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Scientific letters

Clostridium colicanis bacteraemia in an asthmatic patient diagnosed as acute respiratory infection

Bacteriemia por Clostridium colicanis *en una paciente asmática diagnosticada de infección respiratoria aguda*

Although obligate anaerobes are seldom isolated from patients with bacteraemia, the genus *Clostridium* is in second place behind the genus *Bacteroides* and represents approximately 1% of all positive blood cultures, with *Clostridium perfringens* being the most commonly isolated species. The risk factors associated with its isolation in blood are haemolysis, malignant intestinal neoplasia, inflammatory bowel disease and immunosuppression.[1] In most cases, its clinical meaning is unclear, representing contamination or transient bacteraemia, and its pathogenicity and virulence continue to be a subject of debate. *Clostridium colicanis* is a *Clostridium* species that has been rarely isolated in the blood, the first time in 2008 by Simmon et al., although it was not documented the episode.[2] Thus, we report the first documented case of bacteraemia by this microorganism in an immunocompetent patient diagnosed as acute respiratory infection.

We present the case of a 77-year-old woman with asthma and anticoagulation who was admitted to the Emergency Department due to symptoms of fever of up to 39 °C, chills, malaise, asthenia, dyspnoea, cough and decreased level of consciousness. The abdominal anamnesis was anodyne, and the patient presented no urinary symptoms, heart failure or oedema. The physical examination revealed a blood pressure of 120/63 mm Hg, a temperature of 37.7 °C and an oxygen saturation of 95%. The most noteworthy laboratory data were as follows: leukocytes count of 23.7×10^9/L [4–11] with 89.4% [40–80] granulocytes and 4.6% [20–50] lymphocytes, prothrombin activity of 42% [70–120], international normalised ratio of 1.85 [0.8–1.85], total bilirubin of 1.7 mg/dL [0.2–1.2] and C-reactive protein of 41.9 mg/L [0–5]. Upon her arrival, the patient underwent blood cultures, influenza A/B virus detection using polymerase chain reaction in a nasopharyngeal exudate (negative) and urine culture (negative). Treatment was started with intravenous cefotaxime (1 g/8 h for 10 days) and oral levofloxacin (500 mg/day for 7 days), which resulted in the disappearance of the fever.

The blood cultures were processed in the BD BACTEC™ 9240 system (Becton-Dickinson and Company, NJ, USA). The two anaerobic bottles were positive after 26 h of incubation. Gram staining revealed the presence of long Gram-positive bacilli with straight ends (Fig. 1), which were isolated under anaerobic conditions (Oxoid™ AnaeroGen™ 2.5-L sachet, ThermoFisher Scientific) in Schaedler agar at 48 h. The colonies were round, somewhat irregular, white-grey, catalase-negative measuring approximately 3 mm in diameter. The strain was identified as *C. colicanis* (log score: 2.122) using matrix-assisted laser desorption ionisation time of flight mass spectrometry (MALDI Biotyper® Microflex LT, Bruker Daltonik GmbH), and 16S rRNA sequencing (99%, GenBank accession number FJ957867.1). Antimicrobial susceptibility testing was carried out by the Etest gradient diffusion method (bioMérieux, Marcy létoile, France) using a 0.5 McFarland bacterial suspension and *Brucella* blood agar with hemin and vitamin K1. The plates were incubated anaerobically for 48 h at 35–37 °C. The minimum inhibitory concentration (μg/mL) was interpreted as susceptible according to the recommendations for anaerobic bacteria (EUCAST and CLSI criteria)[3,4]: penicillin (0.016), amoxicillin-clavulanic acid (0.094), cefotaxime (0.015), piperacillin-tazobactam (0.016), clindamycin (2), metronidazole (1), meropenem (0.002) and tetracycline (1.5). After isolating *C. colicanis*, an abdominal ultrasound was requested, which showed no significant abnormalities. The patient progressed favourably and was discharged from the hospital.

