Clinical symptoms of central AI can be non-specific: fatigue, nausea, abdominal pain, hypotension, hypoglycemia, can be seen[8]. AI often goes undetected when not specifically thought of.

The clinical and analytical response of a patient with central AI started on GC therapy is dramatic. Negative GC feedback on parvocelular AVP secretion rapidly inhibits its secretion, and marked aquaresis ensues, with a risk for overcorrection of SNa during the first 24 to 48 hours of GC treatment[9]. Given the risk for overcorrection inducing Osmotic Demyelination Syndrome, patients' rise in SNa must be closely monitored during initial treament.

We present this case to highlight the importance of considering central AI in patients whose chronic CG therapy has been discontinued, and to underline the fact that other causes of euvolemic hyponatremia must be ruled out before establishing a diagnosis of SIADH.

Conflicts of interest

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

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Hypersensitivity to Synthetic Hemodialysis Membranes

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To the Editor,

With the development of synthetic membranes and the fact that ethylene oxide is no longer used as a sterilizing agent, hypersensitivity reactions in haemodialysis have decreased considerably. However, they still exist. We describe a particular case from which we learnt some interesting things.

Ours was an 86-year-old male patient, smoker, on haemodialysis due to chronic interstitial nephropathy, with hypertension, ischaemic heart disease, type 2 diabetes mellitus, chronic obstructive pulmonary disease (COPD) and dyslipidaemia. He was on treatment with amlodipine, doxazosin, omeprazole, saxagliptin, pravastatin, paricalcitol, and beta 2 combined with inhaled steroids and during dialysis intravenous iron sucrose (Feriv®) and alpha erythropoietin (Eprex®). With no known history of allergies, urticaria or eosinophilia. He began haemodialysis during an episode of overall cardiorespiratory failure due to COPD decompensation with haemodynamic instability, volume overload and hypotension. He required treatment with steroids. He was dialysed with ultrapure water through a Poliflux21H® dialyser (polyamide polymer combination, polyarylethersulfone and high permeability polyvinylpyrrolidone, heat sterilised) and for one month suffered hypotension and clinical angina during dialysis. Once he had overcome these symptoms, steroid treatment was suspended and a week later, five minutes after connection, he suffered severe bronchospasm and hypotension with a generalised burning sensation, which responded to the administration of expanders, high flow oxygen, inhaled bronchodilators, antihistamines and intravenous steroids; it was not necessary to interrupt dialysis. During the next three dialysis sessions he suffered a repetition of these symptoms. No eosinophilia was seen. Antiheparin antibodies were negative. Dialysis fluid culture and endotoxins were negative. IgE was normal. No contact with ethylene oxide in any material. No thrombocytopaenia. In the following session, he was pre-treated with dexchlorpheniramine and methylprednisolone and the dialyser was replaced by NEPHRAL® (polyacrylonitrile AN 69 sterilised by gamma rays). The symptoms did not reappear, so prophylaxis was stopped after a month. During the next month, after six months on the programme, he

