Table 1 – Coagulation study performed with Sysmex CS-5100 automated analyser, with Pathromtin® reagent.

	Case 1	Case 2	Case 3	Case 4	Normal range
aPTT; s	90.3	64.3	86.6	71.3	25–38
aPTT; ratio	2.91	2.07	2.89	2.38	0.5–1.2
Prothrombin time; s	13.7	15.7	12.5	11.8	
Quick ratio; %	79	61	83	100	70–100
INR	1.1	1.3	1.1	1	1–1.3
Clotting factors; %					
Factor II	84.6	NA	99	110	70–120
Factor V	155.9		184	118	70–120
Factor VII	197.1		185	166	55–170
Factor VIII	285.0		315	314	60–150
Factor IX	241.9		182	219	60–150
Factor X	121.5		115	118	70–120
Factor XI	186.3		118	210	60–150
Factor XII	127.3		171	171	60–150
Von Willebrand factor; %					
Activity	249.7	NA	349	192	40–150
Antigen	271.2		482	178	45–150
Platelet function; s					
Collagen/adrenaline	101	NA	170	150	85–165
Collagen/ADP	71		104	129	71–118

ADP: adenosine diphosphate; aPTT: activated partial thromboplastin time; INR: international normalised ratio; NA: not available.

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Skin reactions with the use of icodextrin in peritoneal dialysis patients☆

Reacciones cutáneas debido al uso de icodextrina en pacientes en diálisis peritoneal

Dear Editor,

Icodextrin is a starch-derived glucose polymer which is metabolised into maltose. Its use in peritoneal dialysis (PD) has been an alternative to glucose as an osmotic agent, obtaining greater ultrafiltration (UF) for long dwells.

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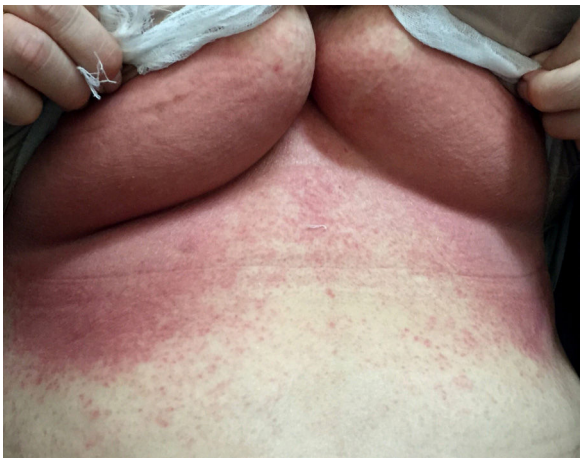

Fig. 1 – Inframammary folds.

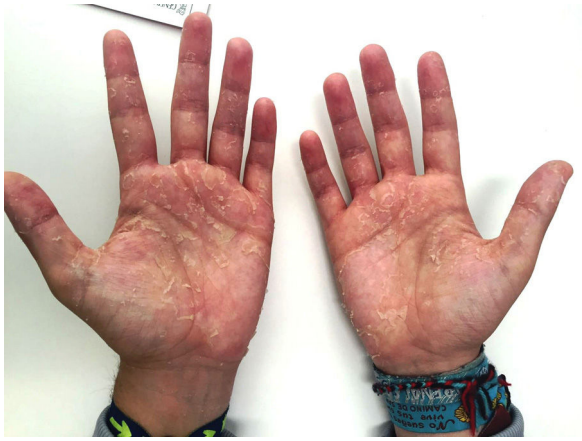

Fig. 2 – Palms of the hands.

Icodextrin is generally well tolerated by patients, but skin reactions associated with its use have been reported. We present our experience with two patients who had skin reactions to icodextrin.

First case: a 73-year-old woman with chronic renal failure secondary to interstitial nephritis due to recurrent urinary tract infections. She had a history of severe toxicoderma caused by furosemide. She started PD in November 2013. After two years on PD, icodextrin was added to increase the UF. Seven days later, the patient consulted with generalised pruritus, poor general condition, sensation of dysthermia and the development of confluent desquamating maculopapular exanthema, mainly in inframammary folds (Fig. 1). She had no limb involvement or mucosal lesions. The patient was diagnosed with drug-induced toxicity and it was decided to withdraw the icodextrin, replacing it with 2.3% glucose. Dermatology performed epicutaneous allergy tests on icodextrin which came back negative. Seven days after the withdrawal, there was significant improvement of the lesions on her trunk, but still residual areas of desquamation on the palms of her hands. Fourteen days after discontinuing icodextrin the patient was asymptomatic.

Second case: a 26-year-old man with no known allergies with renal failure due to episode of thrombotic microangiopathy secondary to methylmalonic acidaemia. In April 2016, he started PD from haemodialysis with three daytime 1.27% glucose exchanges and one nocturnal icodextrin exchange. After three weeks, the patient consulted with generalised pruritus, predominantly at night and the development of erythematous-desquamative lesions on the palms of his hands (Fig. 2) and soles of his feet, with no mucosal involvement. He was referred to dermatology and was diagnosed with drug-induced toxicoderma in the post-critical phase. In view of the possible relationship between the lesions and the use of icodextrin, it was discontinued. Fifteen days after withdrawal, the pruritus and skin lesions had disappeared.

