lipid-lowering agents and azathioprine, azoles were avoided to reduce the risk of interactions), the tinea was eradicated. Like azoles or amphotericin B, terbinafine causes a decrease in ergosterol; in this case, by inhibiting squalene epoxidase in the cell membrane.[10]

The lack of literature and of cut-off points for the sensitivity of antifungals underline the need for further studies to investigate treatments for this type of infection.

The *Prototheca* species cause a wide range of infections in humans. These infections can occur in both immunocompetent and immunosuppressed patients, although the most severe and widespread infections usually occur in immunocompromised individuals. In view of their similar appearance to yeasts in routine media, but with very different implications for prognosis and treatment, both clinicians and microbiologists have to be aware of these organisms and work together to ensure that they are correctly diagnosed and the proper treatment provided.

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2529-993X/

Cryptosporidium hominis IbA12G3: First report of a rare sub-genotype in Spain

Cryptosporidium hominis *IbA12G3: primera descripción de un subgenotipo infrecuente en España*

Members of the genus *Cryptosporidium* are major contributors to the burden of diarrhoeal disease globally.[1] Cryptosporidiosis primarily affect children in resource-poor settings with unsafe drinking water and inadequate sanitary facilities, but also represents a significant health concern in developed nations.[2] *Cryptosporidium hominis* and *Cryptosporidium parvum*, particularly the former, are the two major causative species of human cryptosporidiosis. To date, at least nine subtype families (Ia, Ib, Id, Ie, If, Ig, Ih, Ii, Ik) have been identified within *C. hominis* by sequence analysis of the 60-kDa glycoprotein (*gp60*) gene.[3] Among them, Ia (1.7%), Ib (92.2%), Id (5.1%), and Ie (0.9%) were the subtype families found ($n = 529$) in the four largest molecular epidemiological surveys conducted so far in Spain.[4–7] In these studies, all the Ib samples ($n = 152$) molecularly characterized at the sub-genotype level were assigned to IbA10G2.

In December 2017 an 18-month-old male infant complaining of gastrointestinal symptoms including altered intestinal transit, distended abdomen, cramps, and acute, non-bloody watery diarrhoea associated to weigh loss (below 25th percentile) and anaemia (serum iron: 24 μg/dL) was admitted to the outpatient clinic of the University Hospital Puerta de Hierro Majadahonda (Madrid) for routine coproparasitological examination. The patient had a normal immune status, no contact with pet animals, and no relevant record of recent travelling abroad, although his father reported travelling to Romania during the same period. A single, concentrated stool sample tested positive for the presence of *Cryptosporidium* oocysts by a rapid immunochomatographic test (Cer Test Biotec S.L., Zaragoza, Spain) and by microscopic examination of a fresh faecal smear stained with the modified Ziehl–Neelsen method. As part of an ongoing research project, a new, fresh aliquot of the faecal material was sent to the National Centre for Microbiology at Majadahonda (Madrid) for genotyping analyses. Total DNA was extracted and purified using the QIAamp® DNA stool mini test kit (Qiagen, Hilden, Germany). Molecular characterization of the sample was achieved by PCR amplification of the *gp60* marker.[8] Amplicons of the expected size (~870 bp) were directly submitted for sequencing. Subsequent sequence analyses confirmed the presence of *C. hominis* 1bA12G3, a sub-genotype not previously identified in Spain. A representative nucleotide sequence of the sub-genotype obtained was submitted to the GenBank® public repository under the accession number MH161561.

Remarkably, a search using the BLAST tool of the National Centre for Biotechnology Information (NCBI) revealed that only six additional IbA12G3 sequences have been previously deposited in GenBank®. This particular *C. hominis* sub-genotype was initially described in a human specimen from UK in 2010.[9] Further molecular research conducted in the Sonora state (Mexico) allowed the identification of IbA12G3 in four children attending hospital settings, three of them presenting with gastrointestinal and/or nutritional disorders and the remaining one asymptomatic at the moment of diagnosis.[10] Finally, IbA12G3 has also been reported in seven rhesus macaques (*Macaca mulatta*) housed on monkey farms in China, a finding that may be indicative of potential zoonotic transmission.[11] Fig. 1 shows the phylogenetic relationships among the IbA12G3 sequences generated in the present study and those reported in the surveys mentioned above. Appropriated reference sequences and representative sequences of the most frequent *C. hominis* family subtypes circulating in Spanish human populations were retrieved from NCBI and included in the analysis for comparative purposes. As expected, sequences belonging to the family subtype Ib grouped together in a well-defined cluster. Within this group, the IbA12G3 sequence of non-human primate origin was

