Table 1. Aetiology of cases of peritonitis that caused abandonment of peritoneal dialysis.

	Period A	Period B
Candida	5 (38%)	1
Gram-	5 (38%)	2
Pseudomonas	2	1
S. epidermidis	1	1
St. aureus	-	6 (43%)
Xanthomonas	-	1
Streptococcus	-	1
Pantoea aglomerans	-	1

In our study, the change in antibiotic protocols, with the advent of the use of intra-peritoneal ciprofloxacin, has produced a change in the relative frequencies of the various microorganisms responsible for the most aggressive types of peritonitis, thus significantly decreasing the rate of infection by gram-negative bacteria.

Cases of peritonitis caused by *S. aureus* are, in general, the most severe types of peritonitis caused by gram-positive bacteria, and these occur most commonly in patients with nasal *S. aureus* carriage or colonisation of the skin and hands, or in relation to colonisation and infection of the catheter outflow orifice.[3]

In our experience, we also observed an increase in the virulence of *S. aureus* that produced peritonitis in the second period, as this bacterium became the primary infectious cause of abandoning PD. We believe that resistance to vancomycin was the primary factor in impeding the resolution of these infections and thus the continuity of PD.

This increase in the resistance to vancomycin will also require reformulating the antibiotic protocol, orientating treatment for the best coverage of gram-positive infections.

Fungal peritonitis constitutes between 1% and 15% of all peritonitis episodes occurring in patients on PD, although its incidence has decreased notably with the use of adequate preventative measures, such as the administration of fluconazole whenever antibiotics are prescribed to the patient for any reason.[4] This led to the decrease in the percentage of cases of peritonitis produced by fungi that caused patients to abandon PD from 38% in the first period to 7% in the second period.

The epidemiology of peritoneal infections is heavily influenced by the antibiotic regimens utilised in each hospital department, causing the frequency and resistance of the microorganisms responsible for these infections to vary over time. This requires a critical regimen of periodical changes to antibiotic protocols, changing and adapting them as needed to emerging scenarios of causative microorganisms and their acquired resistances.

Conflicts of interest

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

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C) BRIEF CASE REPORTS

Proliferative glomerulonephritis with monoclonal IgG deposits in multiple myeloma

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To the Editor:

Glomerular deposits of monoclonal immunoglobulins can arise as a condition secondary to a number of different entities, including AL amyloidosis, Randall type monoclonal immunoglobulin deposition disease (MIDD), type 1 cryoglobulinaemia, immunotactoid/fibrillar glomerulonephritis, and the most recently described, non-Randall type proliferative glomerulonephritis with monoclonal IgG glomerular deposits (MIgG PGN).[1]

Here we described the case of a patient with nephrotic syndrome and renal failure, whose examination led to the diagnosis of multiple myeloma and MIgG PGN.

Our patient was a 76-year old male who sought treatment due to renal failure and oedema, with two months evolution. The patient's medical history only mentioned arterial hypertension. A physical examination revealed pitting oedema in the legs up to the knees and no other relevant findings. Complementary tests revealed: haemoglobin: 7.7g/dl; creatinine: 3.4mg/dl; albumin: 2.1g/dl; total cholesterol: 232mg/dl; IgG: 304mg/dl; IgA: 958mg/dl; IgM: 40mg/dl; C3: 79mg/dl; all other tests, including C4, rheumatoid factors, antinuclear antibodies, anti-DNA antibodies, anti-neutrophil cytoplasmic antibodies, anti-GBM, and cryoglobulin results were all normal/negative. Serology tests for hepatitis B and C virus and human immunodeficiency virus were all negative. Urine analysis: proteinuria: 11g/24h; urinary sediment: 6-12 leukocytes per field, 250 red blood cells per field, and negative urine culture. High resolution electrophoresis of urine and blood samples revealed a lambda IgA monoclonal component. A bone marrow core sample revealed a hypercellular medulla with 13% plasma cells of aberrant morphology, compatible with the diagnosis of lambda IgA type multiple myeloma. A chest x-ray, abdominal ultrasound, and computed axial tomography of the chest, abdomen, and pelvis all failed to produce relevant findings.

