Acinetobacter baumannii orofacial cellulitis: report of 2 cases

Thaís dos Santos Fontes Pereira, DDS, PhD,[a] Denise Vieira Travassos, DDS, PhD,[b]
Raissa Cristina Costa Silva, DDS,[a] Laiz Fernandes Mendes Nunes, DDS,[c] Maria Edileusa Santos, DDS,[c]
Célia Regina Lanza, DDS, PhD,[a] Gabriela Assunção Goebel, MD,[d]
Fabiana Maria Kakehasi, MD, PhD,[e] and Tarcília Aparecida Silva, DDS, PhD[a]

Acinetobacter baumannii infection of skin and soft tissues is uncommon and usually associated with trauma. The present report describes 2 pediatric cases of cellulitis in the orofacial region, caused by *A. baumannii* infection with a fatal outcome. A 12-year-old male patient, diagnosed with acute promyelocytic leukemia, presented with an ulcerated lesion on the lip suggestive of local trauma. The condition progressed to cellulitis, epithelial necrosis, and nonspecific vesicles and blisters. The second case occurred in a 10-year-old male patient with a diagnosis of Burkitt lymphoma. The patient's condition progressed to World Health Organization Grade IV mucositis and cellulitis. In both cases, hemoculture was positive for multidrug-resistant *A. baumannii*. In conclusion, *A. baumannii* should be considered a potentially multidrug-resistant pathogen in the presence of skin and soft tissue cellulitis. Ulcerated oral lesions may place hospitalized pediatric patients at risk for *A. baumannii* infection. (Oral Surg Oral Med Oral Pathol Oral Radiol 2019;127:e118–e122)

Acinetobacter baumannii is a gram-negative microorganism that can cause serious infections, including meningitis, pneumonia, endocarditis, and necrotizing fasciitis.[1-4] *A. baumannii* infection is more likely to occur in critically ill patients, with longer stays in intensive care units (ICUs) and longer ventilator dependence and in those treated with broad-spectrum antibiotics.[5,6] Although *A. baumannii* is frequently detected in human clinical settings, a wide variety of *A. baumannii* has also been isolated from pets and the environment.[7]

A. baumannii is found in conjunction with periodontal pathogens. This organism is associated with sites of periodontitis with increased probing depth and clinical attachment level. The presence of *A. baumannii*, *Aggregatibacter actinomycetemcomitans*, *Pseudomonas Aeruginosa*, and red complex increases the likelihood of aggressive periodontitis.[8] In has been increasingly shown that oral pathogens have relevance in medical disorders. Dental biofilm is a reservoir of bacteria known to cause nosocomial pneumonia in susceptible individuals. *Staphylococcus aureus*, *P. aeruginosa*, *A. baumannii*, and *Enterobacter cloacae* are found in the dental biofilm of hospitalized patients with chronic lung disease significantly more often than in outpatients.[9]

Multidrug resistance of bacterial pathogens is becoming a serious public health threat. The clinical relevance of *A. baumanni* infection has increased because the microorganism has developed resistance to carbapenems, which are broad-spectrum β-lactam antibiotics that are used as last-resort drugs to treat infections caused by multidrug-resistant (MDR) agents.[10-12] This pathogen is causing global outbreaks of infection, and management, so far, is based on antibiotic combination therapy to eliminate MDR strains.[13]

An unusual pattern of skin and soft tissue infection associated with war wounds was reported during an infection outbreak at a U.S. Navy hospital in 2003. The cases were associated with *A. baumannii* bacteremia, revealing the pathogenic role of this bacterium in the specific pattern of skin and soft tissue infection.[14] More recently, cases of *A. baumannii* infection were reported in patients with comorbidities, such as morbid obesity, alcoholic liver disease, and paranoid schizophrenia.[15,16] *A. Baumannii* was also the most isolated organism in cultures of skin burn injuries and in the blood cultures of these patients.[17] The rupture of the protective barrier of the skin and mucosa is an important factor in invasive infections, sepsis, and increased mortality. The description of clinical cases in this context is important to better define diagnosis, risk factors, and prognosis. Here, we report 2 cases of cellulitis in the oral and perioral regions, associated with MDR

Statement of Clinical Relevance

Acinetobacter baumannii infection originated from oral lesions may be associated with bacteremia and fatal outcome in oncologic pediatric patients.

