Enfermedades Infecciosas y Microbiología Clínica 40 2022 275–283

Enfermedades Infecciosas y Microbiología Clínica

www.elsevier.es/eimc

Scientific letter

Bacteremia due to C. jejuni in kidney transplant patients. Is immediate post-transplant immunosuppression a risk factor?☆

Bacteriemia por C. jejuni en el paciente trasplantado renal. ¿Es la inmunosupresión del postrasplante inmediato un factor de riesgo?

We report the case of a 52-year-old man with terminal chronic kidney disease secondary to autosomal dominant polycystic kidney disease who underwent a kidney transplant in July 2020, for which he was initially receiving quadruple immunosuppressant therapy with thymoglobulin (425 mg total), tacrolimus, mycophenolate mofetil and prednisone. Subsequently, given his poor kidney function secondary to extensive post-transplant kidney infarction, a decision was made to start belatacept (900 mg once every other week for two months, then once monthly, down-titrating the dose to a steady 500 mg monthly at present) with gradual withdrawal of tacrolimus. He received prophylaxis with trimethoprim/sulfamethoxazole and valganciclovir for six months.

In September 2020, he visited the accident and emergency department due to fever and diarrhoea with up to seven bowel movements in the past 24 hours. Laboratory testing revealed results for polymerase chain reaction (PCR) testing of 113 mg/l and leukopaenia of 2,350. He was initially diagnosed with acute gastroenteritis. After stool and blood cultures were taken, he was started on empirical therapy consisting of intravenous levofloxacin 500 mg/day. Subsequently, he remained afebrile and diarrhoea-free as of his second day of admission.

Stool PCR and stool culture detected *Campylobacter jejuni*. Given these results, levofloxacin was suspended and oral azithromycin 500 mg was administered for three days. At the same time, three days after incubation, quinolone-resistant *C. jejuni* was isolated in the blood cultures, and a decision was made to administer treatment with meropenem and order a full-body computed tomography (CT) scan to screening for malignancy; the scan was negative for neoplasms.

Once the patient's signs and symptoms resolved, his blood culture results became negative and he followed a good clinical course. After he had completed seven days of treatment with meropenem 1 g, he was discharged with a five-day regimen of oral amoxicillin/clavulanic acid 500/125 mg up to 14 days of treatment. In follow-up (six months), the patient has remained asymptomatic apart from presenting replication in the eight month of the post-transplant period.

Discussion

C. jejuni infection causes signs and symptoms of enteroinvasive diarrhoea and accounts for less than 1% of cases of bacteraemia. Such cases generally occur in the immunosuppressed population. Among patients with bacteraemia due to *C. jejuni*, 38% may have tumours.[1] Other commonly associated factors are human immunodeficiency virus (HIV), liver disease and hypogammaglobulinaemia.[2] In our case, we did not test for immunoglobulin levels, and therefore we are unaware of their potential contribution. However, we found only three reported cases in the literature of bacteraemia due to *C. jejuni* in kidney transplant patients.[3–5] All of them occurred in the first year of the post-transplant period, as in our case. This is probably related to high levels of immunosuppression during this period.[3]

With regard to immunosuppression, a higher prevalence of infections has been reported in patients induced with thymoglobulin versus basiliximab, with bacterial infections being significantly more common.[6] In addition, the use of belatacept was linked to an increase in cytomegalovirus (CMV), *Pneumocystis jirovecii* and *Mycobacterium tuberculosis* infections.[7–9] Age and low glomerular filtration rate were identified as risk factors for infection.[8,10]

The use of belatacept, though it improved the patient's glomerular filtration rate from 7 ml/min to 22 ml/min, might have been a risk factor for bacteraemia due to *C. jejuni*. However, in other published cases of bacteraemia in kidney transplant patients, the immunosuppressants used were different, which would make this the first reported case with belatacept.

Regarding antibiotic therapy, quinolones should be avoided since *C. jejuni* is quinolone-resistant in 50% of cases.[2] In our case, the patient was on prophylaxis with trimethoprim/sulfamethoxazole; nevertheless, it has been reported that 21%-25% of *C. jejuni* strains may be resistant.[2] In addition, there have been almost no cases of resistance to carbapenems.[2] The mortality rate in these patients is 15%, and the factors associated with a poor prognosis are delayed initiation of antibiotics and deficient antibiotic coverage.[1]

In conclusion, bacteraemia due to *C. jejuni* is a very uncommon infection in kidney transplant patients that may be fostered by the choice of immunosuppression, as well as other factors such as hypogammaglobulinaemia. Therefore, suitable antibiotic coverage is important given the potentially fatal nature of this infection.