A percutaneous renal biopsy (23 glomeruli) revealed mesangial and endocapillary proliferation with splitting/thickening of the capillary walls (Figure 1); immunofluorescence revealed evidence of IgG, C3, C1q, and lambda chain deposits in the glomeruli studied; kappa chain stain test was negative; no relevant deposits were observed in the tubules; and Congo red stain was negative. In the electron microscope analysis, we observed fused foot processes, mesangial interpositioning, and subendothelial electrodense deposits; there were no fibrils or microtubules. These findings were compatible with the diagnosis of MIgG PGN. We treated the patient with dexamethasone and bortezomib. Three months later, the patient sought treatment for acute enteritis and bacteraemia from *Escherichia coli*, with deteriorated renal function that did not recover, and the patient was started on haemodialysis.

Nasr et al.[2] considered MIgG PGN as an entity defined by glomerular deposition of monoclonal IgG (predominantly IgG3), along with a light chain isotype, absence of tubular deposits, and electron microscope findings similar to those from cases of immunocomplex glomerulonephritis; in addition, these authors defined this condition as involving an absence of clinical and laboratory evidence of cryoglobulinaemia. In a 37-patient study, the most common histological forms were membranoproliferative GN and endocapillary proliferative GN; nephrotic syndrome and renal failure were the most common forms of presentation. In 30% of cases, there was also a monoclonal component encountered in serum samples, but only one case of myeloma; 10 patients had hypocomplementaemia. In another study,[3] membranous GN was the predominant form, and other haemopathies were encountered in addition to myeloma, such as chronic lymphatic leukaemia and non-Hodgkin's lymphoma. Sethi et al.[4] also described patients with membranoproliferative GN, associated with monoclonal gammapathy, with glomerular deposits of monoclonal IgG and IgM.

The differential diagnosis of MIgG PGN must take into account the aforementioned diseases, and especially Randall type MIDD (heavy/light chain deposition type).[5] In this disease, the most typical glomerular involvement is nodular glomerulosclerosis, although membranoproliferative GN can also be observed, tubular deposits are practically a constant, and the electron microscope findings would be different. However, some authors include MIgG PGN within the spectrum of MIDD.[6] For now, it is still not clear whether considering MIgG PGN as a distinct entity may produce therapeutic repercussions.

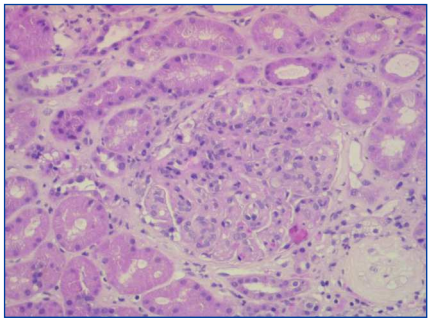

Figure 1. Light microscope image. HE x10. Glomerulus with mesangial and endocapillary proliferation and thickening of capillary walls.

In our patient, the immunofluorescence result of a unique light chain isotype suggests a type of nephropathy from monoclonal immunoglobulin deposition; the absence of tubular deposits and electron microscope findings were in favour of the diagnosis of MIgG PGN. There was a discrepancy between the monoclonal peak encountered in serum samples (lambda IgA) and in deposits (lambda IgG), which has already been described in another case of MIgG PGN,[7] and which would be due to a rapid tissue precipitation of the IgG component, or concentrations in serum samples that are below the detectable threshold.

To conclude, MIgG PGN must be considered as a possible diagnosis, among others, in patients with glomerular deposits of monoclonal immunoglobulin.

