Sanidad y Dependencia, Dirección General de Gestión del Conocimiento y Calidad Sanitaria.
3. Guías de Acceso Vascular en Hemodiálisis. Nefrología 2005;25(1).
4. Clinical Practice Guidelines for Vascular Access. Update 2006. Am J Kidney Dis 2006;48(1):S176-S247.
5. Rodríguez Hernández JA, López Pedret J, Piera L. El acceso vascular en España: análisis de su distribución, morbilidad y sistemas de monitorización. Nefrología 2001;21(1):45-51.
6. Pisoni RL, Young EW, Dykstra DM, Greenwood RN, Hecking E, Gillespie B, et al. Vascular access use in Europe and United States: Results from the DOPPS. Kidney Int 2002;61:305-16.

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C) BRIEF CASE REPORTS

CMV disease resistant to Ganciclovir. Should valganciclovir plasma levels be monitored in high risk patients?

Nefrología 2009;29**(2)**:180-181.

Dear Editor,

We report the case of a patient who received a kidney transplant in June 2007. The donor was Ig G CMV positive and the recipient was negative. The immunosuppression consisted of steroids, mycophenolate (1g/day) and tacrolimus (0.1mg/12 h). Borderline/acute rejection occurred on the 11th day post-transplant and was successfully treated with steroids.

The patient was discharged with good renal function, Cr 0.9mg/dl on prophylactic treatment with valganciclovir, for six months having to repeatedly adjust the doses because of leucopenia. CMV viral load was negative whilst on treatment.

10 days after discontinuing treatment, the patient presented with diarrhoea, abdominal pain, fever, leucopenia and thrombocytopenia, and deterioration of renal function, blood and urine cultures were negative and CMV- PCR was positive, 101,000 copies/ml.

Treatment was started with valganciclovir and the dose of MMF was reduced. The fever subsided and the CMV viral load began to decrease.

The patient was discharged with stable kidney function (1mg/dl), without leucopenia and on treatment with valganciclovir.

A few days later, the patient presented with low fever, abdominal pain and persistent CMV viral load (4,900cop/ml). The patient received treatment with intravenous ganciclovir during 20 days until the CMV viral load was negative in blood. The patient was discharged without symptoms, with normal leucocyte count and on valganciclovir treatment.

Three days after discharge, the patient developed fever, epigastrium pain and leucopenia again. CMV- PCR was negative in blood. Screening for acute febrile illness was negative except for discreet hepatosplenomegaly. The upper endoscopy showed normal mucosa of which biopsies were taken. The qualitative PCR of the gastric tissue was positive for CMV and HSV 6. Intravenous gancyclovir was reinitiated. Non-specific abdominal pain persisted along with anaemia and leucopenia, needing treatment with granulocyte colony-stimulating factor, transfusion of erythrocyte concentrate. The patient continued to have low-grade fever. The CMV serum PCR remained negative.

Suspecting CMV disease resistant to ganciclovir, a drug resistance test was carried out on the gastric specimen. An L 595F mutation in UL97 was found.

The UL97 gene regulates the phosphorylation of the ganciclovir associated with resistance to it. We then began treatment with intravenous foscarnet and specific anti-CMV immunoglobulin 200mg/kg every three weeks. The patient became asymptomatic after 10 days of treatment with normal leukocyte count. The complications of this treatment included transient acute kidney failure at two weeks, with hypomagnesaemia and hypokalaemia.

The treatment continued for one month, followed by valganciclovir and foscarnet every 48 hours until the therapeutic levels of valganclovir were confirmed. Since then, the patient has remained asymptomatic and with excellent kidney function, six months after the episode.

The difficulty in diagnosing CMV Diease in this case was because of the false negative CMV viral load test requiring the confirmation of CMV and its resistance on gastric tissue. The resistance of the treatment was probably enhanced by inadequate doses of valganciclovir, which stresses the importance of monitoring vanganciclovir levels to ensure an adequate treatment thus avoiding the development of resistance to the treatment.

1. Eid AJ, Arthus SK, Deziel PJ, Wilhelm MP, Razonable RR.Emergence of drug-resistant cytomegalovirus in the era of valganciclovir prophylaxis: therapeutic implications and outcomes.Clin Transplant 2008;22(2):162-70.
2. Nogueira E, Ozaki Ks, Vomitaba H, Granato CF, Camara MO, Pacheco-Silva A. The emergence of cytomegalovirus resistance to ganciclovir therapy in kidney transplant recipients. Int Immuno-pharmacol 2006;20(6):2031-7.
3. Boivin G, Goyette N, Gilbert E, Covington E. Analysis of Cytomegalovirus DNA Polymerase (UL54) Mutations in Solid Organ Transplant Patients Receiving

