**Table 1.** Evolution of metformin levels

| Evolution | Metformin levels (mg/l) |
|---|---|
| Upon admission (predialysis) | 34.4 |
| 24 hours | 21.3 |
| 48 hours | 10.2 |
| 96 hours | 4.8 |

deteriorate. We therefore decided to perform her first 2-hour haemodialysis session without ultrafiltration. Having confirmed hyperlactacidaemia (10.7mmol/l), high metformin levels (34.4mg/l; therapeutic levels 1.3-5) and symptoms of heart overload with haemodynamic disorder, we decided to perform dialysis for four days and then every 48 hours until reaching a constant lactate decrease and non-toxic levels of metformin (Table 1). She received 7 sessions in total. She received empirical antibiotic therapy with third-generation cephalosporin; the urine and faecal cultures were negative.

She was discharged without any neurological and renal symptoms, with creatinine at 1.6mg/dl and the following treatment: carvedilol at a dosage of 6.25mg/24hrs, repaglinide at a dosage of 1.5mg/8hrs, telmisartan, atorvastatin, torsemide at a dosage of 10mg/24hrs and omeprazole at 20mg/24hours.

She currently presents with 1.26mg/dl creatinine and is neurologically stable.


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## Warning against unexpected medication in haemodialysis


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**To the Editor,**

As nephrologists we are used to having to be extremely careful with the medications given to advanced kidney failure patients. However, our patients tend to have multiple disorders, meaning that we must obtain second opinions from other specialists. There are different types of consultations, and often very complex patients have a reduced visit time. Drugs are prescribed, sometimes correctly, but on other occasions without considering the degree of the renal function. It is our responsibility to supervise the dose of these drugs that are added to their usual medication, to prevent any surprises from occurring, such as the ones that we are to describe.

Seventy-eight year old male, diagnosed with end-stage kidney failure, undergoing regular haemodialysis. He arrived at the emergency department because of motor discoordination. Neurologically, he presented with ataxia, motor aphasia and visual hallucinations. Five days before, he presented pain in his left side, and erythematous and vesicular lesions in the same area. We prescribed acyclovir at a dosage of 400mg, and subsequent dosages of 200mg every 24 hours. The patient incorrectly ingested 400mg every 8 hours. After receiving a haemodialysis session, he improved rapidly and was discharged the following day. Alcohol poisoning was first suspected in the emergency department, but was dismissed given that ethanol levels were zero.

Seventy-five year old male undergoing regular haemodialysis for diabetic nephropathy, but who needed daily haemodialysis due to intense cramps, especially during the sessions. Suffering from polyneuritis, a specialist visit was arranged and he was prescribed treatment with baclofen (muscle relaxant). Two days after starting the treatment, he arrived at the emergency department presenting with intense tremors. We observed greatly intense fixed miotic and myoclonic pupils, which initially ceased with clonazepam. He then underwent dialysis and his pupils improved. He was discharged in 24 hours. The first diagnostic suspicion at the emergency department was uraemic myoclonus.

With these two cases, which are repeatedly referred to in the literature[1-6] and reported in our journal NEFROLOGÍA,[3,4] we aim to remind readers of how easily our patients become intoxicated and how difficult it is to reach a diagnosis in the emergency department. Unnecessary examinations are required (cranial computerised tomography, etc.) if data

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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