Skin involvement associated with the use of icodextrin can present in many different ways; from localised pruritus and small desquamative areas on palms and soles[1] to generalised pruritus and exfoliative lesions affecting the trunk and extremities, which can be potentially serious.[2] As a result of the diverse forms of presentation, the incidence of icodextrin-induced skin lesions ranges from 7.8%[3] to 17%[4,5] according to the different series reported.

The mechanism by which icodextrin causes cutaneous toxicity is not fully understood. There are suggestions that it may be the result of a type IV hypersensitivity reaction, similar to that caused by the use of dextran solutions, given that the chemical composition of both polymers is very similar.[1,4] The skin reaction usually occurs shortly after starting treatment with icodextrin, and it disappears rapidly after discontinuation.[1,3,4] In most situations[1,2,4] withdrawal of icodextrin is necessary for complete resolution of the condition. However, where the reaction was mild, the lesions are reported to have disappeared without the need to discontinue icodextrin.[3]

Despite the limitations in our two cases, as both lacked a skin biopsy, we think that nephrologists need to be aware of the risk of skin reactions associated with the use of icodextrin and their form of presentation in order to suspect them should they occur in PD patients.

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Acute kidney disease related to leptospirosis☆

Fracaso renal agudo asociado a leptospirosis

Dear Editor,

Leptospirosis is a zoonotic disease, which occurs all over the world and is related to direct or indirect contact of humans with the urine of infected animals.[1] The clinical manifestations are highly variable, from flu-like symptoms to very serious forms (Weil's disease with liver, kidney and meningeal involvement and alterations in haemostasis).[1–3] Leptospirosis causes acute interstitial nephritis which often presents either as asymptomatic urinary abnormalities (mild proteinuria, microhaematuria); acute kidney injury (AKI), which is typically non-oliguric and hypokalaemic, like a haemolytic uraemic syndrome (HUS); or chronic kidney disease (CKD) of unknown aetiology.[4] Although the diagnosis of leptospirosis is not common, severe cases with AKI are the main reason for requiring nephrology treatment. For that reason, we carried out a retrospective descriptive study of all the cases of leptospirosis diagnosed in our hospital in the period 2007–2017. Our study had the approval of the Continuing Education, Teaching and Research Commission. The immunodiagnostic techniques used for the screening were serum detection of total antibodies by indirect haemagglutination and/or IgM antibodies by ELISA, with positive cases confirmed by microscopic agglutination test (MAT) in serum and/or detection of *Leptospira* sp. DNA in urine by real-time PCR. We used the diagnostic criteria of the Centers for Disease Control and Prevention (CDC), classifying the cases as confirmed or probable. In total, 10 cases of leptospirosis were diagnosed (8 men and 2 women). Table 1 shows the clinical-analytical data of the patients studied, the possible sources of infection and the form of presentation. Six of the eight males had AKI. Of these, only two required haemodialysis; both were transferred to intensive care for inotropic support and assisted ventilation, these two being the cases with the highest levels of serum bilirubin. All patients survived. Eight patients had suggestive epidemiological factors (some more than one factor) and in two cases they were not known. In terms of clinical presentation, two presented with flu-like symptoms, two with Weil's disease and six with atypical manifestations. Table 2 shows the microbiological diagnostic techniques used and the titres of the different varieties. Of the 10 patients studied, four met diagnostic criteria, with an antibody titre by MAT >1:800 and/or detection of *Leptospira* DNA in urine by real-time PCR. The remaining six patients had positive screening for indirect haemagglutination and/or IgM ELISA without diagnostic confirmation criteria. MAT is the "gold standard" in the immunodiagnosis of leptospirosis due to its specificity (serovariety/serogroup). As MAT requires isolation of the microorganism by culture and experience in the isolation, it is performed in reference laboratories and is therefore not always available. The serovarieties was *icterohaemorrhagiae* (rats) in three of the cases determined by MAT. Leptospirosis-associated AKI occurs in 44–66% of affected patients,[5] and our series was 60% which is n the same line with that range with developing AKI. According to the KDIGO (Kidney Disease: Improving Global Outcomes) guidelines for AKI,[6] four had stage 3 and two, stage 2. As in this series, most cases of AKI are associated with jaundice.[4] Although the most common is non-oliguric AKI, in cases associated with jaundice and/or hypotension, oliguric or anuric AKI can occur and if necessary, both haemodialysis and peritoneal dialysis can be given. Most patients recover renal function, but in some patients a degree of renal failure may persist and progress to CKD. In a recent article in which renal biopsy was performed after recovery of the patient with antibiotics, inflammatory infiltration was demonstrated in both the tubules and in the interstitium, where most of the cells were monocytes or CD68-positive macrophages predominantly of the M1 phenotype.[7] After treatment with oral corticosteroids, the patient recovered renal function, which is why the authors suggest that corticosteroids may be a therapeutic option for some patients with sustained tubulointerstitial nephritis after surviving severe leptospirosis.[7]

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