We report two cases of live donor RT with low ABO incompatibility who developed PLS.

CASE 1

A 23-year-old male with stage V chronic kidney disease (CKD-V) due to polycystic kidney disease. He received a live donor RT on 6 June 2011, with the donor being group O+ and the recipient being group A+, without initial complications and with functional graft at discharge (plasma creatinine [Pcr] 1.1mg/dl) and haemoglobin 10g/dl. Immunosuppressive therapy consisted of tacrolimus, mycophenolate, and steroids.

Fourteen days after transplantation, laboratory tests were performed and we observed haemoglobin of 5.8 g/dl with stable renal function (Pcr 1.2mg/dl) and the remaining complementary tests (including CT and abdominal ultrasound) were within the normal range.

The anaemia work-up showed haemolytic anaemia (low haptoglobin and high reticulocytes and direct bilirubin) with positive direct Coombs test and appearance of anti-A antibodies in the patient's red blood cell preparation, compatible with alloimmune haemolytic anaemia (PLS). The patient was treated with transfusion of 4 packed red blood cell units and methylprednisolone (1mg/kg/day) with subsequent gradually decreasing amounts. Tests at discharge: Pcr 0.7mg/dl and haemoglobin 9.6g/dl. In the following weeks, there was complete resolution and no recurrence.

CASE 2

A 42-year-old male with CKD-V due to diabetic nephropathy. He received a live donor RT on 10 September 2012. The donor was blood group O+ and the recipient was A+. The initial immunosuppression included tacrolimus, mycophenolate and steroids. After the RT progression was very good. At discharge he had haemoglobin of 11.4g/dl and Pcr of 1.4mg/dl.

Fourteen days later, he sought consultation due to general malaise and severe haemolytic anaemia was detected (haemoglobin 4.3g/dl). Complementary tests ruled out active bleeding. The blood smear did not show schistocytes and the direct Coombs test was positive with presence of anti-A antibodies. He was diagnosed with alloimmune haemolytic anaemia (PLS). He was treated with transfusion of 12 packed red blood cell units and high-dose steroids (methylprednisolone 1mg/kg/day) with a progressive decrease in dosage. The blood abnormalities cleared within ten days and there was haemoglobin stability, without further transfusions being required. 31 days after transplantation, he was diagnosed with II-B acute rejection with negative donor-specific antibodies, and treatment with thymoglobulin was required. Renal function subsequently stabilised, with Pcr of 3.2mg/dl.

PLS must be suspected for sudden anaemic symptoms in the first-second week after transplantation, in SOT with low ABO incompatibility or different Rh.[1-5] Its duration is limited in time (about 3 months).[1-3] Blood transfusion of the donor group and steroid administration are recommended. In severe cases, rituximab and/or plasmapheresis have been used.[1-5] Immunosuppressive therapy with mycophenolate is recommended for its effect on B cells.[1,5] Prevention measures, such as careful graft perfusion and removal of lymph nodes from perirenal fat[1,3] are particularly important. In our cases, the two grafts came from living donors. It is possible that the lower cold ischaemia time and higher speed in the implantation process also favoured the development of PLS, due to the greater number and viability of the donor's lymphocytes.

Conflicts of interest

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

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Bariatric surgery, a new cause of acute renal failure

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To the Editor:

Obesity, defined by a body mass index (BMI)> 30 kg/m^2, is a global public health problem, on an epidemic scale. The prevalence of obesity in the Spanish adult population (25 years-60 years) is 14.5%, while the figure for overweight is 38.5% and it increases each year. Obesity is associated with other cardiovascular risk factors such as high blood pressure (HBP), insulin resistance, type 2 diabetes mellitus (DM), dyslipidaemia and coronary disease, and

can lead to progressive renal failure (RF). RF associated with obesity is a glomerulopathy with variable proteinuria and histopathological findings of focal segmental glomerulonephritis.[1] Currently, the treatment for obesity includes bariatric surgery when the ponderal target is not achieved with medical treatment, and even in RF associated with obesity. But it is not an innocuous surgical procedure and can cause RF *per se*.[2] We report two cases of acute renal failure (ARF) following bariatric surgery, recorded at our service.

