neuroimagen, cumpliendo los criterios de mielitis transversa antes mencionados.

La mielitis transversa aguda puede ser resultado de procesos autoinmunes[4], posvacunal[7] o en la mayoría de casos posterior a procesos infecciosos de etiología viral, entre los más comunes se han reportado citomegalovirus, herpes virus, Epstein Bar virus y varicela zoster[8,9]. Una revisión sistemática realizada en Europa, que evaluó las manifestaciones neurológicas de 129 casos debidos a infección por parvovirus B19, documentó solo 2 casos de mielitis transversa y uno de mielitis parainfecciosa asociados a este virus y confirmados serológicamente por la presencia de IgG e IgM en el LCR de pacientes entre 1970 y 2012[2,10,11]. Susuki et al. Publicaron el caso de un niño de 9 años con diagnóstico de mielitis transversa aguda asociado a parvovirus B19 confirmado serológicamente por presencia de ADN en suero y líquido cefalorraquídeo[12]. La búsqueda en la base de datos PubMed no reportó más casos hasta la fecha.

El parvovirus B19 es un virus ADN de cadena simple, con tropismo hacia eritroblastos de la médula ósea en humanos, quienes son sus únicos huéspedes[1]. En el adulto inmunocompetente las principales manifestaciones clínicas de la infección por parvovirus B19 encontradas en el estudio de Parra et al., realizado en Francia, incluyen fiebre (65%), artralgias (62%), erupción maculopapular en el tronco, las extremidades y la cara (47%), mialgias (38%), adenopatías (38%), edema difuso de manos (32%), prurito (16%) y parestesias(4%)[13]. Debido a un brote de parvovirus B19 en un hospital pediátrico de la zona, orientamos nuestro caso hacia una etiología viral, por lo cual dentro de los virus a investigar se solicitó anticuerpos para parvovirus B19, que resultaron positivos en suero y en el LCR.

En conclusión, los trastornos desmielinizantes agudos por parvovirus B19 constituyen una manifestación clínica poco común de la infección por esta enfermedad. Nuestra paciente presentó todos los criterios clínicos de una mielitis transversa, aunque el reporte de la resonancia magnética muestra un engrosamiento de la raíz neural, la misma excluye una etiología compresiva. Como punto importante debemos tener en cuenta que el diagnóstico de mielitis transversa se basa en los hallazgos clínicos, además en nuestro caso se demostró inflamación en la médula espinal por la presencia de pleocitosis con predominio de mononucleares en el LCR. Finalmente, el cuadro respondió favorablemente a la administración de corticoides intravenosos y orales.

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Ribotype 027 *Clostridioides difficile* infection presented as a traveller's diarrhoea

Infección por Clostridioides difficile *Ribotipo 027 presentada como diarrea del viajero*

A 34-year-old Spanish man with no previous medical history presented with diarrhoea during a tourist trip to Cuba, where he stayed for 15 days. Five days after arriving to Cuba, he started with watery diarrhoea – up to 14 stools per day without pathological products –, abdominal pain and temperature up to 37.3 °C. For this reason, he received antibiotic treatment twice: firstly, in Cuba for 3 days (unknown active principle), and secondly after returning from the trip, with ciprofloxacin 500 mg/12 h for 7 days. Given the lack of improvement, he decided to consult to our International Health Department. During the trip, he had eaten street-food, uncooked vegetables, peeled fruit and ice with drinks. The patient denied having had unprotected sex and he had not visited any healthcare facility. At our visit, the physical exam revealed abdominal pain at palpation of left lower quarter but peritonism and blood test only showed elevated acute-phase reactants.

From a microbiological point of view, no trophozoites or ova were observed in two microscopic coproparasitological exams and no enteropathogenic microorganisms were isolated in a bacterial culture. The patient was then tested for *Clostridioides difficile* infection (CDI). Glutamate dehydrogenase (GDH) was detected by immunochromatography (Health & Research *C. difficile* GDH, Vegal Farmaceutica, Spain). Detection of three targets: Toxin B, Binary Toxin (cdtA), and tcdC deletion, for presumptive identification of the 027/NAP/BI epidemic strains of *C. difficile*, were determined by real time polymerase-chain reaction (Xpert® C. difficile, Cepheid, CA, USA), with Ct values of 23.3; 23.2; 23.1, respectively. Faecal specimen was sent to a reference laboratory, where ribotype 027

was confirmed by ribotyping and sequencing of gene tcdC, that showed 18pb delection in position 117.

