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, haematocrit (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) 4637IU/l (Figure 1). Iron: 242µg/dl; ferritin: 2754mg/ml; Urea: 242mg/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 anuria and acute renal failure. Haemolysis stopped after two plasmapheresis sessions. Upon admission he presented with Hb: 10.6g/dl and LDH: 1500-2000IU/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 500IU/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 closure and chronic haemolysis. He had baseline Hb: 10.6g/dl and LDH: 1500-2000IU/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 500IU/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.
Comparing evolution of creatinine figures in two patients.

**Figure 2.** Comparing evolution of creatinine figures in two patients.

HD patients submitted to kidney transplantation and treated with immunosuppressive agents are at higher risk of developing tuberculosis, commonly being atypical and extrapulmonary. We present a case of two patients submitted to kidney transplantation with extrapulmonary tuberculosis in an uncommon localisation.

A 66-year-old female, with chronic kidney failure secondary to hepatorenal polycystosis, which received a deceased-donor kidney transplant and treatment with basiliximab, steroids, mycophenolate mofetil and tacrolimus. She suffered a type IIb cortical-resistance acute rejection and needed treatment with OKT3. After four months she was admitted for fever, general discomfort and intense asthenia. She was diagnosed with suspected pulmonary tuberculosis by chest computed tomography (CT) and fibrobronchoscopy, confirmed by Ziehl-Neelsen staining and Löwenstein culture. She was prescribed treatment with rifampicin, isoniazid and pyrazinamide for two months, followed by rifampicin and isoniazid for four months. After 15 days she was readmitted for confusion, occipital cephalgia and visual alterations. In a brain resonance, multiple hypertensive nodules were seen in T2, with nodular focal contrast in the right frontal, subcortical, suprasylvian, right occipital areas and in cerebellar peduncles, indicative of granulomatous inflammation secondary to tuberculosis (Figure 1). Treatment with isoniazid and rifampicin was extended to nine months and the patient recovered.

A 41-year-old male, diagnosed with hepatorenal polycystosis received a deceased-donor kidney transplant with immunosuppression with cyclosporin and steroids. He suffered a grade IIb acute interstitial rejection, treated with...