Table 1. Increased kidney volume in patients assigned to the placebo group in large observational studies and clinical trials and in the patient reported

	Increase in total kidney volume	Decrease in eGFR
Grantham (initial volume >1500ml and =30 years)[12]	144ml/year, 6.7%/year	-5.04mL/min/year
Torres[5]	4.4%/year if initial volume <1500mL 6.7%/year if initial volume >1500mL	-3.81mg/mL/year
Walz[7]	319mL/2 years (159mL/year)	-3.8mL/min/year
Serra[8]	140mL/18 months (93mL/year)	-2.33mL/min/year
Caroli[10]	460mL/3 years (153mL/year)	-4.95mL/min/year
This case	1938mL/12 months, 70.6%/year	-13.2mL/min/year

not a definitive observation. In fact, it may be argued that the existence of a systemic inflammatory disease could have contributed to accelerating the disease. Nevertheless, anti-TNF treatment was effective in controlling the activity of ankylosing spondylitis.

In conclusion, we reported the case of a patient with ADPKD treated with anti-TNF therapy due to a concomitant rheumatic disease, whose kidney disease progressed quickly. This case report argues against the efficacy of anti-TNF therapies for treating the human form of ADPKD.

Conflicts of interest

The authors declare that they have no conflicts of interest related to the contents of this article.

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Non-infectious cloudy peritoneal fluid secondary to lercanidipine

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To the Editor,

It is well known that certain calcium channel blockers (CCB) can cause cloudy peritoneal fluid not associated to infection (NICPF).

We report what we believe to be the first case of cloudy peritoneal fluid in relation to lercanidipine identified in our country and possibly in Europe.

CASE REPORT

The patient was a 59-year-old Caucasian female with chronic renal failure secondary to mesangial proliferative glomerulonephritis. She had high blood pressure and was on treatment with 320mg/day valsartan

and 4mg/day doxazosin and was on peritoneal dialysis. She presented with cloudy peritoneal fluid. She did not complain of abdominal pain, fever or any other abdominal symptoms. Three days previously, 5mg/day lercanidipine had been added to her treatment. The appearance of the peritoneal fluid was milky and it did not contain fibrin. The cell count in the dialysate and the subsequent culture were negative. Plasma values for cholesterol and triglycerides (TG) were 189 and 175mg/dl, respectively and the dialysate value for TG was 20mg. Having ruled out bacterial peritonitis and given the potential relationship with lercanidipine, this drug was discontinued and 24 hours later, clear dialysate drainage was confirmed.

A few weeks later, the patient changed to automated peritoneal dialysis with a dry day, and with her consent, 5mg/day lercanidipine was reintroduced into her night dose.

One month later, she presented again with cloudy fluid without associated symptoms. Again, the cell count and culture of the dialysate were negative. On this occasion, TG was not measured in dialysate, but its plasma value was 145mg/dl (lower than in the previous episode). With the diagnosis of NICPF related to CCB, lercanidipine was discontinued. Again, the dialysate became clear over the next few hours.

DISCUSSION

NICPF is uncommon in peritoneal dialysis and its occurrence obliges us to carry out a differential diagnosis that must distinguish between clinical profiles with an increase in cellularity and acellular causes[1,2]. The absence of cells rules out infection, inflammation, allergic reaction and bleeding. Fibrin, TG and drugs have been reported as the most common causes of acellular NICPF

CCB are amongst the drugs included. The oldest case was published in 1993 in relation to manidipine[3], the drug most commonly involved, although other CCB, most of them dihydropyridines, can also trigger it[4]. Since the appearance of new CCB, cases of secondary NICPF have been published fairly frequently.[5,6]. Curiously, classic types of CCB do not cause it or do so less frequently[7]. This has led us to believe that the characteristics of the different CCB may influence the occurrence of NICPF.

CCB-induced NICPF is defined as[4]: cloudiness of the dialysate that appears 48-72 hours after the administration of the drug, with the absence of peritoneal inflammatory signs and symptoms and a negative cell count and peritoneal fluid culture, which disappears spontaneously after the drug is discontinued. Unlike infectious peritonitis, some authors have noted an increase in ultrafiltration during the episode[8].

The cloudiness of the peritoneal fluid, which is characteristically milky, is due to the presence of TG (normally almost non-existent in the dialysate if there is no other cause), although they do not always reach chyloperitoneum levels (>100mg/dl). The mechanism whereby TG increase is still unknown, although there has been speculation that it may be due to a disorder of its degradation in the peritoneum or the reduction of lymphatic stomata in the diaphragmatic peritoneum[3,8]. It has been noted that TG in dialysate seem to have a direct relationship with TG levels in blood[9]. Lercanidipine acts both in the smooth muscle cells of blood vessels and of the lymphatic system and the digestive tract, which may explain its effect[9].

Reports of NICPF secondary to lercanidipine have a curious geographic distribution: the few cases reported have occurred in Asia[8-10], and the closest to Europe are located in the Asian region of Turkey[11]. Although it has been much used in Europe for years, we have not found reported cases of NICPF related to its use (in contrast to the high incidence of cases related to manidipine). This has made us consider that racial factors or genetic predisposition may be involved[8,9].