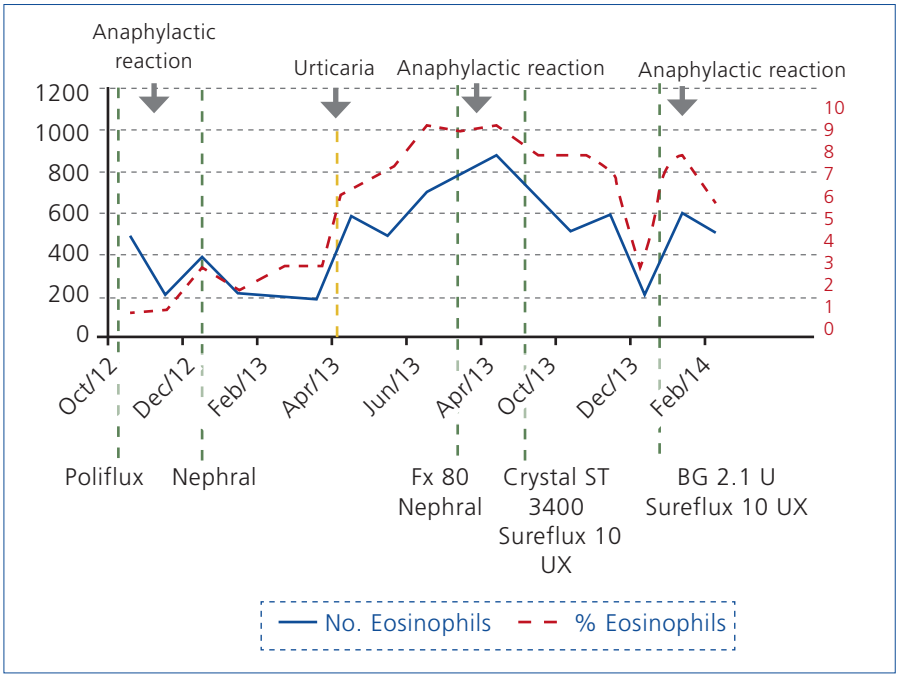

Figure 1. Analytical evolution.

developed generalised urticaria, and peripheral eosinophilia. At different stages his medication was changed without any clinical or analytical improvement. The dialyser was changed to a FX80M® (heat sterilised helixone) and hypersensitivity reappeared in the first session. Since then, again with NEPHRAL®, which was well tolerated, but urticaria and eosinophilia became constant. At 12 months, in a single session, a plate dialyser Crystal ST 3400® (polyacrylonitrile AN 69 without polyvinylpyrrolidone [PVP] as amalgamating agent) was used, and isolated severe hypotension reappeared without respiratory symptoms, explained by the high surface area of the dialyser, so in the next session we used Sureflux 19 UX® (cellulose triacetate sterilised by gamma rays), and we continued with this dialyser for five months. Pruritus partially disappeared and eosinophilia decreased (Figure 1). After 15 months, we used a 2.1 U® (polymethylmethacrylate [PMMA] sterilised by gamma rays) dialyser, resulting in new hypersensitivity reaction within minutes of connection. Since then, this patient has been dialysed with Sureflux 19® and has suffered no new episodes of hypersensitivity.

The patient had a type A[1] anaphylactoid reaction related to polyamide, helixone and PMMA membranes. Urticaria and eosinophilia with polyacrylonitrile. Haemodynamic instability with polyacrylonitrile on plates without PVP and good tolerance to cellulose triacetate membranes.

In the literature, there are examples of anaphylactic and anaphylactoid reactions or tolerance to different types of synthetic membranes in the same patient[2-7], which are attributed to differences in the manufacturing of these membranes and the amount of PVP used to hydrophilise the membranes, but in none of these cases a change of dialyser lead to the disappearance of symptoms and the development of chronic urticaria with eosinophilia. In this regard, the absence of anaphylactoid reaction when using a polysulphone dialyser with plates with no PVP as amalgamating agent suggests a significant role of this product as the cause of the patient's disorders. Other causes that should be assessed are endotoxin backfiltration in highly permeable membranes (absent in low permeability membranes), hypersensitivity to intravenous iron or the use of angiotensin-converting enzyme (ACE) inhibitors. We do not believe this to be the case, since there were no reactions with high-flux dialysers that did not have PVP as an amalgamating agent. Cultures ruled out the presence of endotoxins, no ACE inhibitors were used and the modification or suspension of intravenous iron did not cause clinical changes. It is necessary to take into account the severity of these reactions, which did not always occur at the beginning of the session, thus decreasing clinical suspicion. This case illustrates a gradation in clinical manifestations, a variation on previously published cases, and provides new information on the effectiveness of cellulose triacetate membranes in intolerance to other synthetic membranes.

Conflicts of interest

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

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Acute renal failure with low complement levels: check for tubulointerstitial nephritis

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To the Editor,

A 75 year-old male came to the emergency department due to epigastralgia of five days of evolution. Two weeks before, he had received non-steroidal anti-inflammatory drugs (NSAIDs), due to a trauma of his right wrist, which he had to suspend due to the digestive symptoms described above.

As personal history of significance, he had undergone a radical prostatectomy for a tumour and suffered sphenoid sinus aspergillosis in October 2011.