C. colicanis is a bacillus measuring approximately $0.9–1.0 \times 3–10$ μm and is Gram-positive, obligate anaerobic, sporulating, nonmotile, and catalase-negative. It can use a considerable number of substrates, producing various acids from glucose, lactose, maltose, mannose, ribose, cellobiose and galactose and can reduce nitrates to nitrites.[5] Its genome consists of a single chromosome (2.6 Mpb) and contains approximately 2160 protein-encoding genes.[6] The colonies measure 3–5 μm in diameter and are round with rippled edges, slightly convex, opaque and white-grey. Its optimal growth temperature is 37–40 °C.

This microorganism was first described after its isolation in the faeces of a male Labrador dog in 2003.[5] The microorganism is closely related phylogenetically with *C. absonum*, *C. baratii* and *Eubacterium multiforme*. *C. colicanis* bacteraemia was first reported in humans in 2008 in a scientific article that studied the genotypic diversity of anaerobic isolates from bloodstream infections.[2] This

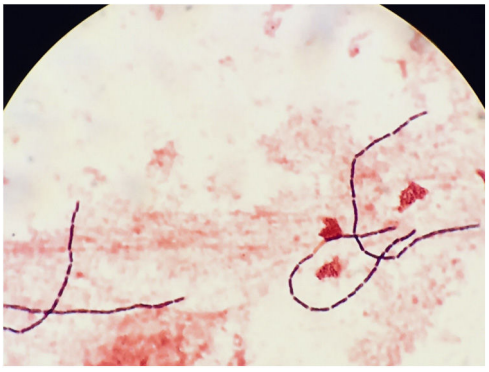

Figure 1. Gram stain of anaerobic blood culture (1000×): **Gram positive** long and straight-ended bacilli.

bacillus was subsequently encountered in 2014, in a study that compared the faecal microbiota of 13 Thai vegetarians and non-vegetarians and was found in a 61-year-old vegetarian who did not eat either yoghourt or eggs but did drink milk.[7] A recent study reported that, in more than half of patients with gastric cancer, the most prevalent microorganisms in the gastric epithelium were bacteria of the species *Fusobacterium nucleatum* (whose pathogenic role in colorectal cancer is well-known) and *C. colicanis*, suggesting a possible contribution of these bacteria in the development or progression of stomach cancer.[8] Our case corresponded to transient bacteraemia in a patient with laboratory data suggesting infection, and to date no signs of gastric or colon neoplasia have been found.

In conclusion, we reported the first documented case of *C. colicanis* bacteraemia in an immunocompetent patient, highlighting the importance of *C. colicanis* as a human pathogen. Further studies are needed to elucidate the pathogenesis and risk factors of *C. colicanis*-related invasive infections such as bacteraemia.

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Conflicts of interest

The authors declare no conflicts of interest.

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Foruncular myiasis. A biting tumor

Miasis foruncular. Un tumor mordiente

Foruncular myiasis is a rare entity in our environment and, as it is usually an imported disease, it is poorly known and suspected in our country. Hence the importance of reporting the very few cases that come across in our health system.

We report the case of a 24-year-old female who presented with a frontal scalp tumor that had grown gradually during the month prior to her admission. She had traveled to Peru a month before and denied fever or other symptoms, except for itching within the lesion. She was assessed by a plastic surgeon showing a lump with a hole which resembled an epidermoid cyst and was scheduled for surgical excision of the mass (Fig. 1a). After incision, a maggot was found (Fig. 1b), and resection was performed without incidents. It was directly sent to the Microbiology Department where it was identified as a *Dermatobia hominis* larva, based on the characteristics of its posterior spiracle (Fig. 1c), with three spiracular slits, each spiracular plate has three split curves directed toward the belly and slightly toward the middle.[1] After extraction, patient was discharged with amoxicilin/clavulanic acid as preemptive treatment of secondary bacterial infection of the wound, presenting no further complications.

