multiple aetiologies, in parallel to the diagnosis of glomerular disease, with a prevalence of 5.5 % (with the expected being 3.1 %). After a 10-year follow-up, the incidence of neoplasm in this population was 1.8 times higher than expected, without being able to rule out the influence of the immunosuppressive treatment received (both by the treatment itself or by accelerating the existing neoplastic process).[2]

Given our case and the evidence in the literature, we wanted to reconsider on how nephrologists perform the screening of neoplasms in a patient with new diagnoses of glomerulonephritis. The KDIGO guidelines[5,6] recommend the screening of neoplasms in the case of membranous nephropathy, GEFyS and IgA nephropathy, but not in other forms, and without specifying the recommended tests to perform. Some authors propose an exhaustive study that includes a first level: chest x-ray, skin examination, breast/testicular examination, abdominal and cervical ultrasound, faecal occult blood (if positive: colonoscopy/gastroscopy). If everything is negative on a second level: mammography and gynaecological review; cystoscopy if haematuria; PSA and rectal exam (prostate biopsy if neoplasia is suspected). If everything is negative and in patients with a high risk (> 60 years, smokers, alcoholic; thromboembolism; HIV/HBV/HCV infection; prolonged immunosuppressive treatment; negative anti-PLA2R antibodies in Membranous GN): CT scan, colonoscopy, fibroendoscopy should be performed. And they recommend repeating the screening in patients who have received or receive prolonged immunosuppressive treatment, every 5 years if patients are < 60 years and every 3 years if> 60 years.[7]

According to the evidence described in the current study[2] with the various forms of glomerulonephritis and the multiple associated cancer, we suggest that the suspicion and investigation of the nephrologist should be greater, so as not to delay the diagnosis of an underlying cancer and to avoid the harmful consequences of an immunosuppressive treatment that can accelerate the development of neoplasia.

REFERENCES

1. Lee JC, Yamauchi H, Hopper J Jr. The association of cancer and the nephrotic syndrome. Ann Intern Med. 1966;64:41–51.
2. Heaf JG, Hansen A, Laier GH. Quantification of cancer risk in glomerulonephritis. BMC Nephrology. 2018;19:27.
3. Birkeland SA, Storm HH. Glomerulonephritis and malignancy: a population based analysis. Kidney Int. 2003;63:716–21.
4. Kuppers R, Schwering I, Brauninger A, Rajewsky K, Hansmann ML. Biology of Hodgkin's lymphoma. Ann Oncol. 2002;13 Suppl 1:11–8.
5. KDIGO Clinical Practice Guideline for Glomerulonephritis. Kidney International supplements. 2012;2:241–74.
6. Rovin BH, Caster DJ, Cattran DC, Gibson KL, Hogan JJ, Moeller MJ, et al. Management and treatment of glomerular diseases (part 2): Conclusions from a Kidney Disease: Improving Global Outcomes (KDIGO) Controversies Conference. Kidney Int. 2019;95:281–95.
7. Pani A, Porta C, Cosmai L, Melis P, Floris M, Piras D, et al. Glomerular diseases and cancer: evaluation of underlying malignancy. J Nephrol. 2016;29:143–52.

Elena Monfá[a,*], Irene Padilla[b], Benjamin de León[a], Maria Jesús Vidal[b], Carmen Barnes[a], Verónica Barcía[a], Catherine Martinez[a], Cristina Lucas[a], Aranzazu Sastre[a], Jorge Stefan[a], Mario Prieto[a]

[a] *Servicio de Nefrologí, Complejo Asistencial Universitario de León, León, Spain*

[b] *Servicio de Hematología, Complejo Asistencial Universitario de León, León, Spain*

[*] *Corresponding author.*
E-mail address: elenamonfa@gmail.com (E. Monfá).

25 February 2019

31 March 2019

https://doi.org/10.1016/j.nefroe.2019.03.011

Aureimonas altamirensis: The first case of peritonitis on peritoneal dialysis☆

Aureimonas altamirensis: primer caso de peritonitis en diálisis peritoneal

Dear Editor,

Aureimonas altamirensis (*A. altamirensis*) is a bacterial specie from the caves of Altamira, Cantabria, Spain; considered as an environmental pollutant, but isolated for the first time in biological samples in 2008 by Luong et al., it is a potential human pathogen, with a variable clinical presentation.[1,2]

A case of peritonitis due to *A. altamirensis*, isolated in the ascites fluid of an oncological patient, has been described in the literature,[3] no cases of peritonitis due to this micro-

DOI of original article:
https://doi.org/10.1016/j.nefro.2019.03.007.

☆ Please cite this article as: Andrade López AC, Bande Fernández JJ, Cortés EA, Gúrpide BD, Suárez CR. Aureimonas altamirensis: primer caso de peritonitis en diálisis peritoneal. Nefrologia. 2019;39:675–677.

organism have been reported in patients on peritoneal dialysis (PD).

Below, we present a case of peritonitis due to *A. altamirensis*, in a patient on PD and we describe its clinical importance.

