deficit of CD19-B in our patient; however, this was not the case of other immunoglobulins such as IgE, which promote immediate mast cell degranulation when coming into contact with the antigenic factor[7,8]; and 3) there was a tendency to develop infections and autoimmune mechanisms in these patients, where the uncontrolled external antigenaemia by IgA dimers was capable of inducing a direct cytotoxic response or a response mediated by Ag-Ac complexes.[6]

In this area of immunopathology, the proposed treatment was directed primarily at inhibiting the reaction of the extracorporeal blood-circuit interface, for which clinical experiences in the literature on pH-metric modulation of intradialytic anaphylaxis were reviewed.[9] This was used to establish a screening protocol for patients with 1/6M-bicarbonate bloodlines (prior to connection during each session) resulting from the onset in an alkalising effect that resolved the episodes of angioedema and haemodynamic instability. Secondly, we also adopted other therapeutic and hygiene measures for the prevention of infectious complications in the patient, such as the creation of an arteriovenous fistula, which was preferable over the central venous catheter that was being used, or even the early administration of antibiotic therapy in the case of respiratory infection. However, we did not consider correcting the IgA deficiency with exogenous immunotherapy, as severe cases of HPS secondary to the formation of anti-IgA antibodies have been described.[10]

Conflicts of interest

The authors declare no conflict of interest related to the content of this article.

1. International Union of Immunological Societies Expert Committee on Primary Immunodeficiencies, Notarangelo LD, Fischer A, Geha RS, Casanova JL, Chapel H, et al. Primary immunodeficiencies: 2009 update. J Allergy Clin Immunol 2009;124(6):1161-78.
2. Yel L. Selective IgA deficiency. J Clin Immunol 2010;30(1):10-6.
3. Ebo DG, Bosmans JL, Couttenye MM, Stevens WJ. Haemodialysis-associated anaphylactic and anaphylactoid reactions. Allergy 2006;61(2):211-20.
4. Arenas MD, Gil MT, Carretón MA, Moledous A, Albiach B. Efectos adversos a polisulfona durante la sesión de hemodiálisis. Nefrologia 2007;27(5):638-42.
5. Arenas MD, López-Collado M, Niveiro E, Cotilla de la Rosa E, Morales A, Moledous A, et al. Alergia al látex y trombosis repetida de accesos vasculares para Hemodiálisis. Nefrologia 2009;29(4):365-6.
6. Szczawinska-Poplonyk A. An overlapping syndrome of allergy and immune deficiency in children. J Allergy (Cairo) 2012;2012:1-9.
7. Kuo MC, Chanq JM, Tsai JC, Chen HC, Tsai WC, Lai YH, et al. Decreased B cells and IgA-secreting B cells partially explain the high prevalence of IgA deficiency in dialysis patients. Clin Nephrol 2006;66(4):240-6.
8. Nechvatalova J, Pikulova Z, Stikarovska D, Pesak S, Vlkova M, Litzman J. B-lymphocyte subpopulations in patients with selective IgA deficiency. J Clin Immunol 2012;32(3):441-8.
9. Brophy PD, Mottes TA, Kudelka TL, McBride KD, Gardner JJ, Maxvold NJ, et al. AN-69 membrane reactions are ph-dependent and preventable. Am J Kidney Dis 2001;38(1):173-8.
10. Rachid R, Bonilla FA. The role of anti-IgA antibodies in causing adverse reactions to gamma globulin infusion in immunodeficient patients: A comprehensive review of the literatura. J Allergy Clin Immunol 2011;129(3):628-34.

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Severe levofloxacin-induced hypoglycaemia: a case report and literature review

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To the Editor,

We report a case of severe hypoglycaemia secondary to quinolones in a haemodialysis patient.

Our patient was a 72-year-old man who received haemodialysis three times a week with a tunnelled catheter. He was admitted for severe shivering during a dialysis session. Blood and catheter cultures showed *S. maltophilia* and *E. casseliflavus*. The catheter was removed, and antibiotic treatment was initiated with levofloxacin, at 250mg every 48h, and cotrimoxazole once a day.

The patient's renal failure was then treated medically. The sepsis developed without complication until the second week, when severe hypoglycaemia was detected, with neurological symptoms that persisted for three days. The patient received boluses of glucose at 30% via intravenous administration, and glucose infusion at 10%. Suspecting erroneous intake of oral hypoglycaemic agents, we performed a drug test but found no traces of these drugs. On the other hand, it did reveal the presence of toxic levels of levofloxacin.

The patient's underlying nephrological disease was a primary membranous glomerulopathy. The patient had been on haemodialysis for more than ten years, and had lost all arteriovenous fistulas. He also had undergone arthrodesis of the right knee, and had multiple infections of this joint. He was hypertensive, and did not suffer diabetes mellitus.

The patient was under normal treatment with water-soluble vitamins, amlodipine, and Venofer.