[a]Department of Oral Surgery and Pathology, School of Dentistry, Universidade Federal de Minas Gerais, Minas Gerais, Brazil.
[b]Department of Social and Preventive Dentistry, School of Dentistry, Universidade Federal de Minas Gerais, Minas Gerais, Brazil.
[c]Multiprofessional Integrated Residency in Health, Hospital das Clínicas, Universidade Federal de Minas Gerais, Minas Gerais, Brazil.
[d]Residence in Pediatrics, Hospital das Clínicas, Universidade Federal de Minas Gerais, Minas Gerais, Brazil.
[e]Department of Pediatrics, School of Medicine, Universidade Federal de Minas Gerais, Minas Gerais, Brazil.
Received for publication Sep 19, 2018; returned for revision Jan 18, 2019; accepted for publication Feb 12, 2019.

2212-4403/$-see front matter
https://doi.org/10.1016/j.oooo.2019.02.012

A. baumannii in hospitalized pediatric patients. The cases highlight early clinical features and diagnosis.

CASE REPORT 1

A 12-year-old male patient with a primary diagnosis of acute promyelocytic leukemia (AML M3) was treated according to the Nordic Society of Pediatric Hematology and Oncology-Dutch-Belgian-Holland AML Protocol.[18] A year and a half after the initial diagnosis, the patient presented with a febrile peak and was hospitalized again. On this occasion, a fusion study of the *PML* and *RARα* genes was performed, and molecular recurrence of AML M3 was diagnosed through nested reverse transcription–polymerase chain reaction. Chemotherapy with cytosine arabinoside, all-trans retinoic acid, and mitoxantrone was initiated, and a good response was obtained. A year and a half after the treatment, the patient presented again with seizures and disease recurrence in the central nervous system. Chemotherapy was reinitiated for the induction of remission, with intravenous cytarabine and intrathecal chemotherapy (methotrexate and dexamethasone). Because of the risk of chemotherapy-induced oral mucositis, prophylactic daily application of low-power laser therapy was performed.[19] The protocol consisted of low-power laser therapy at 22 points of the oral cavity, for 10 seconds at each point, by using a 100 mW, 660-nm laser apparatus (MMOptics Ltda, São Carlos, Sao Paulo, Brazil).

Ten days after the later chemotherapy protocol, the patient presented with an ulcerated lesion on the lower lip mucosa, with firm edema and nonspecific vesicles, suggesting a traumatic lesion after a seizure and overlying herpes simplex virus infection. The initial lesion was restricted to the right side of the lower lip mucosa (Figure 1). The day after the beginning of the lip lesion, the patient was admitted to the ICU for persistent fever, hypotension, diaphoresis, tachycardia, and neutropenia (absolute neutrophil count <500 cells/μL). Chlorhexidine at 0.12% was used for lesion antisepsis. To control the pain and to promote healing of the ulcerated lesion, low-power laser therapy was performed at 8 points, for 20 seconds at each point (100 mW, 660-nm) (MMOptics Ltda, São Carlos, Sao Paulo, Brazil). The initial treatment consisted of empiric antibiotic therapy with vancomycin, meropenem, polymyxin E, and acyclovir.

The edema increased, involving the submandibular region on the right side (Figure 2). The patient's condition worsened with occurrence of edema and yellowish secretion, followed by epithelial necrosis. Complementary examinations were requested to better target the treatment. Ultrasonography revealed signs of cutaneous soft tissue infection, but no signs of obvious abscess, suggesting lip and face cellulitis. Hemoculture was positive for *A. baumannii*, which was susceptible only to tigecycline. Thereafter, treatment with tigecycline (50 mg every 12 hours) and polymyxin (2500 IU/kg/day) was implemented.

