Diabetic Foot Ulcer with *Alcaligenes faecalis* Infection

Chienhsiu Huang

Department of Internal Medicine, Dalin Tzu Chi Hospital, Dalin Town, Taiwan

Keywords

Alcaligenes faecalis · Diabetic foot ulcer · Extensive drug resistant

Abstract

Background: Diabetic foot ulcers are an increasingly common complex problem and are associated with a very considerable health care burden. Diabetic foot ulcer with *Alcaligenes faecalis* infection is rarely reported in the literature. We report a case series of diabetic foot ulcer with *A. faecalis* infection treated at our facility. **Methods:** We conducted a retrospective analysis of all patients with diabetic foot ulcer with *A. faecalis* infection seen from January 2014 to April 2019. We analyzed the clinical characteristics, ulcer lesion classification, comorbidities, prior intravenous antibiotic use within 3 months, wound culture, antibiotics sensitivity test, and clinical outcomes of these patients. **Results:** Eight cases of diabetic foot ulcer with *A. faecalis* infection were seen in 5 males and 3 females. Mean age was 54.6 years. All patients had other comorbidities, and all ulcer lesions were of chronic duration (>14 days). All wound cultures revealed polymicrobial infection, with 2 cases of diabetic foot with extensive drug-resistant *A. faecalis* infection found in 2019. All patients needed intravenous antibiotic therapy and surgical interventions for the chronic ulcer lesion. The wound failed to heal in 3 patients. **Conclusions:** All diabetic foot ulcers with *A. faecalis* infection were of chronic duration (>14 days) and had polymicrobial infection. Extensive drug-resistant *A. fae-*

calis emerged in 2019. Definitive antibiotic therapy is necessary for all infected wounds and should be based on both the culture results and susceptibility data. All patients will need appropriate wound care, and most will need rapid surgical intervention for an optimal outcome.

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Introduction

Diabetic foot ulcers are an increasingly common complex problem and are associated with a very considerable health care burden [1]. Foot ulcer is the most frequent diabetes-related cause of hospitalization. The global prevalence of diabetic foot ulcer is 6.3% [2]. People with diabetes have an about 25% chance of developing a foot ulcer in their lifetime [3]. About half of all diabetic foot ulcers are infected at presentation [4]. Acute infections in patients who have not recently received antimicrobials are often monomicrobial. Gram-positive cocci are the predominant organisms, and *Staphylococcus aureus* is the most commonly isolated pathogen. Chronic infections are often polymicrobial, including Enterococci, Enterobacteriaceae, *Pseudomonas aeruginosa*, other non-fermenting Gram-negative bacilli, and obligate anaerobes [5–9].

Alcaligenes faecalis is a Gram-negative, obligate aerobe, oxidase-positive, catalase-positive, and non-fermenting bacterium commonly found in soil, water, and hospital environments. The organism has been isolated from a range

Table 1. Clinical characteristics and treatment outcome in 8 cases of diabetic foot ulcer with *Alcaligenes faecalis* infection

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8
Year	2014	2015	2015	2017	2018	2018	2019	2019
Duration of wound	14 days	5 months	1 month	3 months	14 days	12 months	14 days	18 months
Comorbidities	DM HTN	DM HTN ESRD CVA	DM CKD CVA	DM HTN	DM PAD ESRD CAD	DM CKD	DM HTN ESRD PAD	DM PAD CKD
Ulcer wound	+	+	+	+	+	+	+	+
Abscess	Nil	+	Nil	+	Nil	+	+	Nil
Cellulitis	+	+	Nil	Nil	Nil	Nil	Nil	+
Necrotizing fasciitis	Nil	+	+	Nil	+	Nil	+	+
Osteomyelitis	Nil	Nil	+	+	Nil	Nil	Nil	Nil
Gangrene	Nil	+	Nil	Nil	+	Nil	+	+
Texas wound classification system [21]	2B	2D	3B	3B	2D	2B	2D	2D
IDSA classification of infected DFU [22]	MOD Class B	MOD Class B	MOD Class B	MOD Class B	Severe Class C	MOD Class B	MOD Class B	MOD Class B
Treatment	SAM	MPM VAN	VAN CAZ	OXA CMZ	MPM VAN	CAZ TEC	CAZ VAN	COL
Operation	DEB	DEB	SEQ	DEB	AMP	DEB	DEB	DEB
Outcome	Heal	Heal	Heal	Heal	No heal	No heal	Heal	No heal

DM, diabetes mellitus; HTN, hypertension; ESRD, end-stage renal disease; CVA, cerebral vascular accident; CKD, chronic kidney disease; PAD, peripheral artery disease; CAD, coronary artery disease.

