C) BRIEF CASE REPORT

Cloudy peritoneal effluent and diarrhoea due to Clostridium difficile

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To the Editor:

Intraperitoneal viscera inflammation favours the translocation of polymorphonuclear leukocytes to the peritoneal cavity, with some cases of cloudy peritoneal effluent being reported alongside infection of the colon by *Clostridium difficile*.[1]

We report the case of an 87-year-old male with a good quality of life, who had long-term high blood pressure, chronic obstructive pulmonary disease with bronchiectasis and stage 5 chronic kidney disease secondary to nephroangiosclerosis, with a baseline glomerular filtration rate of 15ml/min. He came to our hospital due to deterioration in his general condition, asthenia and anorexia, and his blood tests revealed a decrease in his glomerular filtration rate (7ml/min). There was no change in his normal medication or focal infection. In the following days, he did not respond to conservative measures and, with the patient's consent, we started renal replacement therapy by peritoneal dialysis, with a double-cuff straight Tenckhoff peritoneal catheter being introduced, without complications during the procedure. In the hours following the procedure, the patient presented with fever and increasing coughing and expectoration. The X-ray was suggestive of right lower pulmonary lobe consolidation, and as such, given the suspicion of nosocomial pneumonia, we started treatment with meropenem and ciprofloxacin. He started automatic peritoneal dialysis (APD) a week later, given the progressive deterioration of kidney function. He was discharged ten days later having recovered his baseline clinical condition, with oral antibiotic treatment and the indication of continuing APD in the peritoneal dialysis unit while awaiting training. The microorganism was not isolated in the blood cultures or in the sputum analysis.

Four days later, after the weekend period, he came to the hospital complaining of watery diarrhoea, without pathological products, lasting 24 hours. He had abdominal discomfort and fever and cloudy peritoneal effluent was observed. The fluid analysis revealed 408 cells/ul with a predominance of polymorphonuclear cells (79%). Using clinical and cytological criteria, the patient was diagnosed with peritonitis and empirical treatment was started with intraperitoneal ceftazidime and vancomycin.[2] Given the lack of response to treatment or growth of the microorganism in the peritoneal dialysate culture, after three days we received the stool culture result, which revealed the presence of *Clostridium difficile toxin and antigen*.

Given the absence of clinical improvement and the persistence of cloudy dialysate, the case was reassessed and we considered other causes of cloudy peritoneal effluent with polymorphonuclear predominance.[3] We unsuccessfully searched for fungi and mycobacteria. The patient had not received treatment with vancomycin or amphotericin B before symptoms started. The results of the studies to determine the presence of *Clostridium* difficile toxins or antigens in peritoneal dialysate were negative. Other symptoms that may include the presence of sterile cloudy liquid with eosinophil predominance[3] were not suggestive in this patient, based on the chronology with regard to catheter insertion. The X-ray of the abdomen did not display pneumoperitoneum. The existence of an inflammatory intestinal process and the negative results of tests carried out led us to consider the presence of sterile cloudy peritoneal dialysate to be secondary to colitis due to *Clostridium difficile*. We discontinued intraperitoneal antibiotic administration and started the patient on 500mg of metronidazole administered orally every 8 hours, with little response. As such, we decided to combine it with 500mg of vancomycin administered orally every 8 hours, with the patient displaying a gradual improvement of the process, a decrease in the frequency of bowel movements and an increase in density of stools. The transparency of the peritoneal dialysate was simultaneously recovered.

The fact that no *Clostridium difficile* toxins or antigens were detected in the peritoneal dialysate suggests that the aetiology of the cloudy dialysate corresponds to the passage of leukocytes and not to phenomena of bacterial translocation with secondary peritonitis. The fact that symptoms only improved with the introduction of oral *vancomycin in the treatment* supports this hypothesis. There are studies that report that the exposure of intestinal epithelial cells to the toxin *Clostridium difficile* modulates the epithelial expression of IL-8 and the intercellular adhesion molecule 1 (ICAM-1), involved in the chemoattraction and adhesion of leukocytes.[4] Furthermore, the data suggest that the transmigration of leukocytes to the peritoneal cavity is related to the upward regulation of ICAM-1 receptors.[5] These results together may offer an explanation of the pathophysiology of the symptoms described.

