Las neumonías por SARM presentan con frecuencia consolidación unilateral, neumatoceles y derrame pleural[8], y son más frecuentes en lactantes pequeños. En nuestro hospital no encontramos NAC por SARM en lactantes mayores de 6 meses. Cuando se presentan en niños mayores de 2 años, los hallazgos radiológicos, necesidad de cirugía o complicaciones son similares a los lactantes[6].

El estado de portador de SARM incrementa hasta 6 veces el riesgo de neumonía por esta cepa[3], siendo la transmisión intrafamiliar muy alta[6]. La colonización en España es más frecuente en la población inmigrante, especialmente procedente de Sudamérica[9]. Existe actualmente un amplio debate sobre la necesidad de descolonización en niños y sus convivientes. Parece prudente llevarlo a cabo en niños que presenten factores de riesgo, como grandes prematuros, inmunodeprimidos o pacientes ingresados en unidades de cuidados intensivos, así como en sus familiares.

La Academia Americana de Pediatría recomienda el uso de vancomicina o clindamicina en el caso de neumonías con sospecha de SARM[10]. Clindamicina tiene la ventaja de tener actividad frente a la LPV, pero la experiencia en neumonías es muy limitada, y puede haber aislamientos resistentes. Vancomicina tiene una pobre penetración tisular y su toxicidad obliga a la realización de niveles seriados. Linezolid es una excelente alternativa, ya que tiene una actividad similar a vancomicina frente a SARM, adecuada penetración en tejido pulmonar y actividad antitoxina. Aunque el uso de linezolid en pediatría es *off-label*, estudios farmacocinéticos y de seguridad, han demostrado que es un fármaco bien tolerado, con unos niveles plasmáticos adecuados con las dosis establecidas, y que permite además un tratamiento secuencial debido a su excelente biodisponibilidad oral.

En conclusión, debe sospecharse SARM como agente causal de las NAC con empeoramiento clínico y radiológico progresivo (necrosis y derrame pleural) a pesar del tratamiento antibiótico empírico convencional, especialmente en lactantes hijos de familias inmigrantes. Linezolid constituye una buena alternativa a vancomicina en estos pacientes.

Anexo. Material adicional

Se puede consultar material adicional a este artículo en su versión electrónica disponible en doi:10.1016/j.eimc.2018.11.005.

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Imported cryptosporidiosis caused by *Cryptosporidium hominis* IbA13G3 in Spain. The relevance of molecular-based surveillance

Criptosporidiosis importada por Cryptosporidium hominis *IbA13G3. La relevancia de la vigilancia basada en métodos moleculares*

Cryptosporidium is the most common diarrhoea-causing protozoan parasite globally. *Cryptosporidium* infections primarily affect children aged <24 months in the sub-Saharan Africa and South Asia, where three out of four reported cases were due to *C. hominis*.[1] Based on sequence analysis of the 60-kDa glycoprotein (*GP60*) gene, *C. hominis* has been demonstrated to comprise nine (Ia-Ik) subtype families with marked differences in geographical distribution.[2,3]

In middle August 2018 two Spanish-borne siblings complaining of gastrointestinal symptoms (a 2 years-old boy and a 12-years old girl) from a Nigerian family were attended at the María Jesús Hereza primary health centre (Leganés, Madrid). Both children had just returned from a 2-month family trip to Nigeria, where acute, non-bloody watery diarrhoea initiated. Three consecutive, soft stool specimens were obtained from each patient and submitted to the Microbiology Service of the University Hospital Severo Ochoa (Leganés, Madrid) for routine coproparasitological investigation including copro-culture and a commercially available solid phase qualitative immunochromatographic test for giardiosis/cryptosporidiosis (X/Pect *Giardia*/*Cryptosporidium*, Santa Fe, USA). Copro-cultures were negative but all six samples tested positive to *Cryptosporidium* by the rapid screening test. No microscopic examination was conducted. Pooled aliquots of the stool samples