Apart from the unsettling effect, the presence of NICPF does not seem to have any negative clinical repercussions. In fact, Yang reported two patients who remained on lercanidipine in whom the cloudiness gradually disappeared[9].

In our case, all the data support our diagnosis of NICPF secondary to lercanidipine. In the second episode, the delay in the reappearance of peritoneal cloudiness was surprising, which we thought could be related to the absence of dialysate in the peritoneal cavity during the daytime.

In summary, in the presence of acellular NICPF, we must rule out the potential relationship with CCB in order to avoid unnecessary antibiotic treatment.

Conflicts of interest

The authors declare that they have no conflicts of interest related to the contents of this article.

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The importance of vascular access for haemodialysis in Hallopeau-Siemens dystrophic epidermolysis bullosa

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To the Editor,

Bullous or ampullary epidermolysis is defined[1] as a group of rare inherited skin diseases characterised by a tendency of the skin and mucous membranes to separate from the underlying tissues after minimal trauma; it is transmitted as both dominant and recessive factors and is caused by COL7A1[2] gene mutations: The different varieties can be grouped into three main[1,3] types: epidermolysis bullosa simplex of dominant autosomal inheritance, junctional epidermolysis bullosa of autosomal recessive inheritance and dystrophic or dermolytic epidermolysis bullosa. Furthermore, the latter includes three forms: dominant, mild recessive, and severe recessive or Hallopeau-Siemens.

Hallopeau-Siemens dystrophic epidermolysis bullosa is the most widely severe form[4,5]. It begins at birth with blistering of skin and mucous membranes. Blisters and vesicles heal forming atrophic scars. Extracutaneous[4] manifestations include: dystrophy or absence of nails, sparse head hair or alopecia, excessive dental caries, microstomia, mouth ulcers, lingual tip fixation to the floor of the oral cavity, pseudosyndactylia, syndactylia and deformities of hands and feet. Furthermore the surface epithelium of the respiratory tract, gastrointestinal tract and ocular system are affected by blistering. These patients usually suffer from anaemia, malnutrition and stunting. One of the major complications of the disease is the development of chronic terminal kidney disease[3], which causes early death and with the main complication of establishing an appropriate access route for dialysis.

CASE REPORT

A 31-year-old female patient was referred to dermatology for evaluation of renal function after having been diagnosed with epidermolysis bullosa at birth and dysphagia for solid food that did not require endoscopic treatment. The endocrinology department ruled out associated hypothyroidism and suprarenal insufficiency. No previous diagnosis of chronic kidney disease in the last year, she was referred due to azotaemia. On admission: weight 45kg, height 1.55m, cutaneous pallor +, partial alopecia, a few scattered haematic crusts in the oral cavity, microstomia with lingual fixation to floor of oral cavity, generalised erythema, dermabrasion areas with blistering that did not affect the cervical region, deformities of both hands and bilateral scars and erythema (Figure 1).

Relevant laboratory abnormalities: haemoglobin 9.1g/dl, K 7.2mmol/l, urea 345mg/dl, creatinine 9.5 mg/dl, albumin 2g/dl; Urinalysis (GUE) urinary density (UD) 1009, pH 5.5, proteins +, erythrocytes 150 cells/ul; arterial blood gases: pH 7.21, PCO2 27, PO2 92%, 92% Sat, HCO3 7.6; 24h proteinuria 1.8g/day; urine volume: 1500cc; right kidney ultrasound 7.5 x 2.5cm and left kidney 7.1 x 2.6cm, loss of corticomedullary ratio. Electrocardiogram symmetrical peaked T waves and isolated PVCs in all precordial leads. We decided to place an indwelling tunnelled catheter due to the increased risk of infection with the Tenckhoff catheter.

Subsequent to cardiac monitoring, monitoring of non-invasive blood pressure and pulse oximetry, intravenous sedation and analgesia

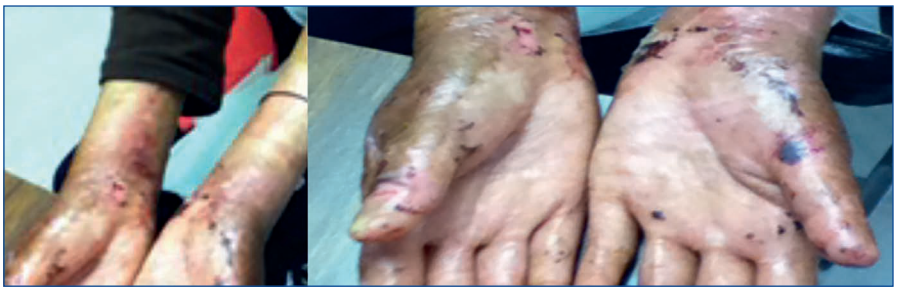

Figure 1. Epidermolysis bullosa dermatological lesions seen on patient with chronic kidney disease on admittance.