Physical examination: the patient was in a mild stupor; blood pressure: 120/80; heart rate: 80 beats/min; respiratory rate: 18 breaths/min; jugular: 5cm at 30°. Heart: RR interval 2 times with II/VI grade systolic murmur in the aortic area (no radiation). Lungs: basal crackles. Abdomen was soft, depressible, and painless, with no visceromegaly. Limbs: oedema and arthrodesis of the right knee. Symmetric pulses. No embolic injuries.

Laboratory tests: [Na^+]: 136mM; [K^+]: 4.5mM; [Cl^-]: 100mM; [HCO_3^-]: 20mM; haemoglobin: 9g/dl; blood urea nitrogen (BUN): 100mg/dl; creatinine: 10mg/dl; thyrotropin (TRH) 1.79mIU/ml; toxicology: 32ug/ml levofloxacin (therapeutic levels: 6ug/dl).

Hypoglycaemia secondary to levofloxacin is a very rare adverse reaction (<0.01%) according to the manufacturer.[1] Several authors have described it in elderly patients with advanced chronic renal failure.[2-4] The mechanism of this severe complication is caused by an inhibition of the K_{ATP} channels in the islets of Langerhans, which enhances calcium influx and the release of insulin filled vesicles. This effect has been demonstrated in a mouse model, and it is similar to that produced by sulfonylureas.[5] Our first suspicion was the use of oral hypoglycaemic agents, which is well known to cause severe hypoglycaemia. However, this was ruled out by drug testing, which revealed the presence of high levels of levofloxacin, despite having adjusted the dose for the renal failure in our patient.

We conclude that, in patients with advanced renal failure, quinolones may cause hypoglycaemia as severe as that produced by oral hypoglycaemic agents.

Conflicts of interest

The authors affirm that they have no conflicts of interest related to the content of this article.

1. Sanofi Aventis. Tavanic® (folleto de información al profesional).
2. Singh N, Jacob JJ. Levofloxacin and hypoglycemia. Clin Infect Dis 2008;46:1127.
3. Gibert AE, Porta FS. Hypoglycemia and levofloxacin: a case report. Clin Infect Dis 2008;46:1126-34.
4. Kelesidis T, Canseco E. Levofloxacin-induced hypoglycemia: a rare but life-threatening side effect of a widely used antibiotic. Am J Med 2009;122(3):e3-4.
5. Saraya A, Yokokura M, Gonoi T, Seino S. Effects of fluoroquinolones on insulin secretion and h-cell ATP-sensitive K channels. Eur J Pharmacol 2004;497:11-7.

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Membranous glomerulonephritis secondary to neoplasia. Different forms of presentation

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To the Editor,

The relationship between membranous glomerulonephritis (MGN) and cancer is no accidental; this is a classic paraneoplastic phenomenon. Approximately 25% of these cases are secondary (10% neoplastic).[1] Here, we describe two clinical cases of membranous glomerulonephritis secondary to lung cancer with different patterns of presentation.

Case 1. Our first patient was a 49-year-old male with hypertension, sleep apnoea-hypopnea, who smoked 40 cigarettes per day. He was admitted due to anasarca, showing nephrotic syndrome (proteinuria: 19g/day), no haematuria, and normal renal function. An immunological analysis was negative for antinuclear antibodies and anti-neutrophil cytoplasmic antibodies; C3 and C4 levels were normal. There was no monoclonal spike in serum proteinogram, the virological study (HbsAg, HbcAb, HbsAb, hepatitis C antibodies, and HIV antibodies) was negative, and tumour markers (prostate specific antigen [PSA], carcinoembryonic antigen, and alpha-fetoprotein) were normal.

Chest x-ray revealed pleuropulmonary parenchyma and hili without pathological signs, except for very mild bilateral pleural effusion. Renal biopsy was diagnostic of stage I MGN. Immunofluorescence analysis showed subepithelial granular deposits strongly positive for IgG.

The patient was started on conservative treatment with angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARB) and a diuretic drug. After three months, the patient was admitted on two separate occasions due to anasarca and poor response to treatment, and was started on immunosuppression with cyclosporine (CsA). By the fifth month post-biopsy, he showed moderate effort dyspnoea and significant right pleural effusion. We performed a thoracentesis with a positive cytological test for malignancy. A thoraco-abdominal computed axial tomography revealed numerous positive lymph nodes. Bronchoscopy was performed, confirming stage IV lung adenocarcinoma with right pleural metastases. Chemotherapy began with pemetrexed, and the patient died after 2 months.

Case 2. Our second patient was a 59-year-old man, smoker of 15 cigarettes per day, with diabetes mellitus type 2. He was admitted with oedema and had proteinuria at 14g/day, microhaematuria, and preserved renal function. Immunity and complement test results were normal. Virus serology was nega-