (S)	0.25 (S)	2 (S)	1 (S)	0.5 (S)	0.5	1 (S)	0.12
14	1 (I/R)	8 (S)	>128 (R)	1 (S)	16 (S/I)	0.12 (S)	0.25 (S)	2 (S)	1 (S)	0.5 (S)	1	1 (S)	0.25
16	0.5 (S/I)	8 (S)	>128 (R)	1 (S)	16 (S/I)	2 (S)	0.25 (S)	2 (S)	1 (S)	0.5 (S)	0.5	0.5 (S)	0.12
17	1 (I/R)	16 (S)	>128 (R)	1 (S)	32 (S/R)	0.5 (S)	0.25 (S)	2 (S)	1 (S)	0.5 (S)	0.5	1 (S)	0.12
18	0.5 (S/I)	8 (S)	>128 (R)	1 (S)	16 (S/I)	0.25 (S)	1 (S)	2 (S)	1 (S)	0.5 (S)	1	1 (S)	0.12
19	0.5 (S/I)	8 (S)	>128 (R)	1 (S)	16 (S/I)	32 (R)	0.25 (S)	2 (S)	1 (S)	0.25 (S)	0.5	1 (S)	0.12
21	1 (I/R)	8 (S)	>128 (R)	1 (S)	16 (S/I)	8 (R)	64 (R)	2 (S)	1 (S)	0.5 (S)	0.5	1 (S)	0.12
22	0.5 (S/I)	8 (S)	>128 (R)	1 (S)	16 (S/I)	0.25 (S)	0.25 (S)	2 (S)	1 (S)	0.5 (S)	1	1 (S)	0.25
23	0.5 (S/I)	8 (S)	>128 (R)	1 (S)	16 (S/I)	0.25 (S)	0.25 (S)	2 (S)	1 (S)	0.5 (S)	0.5	1 (S)	0.25
24	1 (I/R)	8 (S)	>128 (R)	1 (S)	16 (S/I)	0.25 (S)	4 (I)	2 (S)	1 (S)	0.5 (S)	1	1 (S)	0.25
25	1 (I/R)	16 (S)	>128 (R)	1 (S)	16 (S/I)	0.25 (S)	0.25 (S)	2 (S)	1 (S)	0.5 (S)	0.5	1 (S)	0.12
26	1 (I/R)	8 (S)	>128 (R)	1 (S)	16 (S/I)	0.12 (S)	2 (S)	2 (S)	1 (S)	0.25 (S)	0.5	1 (S)	0.25
27	1 (I/R)	8 (S)	>128 (R)	1 (S)	16 (S/I)	1 (S)	0.25 (S)	2 (S)	1 (S)	0.5 (S)	0.5	1 (S)	0.25
28	1 (I/R)	8 (S)	>128 (R)	1 (S)	32 (S/R)	0.5 (S)	0.25 (S)	2 (S)	1 (S)	0.5 (S)	0.5	1 (S)	0.12
29	1 (I/R)	8 (S)	>128 (R)	1 (S)	32 (S/R)	0.5 (S)	0.25 (S)	2 (S)	1 (S)	0.5 (S)	0.5	1 (S)	0.12
30	0.5 (S/I)	8 (S)	>128 (R)	1 (S)	16 (S/I)	0.25 (S)	0.25 (S)	2 (S)	1 (S)	0.5 (S)	0.5	1 (S)	0.12
Total [no. (%) susceptible]	9 (39)	23 (100)	0	23 (100)	23 (100)	21 (91)	17 (74)	23 (100)	23 (100)	23 (100)	NB	23 (100)	NB

[a] PEN, penicillin; FOX, cefoxitin; CRO, ceftriaxone; AMC, amoxicillin-clavulanate; TZP, piperacillin-tazobactam; CLI, clindamycin; MOX, moxifloxacin; MET, metronidazole; ERT, ertapenem; MEM, meropenem; TGC, tigecycline; VAN, vancomycin; TEC, teicoplanin; S, susceptible; I, intermediate; R, resistant; CLSI/EUCAST results (where interpretations are discordant); NB: no breakpoints available.
[b] CLSI breakpoint only.
[c] EUCAST breakpoint only.

They identified ICU admission and absence of fever on the day of admission as risk factors for increased 30-day mortality, which was much higher than in our series (36% versus 12%). Interestingly, of our 10 patients admitted to ICU, all were alive at 30 days, and of the 4 patients dead at 30 days, 2 were febrile on admission. Lee et al. (13) described 7 patients with *E. lenta* bacteremia. They had a comparatively lower median age of 56, 71% were in the community onset category, and 43% had an underlying malignancy. The report did not identify the source of infections, although 30% had "abdominal symptoms." Lau et al. (5, 6) described 5 cases of *E. lenta* bacteremia, 3 of which were polymicrobial and 2 of which occurred in patients with infected decubitus ulcers.