CASE REPORTS

Case 1

A 60-year-old male with a history of morbid obesity (BMI 47kg/m^2), high blood pressure, type 2 DM, obstructive sleep apnoea syndrome (OSAS), hypercholesterolaemia and acute myocardial infarction ischaemic heart disease was treated with 2 stents after percutaneous-stent transluminal coronary angioplasty. He was transferred to the Surgery department for bariatric surgery, using the laparoscopic sleeve gastrectomy technique. At discharge, he was treated with 10mg/24 hour ramipril, 10/25mg/24 hour bisoprolol/hydrochlorothiazide, 10mg/24 hour atorvastatin, 20mg/24 hour barnidipine and 1/8 hour metformin. After surgery, the patient reported low intake of food and water, occasional episodes of dizziness and loss of 20kg in a month. Given his progressive deteriorating clinical profile and the association with diarrhoea, he was admitted to the Emergency department, where general poor condition, bradypsychia, dry skin, mucous membranes and blood pressure (BP) of 92/65mmHg were observed. Tests were performed: glucose 214mg/dl, urea 403mg/dl, creatinine (Cr) 9.2mg/dl, Na 151mmol/l, Cl 113mmol/l, K 4mmol/l; Complete blood count: haemoglobin (Hb) 15.4g/dl, VH 44.5%, leukocytes 18,800/ul; platelets 287,000/ul; renal function in urine: urea 239mg/dl, Cr 339mg/dl, Na 52mmol/l, K 9.1mmol/l, fractional excretion of sodium (FENa) 0.9%; venous blood gases: pH 7.33, bicarbonate 11.1mmol/l, pCO_2 21mmHg. He was transferred to the Nephrology department with the diagnosis of with prerenal ARF, hypernatraemia and urinary tract infection. Antibiotic treatment and controlled electrolyte replacement was initiated with good response, with creatinine levels of 0.86mg/dl at discharge (Figure 1).

Case 2

A 44-year-old woman with the following history: ex-smoker, HBP, type 2 DM, morbid obesity (BMI 59.19kg/m^2), cholecystectomy, appendectomy, umbilical hernia and caesarean section. She was referred to General Surgery for bariatric surgery using the Larrad technique. She came to the Emergency department two months after surgery, complaining of repeated postprandial vomiting since discharge and syncope. In the physical examination, poor general condition, signs of salt and water depletion, BP 66/48 and CVP 4 cc H_2O were observed. A test was performed: urea 284mg/dl, Cr 6.98mg/dl, Na 119mmol/l, K 2.4mmol/l, Cl 65mmol/l, Pcr 4.8mg/dl, lactate 4.7mmol/l, osmolarity 333mOsm/kg; complete blood count: Hb 14.2g/dl, VH 42%; leukocytes 19,000/ul; platelets 251,000/ul; venous blood gases: pH 7.49, bicarbonate 25.9mmol/l, pCO_2 34mmHg; renal function in urine: FENa 0.11%, urea 276mg/dl, Cr 274.8mg/dl, Na 5mmol/l, K 15.4mmol/l. She was admitted to the Nephrology department with diagnosis of prerenal ARF secondary to volume depletion, hyponatraemia and hypokalaemia. She started on salt and water replacement and progressive electrolytic correction with improvement of renal function, until normalisation (Figure 1). During hospitalisation, the patient developed respiratory failure and septic shock secondary to respiratory infection. She was transferred to the intensive care unit and died after two months.

DISCUSSION

The treatment of morbid obesity includes: a) diet and modification of lifestyle, b) pharmacotherapy and c) bariatric surgery.[2] Bariatric surgery is indicated in patients with BMI ≥40kg/m^2 in whom treatment with diet and exercise, associated or not with drug treatment, is not effective and in patients with BMI>35kg/m^2 and obesity-related comorbidities such as HBP, glucose intolerance, DM, dyslipidaemia, or OSAS.

Bariatric surgery has high morbidity and its mean mortality rate at 30 days is 1%.