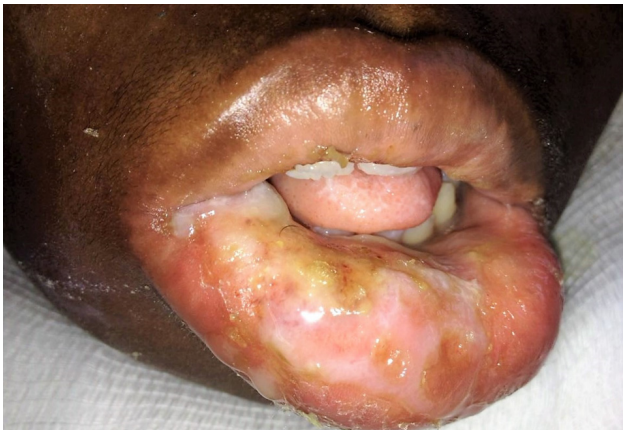

Fig. 1. Cellulitis caused by *Acinetobacter baumannii* on the lips of a 12-year-old male patient (patient 1). The initial clinical presentation consisted of an ulcer in labial mucosa covered by white-yellow pseudomembrane.

Despite the immediate initiation of adequate treatment, signs of sepsis and systemic toxicity rapidly developed, associated with the clinical progression of cellulitis. A significant increase in soft tissue edema was followed by necrosis in the lower lip and spontaneous drainage of a bloody secretion. Gauze soaked in petrolatum was used to soften the necrotic tissue. Fixed analgesia and daily low-power laser therapy sessions were maintained to alleviate severe local pain. During the first 6 days of ICU admission, the patient remained hemodynamically stable, dependent on amines with mechanical ventilation and a high-calorie pediatric enteral diet, but in the following 72 hours, there was clinical worsening associated with refractory thrombocytopenia, anasarca, and signs of multiple organ failure. On the ninth day, hyperkalemia (6.9 mEq/L; reference value <5 mEq/L) and refractory metabolic

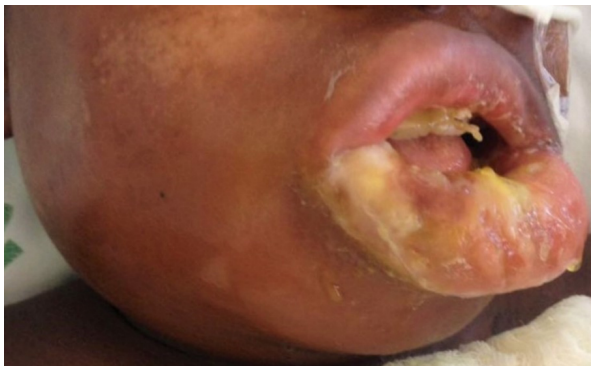

Fig. 2. Progression of cellulitis caused by *Acinetobacter baumannii* (patient 1). Labial edema became tense and tender and progressed with involvement of face and submandibular region.

acidosis occurred; this was followed by a drop in heart rate, ventricular fibrillation with electrical activity without pulse, and death.

A. baumanni infection was diagnosed 4 days after the onset of the lip lesion. The patient died 6 days after the diagnosis.

CASE REPORT 2

A 10-year-old male patient presented with giant cell hepatitis associated with autoimmune hemolytic anemia, with remission achieved only with the combination of tacrolimus and mycophenolate mofetil. This treatment had been introduced in the first year of the patient's life. At age 10 years, he presented with a mass in the upper left quadrant of the abdomen associated with pain. Ultrasonography revealed a heterogeneous and hypoechoic mass suggestive of lymphoproliferative disease. Incisional biopsy of the abdominal mass was performed via median supraumbilical laparotomy. Anatomopathologic examination showed neoplasia characterized by the proliferation of atypical, monomorphic, intermediate-sized lymphocytes with round and hyperchromatic nuclei, interspersed with scattered, tingible, body-laden macrophages, characterizing the "starry sky" pattern. Immunohistochemical staining was positive for CD20, BCL6, and CD10 and negative for CD3, BCL2, CD5, DTT, and CD99. The proliferation index of Ki67 was 100%, leading to a diagnosis of Burkitt lymphoma secondary to immunosuppression for the treatment of giant cell hepatitis.