- Physical examination: blood pressure 195/77mmHg; no other relevant findings.
- Laboratory tests showed: creatinine: 5.38mg/dL, urea: 116mg/dL, amylase: 113IU/L, sodium: 136mEq/L, potassium: 4.7mEq/L, normal blood count.
- Urine analysis: leukocyturia.
- Complement: **C3: 39** (85-180), **C4: <1.4** (10-40).
- Immunoglobulins: IgG: 2460 (680-1530), IgG4: 72 (9-104), IgM: 36 (40-240), IgA: 127 (70-400).
- Antinuclear antibodies, anti-DNA antibodies, ANCA, anti-RO antibodies, anti-La antibodies: negative.
- Cryoglobulins: negative.
- Angiotensin-converting enzyme: 30 (8-50).
- Hepatitis B, Hepatitis C, and human immunodeficiency virus serology: negative.
- Protein in 24-hour urine: 0.5 grams.
- Blood immunoelectrophoresis: normal.
- Urine immunoelectrophoresis: light precipitation band in kappa light-chain.
- Bone marrow: normal.
- Upper digestive endoscopy and colonoscopy were normal.
- Abdominal ultrasound, chest and abdomen X-ray: nothing of note.

Initially, it was interpreted as tubulointerstitial nephritis (TIN) related to NSAIDs. Finding low complement values expanded our differential diagnosis and included suspected lupus nephritis, mixed cryoglobulinaemia, post-infectious and membranoproliferative glomerulonephritis. So we performed a renal biopsy (Figures 1 and 2): renal cylinder with six glomeruli without significant alterations. Inflammatory interstitial lymphocyte infiltrate with few plasma cells. On direct immunofluorescence, a granular deposit of immunoglobulin G and C3 is seen in the tubular baseline membrane and in Bowman's capsule of some glomeruli.

This finding led us to consider a differential diagnosis with systemic lupus erythematosus (SLE), Sjogren, TIN related to Ig4 and idiopathic TIN.

This last term covers three separate entities: TINU (tubulointerstitial nephritis and uveitis), nephritis due to baseline membrane antibodies and low complement TIN. SLE is ruled out due to the absence of antibodies and negative symptoms, Sjogren's syndrome is also ruled out since there is no xerostomia or xerophthalmia, and antibodies are negative.

IgG4-related TIN, name proposed by Saeki et al, is included in a recently described syndrome known as IgG4-related autoimmune disease of unknown origin. It is characterised by elevated plasma IgG4 associated with lymphocyte and plasma cell infiltrates of any organ with a predominance of IgG4-positive plasma cells. In the kidney, TIN is the most characteristic finding with low complement in a high percentage of cases.

We ruled out this entity as IgG4 levels were not high and no other organ was involved.

The patient was diagnosed with low complement TIN. It is a rare entity of unknown pathogenesis. There are several hypotheses regarding the formation of in situ or preformed circulating immune-complex deposits. This disorder usually affects males with a mean age at presentation of 66 years. The most

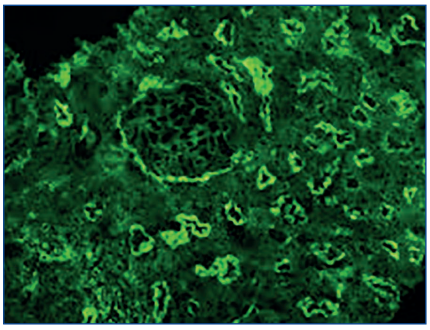

Figure 1. Deposits of IgG and C3 in the baseline membranes of the tubules and in Bowman's capsule with intact glomeruli.

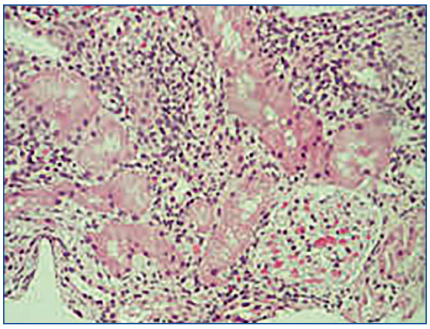

Figure 2. Interstitial infiltrate with healthy glomeruli.