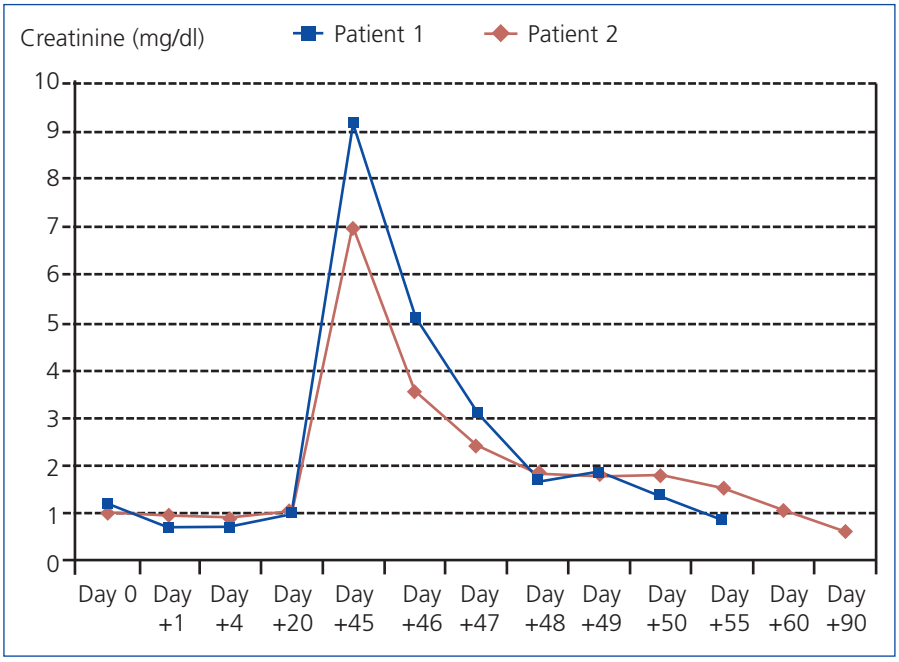

Figure 1. Creatinine progression (mg/dl) after bariatric surgery.

Surgical techniques are divided into restrictive, malabsorptive and mixed. The complications are different according to the surgical technique employed. Early complications of bariatric surgery are bleeding, perforation, deep vein thrombosis, pulmonary thromboembolism and pulmonary and cardiovascular complications, while late complications include cholelithiasis, malnutrition and neurological and psychiatric complications. Although bariatric surgery is an effective treatment for obesity and is even one form of treatment for associated RF, it may result in ARF[3] and very severe, life-threatening water-electrolyte imbalances, especially when the surgical technique used is the Roux-en-Y gastric bypass or a jejunoileal bypass. RF aetiology may be dehydration secondary to diarrhoea and/or vomiting[4] or ARF due to intratubular calcium oxalate crystal deposition. ARF usually appears a month after bariatric surgery and is characterised by high creatinine levels in relation to the patient's weight. The absence of nephrocalcinosis and urolithiasis and the fact that the ARF may have been prerenal and not parenchymal allowed us to rule out the diagnosis of ARF due to intratubular calcium oxalate crystal deposition. The treatment includes progressive hydration and correction of electrolyte abnormalities, haemodialysis in cases of established ARF and even conversion of the surgical technique employed in cases of oxalate-induced nephropathy.[3,4]

In conclusion, bariatric surgery is part of the therapeutic arsenal for obesity, but it is not without risks. Patients should be followed closely after surgery, with early referral to Nephrology if there is renal function deterioration, since it is a reversible form of RF if treated early, or the appearance of electrolyte disturbances that may put the patient's life at risk.

Conflicts of interest

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

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Successful treatment of acute thyroiditis due to Aspergillus spp. in the context of disseminated invasive aspergillosis in a kidney transplant patient.

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To the Editor:

Aspergillosis comprises a range of diseases caused by the fungus *Aspergillus*.[1,2] In 20% of cases, it appears as disseminated invasive aspergillosis.[3] In the case of transplantation, it is associated with high rates of morbidity, mortality and extended hospitalisation.[1] Its incidence varies according to the organ and in kidney transplantation, mortality reaches 70%.[3,4] *Aspergillus* is the fungus that most commonly affects the thyroid gland and it is generally confirmed in the autopsy.[5-7] Reports of invasive aspergillosis that has disseminated to the thyroid gland, diagnosed *ante mortem* and treated in renal transplant patients, as in our case, are extremely rare.

CASE REPORT

A 49-year-old female received a deceased donor renal transplant. Induction therapy: anti-human thymocyte immunoglobulin, 5 doses of 1.5mg/kg/day. She displayed delayed graft function and urinary fistula. A biopsy revealed capillaritis with C4d-positive diffuse peritubular capillaries (50% positive), treated with three pulses of 500mg methylprednisolone and 400mg/kg/day intravenous immunoglobulin for five days. Immunosuppression: tacrolimus 8mg/day, mycophenolate sodium 1440mg/day and methylprednisolone 4mg/day. Prophylaxis for cytomegalovirus and pneumonia due to *Pneumocystis carinii*: valganciclovir and trimethoprim-sulfamethoxazole.

40 days after transplantation, the patient developed urinary tract infections due to *Pseudomonas aeruginosa* and *Escherichia coli*. Fifteen days later, she was admitted with dyspnoea and fever. A thoracic computerised tomography scan revealed bilateral pulmonary infiltrates, and images of consolidation and cavitation in both lungs. We performed bronchoalveolar lavage and a galactomannan antigen assay by ELISA (Platelia® Aspergillus, BioRad, France) with positive galactomannan of 1.20 units. Blood test: haematocrit 27.2%, haemoglobin 9.6g/dl, platelets 149 000/mm^3, leukocytes 691 000/mm^3, blood sugar 103mg/dl, urea 56mg/dl, creatinine 1.46mg/dl. The search for *Pneumocystis jiroveci* (*P. carinii*) with Giemsa and Gram-Weigert stain and