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Lilia Susana Gallardo Vidal[a,*], Adriana Jheny Rodríguez Méndez[b], Mariana Burgos Ochoa[c], Martha Leticia Martínez Martínez[b], Pablo García Solís[a], Maria Elena Villagrán Herrera[a], Ana María Pérez Baza[a]

[a] *Unidad de Medicina Familiar N.°13, Instituto Mexicano del Seguro Social, Tuxtla Gutiérrez, Chiapas, Mexico*
[b] *Facultad de Medicina, Universidad Autónoma de Querétaro, Santiago de Querétaro, Querétaro, Mexico*
[c] *Unidad de Medicina Familiar N.°14, Instituto Mexicano del Seguro Social, México, D.F., Mexico*

* *Corresponding author.*
E-mail addresses: susi2947@gmail.com, susana.gallardo@imss.gob.mx (L.S. Gallardo Vidal).

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Cloudy peritoneal dialysate effluent due to graft intolerance syndrome☆

Efluente peritoneal turbio debido a un síndrome de intolerancia al injerto renal

To the Editor,

The presence of cloudy peritoneal fluid (PF) in patients on peritoneal dialysis (PD) usually occurs in the context of infectious peritonitis. For this diagnosis, two of the following three criteria must be met: (1) the presence of abdominal pain, (2) cloudy fluid with more than 100 leukocytes/μL and more than 50% of them polymorphonucleocytes, and (3) positive culture from PF.[1] There are many causes of cloudy PF due to a high cell count. Inflammation of juxtaperitoneal organs (pancreatitis, cholecystitis, splenic infarct, appendicitis, etc.) can increase the number of polymorphonuclear lymphocytes in

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PF. An increase in PF lymphocytes has also been described with the use of icodextrin,[2] tuberculous peritonitis, viral gastroenteritis,[3] and in one case of acute rejection in graft failure,[4] all with negative PF culture. We describe a new case of cloudy PF with a predominance of lymphocytes and negative culture in a patient with a non-functioning renal graft on PD.

This was a 67-year-old man with chronic kidney disease of unknown aetiology, who started haemodialysis in 2005, and in January 2009 received a renal graft from a cadaveric donor. Six months post-transplant, a renal biopsy was done because of graft dysfunction and a diagnosis was made of nephritis secondary to BK polyomavirus. The patient continued with the dysfunctional graft until March 2012 when he restarted haemodialysis. After 3 months of haemodialysis, the patient expressed his wish to be transferred to PD and in July 2012 he started continuous ambulatory PD (CAPD). At that time, the patient was receiving 1 mg/day of tacrolimus (blood level 4 mg/dL), prednisolone had been stopped 2 months before starting PD, and he had a residual diuresis of 500–700 mL/day. As the patient had had viraemia positive for BK virus, and therefore could not be re-added to the waiting list, we decided to stop tacrolimus to try to get a negative viral load.[5] Two months later, the patient presented with symptoms of diffuse abdominal pain, worst in the zone of the graft, with a low-grade fever, and cloudy PF. Diuresis had decreased to 400 mL/day, and there was no failure of ultrafiltration with his regular CAPD routine. Physical exam revealed mild tenderness on palpation of the graft, with no peritonism. Blood tests showed anaemia despite high doses of erythropoietic agents and a CRP of 185 mg/L, and there were no signs of systemic infection. Urine had minimal proteinuria and haematuria without leukocyturia. The PF cell count showed 300 leukocytes/μL with 35.7% polymorphonucleocytes and 64.3% lymphocytes. Doppler-echo confirmed the presence of graft flow. Due to the apparent clinical picture of graft intolerance and lack of criteria for a diagnosis of infectious peritonitis, we decided to start treatment with corticoids at a dose of 0.5 mg/kg/day without adding intraperitoneal antibiotics. Forty-eight hours later, the patient showed a significant clinical improvement with resolution of pain at the graft site and resolution of the cloudy PF. Subsequently, all cultures, including PF, were confirmed negative. Peritoneal fluid cytology was performed with immunohistochemistry techniques and flow cytometry, showing a predominance of T lymphocytes (CD-3 positive). Finally, in March 2013 we performed graft embolisation, after a biopsy. The biopsy showed humoral rejection with vasculitis, tubulitis, and presence of C4d in the peritubular capillaries. Following a reducing dose of steroids and graft embolisation, the patient had no further cloudy PF, and negative cultures as long as he remained on PD.

Discussion

Cloudy PF in PD can be due to multiple causes. Although the most common cause is infectious peritonitis, other possibilities should be considered. The management of immunosuppression in transplant patients starting PD is controversial; maintenance immunosuppression can increase the incidence of infection and neoplasia, but its withdrawal leads to accelerated loss of residual renal function and the possible occurrence of graft intolerance syndrome. In this patient with significant cumulative immunosuppression, as soon as cloudy PF was noted, infectious peritonitis was the main diagnosis of suspicion. However, as we have seen, if not all the criteria for infectious peritonitis are present, and above all, there is a predominance of lymphocytes in the PF, we must look for other causes of cloudy PF. We think that graft intolerance must be in the differential diagnosis of cloudy PF in patients with a dysfunctional renal graft on PD.

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Sandra Beltrán-Catalán*, Belén Vizcaino-Castillo, Pablo Molina-Vila, Marco Montomoli, Luis Manuel Pallardó-Mateu, Ana Ávila-Bernabeu

Servicio de Nefrología, Hospital Universitario Dr. Peset, Valencia, Spain

* *Corresponding author.*
E-mail address: sanbelca@gmail.com (S. Beltrán-Catalán).

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