Myaisis means invasion of organs and tissues by fly maggots.[2] The most common fly species that cause these affection are *Cordylobia anthropophaga*, original of the African continent, and *D. hominis*, from Central and South America.[3,4] The number of cases of myaisis in countries from continents different to these is increasing due to rise on migration to tropical regions.[5] To our knowledge, there are less than 30 cases of myiasis caused by *D. hominis* reported in Spain.[6]

D. hominis has three forms on its cycle: adult fly, pupa and larva. Only larvae are parasites,[7] and present three different stages. It is interesting that *D. hominis* is unable of biting because of its poorly developed buccal apparatus. Female adult flies capture hematophagous insects of other species and deposit their eggs on them (around 15–20 eggs at a time).[5] As the hematophagous vector bites a mammal, the eggs hatch and larvae fall onto the mammal's skin, where after penetrating and reaching the epidermis, remain growing for 33–41 days. When a 3rd stage larva is under the skin, it fixes its hooks (Fig. 1d) in soft tissue and orientates its respiratory organ, located in its last segment, toward the surface. This respiratory organ is used for species identification,[1,4] based on the morphology of this posterior spiracle (peritrem, button and spiracular slit).[7]

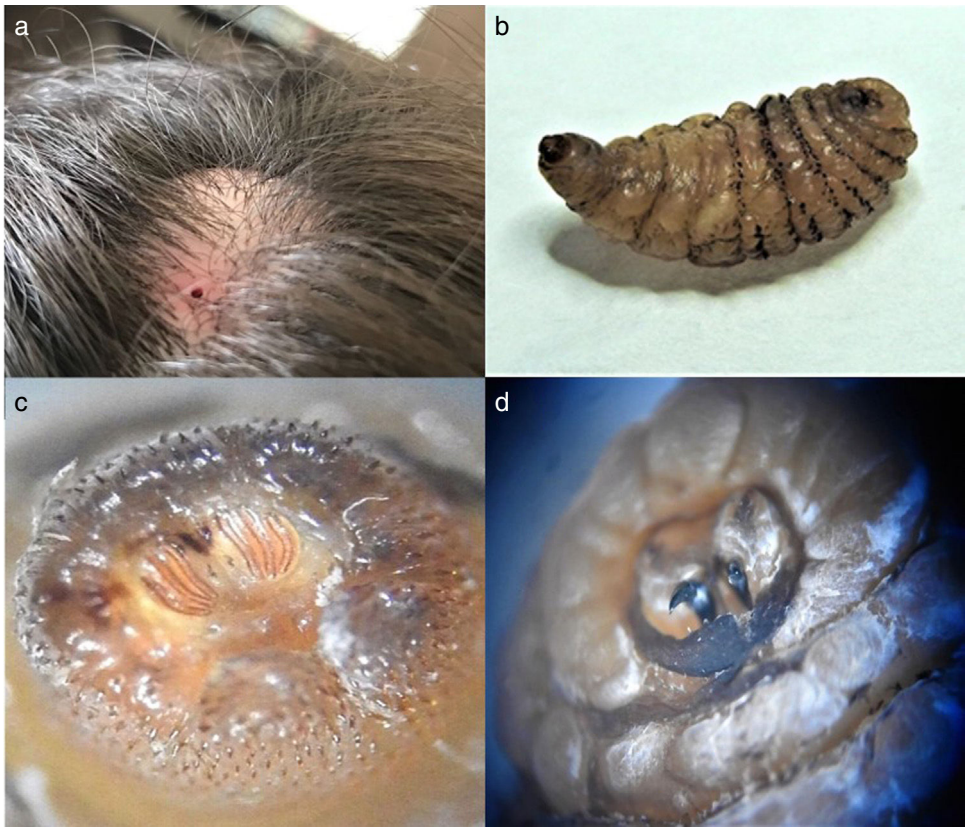

Fig. 1. Skin lesion in the scalp and parasite. (a) Lump with a hole. (b) *Dermatobia hominis* larva. (c) *D. hominis* posterior spiracle. (d) *D. hominis* hooks.