Valganciclovir Ganciclovir Prophylaxis. J Med Virol 2005;77:425-9.
4. Cervera C, Pineda M, Linares L, Marcos MA, Antón A, Cofán F, et al. Impact of valganciclovir prophylaxis on the development of severe late-cytomegalovirus disease in high-risk solid organ transplant recipients. Transplant Proc 2007;39(7):2228-30.
5. Asberga A, Humar A, Rollagc H, Jardined AG, Mouase H, Pescovitz MD, et al. on behalf of the VICTOR Study Group. Oral Valganciclovir Is Noninferior to Intravenous Ganciclovir for the Treatment of Cytomegalovirus Disease in Solid Organ Transplant Recipients. American Journal of Transplantation 2007;7:2106-13.

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Cefepime-induced encephalopathy in patients with renal failure

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Dear Editor,

Cefepime is a fourth-generation cephalosporin that is widely used in hospital settings.[1] Since its approval, isolated cases of encephalopathy have been reported in patients with both normal[2] and impaired kidney function.[3,4] Nonetheless, the information about the clinical manifestations and the prognosis of this adverse reaction is scarce. Therefore, we believe it is important to report seven cases of cefepime-induced encephalopathy in patients with kidney failure. These cases corresponded to 4 males and 3 females with an average age of 63 years. All of the patients had acute or chronic renal failure when cefepime was prescribed. The average value of creatinine at the beginning of treatment was 3.6mg/dl and the initial dose of cefepime was 2.75g/day; in five patients the dose was adjusted for the degree of kidney function. The average time period between beginning of treatment and symptoms was 5.4 days. The most common clinical manifestations were a decreased level of consciousness (71.4%) and myoclonus (71.4%). The EEG was pathological in the six cases where it was carried out, demonstrating a non-convulsive epileptic status in three, slowed global activity with repetitive paroxysm in two, and diffuse affectation with a predominance of triphasic waves in one. The CT scan and the spinal tap were normal in all cases. After diagnosing the encephalopathy, treatment with cefepime was discontinued. Three of the patients received dialysis. Three patients improved (42.9%), one of whom required haemodialysis. The 4 remaining patients (57.1%) died from the encephalopathy.

The use of cefepime in patients with kidney failure, even at adjusted doses, may cause serious encephalopathy, and thus its administration should be avoided or used with close monitoring. The appearance of alterations in the level of consciousness and the myoclonus should alert us to the appearance of a non-convulsive status that requires an EEG as it is the most useful diagnostic test. Haemodialysis does not seem to modify the clinical outcome.

1. Sánchez-Bailén MJ, Navarro Ciruelos N, Rovira García-Marrón R, Anglés Coll. Encefalopatía asociada a la administración de cefepime en una paciente con shock séptico e insuficiencia renal. Rev Clin Esp 2004;204(9):501-4.
2. Maganti R, Jolin D, Rishi D, Biswas A. Non-convulsive status epilecticus due to cefepime in a patient with normal renal function. Epilepsy&Behavior 2006;8:312-4.
3. Chatelier D, Jourdan M, Mangalaboyi J. et al. Cefepime-induced neurotoxicity: an underestimated complication of antibiotherapy in patients with acute renal failure. Intensive Care Med 2002;28:214-7.
4. Barbey F, Bugnon D, Wauters JP. Neurotoxicity of Cefepime in Uremic Patients. Ann Intern Med 2001;135:1011.

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Ileal intussusception by carcinoid tumour in patients with chronic renal failure

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Dear Editor,

In connection with the clinical case presented in number 4, volume 26, of this same journal, we would like to report a similar case of intussusception of the terminal ileum by a carcinoid tumour in a patient with chronic kidney failure as it is an infrequent and little referenced illness in patients with chronic kidney failure.[1]

A 54 year old female patient with a history of chronic renal failure, hyperuricaemia and nephrolithiasis. She attended Accident and Emergency with generalized abdominal pain, nausea, vomiting and diarrhoea of 48 hour duration.

She had distended and tympanic abdomen with diffuse pain and no signs of peritonism.

Air-fluid levels could be seen in the small intestine on plain abdominal x-ray. The CT of the abdomen showed a dilated jejunum-ileum with thickening of the terminal ileum and caecum wall and a 4cm mass.

With the impression of an acute intestinal obstruction, the patient underwent urgent laparotomy. The small intestine was dilated to the terminal ileum where a tumour measuring 5cm was found that had caused the intussusception of the small intestine and the obstruction. A right colectomy and an ileum-colonic anastomosis were performed.

The postoperative evolution was satisfactory. A carcinoid tumour of the ileum measuring 1.8 x 1.5cm was