Treatment consisted of chemotherapy according the Non-Hodgkin Lymphoma−Berlin-Frankfurt-Münster 95 protocol.[20] Low-power laser prophylaxis was performed daily at 22 intraoral points, for 10 seconds at each point, by using a 100 mW, 660-nm laser therapy apparatus (MMOptics Ltda, São Carlos, Sao Paulo, Brazil). On day 12 after chemotherapy course the patient developed World Health Organization Grade I mucositis[21] and neutropenia (absolute neutrophil count <300 cells/μL). Despite low-intensity laser therapy, the mucositis progressed to grade IV 5 days after its onset of mucositis.

Simultaneously, edema of the tongue and lips, initially associated with an anaphylactic reaction to platelet transfusion, developed and persisted despite treatment with antihistaminic drugs and corticosteroids. The clinical presentation of mucositis was accompanied by progressive edema and a yellowish secretion (Figure 3). The patient showed significant neutropenia, and empiric treatment with trimethoprim−sulfamethoxazole (trimethoprim 150 mg/m^2/day, sulfamethoxazole 750 mg/m^2/day), meropenem (40 mg/kg intravenously [IV]; every 8 hours), and filgrastim (5 μg/kg/day) was provided. Hemoculture performed 1 day after the onset of the lip edema confirmed *A. baumannii* infection. The antibiogram defined treatment with meropenem (40 mg/kg IV; every 8 hours) and polymyxin B (2500 IU/kg/day). The oral lesions resolved after 15 days of antibiotics treatment. At this point, the patient's clinical context included partially recanalized portal vein thrombosis, eosinophilic esophagitis, and bloody diarrhea.

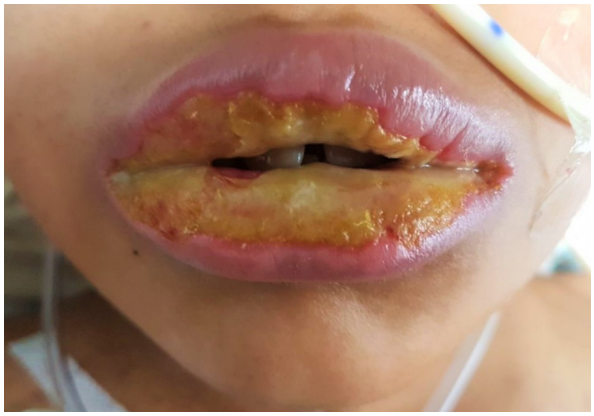

Fig. 3. Cellulitis caused by *Acinetobacter baumannii* on the lips of a 10-year-old male patient (patient 2). The edematous cellulitis was associated with ulcer in the labial mucosa covered by white-yellow pseudomembrane.

Two weeks later, the patient was admitted in the ICU for uncontrolled severe sepsis associated with respiratory insufficiency with pleural effusion, acute renal failure, and multiple organ dysfunctions. Hemoculture was performed 1 day after admission to the ICU, and the result was again positive for *A. baumannii*, susceptible only to polymyxin. On the second day, there was progressive reduction in consciousness level, and 2 cardiac arrests occurred, and cardiopulmonary resuscitation, endotracheal intubation, and a regimen of adrenaline were provided. Despite all efforts, the sepsis progressed to refractory shock associated with hypotension, even with high doses of adrenaline and noradrenaline (2.5 μg/kg/min). The next day, bradycardia, asystole, and cardiorespiratory arrest occurred, resulting in death.

Considering the timeline, there was a window of 33 days from the onset of the lip edema associated with *A. baumannii* infection to the overwhelming sepsis caused by *A. baumannii* bacteremia. The time elapsed from the onset of lip edema to death was 36 days.

DISCUSSION

A. baumannii has emerged as an opportunistic nosocomial pathogen and poses a clinical challenge because of emerging antibiotic resistance. *A. baumannii* infections constitute 18.4% of hospital episodes of gram-negative bloodstream infections.[22] Rupture of the epithelial barrier of the oral cavity, caused by trauma, herpes simplex virus−induced lesions, and chemotherapy-induced oral mucositis, was an important point of entry of *A. baumannii* in the 2 reported cases of hospitalized pediatric patients.