Conflicts of interest

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

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Amyloidosis AL with severe renal and cardiac involvement: a very rare association of terrible prognosis, two case reports

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To the Editor:

Amyloidosis is an uncommon disease produced by the deposition of fibrillar material that precipitates in the body tissues. The most commonly affected organs are: the kidneys (50%), heart (40%-50%), and peripheral nerves (25%), although it can affect any organ.[1] This disease implies a poor prognosis, with 80% mortality two years after diagnosis, despite treatment.[2]

Here we present two cases of primary amyloidosis that appeared initially in the form of heart failure (HF), hypotension, and progressive renal failure (RF): an uncommon form of evolution for this disease.

Both cases were female patients (aged 58 and 57 years) who sought emergency treatment due to symptoms of HF: one with right HF and the other with left HF. Both patients also had hypotension and mild oedema upon physical examination, and an initial laboratory analysis revealed previously undiagnosed RF (Table 1) with conserved diuresis. In both cases, an electrocardiogram revealed low-voltage sinus rhythm. Given the state of hypotension and signs of heart failure, both patients underwent electrocardiography that revealed a restrictive pattern of mitral filling, suggestive of hypertrophic cardiomyopathy (as opposed to restrictive). Simultaneously, we performed an analysis of RF, with ultrasound images revealing the kidneys to be morphologically normal. We determined the protein/creatinine (Cr) ratio, which was 2500mg/g Cr in one patient, and almost normal (66mg/g Cr) in the other patient. Given the finding in both cases of normochromic normocytic anaemia, with elevated sedimentation rates and renal failure, negative sediment results, and a restrictive pattern in ultrasound analyses, we established the preliminary diagnosis of a systemic infiltrative pathology such as amyloidosis, which led to tests for immunoglobulins and light chains in blood and urine samples. The results from these tests revealed a monoclonal gammapathy. We administered myelograms that confirmed the diagnosis of multiple myeloma (in the first case, lambda IgA, and in the second, lambda IgG), with 24% and 22%-

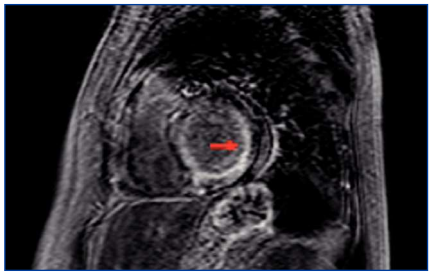

Figure 1. Cardiac magnetic resonance. Delayed gadolinium enhancement in the subendocardium.

40% infiltration, respectively. Given the suspicion of associated amyloidosis, we performed biopsies of the rectal submucosa, which were positive for Congo red stain tests and birefringence, confirming the diagnosis of AL amyloidosis. Both patients started treatment with bortezomib and prednisone, but the first patient experienced a poor evolution, requiring renal replacement therapy followed by the development of acute pulmonary oedema with cardiogenic shock that was not improved by vasoactive drugs, followed by death after a few weeks.

Amyloidosis is a systemic disease that affects several organs at the moment of diagnosis. In primary amyloidosis, the protein deposits include light chains from the immunoglobulins produced by clonal proliferation of plasma cells, primarily due to multiple myeloma. Asymptomatic deposits of amyloid material can be observed in 30% of patients,[1,2] and 10%-15% develop symptomatic AL amyloidosis.[3,4] Both myeloma and amyloidosis can produce renal manifestations: renal involvement in multiple myeloma is multi-factorial, although the most common finding is referred to as "myeloma kidney" (60% of cases), which is characterised by tubulo-interstitial damage that is clinically expressed as acute or chronic RF due to tubular light chain precipitation. The majority of patients progress with proteinuria, which is non-selective in 90% of cases, and 25% of these patients develop nephrotic syndrome. Patients with vascular involvement develop only mild proteinuria, but RF continues to progress due to the decrease in renal flow.

The heart is another organ often implicated in amyloidosis. Cardiac involvement can