our patient we ruled out susceptibility due to absence of factor H deficiency, but the 3 conditions coexisted: FGN, vasculitis, and TMA.[10] FGN probably associated with GBMD with ANCAp+ and TMA has not been previously described in the literature, to our knowledge.

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Bilateral hip osteonecrosis and cholelithiasis after eculizumab discontinuation in atypical haemolytic uraemic syndrome

Osteonecrosis bilateral de cadera y colelitiasis tras la interrupción de eculizumab en sindrome hemolitico uremico atípico

Dear Editor,

We describe the course of a boy with atypical haemolytic uraemic syndrome (aHUS) with no mutation in the complement system, who presented with two unusual extrarenal complications; osteonecrosis of both femoral heads and cholelithiasis, resulting from a relapse of aHUS caused after suspending eculizumab treatment.

The case is an 8-year-old boy with a neonatal onset of aHUS with severe and multisystemic disease. By administering eculizumab, it was achieved full haematological and renal recovery.[1] He continued the treatment with eculizumab from the neonatal period to the age of three years and six months, and the disease was maintained in complete remission.

Exhaustive testing of all the known genes that could cause aHUS was carried out and no mutations were found. There-

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Table 1 – Laboratory test data over time.

Age (years)	1	3	3.5	3.6	4	6	8
Creatinine, plasma mg/dl	0.34	0.28	0.99	0.33	0.31	0.49	0.56
Urea, plasma mg/dl	35	36	149	35	41	42	42
Cystatin mg/l		0.93	RELAPSE	0.89		0.85	
Haematocrit %	38	38	26	30	39	40.2	40
Platelets $10^3/\mu l$	239,000	166,000	13,000	174,300	180,000	204,000	181,000
LDH U/l	262	303	2218	342	243	203	215
Prot/Cr in urine	0.51	0.45	6.1	3.55	0.25	0.16	0.22
Complement C3 (g/l)	1.16	1.12	1.18	1.37	1.02		1.1

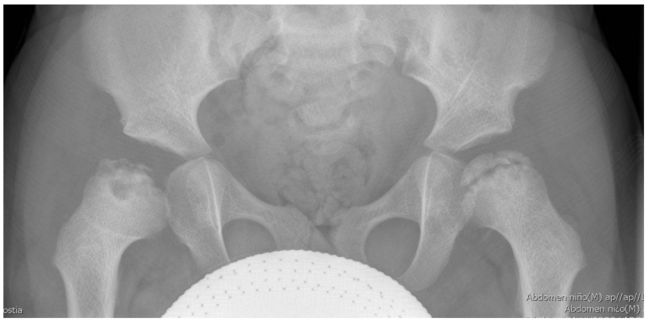

Figure 1 – X-ray of both femoral heads at diagnosis.

fore the treatment was discontinued. The genes studied were: ADAMSTS13, ARMS2, C1S, C2, C3, C3ARI, C4BPA, C4BPAP1, C4BPAP2, C4BPB, C5, C5ARI, C6, C7, C8A, C8B, C8G, C9, CD46, CD46, CD55, CD59, CFB, CFD, CFH, CFHR1, CFHR2, CFHR3, CFHR4, CFHR5, CFI, CFP, CLU, CPB2, CR1, CR1L, CR2, CRP, DGKE, F12, F2, F3, FCN1, HTRA1, KDR, MASP1, MASP2, MBL2, NR5A2, PHG, PIGA, PLG, PROC, PROCR, PROS1, PTX3, RP1, RP1L1, SELP, SERPING1, TFP1, THBD, THBS1, VSIG4, VTN, and VWF. No variants (haplotypes) of risk in the MCP gene or the CFH gene were found.

Three months later, after mild cold symptoms, he was admitted for acute kidney failure. Treatment was resumed with eculizumab, and all parameters wee normalized within a few days (Table 1).

One year after the last relapse, it was detected by chance, synchronic and asymmetric bilateral necrosis of both femoral heads (Fig. 1). Based on the stage of progression at which the lesions were found, the osteonecrosis occurred during the relapse caused after withdrawing the medication. The absence of a history of degenerative coxofemoral joint disease and other secondary causes of osteonecrosis enabled other possible aetiologies to be ruled out.

In the same study, asymptomatic gallstones were detected.

Five years later, the child continues to receive treatment with eculizumab, with good tolerance (Table 1), without presenting evidence of thrombotic microangiopathy (TMA) activity.

