in the diagnostic approach to the patient. In the event of abducens nerve palsy, whether uni- or bilateral, it is necessary to consider the possibility of intracranial hypertension and to request the necessary complementary tests (neuroimaging and lumbar puncture) to establish its aetiology.

References

1. Kodsi RS, Yoinge BR. Acquired oculomotor, trochlear, and abducent cranial nerve palsies in pediatric patients. Am J Ophthalmol. 1992;114:568–74.
2. Lee MS, Galetta SL, Volpe NJ, Liu GT. Sixth nerve palsies in children. Pediatr Neurol. 1999;20:49–52.
3. Larner AJ. False localizing signs. J Neurol Neurosurg Psychiatry. 2003;74:415–8.
4. Sabatini C, Bosis S, Semino M, Senatore L, Principi N, Esposito S. Clinical presentation of meningoccocal disease in childhood. J Prev Med Hyg. 2012;53:116–9.
5. Dass Hazarika R, Deka NM, Khyriem AB, Lyngdoh WV, Barman H, Duwarah SG, et al. Invasive meningoccocal infection: analysis of 110 cases from a tertiary care centre in North East India. Indian J Pediatr. 2013;80:359–64.

Ana Camacho Salas[a,b,*], Pablo Rojo Conejo[b,c], Noemí Núñez Enamorado[a], Rogelio Simón de las Heras[a]

[a] *Paediatric Neurology Section, Neurology Department, Hospital Universitario 12 de Octubre, Madrid, Spain*
[b] *School of Medicine, Complutense University of Madrid, Madrid, Spain*
[c] *Paediatric Infectology Section, Paediatric Department, Hospital Universitario 12 de Octubre, Madrid, Spain*

* Corresponding author.
E-mail address: acamachosalas@yahoo.es
(A. Camacho Salas).

2529-993X/

Bacteremia due to *Leptotrichia trevisanii* after an allogeneic bone marrow transplant☆

Bacteriemia por Leptotrichia trevisanii *en una paciente sometida a trasplante alogénico de médula ósea*

We present the case of a 62-year-old woman diagnosed with a haematological disease, monoclonal gammopathy, in 2006, which developed into quiescent multiple myeloma in 2009 and symptomatic myeloma (IgG kappa, ISS 1) in 2011. She underwent autologous transplant of peripheral blood haematopoietic progenitors (AHSCT), after which she suffered a relapse in October 2014, with a discreetly hypocellular bone marrow, with 7% intermediate maturation plasma cells. After receiving 10 sessions of radiation therapy and 4 cycles of VTD (bortezomib + thalidomide + dexamethasone), the patient was admitted to the Haematology Department of the Hospital Universitario Central de Asturias in April 2015 to receive an allogeneic transplant of haematopoietic precursors from a HLA-identical sibling, conditioned with fludarabine. Furthermore, at the time of admission, she had a persistent catarrhal clinical picture with cough, without expectoration, fever, or dyspnoea, so she was given levofloxacin (500 mg IV/24 h).

The procedure was performed without incident and prophylaxis for graft-*versus*-host disease was initiated with methotrexate and cyclosporin. In the history recorded on the following days, the patient reported oral pain, with a WHO grade 3 mucositis being detected that required parenteral nutrition. On the sixth day post-transplant, she started with a fever of 39 °C, and the blood work showed a deep medullar aplasia (leukocytes $0.00 \times 10^3/\mu l$, red blood cells $2.76 \times 10^6/\mu L$, haemoglobin 8.7 g/dl, platelets 13,000/μL), with liver and kidney function tests showing values within normal limits. Blood cultures were taken (BD BACTEC® Plus Aerobic/Anaerobic F), and treatment with piperacillin-tazobactam was started (4 g IV/6 h), according to the hospital's antibiotic therapeutic protocol for febrile neutropenia.

At 32 h, the anaerobic bottles were positive, while the aerobic bottles were positive at 57 h, gram-negative bacilli with a spindle-shaped appearance were observed in staining. In subcultures in a *Brucella*, chocolate blood agar some greyish colonies with dry appearance grew at 18 h. Identification was performed using MALDI-TOF, obtaining a score of 2.1 for *Leptotrichia trevisanii*. This result was confirmed through 16S rRNA sequencing, comparing the sequence obtained with the GenBank® database and using the BLAST program (https://blast.ncbi.nlm.nih.gov/Blast.cgi).

