by protein expression induced by intratubular-free haemoglobin, by tubular lesions,[5] and sometimes by association with interstitial nephritis.[6] Praga, at al[1] described the relationship between acute tubular changes and the percentage of haematic casts and MH duration, although other mechanisms may exist, such as the presence of necrotising glomerular or extracapillary proliferative lesions[2,7] as possible triggers of AKF in the MH episode.

Progress is favourable by stopping macroscopic haematuria[3,6]; however, some patients benefit from steroids if they have prolonged haematuria, are over 50 years of age, or have had previous kidney damage.[8] Immunosuppressive drugs are not recommended, unless a massive extracapillary proliferation or signs of acute vasculitis coexist.

In summary, MH in cases of glomerulonephritis different to IgA can become complicated with acute failure due to tubular necrosis and intratubular haematic casts. Its pathogenesis is not fully known and it seems that steroids may be effective in the most serious cases.

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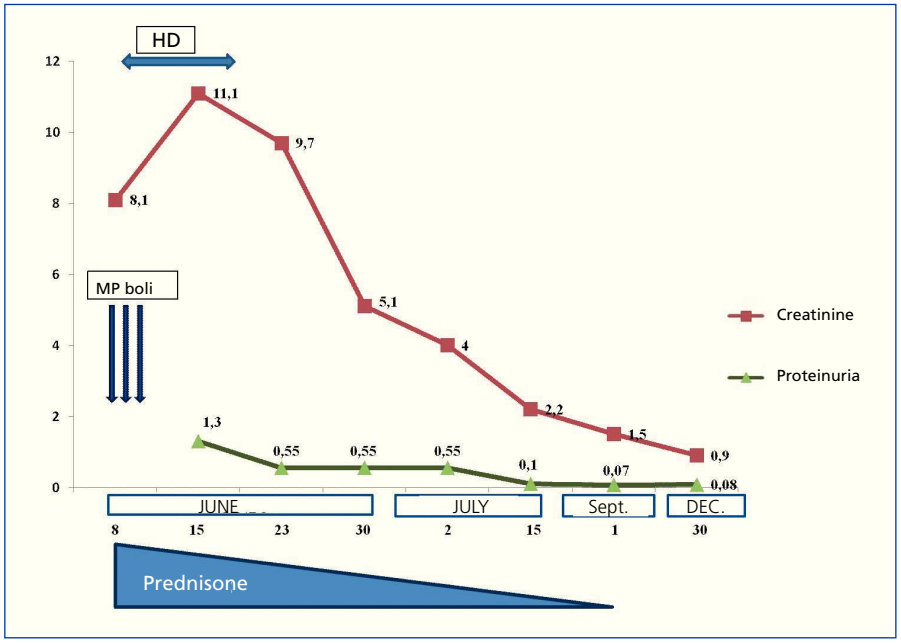

Figure 2.

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Libman-Sacks endocarditis and severe aortic regurgitation in a patient with systemic lupus erythematosus in peritoneal dialysis

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To the Editor,

Libman-Sacks endocarditis is the most classic heart disorder associated with systemic lupus erythematosus (SLE) and is a serious cause of morbidity and mortality. For some patients undergoing peritoneal dialysis (PD) lupic activity markers remain positive after having started treatment, with accompanying clinical symptoms, especially serositis or vasculitis.

We present the case of a 46-year-old female, affected by advanced type IV lupus nephropathy, undergoing a PD pro-

gramme since February 2009. Since then, she has presented with positive markers. She was suffering flare on her skin and joints, and received sodium mycophenolate at a dosage of 180mg and prednisone at 5mg daily. She is on the waiting list for a kidney transplant. The only PD complication that she experienced was an episode of peritonitis, in June of the same year.

She was admitted because of dyspnoea and general progressive discomfort, which had lasted for 15 days. During the last days she had chest pain in the left hemithorax, which increased when she breathed deeply and improved relatively in anteversion. She did not have a fever or any other clinical symptoms.

In the physical examination, a diastolic murmur was found in the aortic area, which extended to the carotids, with a significant pericardial friction, but there were no signs of heart failure. The rest of physical examination was normal.