IDSA, Infectious Disease Society of America; DFU, diabetic foot ulcer; MOD, moderate.

SAM, ampicillin-sulbactam; MPM, meropenem; VAN, vancomycin; CAZ, ceftazidime; OXA, oxacillin; CMZ, cefmetazole; TEC, teicoplanin; COL, colistin.

DEB, debridement; SEQ, sequestrectomy; AMP, amputation.

of clinical materials such as urine, blood, wound discharge, stool, cerebrospinal fluid, and respiratory secretions [10–12]. *A. faecalis* has been associated with endocarditis, bacteremia, meningitis, endophthalmitis, skin and soft tissue infection, urinary tract infection, otitis media, peritonitis, and pneumonia [13–20]. We here report a series of patients with diabetic foot ulcer with *A. faecalis* infection, in order to improve the clinical care of such patients.

Methods

We conducted a retrospective analysis of all patients presenting with diabetic foot ulcer with *A. faecalis* infection who were admitted to Dalin Tzu Chi Hospital, Taiwan, from January 2014 through April 2019. Patients were diagnosed with diabetic foot ulcer with infection when their clinical symptoms and signs indicated skin and soft tissue infection, such as redness, warmth, induration, tenderness, ulcer lesion, abscess, gangrene, fasciitis, or osteomyelitis. Ulcer lesion classification was done by using the University of Texas wound classification system [21], and the infected diabetic foot ulcer classification system of the Infectious Disease Society of America [22]. The medical records of all patients were reviewed for demographic information, clinical symptoms and signs, pharmacological therapy for blood sugar, admission HbA1c values, blood sugar val-

ues (at 6 a.m., 11 a.m., 5 p.m., and 9 p.m.), comorbidities, use of intravenous (IV) antibiotics within 3 months, wound culture, antibiotics sensitivity test, and clinical outcomes. Superficial wound swab specimens were collected for culture. Specimens were obtained only after the wound was cleaned using povidone iodine solution and sterile saline. Wound swab specimens for antibiotic susceptibility were done using the Vitek II system with ASTGN87 cards (BioMérieux, Marcy-l'Étoile, France) with the interpretive criteria M100-25th of the Clinical and Laboratory Standards Institute.

Wound swab specimens were cultured as soon as they were collected, on chocolate agar, blood agar, eosin methylene blue agar, colistin-nalidixic acid agar plates, and thioglycollate broth. Gram staining and microscopy were prepared and read. The 5 culture mediums were incubated in a carbon dioxide-enriched atmosphere for 16–18 h. Then, a sterile swab stick was used to transfer a sufficient number of colonies of pure culture and to suspend the microorganisms in 3.0 mL of sterile saline (aqueous 0.45–0.50% NaCl, pH 4.5–7.0) in a 12 × 75 mm clear plastic test tube. The turbidity was adjusted to 0.50–0.63 McFarland Equivalence Turbidity Standard. Identification cards were inoculated with microorganism suspensions using an integrated vacuum apparatus. A test tube containing the microorganism suspension was placed into a cassette. One GN identification card (automated identification of 135 taxa of the most significant fermenting and non-fermenting Gram-negative bacilli) and another VITEK II AST-N322 card (for susceptibility testing of aerobic Gram-negative bacilli against specified antimicrobials) were placed in the

Table 2. Bacteriology in 8 cases of diabetic foot ulcer with *Alcaligenes faecalis* infection

