on medication prescribed *de novo* to the patient is not considered or is missing. It is not ridiculous that the first case was considered to be alcohol poisoning and the second case the symptoms were thought to be secondary to uraemia. With respect to the second case, severe poisoning has been reported with low doses of baclofen [6] and it is even considered a contraindication for these patients. Although clinical symptoms vary greatly, myoclonus twitching/convulsions and mental confusion is reported. In our hospital, patients are constantly reviewed every 2 days by the dialysis staff. Furthermore, the diagnostic and therapeutic value of this treatment is considered, as occurred in both of these cases. Improvement was especially spectacular and sustained in the second case, once the drug was supposedly withdrawn by the end of the session.

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Effect of fluorescein on renal function among diabetic patients

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To the Editor,

One of the most important complications of diabetes is retinopathy. Intravenous fluorescein angiography has been widely used for evaluation of diabetic retinopathy. Although numerous reports have been published about the iodinated contrast media induced nephropathy[1-3], there is a few researches about renal injury secondary to fluorescein (as a noniodinated contrast media)[4]. In this investigation, we have been tried to evaluate effect of fluorescein sodium on the renal function among diabetic patients who have more susceptible to the renal injury compared with general population[5].

This study was conducted on diabetic patients undergoing fluorescein angiography to assess retinopathy at the Department of Ophthalmology, Imam Khomeini Hospital, Ahvaz, Iran in 2006. Exclusion criteria were pregnancy, lactation, having received contrast media within 7 days of study entry, acute renal failure, endstage renal disease requiring dialysis, history of hypersensitivity reaction to contrast media, parenteral use of diuretics, and use or start of nonsteroidal anti-inflammatory drugs or angitansine receptor binding, or angiotansine converting enzyme inhibitor within 48 h of the procedure. The protocol was approved by the Ahvaz Jundishapur University of Medical Sciences. All patients provided informed, written consent. Upon fluorescein angiography, 500 mg sodium fluorescein solution was injected into the antecubital vein over 5 seconds. Serum creatinine (SCr) was measured before and on days 2 and 3 after the angiography. Renal injury was defined as a relative increase in SCr from the baseline of $\geq$25% or an absolute increase of $\geq$0.5 mg/dl during days 2 and 3. Data was analyzed by SPSS software, version 13. All data are presented as percentages or as mean ± standard deviation. The paired Student's t test was used to compare SCr between various groups; and all p values <0.05 were considered statistically significant.

A total of 44 diabetic patients (22 male and 22 female) met the inclusion criteria and were studied; mean age of participants was 53.1 ± 9.2 years; range 30-72 years (male, 51.8 ± 9.5 and female, 54.3 ± 9.0; p = 0.38). Mean of SCr before fluorescein angiography was 1.09 ± 0.07 mg/dl (male, 1.13 ± 0.56 and female, 1.05 ± 0.40; p = 0.60), and after angiography was 1.16 ± 0.08 mg/dl (male, 1.23 ± 0.62 and female, 1.11 ± 0.50; p = 0.49). Nine patients (20.5%) had an increase in SCr from baseline within 72 hours of fluorescein administration (7 male and 2 female). In the present study, we did not observe any significant adverse effects after fluorescein usage.

Although, Kameda and colleagues use the estimated glomerular filtration rate to show renal injury secondary to fluorescein sodium and did not find any hardly effects on renal function[4], but current study demonstrated that fluorescein could cause to renal injury in diabetic patients following angiography. Because lack of enough data, prospective studies will be required to determine whether fluorescein angiography is associated with higher incidence of adverse effects on renal function especially in diabetic patients.

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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