In addition to these series, there are 6 published case reports of patients with *E. lenta* bacteremia. Two describe severe, disseminated disease with multifocal abscesses requiring extended courses of intravenous antibiotics and/or surgical drainage to cure (12, 38). There are other published cases of severe *E. lenta* infection without definite bacteremia, including reports of frontal sinusitis (43), pyomyositis (44), cutaneous abscesses (45), spondylodiscitis (46), and liver abscess (47). *E. lenta* has also been implicated in the pathogenesis of bacterial vaginosis (48–50) and linked to appendicitis in children (51).

Identification of *E. lenta* by MALDI-TOF was initially not reliable due to the limited number of anaerobic bacterial species included in earlier versions of the databases (28, 52). In 2011, Justesen et al. (30) reported that one *E. lenta* isolate was identified by the Bruker MS method using an updated database but not by the Shimadzu MALDI-TOF MS method. In more recent studies, *E. lenta* has been correctly identified using the Bruker MS method: 2/2 isolates to the species level (15), 8/8 isolates to the genus level and 6/8 isolates to the species level (16), and 8/10 isolates to the genus level and 2/10 isolates to the species level (17). Our study showed that *E. lenta* can be reliably identified using both available commercial MALDI-TOF MS platforms.

Access to anaerobic AST is problematic, however, and a lack of available data has led to breakpoint extrapolation from other anaerobic species to *E. lenta*. Our antimicrobial susceptibility results are consistent with those of previous studies (Table 5), which have shown that isolates are frequently resistant to penicillin and have variable susceptibility to clindamycin and moxifloxacin but remain susceptible to amoxicillin-clavulanate, cefoxitin, metronidazole, and carbapenems. The differences between the EUCAST (susceptible [S], ≤8/4 mg/liter; resistant [R], >16/4 mg/liter) and CLSI (S, ≤32/4 mg/liter; intermediate [I], 64/4 mg/liter; R, ≥128/4 mg/liter) breakpoints for piperacillin-tazobactam are significant and, given the high piperacillin-tazobactam MICs of our isolates, have resulted in significant differences in the interpreta-

TABLE 4 Previously reported case series and case reports of *E. lenta* bacteremia

Category and reference (no. of patients)	Age(s) (range)/no. of patients of indicated sex	Diagnosis	Treatment detail(s)			Outcome
			Procedure	Antibiotic	Duration	
Case series						
14 (25)	36–90/5 M	Decubitus ulcer (8 patients)	NS[a]	4 received piperacillin-tazobactam, 1 metronidazole and ciprofloxacin, 1 ampicillin-sulbactam—amoxicillin-clavulanate, 1 vancomycin, ceftazidime and metronidazole, 1 ceftriaxone and metronidazole	NS	50% survival
	36–83/4 M	Intra-abdominal sepsis (10 patients)	NS	Different regimens, including piperacillin-tazobactam, amoxicillin-clavulanate, ciprofloxacin or ceftriaxone and metronidazole, imipenem/cilastatin	NS	80% survival
	72, 78/F	Urinary source (2 patients)	NS	Ceftriaxone-ciprofloxacin; vancomycin and metronidazole	NS	50% survival
	51/M	Gluteal abscess and left hip osteomyelitis	NS	Piperacillin-tazobactam	NS	Survived
	81/M, 72/F	Not stated (2 patients)	NS	Metronidazole and ciprofloxacin; none	NS	50% survival
	71/F	Gastrointestinal bleed	NS	None	NS	Death
	79/F	Percutaneous endoscopy gastrostomy site infection	NS	Clindamycin	NS	Survival
13 (7)	Median, 56 (51–93)	Two patients had "abdominal symptoms"	NS	2 received penicillins, 4 cephalosporins, 1 metronidazole	NS	3 deaths within 60 days
5, 6[b] (5)	69/F	Lung cancer, intestinal obstruction	NS	Metronidazole	NS	Cured
	74/M	Alcoholic cirrhosis, gastrointestinal bleeding	NS	Ticarcillin/clavulanate	NS	Died
	75/F	Pelvic inflammatory disease	NS	Cefuroxime + metronidazole	NS	Cured
	87/F	Infected decubitus ulcer	NS	Cefuroxime + cloxacillin	NS	Died
	84/F	Infected decubitus ulcer	NS	Cefuroxime + metronidazole + netilmicin	NS	Cured
37 (2)	82/M	Intestinal perforation	Colostomy	Piperacillin-tazobactam and ciprofloxacin, imipenem	1 mo	Favorable
	33/M	Acute appendicitis and parietal abscess	Appendicectomy	Amoxicillin-clavulanate	Unclear	Favorable[c]
Case reports						
12	70/M	Spondylodiscitis, osteomyelitis, meningitis, and multiple abscesses	Drainage of subdiaphragmatic and right psoas collections	Meropenem and linezolid	3 mos	Favorable
38	19/M	Multifocal abscesses—brain, liver, spleen, lung	Laparoscopic cholecystectomy	i.v.[d] penicillin G and oral metronidazole	5 mos	Favorable
39	53/M	Metastatic rectal cancer	None	i.v. cefotaxime and amikacin—oral antibiotics	>4 days	Favorable[c]
40	21/F	Crohn's disease; small bowel obstruction	Ileocecal resection and repair of anastomotic leak	Vancomycin and piperacillin-tazobactam—vancomycin, meropenem and metronidazole—meropenem	3 weeks	Favorable[c]
41	86/F	Infected decubitus ulcer	None	Cefuroxime—amoxicillin	>2 days	Favorable[c]
42	68/F	Tubo-ovarian abscess; postoperative bowel leak	Hysterectomy, salpingo-oophrectomy; sigmoid resection and Hartmann's, drainage of perisplenic collection	Ampicillin, gentamicin, metronidazole—vancomycin, ticarcillin/clavulanate, metronidazole	>1 mo	Favorable[c]