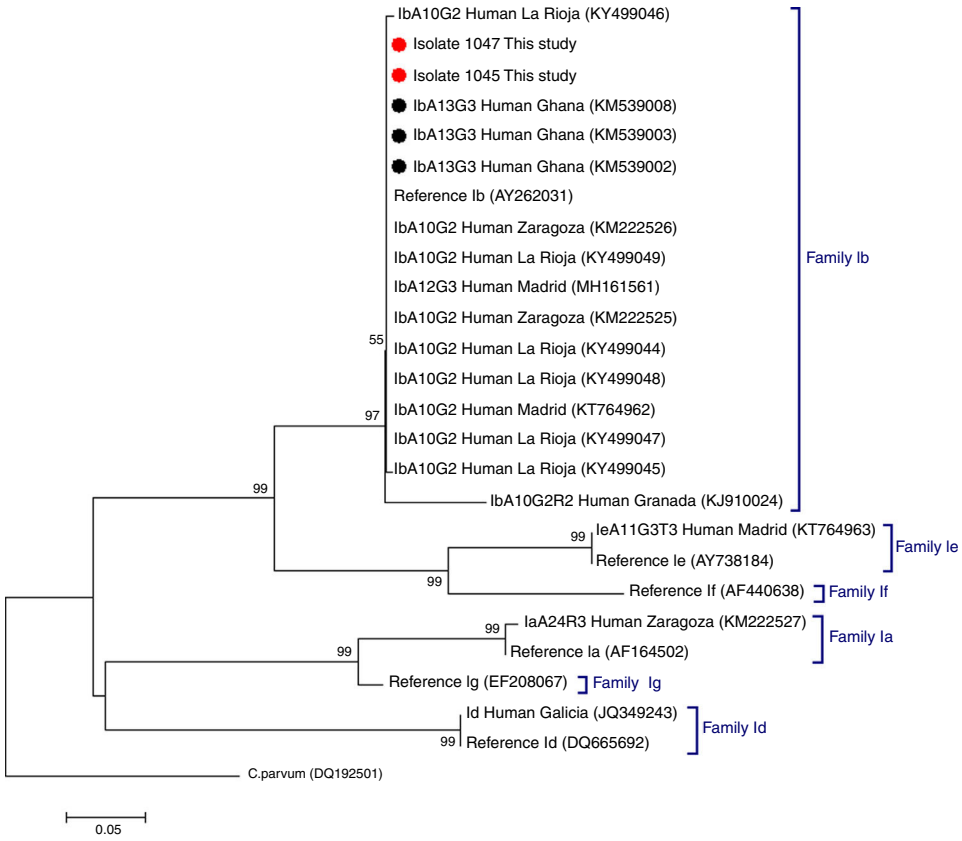

Fig. 1. Phylogenetic tree depicting evolutionary relationships among *C. hominis* IbA13G3 sequences at *GP60* from human isolates generated in the present study (represented by red filled circles) and other available surveys (represented by black filled circles). Representative sequences of *C. hominis* subtype families previously identified in different Spanish regions were also included in the analysis for comparative purposes. The analysis was inferred using the Neighbor-Joining method. Bootstrapping values over 50% from 1.000 replicates are shown at the branch points. The evolutionary distances were computed using the Kimura 2-parameter method. The rate variation among sites was modelled with a gamma distribution (shape parameter = 2). *C. parvum* was used as outgroup taxa.

were shipped to the Spanish National Centre for Microbiology at Majadahonda (Madrid) for diagnosis confirmation and genotyping analyses. Genomic DNA was extracted and purified using the QIAamp® DNA stool mini test kit (Qiagen, Hilden, Germany). Molecular detection and characterisation of the samples was achieved by PCR amplification of the *GP60* marker.[4] Amplicons of the expected size (~870 bp) were directly submitted for sequencing. Sequence analyses confirmed the presence in both samples of *C. hominis* IbA13G3, a very rare subtype not previously reported in Spain to date. A representative nucleotide sequence of the subtype obtained was submitted to the GenBank® public repository under the accession number MK105902. Duration of gastrointestinal complaints lasted for two weeks after diagnosis and remitted spontaneously without specific treatment.

C. hominis IbA13G3 was initially reported in four Peruvian HIV-positive individuals in 2007.[5] Subsequent molecular epidemiological surveys conducted in Nigeria allowed the identification of this subtype in community ($n = 7$)[6] and clinical ($n = 1$)[7] paediatric populations, and in HIV-infected persons ($n = 2$).[8] Additionally, the IbA13G3 subtype accounted for 19% ($n = 17$) of all *Cryptosporidium* genetic variants detected in children from rural Ghana, only second after the IIcA5G3a subtype.[9] Based on these molecular data a West African origin for *C. hominis* IbA13G3 has been suggested, but not conclusively proven, by some authors.[5,7] As expected, our phylogenetic analysis based on the *GP60* gene demonstrated that the IbA13G3 sequences generated in the present study and those available at GenBank from Ghana[9] clustered together in a well-defined group within the family subtype Ib. In order to enable direct comparison, reference sequences and representative sequences of the most frequent *C. hominis* family subtypes circulating in different Spanish human populations were also included in the analysis (Fig. 1).

In summary, we identified the presence of *C. hominis* IbA13G3 in two returning travellers from Nigeria, the geographical area where this *Cryptosporidium* subtype seems more prevalent. To the best of our knowledge the IbA13G3 subtype has been only detected so far in West African countries and Peru, so this is the first report of the occurrence of this *Cryptosporidium* subtype in Europe. These and previous[10,11] findings by our laboratory highlight the relevance of conducting active, molecular-based surveillance for the investigation of travel-related morbidity as an essential component of global public health surveillance.

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Competing interests

The authors declare that they have no competing interests.

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