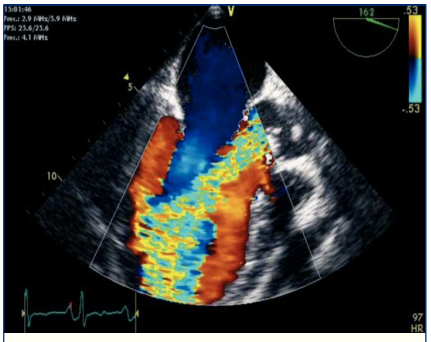
The biochemical tests found: leukocytes: *21.3K/μl (4.4-11.3), band cells: 3%, neutrophils: * 92.0% (50-70), lymphocytes: * 3.0% (25-40), C-reactive protein (CRP): * 17.73mg/dl (0.1-0.5), procalcitonin: * 4.84ng/ml (<0.5). Antinuclear antibodies (indirect immunofluorescence [IIF]): 320-640 u arb (0-80), anti-DNA antibodies (enzyme immuno-assay): 1.3ml (<10IU/ml), anti-DNA antibodies (IIF): 80-640 u arb (0-80), cardiolipin antibodies (IgG): 3.5IU GPL/ml, IgM: 2.5IU GPL/ml (negatives), C3: 118mg/dl (79-152), C4: 24.4mg/dl (16-38), urea 127, creatinine: 8.46mg/dl; haemoglobin (Hb): 9.6g/dl, haemocrit: 29%. Cephalin time test: 27.9/30 seconds (29-31s), positive lupus anticoagulant.

A cardiac ultrasound was performed, showing massive aortic regurgitation (AR) with a preserved left ventricular ejection fraction (LVEF) (69%). Three vegetations were found in the aortic valve, and the largest one measured 20mm. A pericardial haemorrhage was also observed without signs of heart block or thrombi (Figures 1 and 2).

All of the blood and serum cultures were negative. Chest, abdominal and cranial computed tomography (CT) did not show any significant changes.

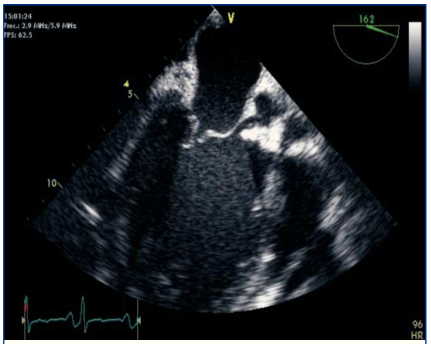
Daily dialysis was indicated given the evidence of pericarditis and the uraemic evidence. With the diagnosis of endocarditis and awaiting the haemocultures, treatment with empirical antibiotics was started with vancomycin + ceftazidime + gentamicin. Given the suspicion of lupus activity, the prednisone dose was increased to 60mg/24 hours and the sodium mycophenolate dose to 180mg/12 hours.

After eight days of treatment, a control echocardiogram was performed which showed that the pericardial leakage had



Regurgitation in the left ventricle compatible with massive aortic regurgitation

Figure 1. Massive aortic regurgitation.



Severe aortic regurgitation with large vegetation adhered to the right coronary sigmoid.

Figure 2. Large vegetation adhered to the right coronary sigmoid

reduced and that the severe AR was persistent, with an image of swaying vegetation. Lastly, blood cultures were negative.

Given the severity of AI, it was decided that the valve should be repaired surgically, and a mechanical prosthesis placed. The anatomopathological diagnosis of the valvular piece informed of an aortic endocarditis with no evidence of microorganisms (Libman-Sacks endocarditis). On the pericardium a fibrinous chronic pericarditis was observed.

At present, two months after heart surgery, the patient has returned to the PD programme, with no clinical signs of lupus activity, maintaining positive immunological markers and undergoing treatment with sodium mycophenolate at a dose of 180mg/12 hours and prednisone at a dose of 50mg/24 hours.

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Intravascular haemolysis and renal failure

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To the Editor,

It is well known that acute haemolysis is a cause of acute renal failure due to tubular damage caused by pigments being deposited in the proximal tubule. Maintained haemolysis can produce chronic renal damage, caused by different mechanisms.