Drug resistance is an epidemiologically useful indicator, classified as MDR—that is, nonsusceptibility of a pathogen to at least 1 agent in ≥3 antimicrobial categories—and *extensively drug-resistant*—that is, susceptibility to ≤2 antimicrobial categories.[23] According to this system, of the cases presented here, case 1 was of the extensively drug-resistant category and case 2 of the MDR category, respectively. Despite this classification, the scale of bacterial resistance has not been consistently associated with prognosis and outcome,[24] and in both cases of *A. baumannii* infection presented here, a fatal outcome was observed.

Antimicrobial resistance may also be evaluated according to an agent's difficult-to-overcome resistance, with treatment-limiting resistance to first-line agents; β-lactams, including carbapenems and β-lactamase inhibitor combinations; and fluoroquinolones. Nonsusceptibility to first-line antimicrobial agents leads to the use of more toxic and less effective drugs.[22] This is especially true of the first clinical case reported here. Susceptibility of *A. baumannii* only to tigecycline represented significant treatment difficulty because tigecycline is a bacteriostatic agent with nonlinear protein binding and low serum levels, which may lead to an unfavorable microbiologic response.[25]

A. baumannii has been reported in polymicrobial infections, usually associated with streptococci in severely ill and immunocompromised patients, and monomicrobial infection is rare.[1,26] Risk factors associated with *A. Baumannii* infection delineate a profile similar to other opportunistic infections—that is, critically ill patients, with previous surgical and invasive procedures; prolonged hospital stay; ICU admission; prior exposure to several antibiotic classes; and immunocompromising conditions.[5,15,26-28] Malignancies, especially hematologic, and chronic obstructive lung disease were reported as major underlying conditions in *A. Baumannii* bacteremia.[29] In the 2 cases presented here, blood cultures showed *A. baumannii*, with multidrug resistance, and the patients were critically ill, with long-term hospitalization and an identified open portal for infection.

The diagnosis of skin and soft tissue cellulitis was based on the definitions of the Infectious Diseases Society of America.[30] Specific aspects of *A. baumannii* cellulitis had been described in the context of war wounds. The lesion is initially characterized by well-demarcated edema, usually circumscribing an infected wound. Progressing cellulitis becomes a diffuse swelling that is warm to the touch, and overlying vesicles may be observed.[14] Blood cultures are recommended in moderate or severe cases. Microscopic examination of swabs or cutaneous aspirate may also be considered in critically ill patients.[30]

Because few cases of *A. baumannii*−associated skin and soft tissue infections have been reported, the prognosis and outcome of these lesions are uncertain. In war-wounded patients, debridement and antibiotic therapy were effective, although overwhelming fatal sepsis has also been reported.[14] Fatal outcomes are also associated with comorbidities and progression to necrotizing fasciitis, which is characterized by necrosis of the fascia and subcutaneous tissue.[1,15]

The presence of septic shock was previously identified as an independent prognostic factor for mortality in *A. Baumannii* bacteremia as well as the severity of the underlying disease.[29] The frequency of septic shock caused by this pathogen ranges from 15% to 25%.[29,31] *A. baumannii* bacteremia commonly has a lethal clinical course, associated with early mortality.[31,32] In both our cases reported here, the outcome was fatal, associated with *A. baumannii* bacteremia and septic shock and a clinical course accelerated by severe underlying conditions. The oral health provider may play a relevant role in reducing the risk of bacteremia by adopting oral care measures and preserving mucosal integrity.

A. baumannii infection is a life-threatening condition that requires early identification and intervention to ensure better prognosis and outcome. An attentive and vigilant multiprofessional team is essential to prevent late diagnoses and, thus, fatal consequences. The severity of the primary disease and diverse risk factors create complexities, however, making control of infection and determination of the prognosis difficult to achieve.

DISCLOSURE

This work was supported the National Council for Scientific and Technological Development (CNPq)/Brazil and Coordination for the Improvement of Higher Education Personnel (CAPES, Finance Code 001). T.A.S and T.S.F.P. are research fellows at CNPq.

PRESENTATION

The abstract was presented at the 44th Brazilian Congress of Oral Medicine and Oral Pathology (44th Congresso Brasileiro De Estomatologia e Patologia Oral − SOBEP 2018).