Conflicts of interest

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

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Acquired perforating dermatosis in patients with chronic renal failure. A report of two cases and a review of the literature

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To the Editor:

Acquired perforating dermatosis (APD) is an uncommon cutaneous perforating disorder. It is characterised by the presence of hyperkeratotic papules and nodules and histologically, by transepidermal elimination of various substances, such as keratin, collagen and elastic fibres. It was first described by Mehregan in 1967. There are two types: hereditary and acquired, the latter is generally associated with diabetes mellitus (DM) and chronic renal failure (CRF).[1]

In the literature, we also found other cases associated with neoplasia,[2,3] liver diseases and endocrine disorders,[4] AIDS,[5] tuberculosis,[6] etc.

There are four classic forms of APD, according to clinical and histological criteria: elastosis perforans serpiginosa, reactive perforating collagenosis, perforating folliculitis and Kyrle disease.

As regards the population with CRF on renal replacement therapy, the only studies of this condition date back to 1982,[7] with a reported prevalence of 4.5%-10% in haemodialysis patients in the United States, and 1996,[8] with a prevalence of 11% in the United Kingdom. Only haemodialysis patients have been reported to date, not peritoneal dialysis patients.

Many treatments have been used in an attempt to manage this disorder, with varying results: topical and systemic steroids, topical and systemic retinoids, vitamin A, light therapy, methotrexate and cryotherapy. In recent times, allopurinol has been considered as treatment, due to its antioxidant effect: inhibiting xanthine oxidase and reducing the production of oxygen free radicals and, therefore, collagen damage.[9,10]

We report two cases in our unit; the first was a peritoneal dialysis patient and the second, a haemodialysis patient.

CASE 1

A 39-year-old female with a history of very debilitating juvenile chronic arthritis from the age of 16 years old. She was not diabetic and had bilateral hip prostheses due to femoral head avascular necrosis, secondary to prolonged steroid treatment. She also had rheumatic mitral regurgitation and was on peritoneal dialysis due to chronic kidney disease. She came to our hospital due to exophytic and hyperkeratotic lesions on the soles of her feet, the pretibial area (Figure 1) and her buttocks. She was transferred to the Dermatology department, the lesions were biopsied and she was diagnosed with reactive perforating collagenosis. After reviewing the literature, treatment was started with allopurinol and a significant improvement was noted in the lesions after one month, with the disappearance of pruritus.

CASE 2

A 50-year-old male with ESRD secondary to diabetic nephropathy on renal replacement therapy with haemodialysis. He had chronic ischaemic heart disease, ischaemic dilated cardiomyopathy, diabetic retinopathy and had suffered a stroke some years before; he was admitted to internal medicine due to an

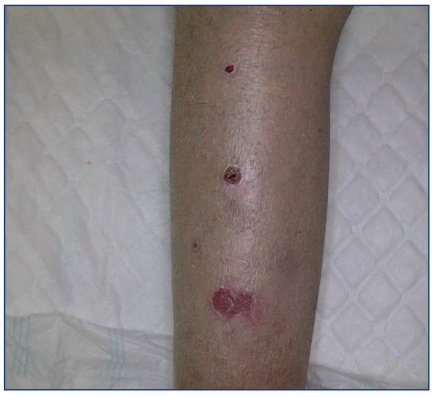

Figure 1. Pretibial lesion.

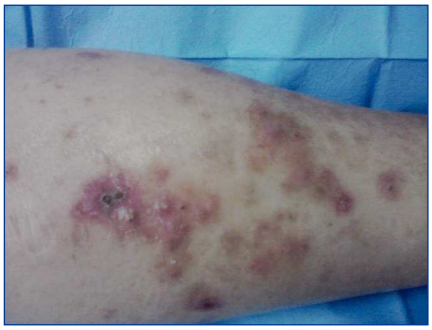

Figure 2. Universally distributed hyperkeratotic and erythematous lesions.