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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Laia Albiach[a,*,1], Daniel Camprubí[b,c,1], Miriam J. Alvarez-Martínez[c,d], Alex Soriano[a]

[a] *Infectious Diseases Department, Hospital Clínic de Barcelona, Spain*
[b] *Tropical Medicine and International Health Department, Hospital Clínic de Barcelona, Spain*
[c] *ISGlobal, Hospital Clínic – Universitat de Barcelona, Barcelona, Spain*
[d] *Microbiology Department, Hospital Clínic de Barcelona, Spain*

* Corresponding author.
E-mail address: albiach@clinic.cat (L. Albiach).
[1] Both authors contributed equally to this article.

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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

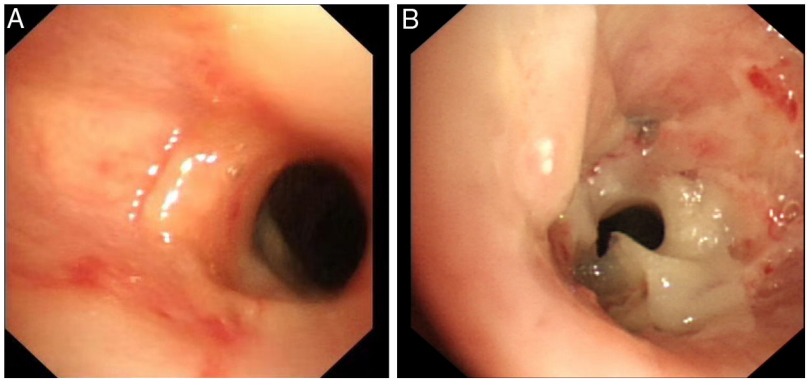

Fig. 1. (A) Fluorescence bronchoscopy showing some necrotic pseudo-membranes at the top of the stent. (B) Fluorescence bronchoscopy showing a great quantity of necrotic pseudo-membranes at the top of the stent which narrowed the lumen.

of bronchial stent. Chest computed tomography (CT) revealed a low-density shadow in left bronchial cavity, left upper atelectasis, and right-sided pneumonia. The patient was initially treated with moxifloxacin and cefotaxime due to elevated WBC and right-sided pneumonia. Meanwhile, she received a fluorescence bronchoscopy for the left upper lobe atelectasis detected through CT. Bronchoscopy revealed prominent scar tissue stenoses in the middle trachea and left main bronchus. The patient was implanted with two metallic bronchial stents at these two locations and her condition improved subsequently. However, she experienced shortness of breath again three days later. A second bronchoscopy revealed a concentration of necrotic pseudo-membranes at the top of the stent (Fig. 1A). Removing the obstructive biofilm relieved the symptoms. One week later, however, the symptoms recurred and could not be alleviated. A third bronchoscopy revealed a large number of pseudo-membranes clustered at the top of the stent, narrowing the lumen (Fig. 1B). Dyspnea was relieved once the necrotic pseudo-membranes were removed. The necrotic specimen was sent to the laboratory and *Corynebacterium striatum* was detected through blood agar plate, Gram staining, and VITEK 2 Compact automatic microbial analysis system. Since *C. striatum* is considered to be a pathogenic bacterium, she was treated with the empirical therapy of vancomycin (1 g every 12 h) for 14 days. Dyspnea resolved, and she had no recurrence of symptoms. She was re-admitted to the hospital for evaluation at one month and three months post-treatment. Bronchoscopies at both follow-ups showed no stent obstruction or new formation of necrotic pseudo-membranes. Furthermore, both bronchoalveolar lavage cultures were negative.

C. striatum, which belongs to Corynebacterium spp, is a part of the normal flora of the human skin and respiratory tract. Although commonly considered a contaminant, it has been increasingly reported as an etiologic agent of various infections, and commonly infects the respiratory tract. *C. striatum* was first reported in 1980 as a pathogenic bacteria causing lung infection in a patient with chronic lymphocytic leukemia.[1] Renom et al. reported the first nosocomial outbreak of *C. striatum* in patients with chronic obstructive pulmonary disease in 2007.[2] Additionally, Roig-Rico et al. described *C. striatum* pneumonia in an HIV patient in 2011.[3] The aforementioned patients were all with severe underlying diseases. In our case, the patient was immunocompromised due to radiotherapy and chemotherapy after surgery for ACCM. Los-Arcos et al. reported that the incidence of Corynebacterium detection was significantly higher in patients with a bronchial stent than in those without (29% *versus* 4%) and *C. striatum* was the main isolated species (48%).[4] However, the average period for the Corynebacterium respiratory isolation after stent implantation was 1036 days.[4] A recent study revealed *C. striatum* played a central role in endobronchial stent biofilm structure and was not generally considered a pathogenic bacteria.[5] However, our patient developed dyspnea within three days of stent implantation and the symptoms recurred in the next few days, indicating that *C. striatum* was a pathogenic bacterium rather than a contaminant.

C. striatum is generally resistant to penicillin but susceptible to other β-lactams and vancomycin. However, recent studies show *C. striatum* is resistant to a variety of antibiotics including daptomycin, macrolides, and cephalosporins.[6,7] In fact, Los-Arcos et al. reported that 16.3% isolates were sensitive only to vancomycin.[4] Hahn et al. reported that the minimum inhibitory concentration (MIC) distribution for vancomycin for *C. striatum* isolates was 0.6 mg/L, which was universally active *in vitro* and could be used as empirical therapy.[8] Topic et al. suggested a need for a prolonged antibiotic treatment (6 weeks) in patients with fever and bacteremia.[9] Fortunately, our patient had only respiratory symptoms and did not present with fever. She was treated with vancomycin for 14 days. The follow-up results of the patient were satisfactory.

In conclusion, our case indicates that *C. striatum* can be a pathogenic bacterium in patients with stent implantation and should be taken seriously in clinical practice.

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Dongyi Zhu [a], Wenyan Zhang [b], Zhibing Luo [a,*]

[a] *Department of Respiratory Medicine, Shanghai East Hospital, Tongji University School of Medicine, Shanghai 200120, China*

[b] *Department of Microbiology, Shanghai East Hospital, Tongji University School of Medicine, Shanghai 200120, China*

* Corresponding author.
E-mail address: luozhibing669@sohu.com (Z. Luo).

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