Clinically, in the first 24 h, a bug-bite lesion usually appears at the inoculation site, which grows wider and deeper within days, producing pruritus at first and then pain, even referring patients a sense of movement.[5] Treatment consists in removal of the maggot. First stage larvae can be removed by expression, but in late stages the tail portion of the larvae is wider than the anterior portion and the presence of spines make the extraction very difficult, resulting in the need of surgical removal.[6,8]

In conclusion, it is important to report the study of these cases, because of their increasing number, to make an accurate treatment. It is also important to instruct travelers about using protective clothing and insect repellent to prevent possible infestations.

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Ocular toxocariasis in a pediatric patient undergoing a bone marrow transplantation

Toxocariasis ocular en un paciente pediátrico sometido a trasplante de médula ósea

We report a 14-year-old male, born in Ecuator, diagnosed of severe idiopathic aplastic anemia who underwent an allogeneic bone marrow transplantation from his HLA-identical sibling donor. The early phase of transplantation was uneventful and the patient achieved full-donor chimerism on day +11 postransplant. He was discharged home on day +13 with prophylactic cyclosporine, acyclovir, fluconazole and trimethoprim/sulfamethoxazole with a hesitant treatment adherence.

On day +70 he presented with a sudden onset of diminished vision of the right eye without other symptoms. Physical examination was normal except his fundus eye that showed a white mass in the posterior pole of the right eye extending toward the macula suggestive of granuloma (see Fig. 1). A cranial CT and laboratory test were normal. He did not have peripheral eosinophilia. Stool parasites were negative. The differential diagnosis was made with toxoplasmosis, toxocariosis, neurocysticercosis, retinoblastoma and virus infections that were discarded. The serum enzyme-linked immunosorbent assay test (ELISA) IgM and IgG were positive for *Toxocara canis* (0.67, normal up to 0.25) and he was diagnosed of ocular toxocariasis. Despite the lack of an optimal treatment for OT, some patients can be treated successfully with anthelmintic drugs and systemic or periocular corticosteroids. Steroids can limit the inflammation or fibrosis in a patient who had a sudden onset of diminished vision. This case was considered a medical emergency because the patient had severe symptoms. Thus, he received treatment with topic steroids and oral Albendazole for 5 days with a good outcome. After the treatment of toxocariasis the fundus eye examination showed a decrease in the size of the granuloma with disappearance of macular involvement.

Toxocariasis is a zoonotic disease caused by the infestation of humans by second stage larvae of the dog nematode *T. canis* or the cat nematode *Toxocara cati*.[1] Prevalence estimates for the United States ranged from 8% to 15%, depending on the age, the region and socioeconomic status.[2] Ocular toxocariasis mainly affects younger children and constitutes 1–2% of uveitis in children. Since many infections were asymptomatic and thus can be misdiagnosed, the global burden of toxocariasis is likely to have been underestimated. The highest number of ocular toxoplasmosis cases has been reported in Japan and Korea, France, Brazil and the USA. Inflammation is typically unilateral. This parasite can cause uveitis, posterior and peripheral retinochoroiditis, endophthalmitis, papillitis,[1,3] and other ocular lesions that often lead to loss of vision in the affected eye. Because of its specificity, serum enzyme-linked immunosorbent assay (ELISA) is helpful in identifying patients with ocular toxocariasis. The test is highly sensitive (78%) and specific (92%), but the sensitivity and specificity vary according to the cut-off titter chosen to define a positive test.[4] Fundus photography, fluorescein angiography, ophthalmic ultrasound and OCT can assist in the detection of eye granulomas and in the differentiation of OT from similar ocular conditions.[5,6]