Given the potential virulence of the strain, treatment with Fidaxomicin 200 mg/12 h for 10 days, was prescribed. The patient presented an excellent clinical evolution with resolution of gastrointestinal symptoms 48 h after treatment initiation and with no further relapses.

C. difficile is a sporulated anaerobic gram-positive bacilli. Although it is the main etiological agent of health-care associated diarrhoea in high income countries, it is increasingly being reported as a cause of community acquired diarrhoea (representing up to 25% of cases of CDI).[1] In the last decade, the rise of a new *C. difficile* strain, known as B1/NAP1/027 brought important changes on the CDI epidemiology, spreading throughout United States of America, Canada and Europe after its first description in Quebec in 2001.[2] Furthermore, its higher virulence – caused by a mutation on the tcdC repressor gene – and the high resistance to fluoroquinolones, have entailed a change on the clinical spectrum of the CDI. As a consequence, infection by *C. difficile* ribotype 027 has been associated with more severe cases, poor response to common antibiotic treatment and higher relapse rates.[1]

Despite ribotype 027 is an uncommon cause of CDI in Spain,[3] some health-care related outbreaks have been reported.[4] Index cases coming from high prevalence countries have usually been identified, but indigenous community-acquired cases have also been identified.[4] Regarding the spread of this strain along Latin America, outbreaks in Costa Rica,[5] Panama[6] and Chile[7] have been reported. However, it is probably a very undiagnosed entity due to the scarce access to diagnostic test for anaerobic bacteria by routine.[8]

Interestingly, it is estimated that about 1–3% of general population can be asymptomatic carriers of *C. difficile* toxigenic strains,[9] being at risk of developing invasive infection due to colon microbiome changes. In fact, not only the overuse of antibiotics in traveller's diarrhoea but also the impact of travelling itself can prompt these microbiome changes, favouring infections by enteropathogens such as *C. difficile* in travellers.[10]

Although acquisition of the CD 027 strain before arriving to Cuba cannot be completely ruled out as initial symptoms started only five days after settling the country, from the best of our knowledge, it is the first reported case of CDI 027 after travelling to Cuba. We consider this case relevant, not only for the possible country of acquisition, but also for its clinical presentation as a traveller's diarrhoea in a healthy patient with no contact with health-care facilities. *C. difficile* 027 has been usually associated with nosocomial outbreaks and it is rarely suspected in healthy patients with no classical risk factors. Although our patient took antibiotic treatment, the beginning of symptoms was previous to the first antimicrobial treatment and he had no contact with any health-care facility. As a consequence, we consider that our case highlights the importance of being aware of CDI in patients with persistent diarrhoea even when they do not fulfilled classical CDI risk factors.

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A case of dyspnea due to pathogenic *Corynebacterium striatum* in a patient with a bronchial stent

Un caso de disnea debida a Corynebacterium striatum *patógeno en un paciente con stent bronquial*

A 67-year-old woman who received surgery, radiotherapy, and chemotherapy six months ago for the treatment of lung adenoid cystic carcinoma (ACCM) was admitted to our hospital with a ten-day history of cough and shortness of breath. She was admitted 17 days after her last round of chemotherapy, during which she developed fever and leucopenia, which improved after treatment. Two days before admission, she experienced shortness of breath and went to a local hospital. Despite treatment, the dyspnea persisted.

After admission to our hospital, a pulmonary examination revealed mild cyanosis, signs of respiratory depression and diminished breath sounds in the left upper lobe. Laboratory tests showed a white blood cell (WBC) count of 17.88×10^9 L-1 (86.6% neutrophils). Sputum cultures were negative before the implantation