[a] NS, not stated.
[b] Two publications describing the same patient cohort.
[c] Alive and well at hospital discharge; long-term follow-up not reported.
[d] i.v., intravenous.

tions of our susceptibility results. Neither criterion is supported by clinical outcome studies, and it may well be that piperacillin-tazobactam has activity against *E. lenta*. Although *vanB* has been detected previously in *E. lenta* in our laboratory (21–23) and high vancomycin MICs have been reported in the literature (53), we did not detect vancomycin resistance gene *vanA* or *vanB* or phenotypic resistance in any of the isolates that were tested.

In summary, *Eggerthella lenta* appears to be a significant human pathogen that is often associated with serious GIT pathology. If the diagnosis is not apparent at the time of isolation of this organism in blood cultures, detailed investigation of the abdomen should be undertaken. *E. lenta* is likely to be increasingly identified given the ease and rapidity of MALDI-TOF MS and the uptake of this technology into many clinical microbiology laboratories worldwide. The most reliable antibiotic treatment options appear to be cefoxitin, metronidazole, amoxicillin-clavulanate, and the carbapenems, with the role of piperacillin-tazobactam currently unclear. However, with rising rates of resistance in anaerobes increasingly reported, AST should be considered to guide therapy, particularly in serious infections.

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TABLE 5 *E. lenta* MIC ranges and susceptibility rates reported in the literature[a]

Reference (yr)	No. of isolates	MIC range (mg/liter), % susceptible[b]											
		PEN	FOX	CRO	AMC	TZP	CLI	MOX	MET	ERT	MEM	TGC	VAN
53 (2013)	20								0.125 to 0.5				1 to 8
54 (2013)	10		8 to 16, 100			16 to 64, 90	0.25 to 0.5, 100		0.5 to 1, 100		1 to 2, 100	0.5	
13 (2012)	8	1 to 4, 0				32 to 64, 87.5	0.5 to >32, 37.5	0.25 to >32, 75	1 to 2, 100		0.5, 100	0.12 to 0.25	0.5 to 2
55 (2009)	17				0.5 to 1	8 to 16	0 to >32		0.25 to 0.5				
33 (2006)	10	≤0.06 to 4, 10	0.5 to 8, 100	≤0.12 to 256, 30	≤0.06 to 1, 100	≤0.12 to 32, 100	≤0.06 to 1, 100		0.12 to 1, 100		≤0.06 to 0.5, 100		
6 (2005)	9	0.12 to 0.5*					0.12 to 0.5						0.5 to 1
56 (2005)	14					≤0.12 to 32						≤0.06 to 1	0.25 to 2
57 (2004)	10	≤0.03 to 0.5*				≤0.03 to 32							0.5 to 1
58 (2004)	20					0.25 to 32, 95	0.12 to 8, 90	0.03 to 1, 100	0.06 to 4, 100				
59 (2003)	8	1*			1		0.06 to 0.25	0.25 to 0.5	≤0.125 to 0.5				1 to 2
60 (2003)	12	≤0.06 to 1*			≤0.06 to 2		≤0.06 to 0.5	≤0.06 to 1	≤0.06 to >64				
61 (2003)	17	0.25 to 2*	1 to 16				≤0.03 to 1		0.125 to 1				0.5 to 2
62 (2002)	16			0.25 to >64		≤0.125 to 32	0.03 to >32		0.25 to >16	0.06 to 2	0.06 to 4		
64 (1998)	Group A, 25	2,* 0		32 to 128, 0			0.5 to 4, 88		1 to 2, 100				
	Group B, 6	0.5,* 100		1, 100			≤0.06, 100		1 to 2, 100				

[a] All studies utilized CLSI agar dilution methodology and CLSI breakpoints.

[b] PEN, penicillin (*, ampicillin); FOX, cefoxitin; CRO, ceftriaxone; AMC, amoxicillin-clavulanate; TZP, piperacillin-tazobactam; CLI, clindamycin; MOX, moxifloxacin; MET, metronidazole; ERT, ertapenem; MEM, meropenem; TGC, tigecycline; VAN, vancomycin.

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