was administered. Right jugular region and the exit site were identified with minimal epithelial damage, and the catheter was tunnelled placing a Permacath catheter, its position was checked by means of a portable chest X-ray. Adequate arterial and venous return was achieved, and the skin (Figure 2) was sutured. Haemodialysis was started with symptom remission, patient was discharged due to clinical improvement and enrolled in the chronic haemodialysis programme.

DISCUSSION

Epidermolysis bullosa is an autosomal recessive hereditary skin disease that involves all organs from birth. Chronic kidney disease is the second leading cause of death[3] in these patients after squamous cell carcinoma; it is relevant to mention that many patients die due to associated dilated cardiomyopathy[6]. Kidney disease is usually due to the development of secondary amyloidosis, IgA nephropathy, post infectious glomerulonephritis, and obstructive uropathy[3,7]. Other factors contributing to the development of chronic kidney disease include: dehydration, dysphagia, vomiting and hypoalbuminaemia[8,9].

When developing chronic kidney disease, access to dialysis therapy is considered a big issue. Skin lesions and the tendency to become infected limit the use of peritoneal dialysis as an option. Another limitation to performance of an arteriovenous fistula is the development of skin lesions and local contractions, which prevent the placement of vascular catheters[7]. Therefore, placement of a tunnelled haemodialysis catheter in a central vein is, at present, considered the best option[10].

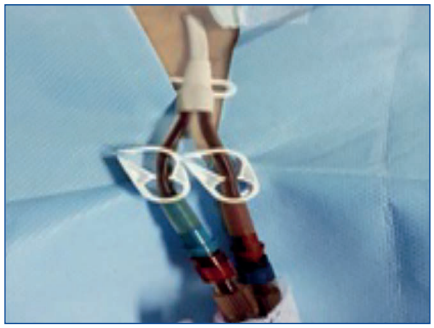

Figure 2. Permacath catheter permeability following corroborated procedure in urgent haemodialysis.

Conflicts of interest

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

1. Fine JD, Eady RAJ, Bauer EA, Bauer JW, Bruckner-Tuderman L, Heagerty A, et al. The classification of inherited epidermolysis bullosa (EB): Report of the Third International Consensus Meeting on Diagnosis and Classification of EB. J Am Acad Dermatol 2008;58:931-50.
2. Varki R, Sadowski S, Uitto J, Pfendner E. Epidermolysis bullosa. II. Type VII collagen mutations and phenotype/genotype correlations in the dystrophic subtypes. J Med Genet 2007;44:181-92.
3. Mann JF, Zeier M, Zilow E, Schärer K, Anton-Lamprecht I, Waldherr R, et al. The spectrum of renal involvement in epidermolysis bullosa dystrophica hereditaria: Report of two cases. Am J Kidney Dis 1988;11:437-41.
4. Fine JD, Mellerio JE. Extracutaneous manifestations and complications of inherited epidermolysis bullosa. Part I. Epithelial associated tissues. J Am Acad Dermatol 2009;61:367-84.
5. Varki R, Sadowski S, Pfendner E, Uitto J. Epidermolysis bullosa. I. Molecular genetics of the junctional and hemidesmosomal variants. J Med Genet 2006;43:641-52.
6. Sidwell RV, Yates R, Atherton D. Dilated cardiomyopathy in dystrophic epidermolysis bullosa. Arch Dis Child 2000;83:59-63.
7. Baloch MS, Fitzwilliams B, Mellerio J, Lakasing L, Bewley S, O'Sullivan G. Anaesthestic management of two different modes of delivery in patients with dystrophic epidermolysis bullosa. Int J Obstet Anesth 2008;17:153-8.
8. Kawasaki Y, Isome M, Takano K, Suyama K, Imaizumi T, Matsuura H, et al. IgA nephropathy in a patient with dominant dystrophic epidermolysis bullosa. Tohoku J Exp Med 2008;214:297-301.
9. Farhi D, Ingen-Housz-Oro S, Ducret F, Rioux-Leclercq N, Cam G, Simon P, et al. Recessive dystrophic epidermolysis bullosa (Hallopeau-Siemens) with IgA nephropathy: 4 cases. Ann Dermatol Venereol 2004;131:963-7.
10. Tammaro F, Calabrese R, Aceto G, Lospalluti L, Garofalo L, Bonifazi E, et al. End-stage renal disease secondary to IgA nephropathy in recessive dystrophic epidermolysis bullosa: A case report. Pediatr Nephrol 2008;23:141-4.

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Non-Hodgkin lymphoma mimicking peritonitis in a patient on peritoneal dialysis

Nefrologia 2014;34(5):686-7
doi:10.3265/Nefrologia.pre2014.Apr.12386

To the Editor,

Cloudy peritoneal fluid in peritoneal dialysis (PD) patients is most commonly due to an increase in leukocytes as an expression of bacterial peritonitis. Occasionally, when there is cloudy fluid, we must differentiate between a peritoneal infection with a negative culture and non-infectious peritoneal inflammation (sterile peritonitis).

