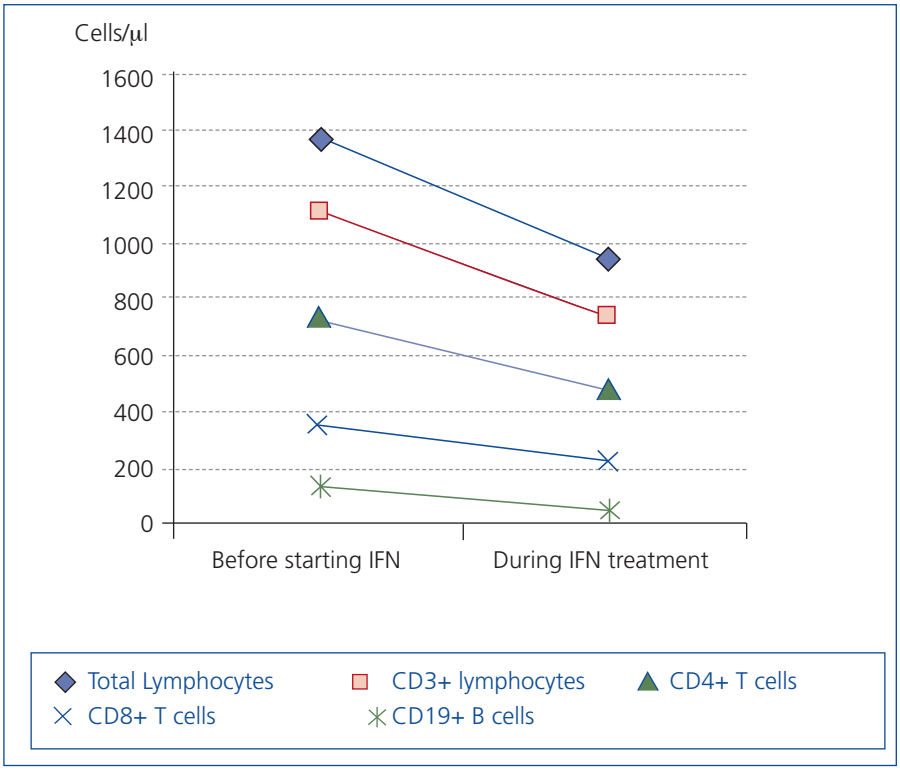

Figure 1. Evolution of lymphocyte populations following treatment with interferon α. IFN: interferon.

toms and autoimmunity test. The growing appearance of new pharmacological therapies, as well as the important pharmacosurveillance of their adverse effects, make the list of agents associated with this condition increasingly comprehensive. For this reason, high clinical suspicion is key, and therefore timing is of great importance.

For our patient, significant lymphocyte depletion induced by IFN-α should also be noted. Although T cell lymphopenia is an effect already described with the use of IFN-α due to thymus' function alteration, this situation, together with the patient's significant B lymphopenia, would mean a dysregulation of the immune response which could be involved in autoimmunity.

Conflict of interest

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

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Baclofen neurotoxicity in a patient with end-stage chronic renal failure

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To the Editor,

Baclofen (4-beta-chlorophenyl-gamma-aminobutyric acid) is a muscle relaxant, used as an antispasmodic in diseases such as multiple sclerosis, medullary trauma and hiccups[1,2].

It is mainly excreted through the kidneys (69%-85%) and has a 2 to 6 hr. half life in healthy people. There is a high risk of neurotoxicity in patients with renal failure, especially if administered with <30ml/min glomerular filtration rate, causing episodes of unconsciousness[3,4].

Baclofen intoxication in dialysis patients is rare, with very diverse forms of clinical presentation[5-9].

We present the clinical case of a patient with baclofen-induced encephalopathy, with atypical clinical evolution; symptoms did not improve with haemodialysis and the patient went into complete remission following the drug's discontinuation.

CASE DESCRIPTION

We present a 31-year-old patient with traumatic spinal injury at 20 and a history of repeated urinary infections related to neurogenic bladder (self-catheterisation) and episodes of urinary obstruction. In 2009 he was referred to the nephrology department due to stage 2 chronic kidney disease, with nephrotic-range proteinuria (1.3mg/dl creatinine, 20g/24h proteinuria). The patient refused renal biopsy and in the same year, he stopped attending nephrology consultations.

In January 2012 he started experiencing episodes of deteriorating levels of consciousness. Cranial computerised tomography and lumbar puncture were performed, both with normal results. Electroencephalogram showed diffuse slowing. At that time, creatinine was 3.3mg/dl. The clinical profile was suggested as secondary to urinary infection.

The same symptoms repeated in April 2012, accompanied by spatial and time disorientation. He presented a new episode in the same month, but this time with behavioural change (infantilism, nervousness, aggressiveness). Cerebral MRI was unremarkable. Following this episode, treatment with clonazepam was started.

In May 2012, due to end-stage renal failure analysis results and symptoms, haemodialysis was started using a temporary catheter in the right jugular vein.

Approximately once a month, in the first two hours after starting dialysis, the patient experienced episodes of reduced levels of consciousness and, at times, psychomotor agitation.

