Table 2. Sensitivity of infection-causing bacteria to treatment prescribed

Year	2008	2009 n = 49	2010 n = 43	2011 n = 43 (5 meses)
No. of episodes	28	9	14	5
MRSA	66% sensitive			100% sensitive
MSSA	100% sensitive		100% sensitive	100% sensitive
Corynebacterium	66% sensitive	0% sensitive	Not tested	
S. epidermidis	66% sensitive	20% sensitive	0% sensitive	100% sensitive
Aerococcus	100% sensitive			
Serratia	100% sensitive			
Klebsiella	100% sensitive			
E. coli	100% sensitive	100% sensitive	100% sensitive	100% sensitive
Micrococcus	100% sensitive			
Prov. stuarte	100% sensitive			
Proteus	1 (intermediate)		0% sensitive	
Enterococcus			Not tested	

MRSA: Methicillin-resistant staphylococcus aureus; MSSA: Methicillin-sensitive staphylococcus aureus

None of the patients presented with fungal infections in the exit site or any other effect that was secondary to topical treatment during the study period.

The percentage of gram-negative peritonitis reduced considerably once the protocol had changed to treat the exit site. Gentamicin probably does not influence the incidence of gram-negative peritonitis whose source is intestinal contamination, but it is related with pericatheter contamination.

The percentage of gram-negative infections did not decrease after changing the protocol, but the overall incidence of ESI did. We should point out that a significant percentage of gram-negative ESI were because a patient did not regularly treat the exit site.

During the study period, we did not observe a significant increase in bacteria being resistant to gentamicin, except in the case of *S.epidermidis*, which presented an elevated resistance during 2009-2010. Only one case occurred due to this bacterium in 2011, which was sensitive to said antibiotics.

To conclude, the use of topical gentamicin to treat the peritoneal catheter exit site could be a good therapeutic measure to prevent ESI and peritonitis. Furthermore, in our sample it was not associated with increased resistance during a 29-month follow-up period, or with any other secondary effect.

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Multidisciplinary treatment. A therapeutic option to treat calciphylaxis

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To the Editor,

Calciphylaxis is a rare but important cause of morbidity and mortality in chronic kidney failure patients undergoing renal replacement therapy. Its prevalence is increasing and ranges between 1% and 4% in patients undergoing dialysis.[1,2] It is characterised by ischaemia and cutaneous necrosis secondary to calcification, fibrodysplasia of the intima and thrombosis of small dermo-epidermic arterioles.[1,2]

Its pathogenesis is not very well known, although it is associated with different risk factors such as female sex, obesity, diabetes, metabolic syndrome and calcium and phosphorus disorders.[3,4] Another factor that may favour this disease is the use of coumarin-based anticoagulant drugs, which favour vascular calcification by means of inhibiting g-carboxylation of vitamin K, depending on the matrix protein Gla (protein that inhibits vascular calcification).[1,4]

We present the case of a 55-year-old male with a personal history of primary antiphospholipid syndrome with oral anticoagulant agents since 2003, renal clear cell carcinoma. He had a pacemaker because of an atrioventricular block, severe mitral regurgitation and aortic regurgitation, lymph node tuberculosis and operated right hydrocele. In 1993, he was included in a haemodialysis programme due to chronic renal failure of vascular origin. He received three kidney grafts, the last being in 1997, later presenting with thrombosis, for which he started peritoneal dialysis in March 1998. He was transferred to haemodialysis in November 1999, because of a peritonitis-related sepsis caused by *Pseudomonas*.

He underwent subtotal parathyroidectomy in 2000, due to severe secondary hyperparathyroidism, manifested as elevated parathyroid hormone (PTH) in the biochemical tests that was treated with vitamin D analogues and cinacalcet.

He was admitted in December 2008 for symptoms of heart failure and data showing protein-calorie malnutrition. In the examination, the patient presented with two ulcers on the heel of his left foot. One was approximately 3.5cmx2cm, ulcerated rounded, with red edges, with a necrotic eschar. It was very painful and had preserved distal pulse.