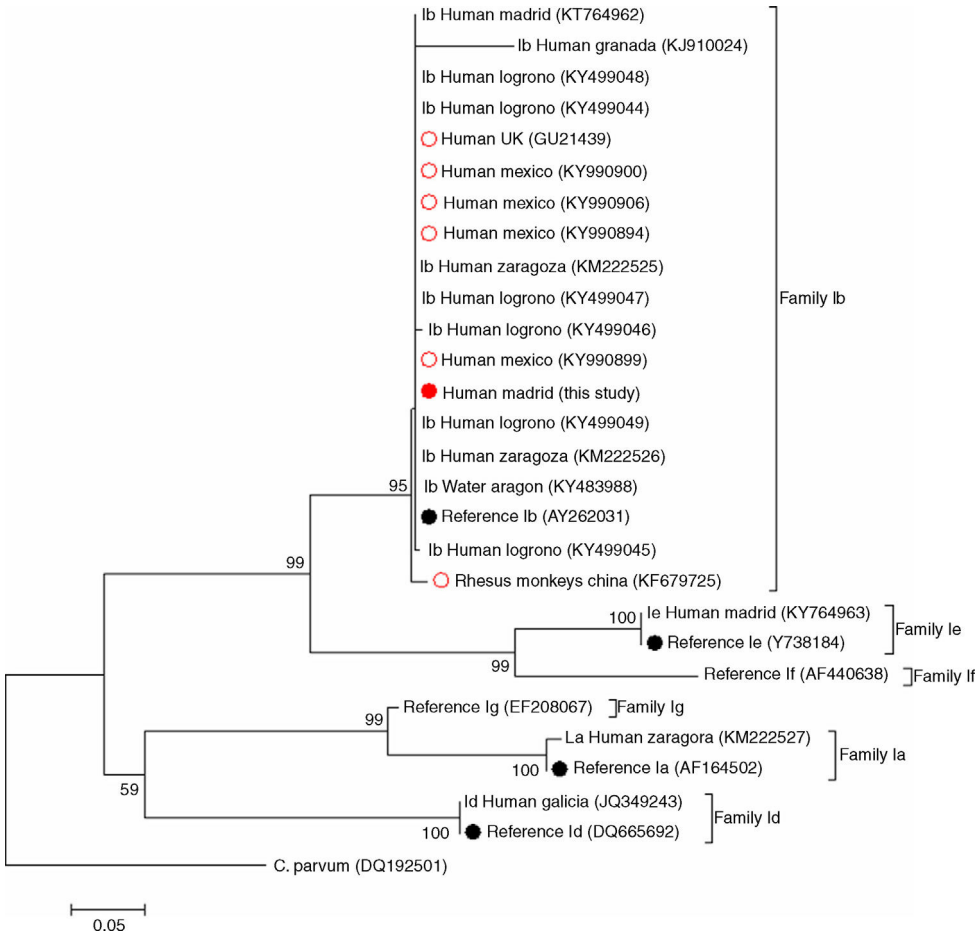

Fig. 1. Phylogenetic tree illustrating the evolutionary relationship of the IbA12G3 sequence generated in the present study (represented by a red filled circle) to other *C. hominis* family Ib sequences previously identified in Spain and other countries at the *GP60* locus inferred by a neighbour-joining analysis. Red empty circles represent all human ($n = 5$) and non-human ($n = 1$) primate IbA12G3 sequences reported globally to date. Black filled circles indicate reference sequences from GenBank®. Bootstrapping values over 50% from 1.000 replicates are shown at the branch points. The evolutionary distances were computed using the Tamura 3-parameter method. The rate variation among sites was modelled with a gamma distribution (shape parameter = 1). *C. parvum* was used as outgroup taxa.

placed into an independent (but closely related) clade, very likely reflecting differences in host adaptation and specificity.

In summary, we show here the first description in Spain of a *C. hominis* human infection by IbA12G3, a very rare sub-genotype only reported before in clinical specimens in UK and Mexico. A host-adapted IbA12G3 variant has been previously identified infecting non-human primates in China, raising doubts about its actual zoonotic potential. Although the origin of the infection in our paediatric case could not be elucidated, we cannot rule out the possibility that this *C. hominis* sub-genotype is naturally circulating in Spain at low frequency rates. This study clearly demonstrates that molecular surveillance of human cryptosporidiosis is key in determining the occurrence or emergence of novel genotypes of the parasite of uncertain pathogenicity and virulence.

Funding

This study was funded by the Health Institute Carlos III (ISCIII), Ministry of Economy and Competitiveness under project MPY 1350/16.

Acknowledgements

The authors acknowledge Marta Hernández de Mingo and María Guerrero for excellent technical assistance, and Dr. Consuelo López for providing relevant epidemiological and clinical information.

