



Figura 1. Resonancia magnética nuclear craneal.

visuales, cefalea y convulsiones, pero se resolvieron en un máximo de 3 semanas sin secuelas ni recidivas al controlar la TA y, en el primer caso, modificar el tratamiento inmunosupresor. En nuestro caso, la paciente debutó a los 4 meses de haber presentado la enfermedad, cuando sus anticuerpos anti-MBG se habían negativizado y sin tratamiento inmunosupresor activo.

En este contexto resulta especialmente importante el control de la HTA para prevenir un cuadro grave que, aunque de curso generalmente benigno, puede provocar una encefalopatía potencialmente severa.

Conflictos de interés

Los autores declaran que no tienen conflictos de interés potenciales relacionados con los contenidos de este artículo.

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Tabla 1. Causas del síndrome de leucoencefalopatía posterior reversible.

- Encefalopatía hipertensiva (HTA inducida por embarazo, HTA maligna)
- Eclampsia
- Insuficiencia renal aguda o crónica
- Insuficiencia hepática aguda
- Sepsis
- Vasculitis y enfermedades autoinmunes: LES, PAN, Wegener, ESP, EMTC, dermatomiositis, poliangeítis microscópica, Schönlein-Henoch, crioglobulinemia
- Enfermedades hematológicas: SHU, PTT, anemia drepanocítica, enfermedades linfoproliferativas
- Porfiria aguda intermitente
- Alteraciones iónicas: hipomagnesemia, hipocalcemia
- Transfusiones sanguíneas
- Exposición a contrastes intravenosos
- Inmunosupresores y citotóxicos: anfotericina B, bevacizumab, altas dosis de corticoides, cisplatino, metotrexate, quimioterapia combinada (CHOP), ciclosporina, citarabina, gemcitabina, interferón alfa, inmunoglobulinas intravenosa, metotrexate, rituximab, sirolimus, sorafenib, sunitinib, tacrolimus y vincristina
- Crisis epilépticas prolongadas
- Tratamiento con eritropoyetina
- Primoinfección por VIH

CHOP: ciclofosfamida, doxorubicina, vincristina y prednisolona; EMTC: enfermedad mixta del tejido conectivo; ESP: esclerosis sistémica progresiva; HTA: hipertensión arterial; LES: lupus eritematoso sistémico; PAN: poliarteritis nodosa; PTT: púrpura trombocitopénica trombótica; SHU: síndrome hemolítico urémico; VIH: virus de la inmunodeficiencia humana.

Catheter-related relapsing peritonitis due to *Kocuria varians* in a patient undergoing Continuous Ambulatory Peritoneal Dialysis

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Dear Editor,

Kocuria varians is a Gram positive non-pathogenic commensal of the mammalian skin that can be present also in soil and water. *Kocuria* spp. was previously classified in the genus *Micrococcus*¹ and identification of the species by automated systems has been reported to be problematic.² Published cases of infections caused by *Kocuria varians* in the English literature are rare and consider patients with serious underlying disease.³ We report here a case of relapsing peritonitis in a patient undergoing Continuous Ambulatory Peritoneal Dialysis (CAPD) that was

resolved after catheter removal.

The patient, a 70-year-old man with chronic renal failure and class III heart failure was admitted to the hospital on 26 July 2010 with turbid peritoneal dialysate, non febrile, having mild abdominal pain and positive Rebound sign at the physical examination. Leucocyte cell count showed the presence of 1200 leucocytes/ μ l (neutrophil count of 90%) in the peritoneal dialysate but subsequent culture of the fluid resulted negative. Treatment started with vancomycin 15mg/kg/5 days i.p. and aztreonam 2gr/d i.p. for a total of 20 days. On 30 July 2010 cell count was 50 leucocytes/ μ l and the patient was discharged.

3 days later however, at the first control visit, the peritoneal dialysate was turbid anew, the patient presented the same clinical findings and cell count revealed the presence of 300 leucocytes/ μ l. A Gram positive, spherical microorganism that occurred in tetrads with circular, smooth, glistening and yellow colonies was recovered from the peritoneal dialysate. The microorganism was identified as *Kocuria varians* by the VITEK 2 system. The isolate was susceptible to gentamycin, erythromycin, clindamycin, tetracycline, glycopeptides and linezolid while it was resistant to levofloxacin by the disc diffusion method. Following laboratory report, the patient was treated with vancomycin alone (15mg/kg/5 days i.p. for a total of 20 days).

Turbidity of the peritoneal dialysate did not reappear until 27 August 2010 when the patient was admitted to the hospital with generalized abdominal pain, positive Rebound sign and cell count of 550 leucocytes/ μ l. *K. varians* was isolated for the second time and removal of the peritoneal catheter followed by insertion of a new one in a different position was considered. Culture of the removed catheter was positive for *K. varians*. Vancomycin i.p. was administered, subsequent cultures were negative and the patient

remained in good clinical condition since then.

Infections related to *K. varians* are uncommon but this species may act as opportunistic pathogen in immunocompromised patients with underlying diseases. Furthermore, *K. varians* is a biofilm forming bacterium⁴ thus probably complicating the antimicrobial treatment of catheter related infections. Erroneous identification of coagulase-negative Staphylococci as *Kocuria* spp. is possible and can be excluded with certainty only with the application of genotypic assays such as 16S RNA.¹ In the present case the Vitek 2 system using the new GP identification card⁵ reported a "very good identification" for all three isolations. This case report aims on emphasizing the importance of careful consideration of the laboratory and clinical procedures when rarely pathogenic microorganisms are isolated in the peritoneal dialysate of patients undergoing CAPD.

Conflicts of interest

The authors declare that there is no conflict of interest associated with this manuscript.

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Enfermedad de Paget poliostótica asintomática asociada a hiperparatiroidismo secundario en un paciente en diálisis peritoneal

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Sr. Director:

La enfermedad ósea de Paget (EP) es un trastorno focal del remodelado óseo que puede afectar a uno o más huesos¹. Es la segunda patología ósea más frecuente después de la osteoporosis y su diagnóstico en la mayoría de los casos se realiza mediante una bioquímica rutinaria, donde se aprecia elevación de la fosfatasa alcalina (FA), o en estudios de imagen realizados por otros motivos¹⁻³.

Su incidencia en pacientes con enfermedad renal crónica (ERC) es desconocida. Pocos han sido los casos comunicados en la literatura^{4,8}, y en algunos de ellos la presencia de EP se ve enmascarada por la presencia de hiperparatiroidismo secundario (HPS)^{4,6}, lo que dificulta su diagnóstico. En