The antibiotic sensitivity was performed by broth microdilution, according to the reference method for anaerobic micro-organisms and the breakpoints indicated in the Clinical and Laboratory Standards Institute (CLSI). The micro-organism was sensitive to the following antibiotics: penicillin (MIC = 0.12 mg/L), amoxicillin (MIC = 1 mg/L), piperacillin (MIC ≤ 16 mg/L), amoxicillin-clavulanic (MIC = 0.5/0.25 mg/L), piperacillin-tazobactam (MIC = 16/4 mg/L), cefoxitin (MIC = 1 mg/L), imipenem (MIC = 0.12 mg/L), chloramphenicol (MIC = 8 mg/L), clindamycin (MIC ≤ 0.5 mg/L), tetracycline (MIC ≤ 2 mg/L) and metronidazole (MIC ≤ 0.5 mg/L). Moxifloxacin (MIC = 8 mg/L) was classed as resistant. There are no breakpoints in the CLSI for erythromycin, but the MIC obtained (64 mg/L) was high.

The first case of bacteremia by *Leptotrichia trevisanii* was described by Tee et al. in a male with acute myeloid leukaemia in 2001,[1] and at least 12 cases have been published since then.[2–8] The patients involved suffered from some type of haematologic disease, except one, who had oesophageal cancer with metastases in the liver, lung and lymph nodes,[2] which shows the opportunistic nature of this micro-organism. All of them described a situation of febrile neutropenia as a predisposing factor of sepsis, accompanied by the appearance of lesions in the oropharyngeal mucosa. These constitute a route for bacterial translocation,[2,3] which can trigger bacteremia in situations of medullar aplasia.

Phenotypic batteries are not able to identify *Leptotrichia* spp., since it presents a low biochemical reactivity.[3,4] In addition, the comparison between the different strains described demonstrates the difficulty in identifying the micro-organism based solely on biochemical tests, due to the variability of the results (Table 1). In our case, the API® Rapid/ID 32 A (bioMérieux®) system was used. The result of the number profile obtained (0411400000) was *Clostridium acetobutylicum* (87.2%), which cannot be correlated with the gram-negative spindle bacilli observed under the microscope.

The MALDI-TOF mass spectrometry has proved to be a cost-effective and rapid tool in the final identification of micro-organisms for which conventional methods are inconclusive. Martín-Gutiérrez et al. managed to correctly identify the *Leptotrichia trevisanii* species just 2 h after the blood culture was

DOI of refers to article: http://dx.doi.org/10.1016/j.eimc.2016.09.010

☆ Please cite this article as: Sabater Cabrera C, Fernández Blázquez A, García Carús E. Bacteriemia por *Leptotrichia trevisanii* en una paciente sometida a trasplante alogénico de médula ósea. Enferm Infecc Microbiol Clin. 2017;35:389–390.

Table 1
Comparison of the results of biochemical tests between different strains of *Leptotrichia trevisanii*.

Test	Our case	Tee et al.[1]	Cooreman et al.[6]	Higurashi et al.,[2] case 1	Higurashi et al.,[2] case 2
Catalase	–	+	–	NT	NT
Oxidase	–	–	–	NT	NT
Urea	–	–	NT	–	–
Indole	–	–	–	–	–
αGAL	+	–	NT	+	–
βGAL	+	NT	+	NT	NT
αGLU	+	+	NT	+	–
βGLU	+	+	NT	+	–
βNAG	+	+	NT	–	–
PAL	+	–	NT	–	–
Esculin	NT	NT	NT	+	+

+: positive; – negative; NT: not tested.

positive,[3] the same as Schmitt et al., who did so in a period of 48 h.[5] In our case, it allowed us to establish the aetiology of the bacteremia within 18 h after the passage of positive blood cultures to solid culture media.

The sensitivity patterns for *Leptotrichia* spp. are not defined, although it has been described as sensitive to most antimicrobials.[1,2,6,7] There is not enough experience to establish a treatment of choice. In the reviewed literature several therapeutic regimens were implemented based on the sensitivity study, with resolution of the clinical picture in all cases. Our patient was treated with piperacillin-tazobactam, the blood cultures being negative on the tenth day after treatment and with resolution of mucositis on day 15. However, Martín-Gutiérrez et al. did not observe clinical improvement using piperacillin-tazobactam, changing the treatment to meropenem.[3]

The use of levofloxacin is inadequate in these cases, since *Leptotrichia trevisanii* shows *in vitro* resistance to fluoroquinolones.[2,6] Schrimsher et al. described 3 clinical cases in which levofloxacin was used as an empirical treatment in febrile neutropenia, which did not prevent the development of bacteremia by *Leptotrichia*.[8] Therefore, it is important that patients with febrile neutropenia and mucositis, more prone to infections by anaerobic micro-organisms, are treated with anaerobicidal antimicrobials.[7,8]