REFERENCES

1. Sinha N, Niazi M, Lvovsky D. A fatal case of multidrug resistant *Acinetobacter* necrotizing fasciitis: the changing scary face of nosocomial infection. *Case Rep Infect Dis*. 2014;2014:1-6.
2. Kunhi M, Sanagar S, Jagadeesh N, Shankar B, Abraham A. Emergency cardiac double valve surgery in active infective endocarditis due to *Acinetobacter baumannii* with aortic root abscess in a patient with dialysis-dependent end-stage renal failure: a rare case report. *J Surg Case Reports*. 2016;2016:1-3.
3. Shah I, Kapdi M. Multidrug-resistant *Acinetobacter* meningitis in children. *J Fam Med Prim Care*. 2016;5:858-859.

4. Karakuzu Z, Iscimen R, Akalin H, et al. Prognostic risk factors in ventilator-associated pneumonia. *Med Sci Monit*. 2018;24:1321-1328.
5. Blot S, Vandewoude K, Colardyn F. Nosocomial bacteremia involving *Acinetobacter baumannii* in critically ill patients: a matched cohort study. *Intensive Care Med*. 2003;29:471-475.
6. Ziółkowski G, Pawłowska I, Krawczyk L, Wojkowska-Mach J. Antibiotic consumption versus the prevalence of multidrug-resistant *Acinetobacter baumannii* and *Clostridium difficile* infections at an ICU from 2014-2015. *J Infect Public Health*. 2018:2-6.
7. Pailhoriès H, Belmonte O, Kempf M, et al. Diversity of *Acinetobacter baumannii* strains isolated in humans, companion animals, and the environment in Reunion Island: an exploratory study. *Int J Infect Dis*. 2015;37:64-69.
8. Da Silva-Boghossian CM, Do Souto RM, Luiz RR, Colombo APV. Association of red complex, *A. actinomycetemcomitans* and non-oral bacteria with periodontal diseases. *Arch Oral Biol*. 2011;56:899-906.
9. Didilescu AC, Skaug N, Marica C, Didilescu C. Respiratory pathogens in dental plaque of hospitalized patients with chronic lung diseases. *Clin Oral Investig*. 2005;9:141-147.
10. Zarrilli R, Giannouli M, Tomasone F, Triassi M, Tsakris A. Carbapenem resistance in *Acinetobacter baumannii*: the molecular epidemic features of an emerging problem in health care facilities. *J Infect Dev Ctries*. 2009;3:335-341.
11. Stoeva T, Higgins PG, Bojkova K, Seifert H. Clonal spread of carbapenem-resistant OXA-23-positive *Acinetobacter baumannii* in a Bulgarian university hospital. *Clin Microbiol Infect*. 2008;14:723-727.
12. dos Santos Saalfeld S, Fukita Viana G, Dias Siqueira V, Cardoso C, Botelho Garcia L, Bronharo Tognim M. Endemic carbapenem-resistant *Acinetobacter baumannii* in a Brazilian intensive care unit. *J Hosp Infect*. 2009;72:365-368.
13. Perez F, Hujer AM, Hujer KM, Decker BK, Rather PN, Bonomo RA. Global challenge of multidrug-resistant *Acinetobacter baumanni*. *Antimicrob Agents Chemother*. 2007;51:3471-3484.
14. Sebeny PJ, Riddle MS, Petersen K. *Acinetobacter baumannii* skin and soft−tissue infection associated with war trauma. *Clin Infect Dis*. 2008;47:444-449.
15. Ali A, Botha J, Tiruvoipati R. Fatal skin and soft tissue infection of multidrug resistant *Acinetobacter baumannii*: a case report. *Int J Surg Case Rep*. 2014;5:532-536.
16. Hiraki Y, Yoshida M, Masuda Y, et al. Successful treatment of skin and soft tissue infection due to carbapenem-resistant *Acinetobacter baumannii* by ampicillin-sulbactam and meropenem combination therapy. *Int J Infect Dis*. 2013;17:e1234-e1236.
17. Güldoğan CE. Clinical infection in burn patients and the consequences. *Turkish J Trauma Emerg Surg*. 2017;23:2-7.