Biochemical tests showed: phosphorus: 4.5mg/dl; corrected calcium: 10.1mg/dl; PTH: 537pg/ml; albumin: 2.2mg/dl; prealbumin: 7.44mg/dl; haemoglobin (Hb) 11.5. *Staphylococcus aureus* and *Clostridium perfringens* were found in the cultures. Given that calciphylaxis was suspected, a skin biopsy was performed, confirming diagnosis.

A multidisciplinary approach was adopted: debridement and treatment of the lesions, starting intravenous antibiotic therapy in accordance with the antibiogram. Calcium-based phosphate binders and vitamin D were withdrawn and cinacalcet dose increased. Given the patient's clinical situation (dyspnoea on minimal efforts and poor tolerance to conventional sessions) and with the aim of intensifying the dialysis dose, long nightly haemodialysis was started on a daily basis. Later, to improve calcaemia control, low-calcium dialysis fluid was used (1.75mmol/l). Once the dialysis session had finished, treatment with 25% sodium thiosulphate (ST) at 25g/l/1.73m^2 was started.

ST treatment was maintained until the lesion was completely cured, which took 9 months of treatment. The only secondary effect was that the patient presented with nausea and vomiting, but this was resolved with prokinetic and antiemetic agents.

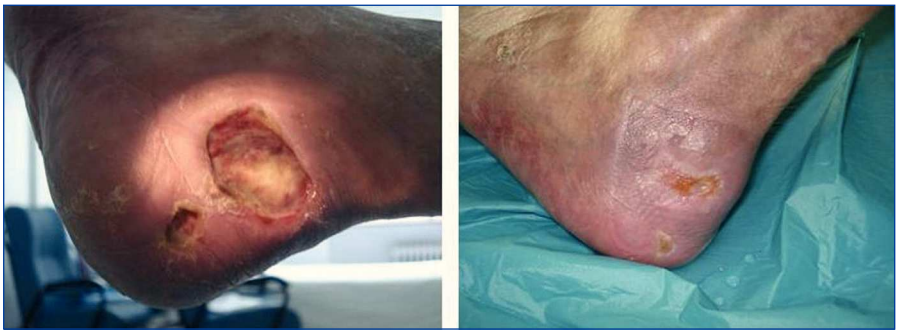

Figure 1. Evolution of the lesion

There is no treatment that is specific for calciphylaxis. Special emphasis is currently made to controlling the calcium/phosphate and hyperparathyroidism products, [4,5] suspending treatment with calcium and vitamin D supplements, increasing dialysis frequency to 5 or 6 weekly sessions, reducing the calcium concentration in the dialysis solution to 1.5-1mEq/l and treating the lesions very carefullly.[1] Also, measures aimed at improving tissue hypoxia were used, such as hyperbaric oxygen therapy.[4,6]

Recently, other treatment lines have become important, such as intravenous ST, an antioxidant that is capable of binding to the nitric oxide synthase enzyme and the calcium binder. In the cases described, ST rapidly alleviated pain but resolved ischaemic ulcers slower.[1-4, 6, 7] Its antioxidant properties correct endothelial dysfunction and favour vasodilatation. Furthermore, together with calcium it forms calcium thiosulphate, which is more soluble than calcium phosphate (present in vascular calcification), which could help eliminate calcium deposits.[1-4, 7] There is no consensus with regards treatment duration but most authors agree that it should be maintained until ulcers have completely healed. Adverse reactions are often mild and includes nausea, vomiting, headaches, nasal discharge and metabolic acidosis.[1] Less common reactions are disorientation, hallucinations, arthralgia, cramps or high blood pressure, among others.[1]

More cases have been reported in the literature, patients treated with biphosphonates whose lesions regressed.[4,7-9] Cinacalcet has also proven to be beneficial. However, current treatment continues to present with poor prognosis, with a mortality rate between 60% and 80%, and sepsis is the most common cause of mortality.[1-4, 7]

From our experience, we believe that multidisciplinary treatment should be used for calciphylaxis.