The patient is an 81-year-old male, with stage 5 chronic kidney disease (CKD) in PD program, with incremental modality (IPD), with a night exchange with 2.3% glucose, who presented abdominal pain of less than 24 h of evolution, along with cloudy fluid in the peritoneal exchange. Not clinical symptoms at any other level.

By Physical exam appears to be haemodynamically stable and afebrile, abdominal tenderness, preserved peristalsis, not painful at palpation and no signs of peritoneal reaction. The exit hole of the peritoneal catheter showed no inflammatory signs.

Among the complementary tests, a cell count was performed in the peritoneal fluid, which showed 827 leukocytes/μl, with 66% of neutrophils, so empirical treatment of peritonitis was established in relation to the technique with vancomycin and intraperitoneal ceftazidime. No imaging tests were performed.

In the culture of peritoneal fluid *A. altamirensis* resistant to gentamicin and tobramycin was isolated; sensitive to piperacillin tazobactam, ceftazidime, ciprofloxacin, trimethoprim sulfamethoxazole and amikacin. According to these results, antibiotic monotherapy with ceftazidime was maintained for 3 weeks with good response, with a cell control in peritoneal fluid of 70 leukocytes/μl, at 48 h.

The genus *Aurantimone,* described by Denner et al.[4] in 2003, covers 4 species from environmental sources, including *A. altamirensis,* a gram-negative aerobic bacillus of marine waters.[5] Initially it was considered a pollutant derived from the environment and/or water sources, but in recent years it has been isolated in biological samples, such as in the sputum of patients with cystic fibrosis, pleural effusion, eye infections (keratitis, corneal ulcers) and even in blood cultures of immunosuppressed patients.[2,6,7]

In a review of the literature, there are only a few cases published in which *A. altamirensis* is identified as a pathogen in humans: peritonitis in a patient with stage IV cholangiocarcinoma with peritoneal carcinomatosis[3]; 2 cases of pleural effusion described by Tellez-Castillo et al., with isolation of *A. altamirensis* in pleural fluid, one of the patients had a gastric adenocarcinoma[8]; lastly, 2 cases of bacteriemia due to *A. altamirensis,* one of them associated with a scrotal infection, in a pluripathological patient with stage 4 CKD[7]; and another in a patient with multiple myeloma, Bence-Jones type.[2]

It seems that all these cases would have as a common feature, an important comorbidity that conditions a situation of immunosuppression of variable importance. *A. altamirensis* could be considered as an opportunistic pathogen, which would cause the disease in patients weakened by an underlying disease.

From the epidemiological point of view, the patient did not report risk behaviours such as contact with animals, intake of contaminated food or untreated water, in our case we would have as the possible factors favouring infection: a dysfunctional immune system, typical of CKD patients on dialysis and the presence of a foreign body (PD catheter), on which, the bacterium has the ability to generate a biofilm, as described in one of the cases of bacteraemia, where the micro-organism was isolated in a blood culture taken from a venous reservoir, of the port-a-cath type.[2]

In our opinion, this is a case of great interest because it is the first peritonitis due to *A. altamirensis* in a patient on peritoneal dialysis, making it clear that, although in the literature it is described as of doubtful pathogenicity in our patients (with a higher comorbidity than usual), this becomes more apparent. It should also be noted that, although there is little experience in the treatment of *A. altamirensis* infections, in our case the pattern of empirical treatment of peritonitis on PD: ceftazidime plus vancomycin, was fully effective, with the resolution of the clinical picture.

REFERENCES

1. Luong ML, Békal S, Vinh DC, Lauzon D, Leung V, Al-Rawahi GN, et al. First Report of isolation and characterization of aurantimonas altamirensis from clinical samples. J Clin Microbiol. 2008;46:2435–7, http://dx.doi.org/10.1128/JCM.00337-08.
2. García-Lozano T, Aznar Oroval E, Juan Bañón JL. Primer aislamiento en España de Aurantimonas altamirensis procedente de un hemocultivo de port-a-cath en un paciente con mieloma múltiple tipo Bence-Jones. Enferm Infecc Microbiol Clin. 2012;30:217–8, http://dx.doi.org/10.1016/j.eimc.2011.11.013.
3. Schröttner P, Rudolph WW, Taube F, Gunzer F. First report on the isolation of aureimonas altamirensis from a patient with peritonitis. Int J Infect Dis. 2014;29:71–3, http://dx.doi.org/10.1016/j.ijid.2014.09.006.
4. Denner EBM, Smith GW, Busse H-J, et al. Aurantimonas coralicida gen. nov., sp. nov., the causative agent of white plague type II on Caribbean scleractinian corals. Int J Syst Evol Microbiol. 2003;53:1115–22, http://dx.doi.org/10.1099/ijs.0.02359-0.
5. Jurado V, Gonzalez JM, Laiz L, Saiz-Jimenez C. Aurantimonas altamirensis Sp. Nov., a member of the order rhizobiales isolated from altamira cave. Int J Syst Evol Microbiol. 2006;56:2583–5, http://dx.doi.org/10.1099/ijs.0.64397-0.
6. Eshaghi A, Shahinas D, Patel SN, Kus JV. First draft genome sequence of aureimonas altamirensis, isolated from patient blood culture. FEMS Microbiol Lett. 2015;362, http://dx.doi.org/10.1093/femsle/fnv016.
7. Mendes RE, Denys GA, Fritsche TR, Jones RN. Case report of aurantimonas altamirensis bloodstream infection. J Clin Microbiol. 2009;47:514–5, http://dx.doi.org/10.1128/JCM.02171-08.
8. Tellez-Castillo CJ, González Granda D, Bosch Alepuz M, Jurado Lobo V, Saiz-Jimenez C, Juan JL, et al. Isolation of aurantimonas altamirensis from pleural effusions. J Med Microbiol. 2010;59:1126–9, http://dx.doi.org/10.1099/jmm.0.019596-0.