18. Abrahamsson J, Hasle H, Abrahamsson J. NOPHO-DBH AML 2012 Protocol: research study for treatment of children and adolescents with acute myeloid leukaemia 0-18 years. EudraCT No. 2012-002934-35. *Sponsored by Västra Götaland Regionen*. 2013;2012. Available at: https://ichgcp.net/clinical-trials-registry/NCT01828489.
19. He M, Zhang B, Shen N, Wu N, Sun J. A systematic review and meta-analysis of the effect of low-level laser therapy (LLLT) on chemotherapy-induced oral mucositis in pediatric and young patients. *Eur J Pediatr*. 2018;177:7-17.
20. Woessmann W, Seidemann K, Mann G, et al. The impact of the methotrexate administration schedule and dose in the treatment of children and adolescents with B-cell neoplasms: a report of the BFM Group Study NHL-BFM95. *Blood*. 2005;105:948-958.
21. Sonis ST. Oral Mucositis. Tarporley, UK: Springer Healthcare Ltd; 2012.
22. Kadri SS, Adjemian J, Lai YL. National Institutes of Health Antimicrobial Resistance Outcomes Research Initiative (NIH−ARORI). Difficult-to-treat resistance in gram-negative bacteremia at 173 US hospitals: retrospective cohort analysis of prevalence, predictors, and outcome of resistance to all first-line agents. *Clin Infect Dis*. 2018;67:1803-1814.
23. Magiorakos A, Srinivasan A, Carey RB, et al. Multidrug-resistant, extensively drug-resistant and pan-drug-resistant bacteria: an international expert proposal for interim standard definitions for acquired resistance. *Clin Microbiol Infect*. 2011;18:268-281.
24. Burnham JP, Lane MA, Kollef MH. Impact of sepsis classification and multidrug-resistance status on outcome among patients treated with appropriate therapy. *Crit Care Med*. 2015;43:1580-1586.
25. Prasad P, Sun J, Danner RL, Natanson C. Excess deaths associated with tigecycline after approval based on noninferiority trials. *Clin Inf Dis*. 2012;54:1699-1709.
26. Charnot-Katsikas A, Dorafshar AH, Aycock JK, David MZ, Weber SG, Frank KM. Two cases of necrotizing fasciitis due to *Acinetobacter baumannii*. *J Clin Microbiol*. 2009;47:258-263.
27. Raad II, Mohamed JA, Reitzel RA, et al. The prevention of biofilm colonization by multidrug-resistant pathogens that cause ventilator-associated pneumonia with antimicrobial-coated endotracheal tubes. *Biomaterials*. 2011;32:2689-2694.
28. Smolyakov R, Borer A, Riesenberg K, et al. Nosocomial multidrug resistant *Acinetobacter baumannii* bloodstream infection: risk factors and outcome with ampicillin-sulbactam treatment. *J Hosp Infect*. 2003;54:32-38.
29. Valero C, García Palomo J, Matorras P, Fernández-Mazarrasa C, González Fernández C, Fariñas M. *Acinetobacter* bacteraemia in a teaching hospital, 1989−1998. *Eur J Intern Med*. 2001;12:425-429.
30. Stevens DL, Bisno AL, Chambers HF, et al. Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014 update by the Infectious Diseases Society of America. *Clin Infect Dis*. 2014;59:e10-e52.
31. Cisneros JM, Reyes MJ, Pachon J, et al. Bacteremia due to *Acinetobacter baumannii*: epidemiology, clinical findings, and prognostic features. *Clin Infect Dis*. 1996;22:1026-1032.
32. Yang S, Sun J, Wu X, Zhang L. Determinants of mortality in patients with nosocomial *Acinetobacter baumannii* bacteremia in Southwest China: a five-year case-control study. *Can J Infect Dis Med Microbiol*. 2018;2018:3150965.

Reprint requests:

Thaís dos Santos Fontes Pereira
Universidade Federal de Minas Gerais
Faculdade de Odontologia
Av. Antônio Carlos 6627
Pampulha − 31270-901
Belo Horizonte
Minas Gerais
Brazil.
Thaissfp@ufmg.br