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C) BRIEF CASE REPORTS

Varicella zoster virus: complications in an ANCA-positive vasculitis

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To the Editor,

Varicella zoster virus (VZV) infection may present with two types of symptoms: the primary infection, known as varicella, with different stages of vesicular lesions mainly on the chest, head and limbs, and subsequent reactivation that causes herpes zoster (HZ). HZ complications are most common in immunocompromised patients with a high incidence in bone marrow transplantation. It may coexist with a visceral dissemination, which is difficult to diagnose if it is not associated with usual skin lesions. Therefore, delayed treatment is associated with high morbidity and mortality, mainly caused by respiratory distress syndrome, fulminant liver failure, pancreatitis or meningoencephalitis.[4,5]

We present the case of a 79-year-old male with antineutrophil cytoplasmic antibodies (p-ANCA). He was diagnosed in 2004 with alveolar haemorrhage and nephritic syndrome treated with corticoids and cyclophosphamide; chronic kidney failure (started haemodialysis in 2006); pulmonary bleeding secondary to a disease flare that was treated with corticoids and cyclophosphamide in 2008; and subsequent maintenance treatment with prednisone at 5mg/day combined with sodium mycophenolate mofetil (MMF) at a dosage of 180mg/12 hours. This treatment is maintained to date. Vesical transurethral resection in 2009 due to vesical neoplasm and atrial fibrillation. His usual treatment was: acetylsalicylic acid 100mg, bisoprolol 2.5mg/24 hours, omeprazole 20mg/24 hours, calcium carbonate 2.5g/24 hours, dacortin 5mg/24 hours and MMF 180mg/24 hours. He was administered Eprex 2000 and weekly Venofer during haemodialysis.

He went to the emergency department with dyspnoea of several days of evolution, coughing and slight expectoration, together with abdominal pain that was variable in intensity. He had nervous fever, but no other symptoms of interest and was sent home. He came to the emergency department again four days later, being referred to our hospital. He had the same clinical symptoms and in the physical examination presented with vesicular lesions limited to the abdomen on dermatomes D9, D10 and D11, which appeared two days later. He had blood pressure of 80/40mm Hg, basal oxygen saturation of 84%, tachypnoea, pain upon deep palpation in the right hypochondrium and intercostal muscle strain. Wheezing could be noted on both sides until the middle fields during auscultation. When he was admitted, the biochemical and radiological data were:

1. Haemoglobin (Hb): 11.7g/dl; leukocytes: 4200 (N: 91.4%, L: 5.3%); platelets: 77 000.
2. Aspartate transaminase (GOT): 6926IIU/l; alanine transaminase (GPT): 3587IIU/l; amylase: 100IIU/l; lactate dehydrogenase (LDH): 1995IIU/l; creatine kinase (CK): 152IIU/l; myoglobin: 708IIU/l; creatinine (Cr): 6.3mg/dl; urea: 88mg/dl; K: 6nmol/l; Na: 144mmol/l; total bilirubin: 2.17mg/dl.
3. pH: 7.21; CHO_3: 12mEq/l; pCO_2: 32mm Hg; pO_2: 59mm Hg.
4. International normalised ratio (INR): 1.7; prothrombin activity: 37%; fibrinogen: 663mg.
5. Chest X-ray: Bilateral interstitial and alveolar pattern with peripheral disposition and cotton-wool like distribution that does not improve following ultrafiltration (Figure 1).

Given that the patient was immunocompromised, and data supported liver failure and acute respiratory failure with skin conditions, VZV was highly probable. We decided to request a serology test for atypical cells; it was VZV-positive, so empirical treatment with acyclovir was started at a dosage of 250mg/12 hours was started, combined with antibiotic treatment (levofloxacin 250mg/48 hours plus cefotaxime 1g/24 hours). HZ diagnosis complicated with visceral dissemination was confirmed

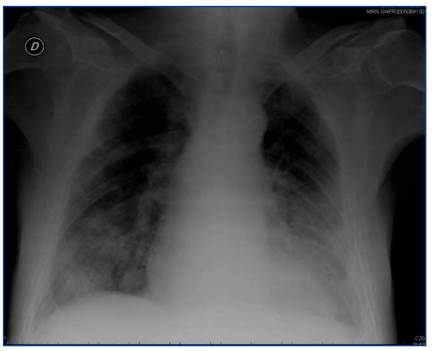

Figure 1. Chest x-ray upon admission