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8
Year	2014	2015	2015	2017	2018	2018	2019	2019
Prior intravenous antibiotics used within 3 months	CIP SAM	PIP CRO VA	Nil	CEF TEC	Nil	CAZ CIP PIP TEC	CAZ VAN	LVX MPM TEC
Results of antibiotics sensitivity test of <i>Alcaligenes faecalis</i>								
GM	S	S	S	R	S	I	R	R
AN	S	S	S	S	S	S	R	R
CAZ	S	S	S	S	S	S	R	R
FEP	S	S	S	I	S	I	R	R
SAM	S	S	S	I	S	S	R	R
TZP	S	S	S	I	S	S	R	R
CIP	S	S	S	R	S	R	R	R
IPM	S	S	S	S	S	S	R	R
MPM	S	S	S	S	S	S	R	R
TGC	NA	NA	NA	NA	NA	NA	NA	S
Mixed infection pathogens								
Enterococcus	V				V			V
<i>Proteus vulgaris</i>		V			V			
<i>Citrobacter koseri</i>			V					
MRSA			V					
<i>Klebsiella pneumoniae</i>						V	V	V
<i>Providencia rettgeri</i>						V		
<i>Acinetobacter baumannii</i>							V	V
<i>Morganella morganii</i>				V				
<i>Staphylococcus epidermis</i>				V				

CIP, ciprofloxacin; SAM, ampicillin-sulbactam; PIP, piperacillin; CRO, ceftriaxone; VA, vancomycin; CEF, cefazolin; TEC, teicoplanin; CAZ, ceftazidime; VAN, vancomycin; LVX, levofloxacin; MPM, meropenem.

GM, gentamicin; AN, amikacin; FEP, cefepime; TZP, piperacillin-tazobactam; IPM, imipenem; TGC, tigecycline; S, sensitive; R, resistant; I, intermediate.

MRSA, methicillin-resistant *Staphylococcus aureus*.

neighboring slots, along with the transfer tube and the corresponding suspension tube. Calculations were performed on raw data and compared to thresholds to determine the reactions for each test.

Statistical Analysis

The difference between wound-healing patients and no-wound-healing patients in terms of blood sugar values (at 6 a.m., 11 a.m., 5 p.m., and 9 p.m.) were evaluated using the Student *t* test for continuous variables. All statistical analyses were conducted using the statistical package SPSS for Windows version 17.0 (SPSS, Inc., Chicago, IL, USA). A *p* value <0.05 was considered statistically significant.

Results

Nine cases of *A. faecalis* skin and soft tissue infection were seen during the study period; One case had a surgical wound infection. Eight cases had diabetic foot ulcer infections, including 5 male and 3 female patients. The mean

age was 54.6 years (range 41–85 years). All patients had other comorbidities. All foot ulcer lesions were of chronic duration (>14 days; range 14 days to 18 months). Two patients had no history of prior IV antibiotics used within 3 months for their chronic ulcer lesion. All wound cultures displayed polymicrobial infection. The antibiotics sensitivity test showed the presence of extensive drug-resistant (XDR) *A. faecalis* beginning in 2019. Two patients did not receive adequate antibiotic therapy. Case 4 was treated with oxacillin and cefmetazole, which do not treat *A. faecalis*. Case 7 was treated with ceftazidime and vancomycin, which do not treat *A. faecalis* and *Acinetobacter baumannii*. All patients needed surgical intervention for their chronic ulcer lesions. The wound did not heal in 3 patients.

Tables 1–3 show the clinical characteristics, pharmacological therapy for blood sugar, admission HbA1c values, 1-week average blood sugar values, treatment outcomes,

Table 3. The 1-week average of blood sugar values and pharmacological therapy in 8 cases of diabetic foot ulcer with *Alcaligenes faecalis* infection

	Pharmacological therapy	HbA1c %	Blood sugar, mg/dL				Wound heal
			at 6 a.m.	at 11 a.m.	at 5 p.m.	at 9 p.m.	
Case 1	Insulin Glargine 14 IU QD NovoRAPID 12 IU TID	7.5	337	403	410	333	Heal
Case 2	Linagliptin 5 mg QD Insulin Glargine 8 IU HS Repaglinide 1 mg TID	6.3	104	127	131	113	Heal
Case 3	Linagliptin 5 mg QD Acarbose 50 mg TID	5.3	113	142	97	128	Heal
Case 4	Metformin 500 mg BID Empagliflozin 25 mg QD Acarbose 100 mg BID	6.2	105	135	120	131	Heal
Case 5	Humalog mix 50, 22 IU TID	6.5	218	214	223	188	No heal
Case 6	Insulin detemir 70 IU QD Insulin aspart 25 IU TID	9.0	316	330	348	328	No heal
Case 7	Repaglinide 1 mg TID Insulin detemir 26 IU QD	7.4	159	159	179	162	Heal
Case 8	Insulin Glargine 30 IU QD	6.1	235	242	288	283	No heal