Funding

The authors declare that they received no funding to conduct this study.

Conflicts of interest

The authors declare that they have no potential conflicts of interest related to the content of this article.

References

1. Pacanowski J, Lalande V, Lacombe K, Boudraa C, Lesprit P, Legrand P, et al. Campylobacter Bacteremia: Clinical Features and Factors Associated with Fatal Outcome. Clin Infect Dis. 2008;47:790–6, http://dx.doi.org/10.1086/591530.
2. Fernández-Cruz A, Muñoz P, Mohedano R, Valerio M, Marín M, Alcalá L, et al. Campylobacter Bacteremia. Medicine (Baltimore). 2010;89:319–30, http://journals.lww.com/00005792-201009000-00006.

☆ Please cite this article as: Santos-Alonso C, González-García E, Ruíz-Carrascoso G, Loeches-Yagüe B. Bacteriemia por C. jejuni en el paciente trasplantado renal. ¿Es la inmunosupresión del postrasplante inmediato un factor de riesgo? Enferm Infecc Microbiol Clin. 2022;40:275–276.

3. Pereira L, Sampaio S, Tavares I, Bustorff M, Pestana M. Bacteremia due to Campylobacter in renal transplantation: a case report and review of literature. Transpl Infect Dis. 2014;16:1007–11, http://dx.doi.org/10.1111/tid.12302.
4. Aggarwal H, Kushnir L, Conti D, Gallichio M, Tobin E. A case of Campylobacter jejuni bacteremia in a renal transplant patient. Transpl Infect Dis. 2010;12:518–20, http://dx.doi.org/10.1111/j.1399-3062.2010.00531.x.
5. Maccario M, Tarantino A, Nobile-Orazio E, Ponticelli C. Campylobacter jejuni bacteremia and Guillain-Barré syndrome in a renal transplant recipient. Transpl Int. 1998;11:439–42, http://dx.doi.org/10.1007/s001470050171.
6. Brennan DC, Daller JA, Lake KD, Cibrik D, Del Castillo D, Thymoglobulin Induction Study Group. Rabbit Antithymocyte Globulin versus Basiliximab in Renal Transplantation. N Engl J Med. 2006;355:1967–77.
7. Karadkhele G, Hogan J, Magua W, Zhang W, Badell IR, Mehta A, et al. CMV high-risk status and posttransplant outcomes in kidney transplant recipients treated with belatacept. Am J Transplant. 2021;21:208–21.
8. Bertrand D, Chavarot N, Gatault P, Garrouste C, Bouvier N, Grall-Jezequel A, et al. Opportunistic infections after conversion to belatacept in kidney transplantation. Nephrol Dial Transplant. 2020;35:336–45.
9. Fishman JA. Mycobacterium tuberculosis in transplantation: Immunity sufficient to perpetuate disease? Am J Transplant. 2019;19:1262–3.
10. Chavarot N, Divard G, Scemla A, Amrouche L, Aubert O, Leruez-Ville M, et al. Increased incidence and unusual presentations of CMV disease in kidney transplant recipients after conversion to belatacept. Am J Transplant. 2020:0–2.

Carlos Santos-Alonso [a,*], Elena González-García [a], Guillermo Ruíz-Carrascoso [b], Belén Loeches-Yagüe [c]

[a] *Servicio de Nefrología del Hospital Universitario La Paz, Madrid, Spain*
[b] *Servicio de Microbiología del Hospital Universitario La Paz, Madrid, Spain*
[c] *Unidad de enfermedades infecciosas del Hospital Universitario La Paz, Madrid, Spain*

* Corresponding author.
E-mail address: Carlos.santos.alonso@gmail.com
(C. Santos-Alonso).

https://doi.org/10.1016/j.eimce.2022.02.009

Tachycardia as an undescribed adverse effect to the Comirnaty© vaccine (BNT162b2 Pfizer-BioNTech Covid-19 vaccine): Description of 3 cases with a history of SARS-CoV-2 disease☆