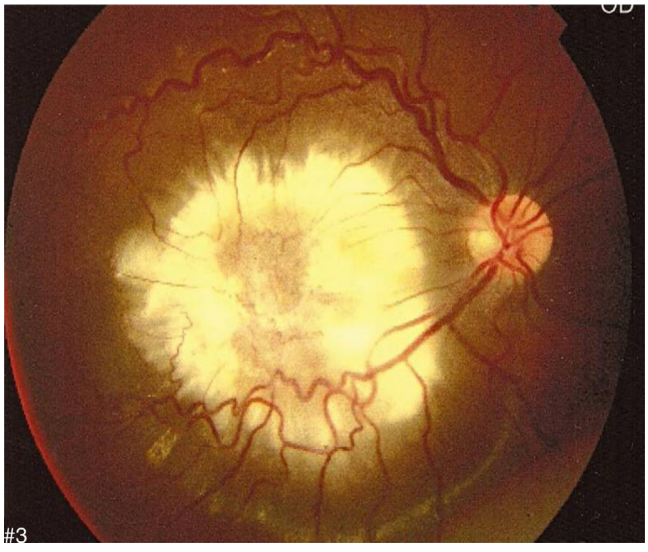

Fig. 1. Ocular toxocariasis: a white mass in the posterior pole of the right eye extending towards the macula.

Prevention of the infection is based on measures such as appropriate health care for dogs and cats, including regular antihelmintic treatments, prevention of contamination of the environment with feces, and responsible pet ownership. Furthermore, precautions based on hygiene are required, and education is important for prevention.[7]

Our patient had very bad social conditions with a poor level of hygiene. He also had a dog at home, so with this clinical history and the typical lesions observed in his fundus eye, the diagnosis of ocular toxocariasis was made. He had a good outcome with the treatment with a partial recovery of vision and a decrease in the size of the granuloma with disappearance of macular involvement. In ocular toxocariasis therapy should be guided according to visual acuity, severity of inflammation or irreversible ocular damage.[1,8]

In conclusion, in an immunocompromised patient who suffers an infection we do not have to consider only typical infections secondary to the poor recovery of the immune system such as toxoplasma or cytomegalovirus, but also imported infections. A good clinical history, keeping in mind the origin of the patient and the socioeconomic status can help us to find the right diagnosis.

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Conflict of interest

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Congenital HIV infection after failure of serological screening. Importance of implementing a second screening for HIV infection during the third trimester of pregnancy☆

Infección congénita por el VIH con cribado serológico gestacional negativo. Importancia de la implementación de la segunda prueba en el tercer trimestre de gestación

HIV infection is still a severe childhood health problem in less developed countries.[1] Although the incidence of new infections has decreased in recent years, 160,000 new cases are still occurring on an annual basis.[1,2] In a setting such as ours, with easy accessibility to the healthcare system and to antiretroviral therapy (ART), the perinatal HIV transmission rate can be reduced to less than 1%.[3] However, even today cases are still being reported, as demonstrated by the six new diagnoses due to mother-to-child transmission recorded in 2016 in Spain, of whom two were of Spanish origin and the rest from other countries, with no data on the prevention protocols applied in the latter (two from Sub-Saharan Africa, one from Latin America and another from Eastern Europe).[4]

Recently, in the latest consensus document from the Grupo de Estudio de Sida [AIDS study group (GeSIDA)] and the Sociedad Española de Ginecología y Obstetricia [Spanish Society of Gynaecology and Obstetrics, (SEGO)], the pregnancy screening protocol has been modified. It is now recommended to do an HIV serological measurement in the third trimester, in addition to the routine one in the first trimester. In high-risk cases, another measurement can also be added in the second trimester.[5] However, the North American guidelines from the *Centers for Disease Control and Prevention* (CDC) only recommend repeating the test in the third trimester in pregnant women with a high risk of contracting HIV, defined as those who receive care in areas with a high incidence of HIV infection.[6] The performance of this second measurement is considered cost-effective, given that the cost of ART far exceeds the cost in the measurement of a second test, which is currently around two dollars per measurement.[7,8]

Not carrying out the second test in the third trimester may lead to errors in the diagnosis of infections acquired during pregnancy which may be passed on to the foetus. In our centre, we recently cared for a male newborn aged 27 days who was brought to the paediatric emergency department due to malnutrition and epistaxis which had started two days beforehand. He was also partly rejecting food, with no other symptoms. The pregnancy was monitored in the mother's local hospital. The first semester serological tests (HIV, syphilis, hepatitis B and C viruses), which were performed in the eighth week of pregnancy, were negative. The baby was delivered through a normal vaginal delivery with amniorrhexis of 46 h at week 38 + 4 of pregnancy. The newborn baby weighed 2775 g (15th percentile). He was admitted at 3 days old due to weight loss and symptomatic hypoglycaemia which were attributed to poor feeding technique by the parents. He was readmitted at 27-days-old with a clinical picture of malnutrition, weighing 2570 g (percentile <3) with a size of 50 cm (3rd percentile).