Discussion

In aHUS, the membrane attack complex causes damage to the endothelium, and this triggers TMA.

Although the aHUS lesions predominantly affect the kidney vessels, the diffuse and systemic nature of TMA leads to microvasculature involvement of other organs (brain, heart, intestines, pancreas, lungs, etc.).[2]

In addition to the triad of haemolytic anaemia, thrombocytopoenia, and acute kidney failure, our patient presented avascular necrosis of both femoral heads, presumably caused by the systemic TMA that occurred during the relapse. Damage to the microvascular endothelium in the terminal circulation of the femoral head would cause tissue hypoperfusion with vascular supply interruption, bone infarction, and ultimately necrosis in both femoral heads.[3] Its association with aHUS has not been reported.

Among gastrointestinal complications, some cases of gallstones have been published in the literature several months after an acute episode of typical HUS[4]; however, this complication is rare in aHUS.

One of the most currently debated topics is the duration of treatment with eculizumab[5,6] since the decision to withdraw treatment is not risk-free, especially in patients with a very severe and life-threatening clinical presentation of aHUS at the start. This controversy is accentuated in the paediatric age, since the common events that lead to complement activation (infections, vaccines, etc.) are common in this age group.

No mutations in the complement genes are identified in 30–40% of patients with aHUS. There is evidence that the severity of aHUS and the response to eculizumab is similar in patients with or without an identified genetic risk,[6] although maintaining the treatment is more difficult in patients in whom no mutations were found in the complement system.

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RT-PCR for SARS-CoV-2 in dialysis effluent in four patients from an ambulatory peritoneal dialysis programme in Mexico City

Hallazgos de RT-PCR para SARS-CoV-2 en eflujo dializante en cuatro pacientes de un programa de diálisis peritoneal ambulatoria de la Ciudad de México

Dear Editor:

After the rapid spread of the COVID-19 pandemic, different reports have been written with contradictory results about the presence of the SARS-CoV-2 virus in peritoneal fluid, as Vischini et al.[1] and Coccolini et al.[2] report in favour, while Candellier et al. and other authors state the opposed.[3–5] For nephrologists it is important to know about the possible presence of the SARS-CoV-2 virus in the dialysis effluent in patients on peritoneal dialysis. Some pathophysiological mechanisms have been mentioned related to the characteristics of the peritoneal membrane and that of the virion itself. The pores of the peritoneal membrane have a maximum diameter of 20–40 nm, while the SARS-CoV-2 virion diameter measures between 60 and 140 nanometres. In theory the virion could enter the peritoneal cavity via haematogenous diffusion or via the peritoneal dialysis catheter after contact contamination.[3]

Specifically, in Latin America, including Mexico, ambulatory peritoneal dialysis predominates as renal replacement therapy.[6] In our experience, we found four patients, two women and two men from the ambulatory peritoneal dialysis programme, three of whom were in continuous ambulatory peritoneal dialysis and one in automated peritoneal dialysis, with an age ranging from 35 to 64 years and with different comorbidities, including diabetes mellitus, hypertension, rheumatoid arthritis, and obesity (Table 1). All cases were diagnosed with COVID-19 by real-time inverse polymerase chain reaction (RT-PCR) test targeting SARS-CoV-2 from samples obtained from the nasopharynx or by CT findings. Samples for RT-PCR were obtained from the peritoneal effluent with a dwell time in the cavity of six hours and the following findings were obtained: three patients were positive for the presence of SARS-CoV-2 in the nasopharynx and in the dialysis effluent, while the fourth patient was negative for the presence of the virus in both samples, despite having CT scan findings highly compatible with the disease. It should be mentioned that in the three patients with positive SARS-CoV-2 RT-PCR tests in the nasopharynx and dialysis effluent, the samples were performed within the first seven days of the onset of COVID-19-related symptoms, and in the fourth patient, the sample was obtained from the peritoneal effluent after more than seven days of the onset of symptoms. All four patients had transparent and colourless peritoneal dialysis effluents. None of the cases reported localised symptoms in the abdominal region. Three of the four patients died during their hospital stay.

The presence of SARS-CoV-2 in the peritoneal fluid continues to be a topic of lively debate in the literature on COVID-19. Positive RT-PCR for SARS-CoV-2 should be confirmed or ruled out using subsequent studies with greater statistical power.

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