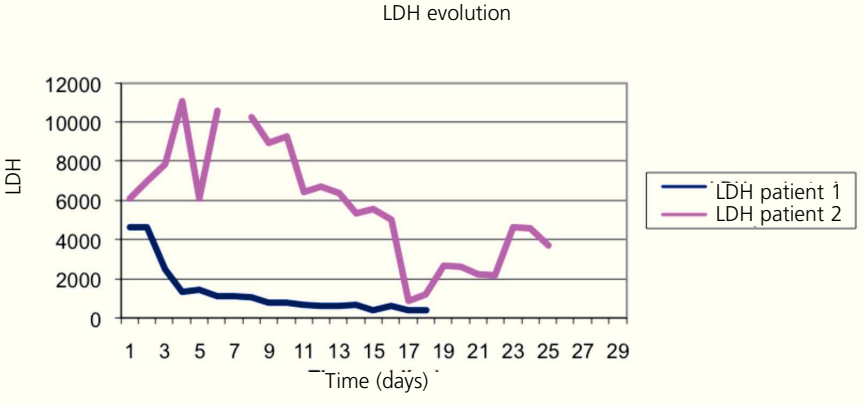
We present two patients with intravascular haemolysis produced by different causes, both with acute renal failure but different progression.

A 57-year-old male, crop-sprayer, was admitted to the haematology department due to non-immune haemolytic anaemia (negative direct Coombs test). He had organophosphate poisoning, which had lasted 4 days, with oligoanuria and acute renal failure. Haemolysis stopped after two plasmapheresis sessions. Upon admission he presented with: haemoglobin (Hb): 6.3g/dl, haemocrit (Ht): 19%, leukocytes 30 630/μl (Ne 77.7%), platelets: 217 000/μl. Blood smear: intense anisocytosis, polychromatophilia, microspherocytes (7-8/field). occasional basophilic stippling, presence of erythroblasts. There were no schistocytes. Haptoglobin: 10.4mg/dl. Total bilirubin: 8.60mg/dl. Conjugated bilirubin: 2.70mg/dl. Indirect bilirubin: 5.90mg/dl. Myoglobin 170.5μg/l, lactate dehydrogenase (LDH) 4637IIU/l (Figure 1). Iron: 242μg/dl; ferritin: 2754ng/ml; Urea: 188mg/dl; creatinine: 2.95mg/dl (Figure 2). Urine: proteins +++. Haemoglobin: ++++, 10 red blood cells/field. Renal function worsened gradually, presenting with anuria and a glomerular filtration rate of 6ml/min 24 hours after, which required 17 sessions of haemodialysis. Renal function was completely recovered after 2 months.

A 71-year-old male, with a metal aortic and mitral prosthesis because of a rheumatic valve disease. He was admitted to the cardiology department to stop a periprosthetic mitral valve leak. He presented with acute haemolytic anaemia secondary to attempting a percutaneous closure and chronic haemolysis. He had baseline Hb: 10.6g/dl and LDH: 1500-2000IIU/l. Baseline renal function: 71.86ml/min, with Cr: 1.85mg/dl. During this episode he presented with Hb: 7.6g/dl; haematocrit: 25.2% ; leukocytes : 7070/μl (Ne: 74.5%) ; platelets: 261 000. Smear: abundant schistocytes. Haptoglobin: <7.56mg/dl. Total bilirubin: 4.80mg/dl; direct bilirubin: 0.6mg/dl; LDH: 10 500IIU/l (Figure 1); normal iron profile; urea: 83mg/dl; Cr: 2.46mg/dl (Figure 2). Urine: proteins: 150mg/dl; haemoglobin: ++++, 47 red blood cells/field. Renal function was maintained stable with conservative treatment. Following the surgical closure, the leak stopped the haemolysis and the renal function recovered up to a glomerular filtration rate of 57ml/min, with Cr 1.7mg/dl.

Intravascular haemolysis of any cause can produce acute tubular necrosis, due to haemoglobinuria. It presents with red/brown urine and plasma, low haptoglobin, elevated LDH, deteriorated renal function and fractional excretion of sodium less than 1%. The incidence is unknown, reaching 50% in massive haemolysis.[1,2]

Haemoglobin is released to the plasma, binds to haptoglobin and is degraded by the reticuloendothelial system. When the haptoglobin is saturated, the free haemoglobin goes from its usual



LDH: lactate dehydrogenase.

Figure 1. Comparing LDH evolution in two patients.