and bacteriology of the 8 patients with diabetic foot ulcer with *A. faecalis* infection. The average blood sugar values at 6 a.m. were significantly different between wound-healing patients and no-wound-healing patients ($p = 0.008$). The average blood sugar values at 11 a.m. were not significantly different between wound-healing patients and no-wound-healing patients ($p = 0.108$). The average blood sugar values at 5 p.m. were significantly different between wound-healing patients and no-wound-healing patients ($p = 0.028$). The average blood sugar values at 9 p.m. were significantly different between wound-healing patients and no-wound-healing patients ($p = 0.009$).

Discussion

The literature shows 12 cases of skin and soft tissue *A. faecalis* infection, including 4 cases of toe web infection [23], 1 case of dog bite cellulitis [24], 2 cases of surgical wound infection [17], 1 case of superinfection in the foot tinea pedis [17], and 4 cases of diabetic foot ulcer infection [13, 15, 17]. Other studies and our case series indicate that diabetic foot ulcer accounts for 57.1% of all *A. faecalis* infections in skin and soft tissue. In 1997, Bizet and Bizet [13] reported the case of a 64-year-old male with

insulin-dependent diabetes and left foot ulcer with *A. faecalis* infection. The patient was treated with IV amoxicillin-clavulanic acid for 10 days [13]. In 2014, Tena et al. [17] reported 2 cases of diabetic foot ulcer with *A. faecalis* infection, all with polymicrobial infection. All strains of *A. faecalis* were susceptible to amoxicillin-clavulanic acid. One case was a cured foot ulcer, and the other case was a recurrence of foot ulcer [17]. In 2019, Chua et al. [15] reported the case of a 63-year-old female with a diabetic foot ulcer with extended spectrum β -lactamase (ESBL)-producing *A. faecalis* infection. They suggested that imipenem and meropenem may be considered as agents of choice for an ESBL-producing *A. faecalis* infection [15].

In our series, all cases had an ulcer lesion of chronic duration (>14 days), and all had mixed infection with other bacteria. The most common additional pathogens were *Enterococcus*, *Klebsiella pneumoniae*, *Proteus vulgaris*, and *A. baumannii*. Gram-negative bacilli account for 71.4% of all mixed infection pathogens. In 2017, the first case with *A. faecalis* resistant to gentamicin and ciprofloxacin was reported. Unfortunately, diabetic foot ulcer with XDR *A. faecalis* infection emerged in 2019, and IV tigecycline or colistin was needed to treat it. There is no literature on XDR *A. faecalis* diabetic foot ulcer infection, but 2 cases of XDR *A. faecalis* pneumonia and 1 case

of pandrug-resistant *A. faecalis* bacteremia have been reported [19, 20, 25]. In 2018, Junejo et al. [19] reported on a 73-year-old male with *A. faecalis* pneumonia. The sensitivity analysis of the sputum culture showed that the isolated *A. faecalis* was resistant to anti-pseudomonas penicillins, carbapenems, aminoglycosides, and quinolones. The patient was treated with polymyxin B and became hemodynamically stable [19]. In 2017, Agarwal et al. [20] reported on a 32-year-old man with Dengue hemorrhagic fever and pneumonia. Bronchoalveolar lavage fluid culture isolated *A. faecalis*, and the antibiotic sensitivity test showed sensitivity to colistin and tigecycline only. Colistin and meropenem were given, but the patient later succumbed to his illness [20]. In our Cases 7 and 8, the antibiotic sensitivity test was the same as in the cases of Agarwal et al. and Junejo et al.