The patient was evaluated in November 2012 by the Neurology and Psychiatry department, with no relevant findings. They diagnosed the condition as secondary to hypoxic metabolic encephalopathy.

Despite presenting correct KT and Kt/V, we increased the number of dialysis sessions to 4/week, without clinical improvement.

In January 2013, we decided to suspend treatment with baclofen (Lioresal®), replacing it with tizanidine and diazepam. After two weeks of tizanidine treatment, the patient stopped the drug because of drowsiness and spasticity was controlled only with diazepam.

10 months after the drug's discontinuation, the patient had not re-experienced neurological symptoms.

DISCUSSION

Spasticity is a classic symptom in spinal injury patients. Baclofen is widely used in these patients, despite their high risk of neurotoxicity, as a result of having reduced renal function due to neurogenic bladder. We also have to bear in mind that, on assessing serum creatinine levels, we overestimated the renal function due to a decrease in muscular mass[10].

Psychomotor agitation is a rare symptom of baclofen-induced encephalopathy. This encephalopathy usually manifests itself as a reduced level of consciousness. Our patient initially presented this symptom, since the same baclofen dose was used as in a patient with normal renal function. Following deterioration in renal function and the start of haemodialysis, the patient experienced the atypical symptom of psychomotor agitation.

In reviewing the literature, we observe that the pharmaco-dynamics of baclofen in dialysis patients is expressed as $C = C_0 + e^{Ket}$, where Ke is dependent on the drug's renal (Kr) and non-renal (Knr) metabolism. In dialysis patients, renal clearance is restricted to clearance during dialysis; thus Kr = Kd, where Kd = 0.291/h and Knr 0.045/h, therefore Ke = Kd + Knr = 0.336/h. Given the drug's excretion, baclofen's half life went from 15.5h in patients with stage 5 chronic renal failure not on dialysis to 2.06h in dialysis patients[11-13].

We suspect that this patient's clinical symptoms can be attributed to a sudden suppression of baclofen levels in blood. It is worth noting that similar cases have been described in sudden withdrawals of this drug in patients with intrathecal perfusions.[14,15]

Despite no clear indications about baclofen in pharmacological guides, we do not recommend the use of this drug in dialysis patients.

Conflict of interest

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

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Achromobacter xylosoxidans in two haemodialysis patients

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To the Editor,

Achromobacter (alcaligenes) xylosoxidans (AX) is a gram-negative, aerobic bacillus, carried by animals (rabbits, ferrets), although it is also present in normal human flora, especially of the skin and gastrointestinal tract[1].

It is an opportunistic bacterium with low virulence, except in immunocompromised patients, in whom it can cause serious infections such as meningitis, endocarditis and, most commonly, bacteraemia[2].

Patients with a catheter are more likely to develop AX and it is more frequent in peritoneal dialysis (PD) than haemodialysis (HD) patients, where there are few published cases; all cases are associated with a central venous catheter (CVC)[3-5]. Contamination of the catheter, the heparin multi-dose vials, the antiseptic solutions and the dialysate itself have been described as possible sources of infection, and the clothes or hands of the health staff as methods of transmission[5].

We present two cases of AX that occurred in our department on the same date in patients undergoing the same HD session.

CASE 1

The patient was a 67-year-old female, from Bulgaria, hypertensive, diabetic, obese, with dyslipidaemia and chronic kidney disease (CKD) possibly secondary to diabetes and/or nephroangiosclerosis, on HD since January 2008. Low socioeconomic status, living with animals and bad personal hygiene.

Left humeral-cephalic arteriovenous fistula (AVF) was performed, with slow recovery, carrying out HD using a temporary CVC (multiple removals and new catheterisations due to infections of the catheter entry site).

The patient was admitted due to fever and shivers following dialysis, symptoms compatible with bacteraemia, with positive blood cultures of Staphylococcus (St.) aureus. There was associated infection in the catheter entry site, for which reason the catheter, which was cultured and resulted positive for AX, St. aureus and Enterococcus faecalis, was removed. The clinical and bacteriological infectious condition disappeared with combined treatment of the three bacteria.

CASE 2

A 46-year-old male patient, hypertensive, with hyperuricemia and CKD possibly secondary to chronic glomerulonephritis (GN), on HD since 1995. He received two cadaveric kidney transplants, with possible early recurrence of membranous GN and restarted HD in 2004.

The patient had multiple vascular accesses, the last being left humero-axillary prosthetic AVF (polytetrafluoroethylene), which resulted in ulceration on the skin close to the anastomosis with serous secretion, leaving the prosthesis exposed. A temporary CVC was implanted and a culture, growing AX, was taken from the ulcer. The patient did not show increase of acute phase reactants nor systemic infection data. He received intravenous antibiotics according to the antibiogram, after which the culture was repeated, with development of AX continuing. He received new courses of antibiotics, without managing to eradicate the bacterium (three AX positive cultures). Thus, surgical removal of the prosthesis was decided upon and the implanting of a new vascular access (femoral saphenous AVF). The culture after the surgical wound tested negative for AX.

CONCLUSIONS

Although AX is not a common bacterium, it can be seen in HD patients.