Septic shock caused by *Leptotrichia buccalis* in a neutropenic patient secondary to chemotherapy☆

Shock *séptico por* Leptotrichia buccalis *en paciente neutropénico por quimioterapia*

We present the case of a 55-year-old male, a major smoker and drinker with T4aN1M0 supraglottic squamous cell carcinoma, a subcutaneous reservoir carrier and peripheral venous access for chemoradiotherapy treatment. A week after receiving the first cisplatin cycle, he came to the emergency department and was transferred to the ICU for septic shock (fever, hypotension, decreased level of consciousness, procalcitonin of 46 ng/ml and lactate of 5.47 mmol/l) with severe hydroelectrolytic disorders (hyponatraemia, hypokalaemia, hypochloraemia, hyperglycaemia and metabolic acidosis), acute renal failure and severe bicytopaenia (leukopaenia of 300, with 60 neutrophils and thrombocytopenia of 19,000) without coagulopathy.

Supportive treatment was initiated with crystalloids, platelet transfusion, vasoactive amines and empirical intravenous antibiotic therapy with piperacillin–tazobactam and subsequently with meropenem, amikacin and linezolid. He presented a torpid evolution, rapid atrial fibrillation and respiratory failure with poor response despite mechanical ventilation and amiodarone infusion. He died within a few hours following a cardiorespiratory arrest and multiple organ failure.

Chest X-ray showed bilateral infiltrate (Fig. 1).

In the blood cultures obtained on admission, a long, fusiform and microaerophilic Gram-negative bacillus grew in all the bottles. Growth was slow and, at 48 h, the colony was small, grey and well-defined. It was identified as *Leptotrichia buccalis* using mass spectrometry (MALDI-TOF).

Leptotrichia spp. is a Gram-negative bacillus; sometimes it has been described as a large variable, immobile, facultative anaerobic, fusiform, carbohydrate fermenter and lactic acid producer. Some species do not grow in conventional media and when they do they are slow growing. Identification by phenotypic methods is difficult, and, therefore, it is believed that their infections are underdiagnosed[1] as they are identified as *Fusobacterium* or *Lactobacillus*. It is currently identified by mass spectrometry or by sequencing the 16S rRNA gene. *L. buccalis* resides mainly in the oral cavity so it usually causes dental diseases and oral abscesses, although in immunosuppressed patients it acts as an opportunistic pathogen.[2] It has a taxonomic similarity to fusobacteria, which is why they formerly belonged to the same family.[1–3] The pathogenicity of *Leptotrichia* spp. is due to the production of LPS and secretion of endotoxins that trigger an inflammatory reaction by cytokines.[2]

In a recent review[4] isolates of this bacterium have been described in cases of sepsis, neutropenic fever, thrombocytopaenia, subacute dyspnoea, halitosis and various dental diseases (pulpitis or pulp necrosis, periodontitis, dental plaques, etc.) and even endocarditis.[5] An increased risk of bacteraemia due to *L. buccalis* has been reported in patients with febrile neutropenia after chemotherapy and mucositis or periodontal infections.[6,7] The patient we present had oral sepsis and missing teeth, in addition to the laryngeal tumour with local invasion.

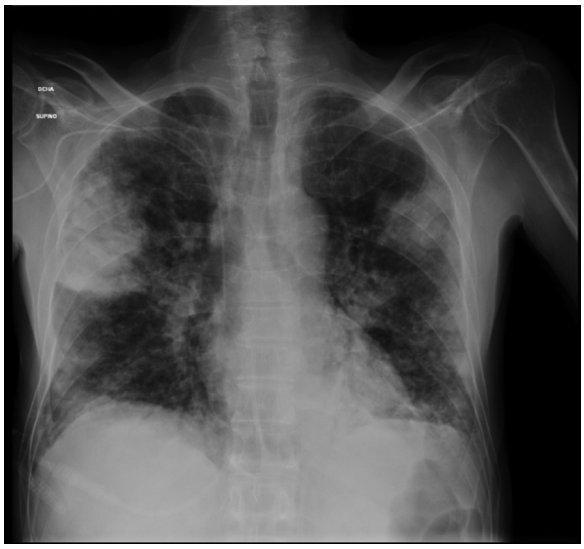

Fig. 1. Bilateral alveolar infiltrate on chest radiography upon admission.

There are published cases of Lemierre's syndrome due to *L. buccalis* in neutropenic patients[8] with bilateral radiological infiltrates and pulmonary abscesses on CT. In our case, after the rapid fatal outcome and the absence of consent for necropsy, the study could not be completed, but, despite its rarity, we do not rule out this possibility. In the differential diagnosis we also include bilateral atypical pneumonia[9] from *L. buccalis* due to severe sepsis, respiratory failure and compatible radiology.