In the study of malnutrition causes in the infant which included, among others, screening for metabolic diseases, congenital heart diseases, blood diseases and congenital infections, a serological test and an HIV viral load measurement were ordered which were positive (RNA HIV-1 6,654,360 copies/ml). HIV-1 infection was also confirmed in both parents, which they were unaware of until that time.

The risk of primary HIV infection is greater during pregnancy due to the fact that, as a result of hormonal changes which occur during pregnancy, there are alterations in the vaginal mucosa and in the immune system, which promote the acquisition of the virus.[9] Furthermore, in these cases, the risk of perinatal transmission increases up to eight times, hence the importance of obligatory compliance with the third trimester screening programmes in all pregnant women.[10]

With this letter, we want to warn of the risk of failure with the first-semester prenatal screening programme, and report a new case of mother-to-child transmission of HIV in our setting, which also displayed an atypical presentation. This case, along with others published in the literature,[10] would potentially be preventable if the recommendation for a second serological test in the third trimester was compulsory and was not performed on an optional basis, as it has been up to now.

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Literature review of mosquito-borne viral infections in non-tropical European Union territories: A cause of concern?

Infecciones virales transmitidas por mosquitos en la Unión Europea: revisión de la literatura. ¿Tenemos que preocuparnos?

Increasing globalization in travel and trade, together with global expansion and the establishment of competent vectors, has enabled the introduction into European Union countries of arboviruses that are widely circulating in tropical areas. Furthermore, several European countries have a close link to highly endemic areas for arboviruses: France has important overseas colonies (Americas, Africa and Oceania), or Portuguese island of Madeira, that has strong social and commercial relations with Brazil. Many European countries, including Spain, receive a huge number of immigrants and travellers from Latin America every year. In that sense, viremic international travel returning from epidemic regions to an area where a competent vector is established can trigger outbreaks, as has happened previously.

Global warming has allowed mosquitoes, such as *Aedes o Culex*, to proliferate, adapt to environmental conditions, migrate and spread to new niche areas in Europe that have become warmer.[1] This fact can contribute both to an increased risk of outbreaks of vector-borne diseases in Europe, such as the Dengue or Chikungunya viruses, and to open the door to new arbovirus infections, such as Zika, Ross River, or Mayaro.

Every year more than 200 seasonal cases of West Nile virus are reported, and sporadic and self-limited outbreaks of Dengue (including the recent report of autochthonous cases in Spain) and Chikungunya viral infection. We conducted an electronic literature search through PubMed (keywords used: [Chikungunya] OR [dengue] or [zika] or [west Nile] or [arboviral infection] AND [European autochthonous cases]) restricted to humans, and also consulted data extracted from the European Centre for Disease Prevention and Control (ECDC), focusing on the most recent outbreaks arboviral infections in non-tropical European Union territories from 2010 to November 2018 (Table 1).

Table 1
Outbreaks of mosquito-borne viral infections in non-tropical European Union territories (2010–2018).