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2529-993X/

Efficacy of albendazole ointment on cutaneous larva migrans in a 2 years child☆

Eficacia del albendazol tópico en el tratamiento de la larva cutánea migrans en un niño de 2 años

Cutaneous larva migrans is an infestation caused by the penetration and migration in the skin of nematode larvae (geohelminths). The most common aetiological agent is Ancylostoma braziliense, although many other species may be involved, such as Ancylostoma caninum and Uncinaria stenocephala,[1] depending on the geographical area. The incidence in Spain has grown, due to the increased numbers of travellers and immigrants from the tropical and subtropical areas in which this condition is endemic.[2,3] The clinical presentation, in the form of serpiginous-looking erythematous lesions which move ahead at one end (creeping eruption), is characteristic and makes it easy to diagnose, without the need for biopsy.[3–5] The lesion is usually limited to the skin, as humans are an incidental host and the parasite is unable to complete its life cycle in humans.[3,5] The course of the infestation is usually benign and, in most cases, self-limiting due to the death of the larva within one to three months.[3,4]

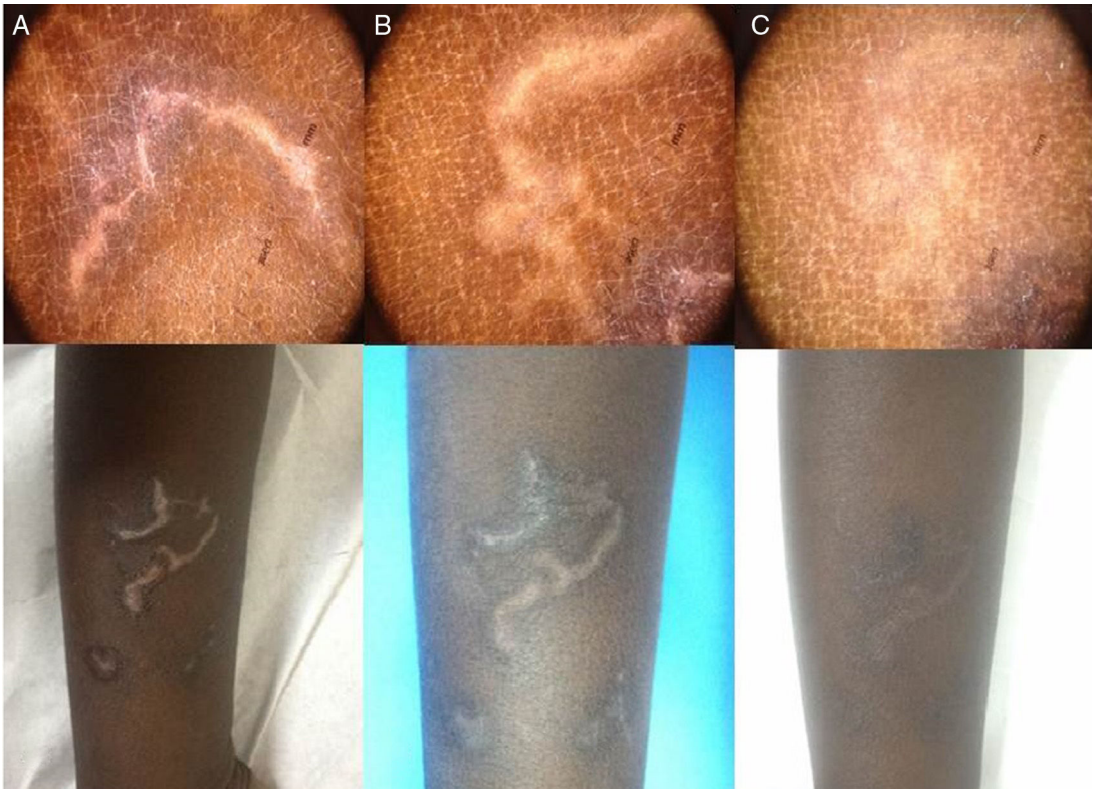

Fig. 1. (A) In the initial image we can see the sinuous trajectory of translucent brown structures, related to the body of the larva. (B) In subsequent check-ups (day 5 of treatment) an improvement of the lesion can be seen. (C) After 10 days of treatment, the structure is no longer evident, but we see an empty trajectory and clinical improvement.

DOI of original article: https://doi.org/10.1016/j.eimc.2018.05.001.
☆ Please cite this article as: Alarcon-Soldevilla F, Gonzalez-Valverde FM, Alonso-Osmer EC, López-Ávila Á. Eficacia del albendazol tópico en el tratamiento de la larva cutánea migrans en un niño de 2 años. Enferm Infecc Microbiol Clin. 2019;37:281–282.