References

1. Tee W, Midolo P, Janssen P, Kerr T, Dyall-Smith M. Bacteremia due to *Leptotrichia trevisanii* sp. Eur J Clin Microbiol Infect Dis. 2001;20:765–9.
2. Higurashi Y, Tatsuno K, Fujimoto F, Kobayashi I, Ida K, Seto Y, et al. Two cases of bacteremia caused by *Leptotrichia trevisanii* in patients with febrile neutropenia. J Infect Chemother. 2013;19:1181–4.
3. Martín-Gutiérrez G, Rodríguez N, Lepe JA, Parody R, Torres MJ, Aznar J. Rapid identification of a *Leptotrichia trevisanii* cateter-related bloodstream infection using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry. JMM Case Rep. 2015, http://dx.doi.org/10.1099/jmmcr.0.000036.
4. Eribe ERK, Olsen I. *Leptotrichia* species in human infections. Anaerobe. 2008;14:131–7.
5. Schmitt BH, Cunningham SA, Dailey AL, Gustafson DR, Patel RJ. Identification of anaerobic bacteria by Bruker Biotyper matrix-assisted laser desorption ionization time of flight mass spectrometry with on-plate formic acid preparation. J Clin Microbiol. 2013;51:782–6.
6. Cooreman S, Schuermans C, van Schaeren J, Olive N, Wauters G, Verhaegen J, et al. Bacteraemia caused by *Leptotrichia trevisanii* in a neutropenic patient. Anaerobe. 2011;17:1–3.
7. Couturier MR, Slechta ES, Goulston C, Fisher MA, Hanson KE. *Leptotrichia bacteriemia* in patients receiving high-dose chemotherapy. J Clin Microbiol. 2012;50:1228–32.
8. Schrimsher JM, McGuirk JP, Hinthorn D. *Leptotrichia trevisanii* sepsis after bone marrow transplantation. Emerg Infect Dis. 2013;19:1690–1.

Christian Sabater Cabrera [a,*], Ana Fernández Blázquez [a], Enrique García Carús [b]

[a] *Microbiology and Clinical Parasitology Department, Hospital Universitario Central de Asturias, Oviedo, Asturias, Spain*
[b] *Internal Medicine Department, Hospital Universitario Central de Asturias, Oviedo, Asturias, Spain*

* Corresponding author.
E-mail address: christiansabaterpes@gmail.com
(C. Sabater Cabrera).

2529-993X/

Activity of *Artemisia annua* infusions on epimastigotes of *Trypanosoma cruzi*☆

Actividad de infusiones de Artemisia annua *sobre epimastigotes de* Trypanosoma cruzi

Chagas disease is an anthropozoonosis caused by the flagellate protozoan *Trypanosoma cruzi (T. cruzi)*.[1] Currently, the treatment against *T. cruzi* is restricted to two drugs of limited effectiveness and high toxicity: benznidazole and nifurtimox.[2] Therefore, it is necessary to find new therapeutic tools. The use of natural products such as *Artemisia annua (A. annua)* represents a novel choice; its active compound is artemisinin, a sesquiterpenoid lactone that crosses the biological membranes.[3] This study evaluated the effect of the *A. annua* infusion on epimastigotes of *T. cruzi*.

Epimastigotes of *T. cruzi* were used (isolated: RHO/Ve/03/RG1 and CHHP). For the preparation of the infusions, dry and crushed *A. annua* leaves from 2 different origins were used: from plants grown in Cumaná (Venezuela) and in Luxembourg. The infusions were prepared at concentrations of 0.4, 0.6, 0.8, 1.0, 2.0 and 3.0% m/v of dry leaves of *A. annua* in the *Liver Infusion Tryptose* (LIT) culture medium.[4] The experiments were performed by adjusting the cultures to a cellular density of 2×10^6 parasites/ml, in the various, previously described concentrations of the *A. annua* infusion. The trials were performed in triplicate and a control culture was included. The cellular viability of the epimastigotes of *T. cruzi*, was determined by the trypan blue dye exclusion method.[5] In order

DOI of refers to article: http://dx.doi.org/10.1016/j.eimc.2016.09.011
☆ Please cite this article as: Berrizbeitia de Morgado M, Cariaco Sifontes Y, Imery Buiza J, Lutgen P. Actividad de infusiones de *Artemisia annua* sobre epimastigotes de *Trypanosoma cruzi*. Enferm Infecc Microbiol Clin. 2017;35:390–392.