In 2019, Hasan et al. [25] reported on a 60-year-old female with pandrug-resistant *A. faecalis* who was treated with double-dose tigecycline and had a successful treatment outcome. It was terrible that pandrug-resistant *A. faecalis* pathogen had been found. We emphasize that *A. faecalis* has emerged as a pathogen resistant to commonly used antibiotics such as anti-pseudomonas penicillins, carbapenems, aminoglycosides, and quinolones. It is too early to know the pathogenesis of emerging multidrug-resistant *A. faecalis*. The medical community urgently needs microbiologists to explore the pathogenesis of emerging multidrug-resistant *A. faecalis*, so as to control this pathogen.

Although Cases 4 and 7 did not receive adequate IV antibiotics to treat the *A. faecalis* infection, they received surgical intervention and wound care. Their diabetic foot ulcer infections were healing. Their diabetic foot ulcers were local infections that could be cured by adequate debridement and appropriate wound care. This result stresses that wound care and surgical interventions are important in treating diabetic foot ulcer infections. In 3 cases, the diabetic foot ulcer did not heal. Case 5 had wound infection complicated by septic shock. This patient had to undergo right lower leg below the knee amputation to save her life. Case 6 had peripheral artery disease (PAD) and chronic kidney disease as comorbidities. The duration of his diabetic ulcer lesion was 12 months. The patient received IV antibiotics with ceftazidime and tigecycline therapy, as well as debridement, but the leg ulcer lesion still failed to heal. Case 8 had PAD, chronic kidney disease, bed-ridden hypoxic encephalopathy, and prolonged mechanical ventilation as comorbidities. The duration of his diabetic ulcer lesion was 18 months. The ulcer lesion showed infection with XDR *A. faecalis*. The patient was treated with IV colistin and wound debridement, but the

ulcer lesion still failed to heal. Two of the 3 cases that failed to heal had PAD comorbidity and the 2 cases did not undergo any revascularization procedures. PAD may be an influential factor of poor wound healing in diabetic foot ulcer. The study by Brechow et al. found that the severity of PAD significantly influences the ability of diabetic foot ulcer to heal [26]. Weck et al. reported that poor wound healing is often aggravated by PAD as comorbidity [27].

Many factors can interfere with diabetic foot ulcer healing, such as wound infection control, blood glucose control, immune function, nutrition, etc. In Christman et al.'s study, it was suggested that glycemia may be an important biomarker in predicting wound-healing rate in diabetic patients [28]. Markuson et al.'s study showed that healing took significantly longer for individuals with lower admission HbA1c values compared with those with higher levels [29]. Our report of diabetic foot ulcer with *A. faecalis* infection consists of only 8 cases, and we could not draw any clinical conclusions by admission HbA1c. We collected our 8 patients' blood sugar level 4 times a day for a week and found that optimizing glucose control is an influencing factor of diabetic foot ulcer healing.

The infection of diabetic foot ulcers may contribute to delayed healing. Optimal management of diabetic foot infections can reduce the incidence of infection-related morbidities and the incidence of limb amputation. Antibiotic therapy is necessary for all infected wounds. Definitive therapy should be based on both the culture results and susceptibility data. It is important to remember that all patients will need appropriate wound care, and most will need rapid surgical intervention.

Limitations

Our case series report of diabetic foot ulcer with *A. faecalis* infection is small and therefore can provide only minimal clinical experience. Additional reports of diabetic foot ulcer with *A. faecalis* infection will add to the knowledge of how to treat this multi-resistant pathogen infection.

Conclusion

Diabetic foot ulcer with *A. faecalis* infection is rarely reported in the literature. XDR *A. faecalis* has emerged recently in our hospital. Diabetic foot ulcer with *A. faecalis* infection is common in those with an ulcer lesion of chronic duration (>14 days), and the infections are commonly polymicrobial. Adequate antibiotic treatment, appropriate wound care, and surgical intervention are all crucial for healing.

Statement of Ethics

The project was approved by Buddhist Dalin Tzu Chi General Hospital's research Ethics Committee (approved IRB No.: B10802024). Written informed consents were obtained from the patients for publication of this report.

Conflict of Interest Statement

The author declares that they have no competing interests.

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Author Contributions

This is a single-author paper. C.H. designed the study, collected the data, analyzed the data, wrote the manuscript, and reviewed the manuscript.

Availability of Data and Materials

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

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