	Main mosquito vector	Outbreaks (year, location, confirmed cases)
Chikungunya fever	*Aedes albopictus*	*First case reported:* 2007; Ravenna, Italy (229[b])[2] 2010; Fréjus, France (2)[2] 2014; Montepellier, France (12)[3] 2017; Anzio, Italy (102)[4], Le Cannet-des-Mares, France (9)[5]
Dengue Fever	*Aedes albopictus, Aedes aegypti*[a]	*First case reported*: 2010; Nice, France (2)[2] 2010; Pejesac, Croatia (17)[2] 2012; Madeira, Portugal (2218[c])[2] 2013; Aix-en-Provence, France (1)[7] 2015; Nimes, France (6)[6] 2018; Provence-Alpes-Côte d'Azur region, France; Murcia and Cadiz, Spain (3)[10]
West Nile fever	*Culex pipiens*	*First case reported*: 1962; Camargue France (2)[8] 2010; Austria, Greece, Hungary, Italy, Romania, Spain[d,9] 2011; Greece (100); Italy (14), Romania (11), Hungary (3)[9] 2012: Greece (161), Italy (50), Hungary (17), Romania (14)[9] 2013: Italy (69), Greece (68), Hungary (31), Romania (24), Croatia (16), Czech Republic (1), Slovenia (1)[9] 2014: Italy (24), Romania (23), Greece (15) Hungary (11), Austria (1)[9] 2015; Italy (60), Romania (19), Hungary (18), Austria (7), Bulgaria (2), France (1), Portugal (1)[9] 2016; Romania (93), Italy (76); Hungary (44); Austria (5), Spain (3), Bulgari (2), Cyprus (1), Croatia (1) [9] 2017; Romania (66), Italy (57), Greece (48), Hungary (21), Austria (5), Croatia (5), France (1), Bulgari (1) [9] 2018; Italy (550), Greece (302), Romania (268), Hungary (212), Croatia (45), France (24), Austria (19), Bulgari (11), Slovenia (3) and the Czech Republic (2)[10]

[a] Aedes aegypty was the main vector un Madeira's outbreak.
[b] Some of the cases were found with active case finding or retrospective serological studies.
[c] Information about number of confirmed/probable cases were only provided at the beginning of the epidemic.
[d] Number of cases were not reported.

The presence of a competent vector, optimal climatic conditions and the possibility of returning patients who are viremic are prerequisites for autochthonous transmission of arboviruses, are currently being met in many countries in Europe. Health professionals should be trained to identify arboviral diseases symptoms and to introduce them into the differential diagnosis, not only among patients returning from the tropics, but also in local patients from countries and areas with the presence of suitable vectors, so that possible outbreaks can be identified before they can have major consequences. Epidemiological and entomological surveillance is necessary to detect and control further outbreaks and to minimize the risk of local transmission in vector-colonized areas.

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Competing interests

None.

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Tularemia: diagnosis of an unexpected oculoglandular case in a non-endemic area by universal PCR

Tularemia: diagnóstico de un caso oculoglandular inesperado en un área no endémica mediante PCR universal

Dear Editor,

Tularemia is a zoonotic disease caused by *Francisella tularensis*, a Gram-negative facultative intracellular coccobacillus[1] with four recognized subspecies[2]: *tularensis* (type A), *holarctica* (type B), *mediasiatica* and *novicida*. Type A is found in North America, while type B is located, but not exclusively, in the northern hemisphere.[3] In Spain, it was an uncommon disease until 1997, when the first tularemia outbreak occurred in Castilla y León.[4] Until now, all cases reported in Spain were caused by *F. tularensis* subsp. *holarctica.* Clinical manifestations of tularemia fall into two main forms: ulceroglandular (>90% of cases in Europe)[2] and typhoidal. However, there are three more clinical forms: oculoglandular, oropharyngeal/gastrointestinal and pneumonic.

We have previously published the first case of ulceroglandular tularemia in a non-endemic area (Asturias, Spain).[5] Here, we present the first reported case of oculoglandular tularemia occurred in the same region which worried us.

An 88-year-old male presented to the emergency department of our hospital in April 2017 for diagnosis and management of pain in his right eye and the presence of conjunctival discharge. He did not have other symptoms, history of trauma, drug intake, or any recently local or systemic infection. His laboratory workup only showed a high value of C-reactive protein (5.5 mg/dL) and his medical and surgical histories were noncontributory. A diagnostic of viral conjunctivitis was done and he was treated with lubricant and anti-inflammatory drops.

