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Assessing resistance to gentamicin following its daily use to treat peritoneal catheter infections

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To the Editor,

Infections continue to be the main problem in peritoneal dialysis (PD). The percentage of gram-positive peritonitis has decreased in recent years as the connection systems have improved. However, gram-negative peritonitis has not changed. Preventing exit site infections (ESI) is crucially important for preventing this type of complication.[1]

There are studies that show that topical gentamicin is more effective than mupirocin at reducing *Pseudomonas* infections, and as effective at reducing *Staphylococcus aureus* infections. Furthermore, there are studies that warn us about mupirocin-resistant *S. aureus* developing.[2]

Topical gentamicin does not have many secondary effects. The most important ones are *Candida* infections, which are generally resolved with oral antifungal treatment with no major consequences.[3] Meanwhile, systemic absorption of topical gentamicin at 0.1% is 2% or less.[4]

We conducted a retrospective study of all types of peritonitis and ESI that occurred in our unit from January 2008 to June 2011.

In January 2009 we decided to change the protocol for treating peritoneal catheter ESI, applying topical gentamicin once a day, with the aim of reducing the incidence of gram-negative peritonitis.

Before changing the protocol, samples were taken from the exudate at the catheter exit site of 44 patients, but no acute infection data were presented. Fourteen percent of the cases were colonised due to a gram-negative bacterium.

Percentages of peritonitis infections were:

In 2008 (51 patients): 33 episodes, 51% gram-positive bacteria, 40% gram-negative and 9% negative culture.

In 2009 (49 patients): 32 episodes, 71% gram-positive bacteria, 22% gram-negative and 8% negative culture.

In 2010 (43 patients): 24 episodes, 58% gram-positive, 29% gram-negative and 13% negative culture.

In 2011 (43 patients), 5-month follow-up: 11 episodes, 90% gram-positive and 10% gram-negative.

The percentages and bacteria responsible for ESI are shown in Table 1.

We assessed the gentamicin-sensitivity of bacteria responsible for ESI during the study period. The results are shown in Table 2.

Table 1. Evolution of bacteria causing exit site infections

Year	2008	2009	2010	2011
	n = 51	n = 49	n = 43	n = 43 (5 months)
No. of episodes	28	9	14	5
Gram-positives	20 (71%)	6 (66%)	11 (78%)	3 (60%)
Gram-negatives	7 (29%)	3 (34%)	3 (22%)	1 (40%)
MRSA	3			1
MSSA	6		4	1
Corynebacterium	6	1	3	
S. epidermidis	3	5	3	1
Aerococcus	1			
Serratia	3			
Klebsiella	1			
E. coli	1	3	1	1
Micrococcus	1			
Prov. stuarte	1			
Proteus	1		2	
Enterococcus			1	

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

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*.