It is usually susceptible to multiple antimicrobial agents such as beta-lactam, carbapenems, clindamycin, metronidazole, rifampin, tetracyclines and chloramphenicol,[1,4,6,7] and resistant to aminoglycosides, macrolides, vancomycin and fluoroquinolones.[4,10]

We believe that our contribution is important because there is no published case of septic shock due to *L. buccalis*. Most of the cases with bacteraemia described in the literature also had post-chemotherapy neutropenia, and the point of entry was the oral cavity. However, all of them had responded to the aforementioned antibiotics, unlike our patient.

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Carbapenemase-producing *Enterobacteriaceae* infections in General Surgery patients: Our experience☆

Infecciones por Enterobacterias productoras de carbapenemasas en pacientes de Cirugía General: análisis de nuestra experiencia

During the last decade, infections caused by carbapenemase-producing *Enterobacteriaceae* (CPE) have been dramatically increased worldwide. Numerous publications have focused on the epidemiology and risk factors for CPE-related infections,[1–3] although studies about surgical patients are scarce. Recognizing patterns in patients admitted to a General Surgery Department (GSD), could be essential to ensure a more rational antibiotic use in this specific setting. We performed a retrospective review including nosocomial CPE infections inpatients admitted to the GSD from January 2013 to December 2016. We analyzed patients with at least one (new) positive culture 48 h after admission for a CPE at any location and associated clinical signs or symptoms of infection. The probable infectious source was defined according to microbiological results and the analysis of clinical findings by two physicians in accordance with Centers for Disease Control definitions.[4] Patient's samples were collected and incubated based on standard recommendations.[5] We investigated the clinical and microbiological characteristics, treatment, complications, antimicrobial susceptibility and risk factors for mortality.

We included 40 patients with a CPE clinical infection: 50% were male, with a mean age of 69.4 ± 13.4 years. Charlson's comorbidity index median value was 3 (range 1–5). The rate of CPE infections in the GSD increased annually from 1.2% in 2013, to 4.7% in 2016.

Carbapenemase-producing *Klebsiella pneumoniae* strains were the most commonly identified (92.5%), all with the OXA-48-like carbapenemase. Prior to the CPE infection, other non-resistant microorganisms isolated were Gram-negative bacteria (52.5%), Gram-positive bacteria (65%), and fungi (40%). Intra-abdominal site was the most frequent source of infection (55%), followed by surgical wound (22.5%). CPE were susceptible to amikacin (100%), tigecycline (97%), and colistin (76%), and showed increased MICs but acceptable susceptibility to meropenem (64.3%) and imipenem (52.5%). CPE isolates showed low susceptibility rates to ertapenem (8.8%) and ciprofloxacin (5.3%).

Median hospital stay was 41.5 days (range 26.2–74.5). A surgical procedure had been performed in 35 patients in the previous 30 days, including emergency surgery in 15 cases. A major complication occurred in 23 patients, with a mortality rate of 17.1% in patients who underwent a surgery. A postoperative intra-abdominal infection (IAI) was present in 19 cases. The reoperation rate was 32.5%, without differences regarding mortality compared to non-reoperation. All patients received antibiotic therapy, of which 28 patients received carbapenem therapy. Table 1 shows the analysis of factors associated with mortality. Six patients received an appropriate empirical antibiotic regime, according to the *in vitro* activity. Appropriate definitive antimicrobial treatment was administered to 32 patients. The mortality rate at 30 days was 15%. Factors associated with mortality were: blood transfusions ($p = 0.021$), and lower rate of major complications (Clavien-Dindo ≥ III) ($p = 0.031$). A combined definitive two-drug targeted scheme was protector for mortality ($p = 0.048$).

This study summarizes the outcomes of patients with CPE infections in a GSD. The specific characteristics of this population were age >65 years, previous comorbidities (solid tumor, diabetes, and renal insufficiency), some risk factors for multidrug-resistant infections (including prior hospitalization and antibiotic therapy), all previously described.[1,2,6] In our series, antibiotic therapy had been previously prescribed in all patients (carbapenem therapy in 70%), with high rates of previous hospitalizations, surgery and endoscopic procedures. These findings agree with those of a recently published study of patients admitted in a surgical ICU from a tertiary-care Spanish hospital.[6] In addition, other investigations detailed that factors related with the acquisition of a CPE infection in solid organ recipients are poor functional status and frequent antimicrobial therapy, which more frequently occurs in the early post-transplant period.[7]

Intra-abdominal location was the most common source of CPE infection in our patients (the majority of patients had undergone an abdominal surgery). A two-drug scheme was a protective factor for mortality, and this combination is recommended by current guidelines on the treatment of IAI.[8,9] Despite this, several studies have not identified these differences in all patients and propose appropriate monotherapy for patients with low-mortality risk scores.[10]

Although this study only represents a review of an experience in a GSD of a single-institution with a limited number of patients, it shows a current vision of an increasing problem in a less analyzed population. As physicians prescribing antimicrobials, we may need to pay attention to patients with a specific clinical profile, using a targeted treatment for each patient. Future studies on CPE-related intra-abdominal infections could determine more definite features and outcomes in specific settings with surgical patients.

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Conflicts of interest

The authors report none conflict of interest to declare for the present study.

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