Two months later, he was brought to the Department of Oral and Maxillofacial Surgery with a painful cervical mass (Fig. 1), weight loss and discomfort. He did not declared a recent travel, contact to ill people or animals although he lives in a rural area. Clinical examination revealed the presence of a right neck mass measur-

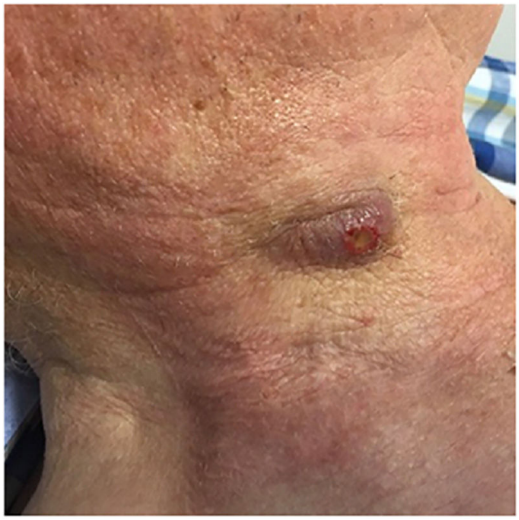

Fig. 1. Cervical mass observed in physical examination one month after conjunctivitis.

ing 30 mm × 20 mm, with gummy consistency, painful on palpation and without other appreciable alterations.

A CT scan was performed and three neck lymph nodes together with an intraparotid lymph node were seen as pathological, showing necrotic areas.

A universal PCR was performed in fine-needle aspiration biopsy (FNAB), based on amplification of the gene coding for 16S rRNA and subsequent sequencing (Bigdye® Terminator, Thermo Fisher Scientific). This evidenced the presence of *F. tularensis* subsp. *holarctica*. A second sample of FNAB was sent 10 days later resulting also positive.

In addition, serum samples were sent to the Spanish National Center for Microbiology (Madrid, Spain) in order to study the presence of antibodies against *F. tularensis* by microagglutination resulting positive with a titer of 1/1024.

Ziehl-Neelsen stain, culture and PCR for mycobacteria were negative. Lymphadenopathy-causing viruses (CMV, EB, HHV-6, HHV-7, HHV-8, Adenovirus, Picornavirus, Enterovirus, Mumps, LCMV) and *Toxoplasma gondii* were undetectable by PCR. Serological tests (ELISA) against *Coxiella burnetii* (IgG), *Rickettsia conorii* (IgG) and *Borrelia burgdorferi* (IgG/IgM) were negative.

Once the diagnosis was confirmed, the patient was treated with a 14-day course of intravenous streptomycin at a dose of 10 mg/kg/12 h, with favorable evolution. No surgical excision of the neck mass was needed.

Oculoglandular form of tularemia is very infrequent in our environment. In Spain some studies show an incidence of this form around 4%[4] but this microorganism should be considered in a patient with Parinaud's syndrome even in non-endemic areas.

The gold standard for the diagnosis of tularemia is the isolation of the causative agent by culture, however, this is difficult (it requires a medium with cysteine) and hazardous for the laboratory staff (Biosafety Level 2 precautions). Therefore diagnosis is based mainly on serology and results became positive between 10 and 14 days after onset of the disease.[6,7]

Genome amplification by polymerase chain reaction (PCR) is more sensitive than culture and provides rapid, sensitive and specific diagnosis of tularemia.[8–10] There are specific targets of *F. tularensis* genes (e.g. *fopA*, *tul4*, *ISFtu2* or RD1 protein-encoding gen) but when there is no suspicion of a specific etiological agent, it is useful to perform a universal PCR. In this case, if we had not performed the 16S rRNA PCR, the patient wouldn't have been correctly diagnosed.

The present case shows the importance of molecular techniques that amplify panbacterial genes, especially useful for diagnosis of rare infections with great difficulty of isolation of the etiological agent like *Bartonella henselae* (also causing Parinaud's syndrome), *Tropheryma whipplei*, *Borrelia* spp. or *Ehrlichia* spp. Also in those cases without bacterial growth due to antibiotic treatment.

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