CASE REPORT

Our patient was 59-year-old, on PD since October 2012 with a history of type 2 diabetes mellitus and chronic kidney disease secondary to diabetic nephropathy.

The patient came to the clinic with abdominal pain and cloudy peritoneal

fluid, referring to a decrease in ultrafiltration. Two weeks earlier, the patient had been diagnosed with a nasal neoplasm. The results of the peritoneal fluid were: cloudy appearance, 700 leukocytes/µl (80% mononuclear and 20% polymorphonuclear); despite the predominance of mononuclear leukocytes, we started empiric antibiotic therapy with intraperitoneal ceftazidime and vancomycin. The abdominal examination did not show signs of peritoneal irritation or masses; the peritoneal catheter outlet and tunnel were normal. The blood tests displayed: leukocytes 37,000/mm^3, platelets 640,000/mm^3, haemoglobin 10.3g/dl, creatinine 5.4mg/dl, urea 180mg/dl, Na 138mEq/l, K 4mEq/l, calcium 9.5mg/dl, phosphorus 4.8mg/dl, albumin 3.9mg/dl. The peritoneal dialysis cultures taken days later were negative. A computerised axial tomography of the skull, thorax and abdomen was carried out to complete the nasal mass study in the following days, with the results showing scattered mediastinal and retroperitoneal lymphadenopathies. The anatomical pathology of the nasal biopsy was reported as non-Hodgkin lymphoma of large B cells and the cytology of the peritoneal fluid showed atypical lymphocytes. The patient began chemotherapy and remained on PD without further dialysis incidents.

The presence of abdominal pain associated with cloudy peritoneal fluid in a PD patient is generally associated with peritonitis. For the diagnosis of bacterial peritonitis, at least two of the following three conditions are required: abdominal pain alone or accompanied by other abdominal symptoms, peritoneal fluid with more than 100 leukocytes/µl, with more than 50% being polymorphonuclear, and the culture or Gram stain showing the existence of micro-organisms[1].

It is difficult to distinguish between a peritoneal infection with a negative culture and non-infectious peritoneal inflammation (sterile peritonitis). A negative peritoneal culture is generally due to technical failures in the processing of samples, and as such, management is complicated given that there is infectious peritonitis without bacterial growth in the culture. It is advised to revise the culture technique when the latter is negative in more than 20% of occasions and ask the patient about previous use of antibiotics[2]. To achieve a quick diagnosis in cases of sterile peritonitis, the Spanish PD guidelines give a general outline, taking into account the presence or absence of cells in the peritoneal fluid and the type of cells. An increase in polymorphonuclear cells may be due to inflammation of the viscera within or around the peritoneum, medication or peritoneal fluid that is contaminated by endotoxins. If eosinophils increase, we must suspect an allergic reaction to the dialysis material, medication and peritoneal irritation due to retrograde blood or after peritonitis due to fungi and parasites. An increase in mononuclear cells may be associated with icodextrin or infection due to mycobacteria or fungi. If there are also red blood cells, the following must be considered: ovulation, retrograde menstruation, ovarian cyst rupture, hypertonic solution, peritoneal adhesions, physical exercise and catheter-induced trauma. When there is a high number of mononuclear and malignant cells, we must consider lymphoma or peritoneal metastases. However, an absence of cells may be due to an increase in fibrin or triglycerides[3,4].

The literature reports some cases of suspected peritonitis in PD associated with tumour processes, amongst them a patient with recurrent renal cell carcinoma diagnosed in the peritoneal fluid cytology[5]. Another patient had sterile peritonitis with a history of lymphoma diagnosed ten years earlier. The peritoneal fluid cytology allowed atypical lymphocytes to be observed and, as in our case, despite peritoneal invasion, the patient continued with the PD technique[6].

In conclusion, we must highlight that in the differential diagnosis of sterile peritonitis, we must not forget the potential existence of a neoplastic process.

Conflicts of interest

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

1. Montenegro J. Peritonitis e infecciones del catéter en la diálisis peritoneal. Available at: http://dx.doi.org/10.3265/Nefrologia.2010.pub1.ed80.chapter2826.
2. Li PK, Szeto CC, Piraino B, Bernardini J, Figueiredo AE, Gupta A, et al. Peritoneal dialysis-related infections recommendations: 2010 update. Perit Dial Int 2010;30:393-423.
3. Montenegro J, Molina A, Rodríguez-Palomares R. Peritonitis en diálisis peritoneal. Guías clínicas de práctica clínica en diálisis peritoneal. Sociedad Española de Nefrología; 2005.
4. de Freitas DG, Gokal R. Sterile peritonitis in the peritoneal dialysis patient. Perit Dial Int 2005;25:146-51.
5. Streather CP, Carr P, Barton IK. Carcinoma of the kidney presenting as a sterile peritonitis in a patient on continuous ambulatory peritoneal dialysis. Nephron 1991;58:121.
6. Vlahakos D, Rudders R, Simon G, Canzanello V. Lymphoma-mimicking peritonitis in a patient on continuous ambulatory peritoneal dialysis (CAPD). Perit Dial Int 1990;10:165-7.

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