Ana C. Andrade López[a,*], José J. Bande Fernández[a], Elena Astudillo Cortés[a], Durá Gúrpide[b], Carmen Rodríguez Suárez[a]

[a] *Área de Gestión Clínica de Nefrología, Hospital Universitario Central de Asturias, Oviedo, Asturias, Spain*

[b] Departamento de Nefrología, Hospital Universitario Puerta de Hierro-Majadahonda, Majadahonda, Madrid, Spain

* Corresponding author.
E-mail address: cris_anlo@hotmail.com (A.C. Andrade López).

27 February 2019

https://doi.org/10.1016/j.nefroe.2019.03.010

Reversible posterior encephalopathy syndrome in renal transplanted patient with refractory arterial hypertension secondary to iliac artery stenosis☆

Síndrome de encefalopatía posterior reversible en paciente trasplantada renal con hipertensión arterial refractaria secundaria a estenosis de arteria ilíaca

ARTICLE INFO

Keywords:
Encephalopathy
Renal transplant
Arterial hypertension

Dear Editor,

With regard to the article published in the journal of Nefrología Ïliac artery stenosis after kidney transplantation as a cause of refractory hypertension and claudication"[1] and a review on posterior reversible encephalopathy syndrome (PRES),[2] published in *NefroPlus*, we present a case that is closely related to both publications.

Vascular problems remain one of the main complications after kidney transplantation, with an incidence of 3%–15%.[3]

Post-transplant arterial hypertension (HTN) secondary to decreased renal blood flow, due to the involvement of the graft artery or less frequently of the proximal aorto-iliac segment,[4] is a correctable form[1] of HTN.

We present the case of a 62-year-old woman with a history of chronic tubulo-interstitial nephropathy and HTN controlled with amlodipine, who had received a kidney transplant and was on immunosuppressive treatment with tacrolimus, sirolimus and prednisone. At 3 weeks, she began with complaints of severe headache, vomiting and blurred vision, associated with refractory HTN, with no evidence of impairement in renal function or proteinuria. The eye fundi showed a grade 2 hypertensive retinopathy. Given the clinical suspicion of hypertensive encephalopathy, a brain CT scan was ordered, with findings compatible with PRES, which was confirmed by brain nuclear magnetic resonance (MNRB) (Fig. 1a and b). Due to poor blood pressure control, hypotensive treatment was intensified; complementary examinations were performed to rule out causes of secondary hypertension and tacrolimus was discontinued, although its plasma concentration remained within the target therapeutic range from admission.

The study of secondary HTN was negative, except for the echo-Doppler of the graft, which reported probable renal artery stenosis (tardus parvus morphology and increased speeds up to 200 cm/s in the proximal portion) (Fig. 2a). CT angiography posed the possibility of arteriovenous fistula, being not very assessable due to artefact (right hip prosthesis), so selective arteriography was performed that evidenced a stenosis of the external iliac artery prior to anastomosis of the artery of the graft (Fig. 2b), with reduction of the calibre of the lumen to 3.5 mm. An endovascular treatment was then performed with angioplasty of the stenosis, achieving a good morphological result. (Fig. 2c), with this procedure with/without stent being the treatment of choice in this type of pathology.[4]

At 24 h blood pressure was better controlled, with gradual disappearance of the symptomatology, which allowed the hypotensive treatment to be progressively reduced.

The MNRB, which was performed one month later, showed a significant improvement in the signs of PRES (Fig. 1c-d). In the control echo-Doppler there were no findings compatible with vascular alteration of the graft and the tacrolimus was reintroduced. The patient is currently asymptomatic and normotensive.

DOI of original article:
https://doi.org/10.1016/j.nefro.2019.02.002.

☆ Please cite this article as: Franco A, González Y, Balibrea N, Díaz M, Planells MC, Pérez Contreras FJ. Síndrome de encefalopatía posterior reversible en paciente trasplantada renal con hipertensión arterial refractaria secundaria a estenosis de arteria ilíaca. Nefrologia. 2019;39:677–679.