Taquicardia como efecto adverso no descrito en la vacuna Comirnaty© (Vacuna COVID-19 mRNA BNT162b2 de Pfizer-BioNTech): descripción de 3 casos con antecedentes de SARS-CoV-2

Multiple side effects were reported in the trial of the efficacy and safety of the Comirnaty© vaccine (Pfizer-BioNTech BNT162b2 mRNA COVID-19 vaccine)[1]. Following the recent vaccination of healthcare professionals, given that 24% of this population has had the disease[2], adverse effects not reported to date in the subpopulation of 540 subjects seropositive for SARS-CoV-2 in which the safety profile was no different from that observed in the general population have begun to be reported in the summary of product characteristics[3].

We report three cases of healthcare professionals, specifically physicians, who received the first dose of the vaccine on 18 January 2021 (1:15 PM). These individuals worked in the same department and developed the disease in March 2020, following an outbreak of SARS-CoV-2 in their workplace; none of them required hospital admission.

Their shared adverse effect, unpublished as of today and also not mentioned in the latest pharmacovigilance report from the Agencia Española de Medicamentos y Productos Sanitarios [Spanish Agency of Medicines and Medical Devices][4], was tachycardia unrelated to hyperthermia as a first sign of the onset of a systemic reaction in all three cases. It was therefore reported to the Centro Autonómico de Farmacovigilancia [Regional Pharmacovigilance Centre] within 48 h of vaccine administration.

On 21 January 2021, the Sociedad Española de Inmunología [Spanish Society of Immunology] prepared a briefing note on reactions to the first dose of the vaccine in individuals having already recovered from the disease, warning that vaccination would act as a second contact with the virus and trigger a more vigorous immune system response in them[5].

Case 1

The first case was a 60-year-old woman with a history of hypertension, dyslipidaemia and total thyroidectomy who was a heterozygous carrier of factor V Leiden mutation. She was on treatment with ramipril, rosuvastatin and levothyroxine. Her latest yearly thyroid check-up had the following results: thyroid-stimulating hormone (TSH) 1.12 μUI/mL (reference range: 0.270–4.200) and free thyroxine (T4) 1.44 ng/dl (reference range: 0.93–1.70).

She presented acute COVID-19 on 8 March 2020, and her signs and symptoms disappeared over the following two weeks. The clinical picture consisted of tiredness, fever, dry cough, muscle pain, joint pain, diarrhoea, scalp hypersensitivity and parosmia.

A polymerase chain reaction (PCR) test was negative. Two months later, a rapid test for SARS-CoV-2 IgG antibodies yielded a positive result.

The result of her latest serology (enzyme-linked immunosorbent assay [ELISA]) was IgG 2.1 U (positive >0.8) on 2 December 2020.

Fourteen hours after receiving the vaccine, she developed observed tachycardia of 110 bpm which spontaneously remitted in approximately 16 h, along with gastrointestinal abnormalities, pain at the puncture site with locoregional lymphadenopathy, tiredness, fever and scalp hypersensitivity.

Case 2

The second case was a 55-year-old woman with a history of Hashimoto's disease on treatment with levothyroxine. Her latest yearly thyroid check-up yielded the following results: TSH 0.608 μUI/mL (reference range: 0.270–4.200) and free T4 1.63 ng/dl (reference range: 0.93–1.70).

She presented acute disease on 6 March 2020; her symptoms consisted of tiredness, chills, fever, muscle pain, dry cough, frontal headache and parosmia, with a duration of 10 days. After eight days with no symptoms, her fever recurred for six additional days.

She had a positive PCR test on 15 March and a negative one on 5 April 2020.

The result of her latest serology (ELISA) was IgG 0.9 U (positive >0.8) on 2 December 2020.

Ten hours after vaccine administration, she developed tachycardia of up to 120 bpm, not co-occurring with hyperthermia, that made it difficult for her to sleep and spontaneously remitted 24 h

☆ Please cite this article as: Marco García MT, Torres Lana Á, Anta Agudo MB, Rufino Delgado MT. Taquicardia como efecto adverso no descrito en la vacuna Comirnaty© (vacuna COVID-19 mRNA BNT162b2 de Pfizer-BioNTech): descripción de 3 casos con antecedentes de SARS-CoV-2. Enferm Infecc Microbiol Clin. 2022;40:276–277.