parathyroid hormone 581pg/ml, 25-OH vitamin D 9.2ng/ml, alkaline phosphatase 221U/l.

One week after the drug was administered, we detected hypocalcaemia (6.7mg/dl corrected calcium) in a routine clinic blood test. The patient was asymptomatic and because of significant history of poor adherence to treatment, she was urged to adhere to treatment as prescribed.

Two weeks later, the patient came to the Emergency department with general weakness, dizziness, tremors and nausea. Corrected calcium of 5.2mg/dl was detected. Physical examination revealed no signs of hypocalcaemia (Trousseau or Chvostek) or peribuccal paresthesia.

It was decided to admit the patient for intravenous treatment, which yielded good clinical and laboratory results.

Bisphosphonates are commonly used to reduce bone loss but they are mainly eliminated through the kidney, and as such, their use is not recommended in patients with severe RF.[2] Denosumab significantly reduces the risk of fractures[3] and it has been shown to increase bone mineral density and reduce resorption markers. Furthermore, preclinical studies have revealed that, unlike bisphosphonate, it is not nephrotoxic at levels of 100µmol/l[4] and is independent of the degree of RF, since its pharmacokinetic and pharmacodynamic profile does not vary significantly according to the degree of renal function.[1]

It has been hypothesised that patients with CKD and bone disease due to hyperparathyroidism may be at greater risk of hypocalcaemia with denosumab, since their serum calcium levels will depend to a greater extent on bone resorption mediated by the parathyroid hormone. As such, inhibition of osteoclast activity following administration of the first dose of denosumab could lead to a syndrome similar to hungry bone, with the rapid decrease in serum calcium levels due to its uptake by bone.[5] At this time, calcium and vitamin D supplements are required, until the formation of osteoclasts leads to proper mineralisation.

In a study[6] conducted on 46 patients with varying degrees of RF, pain in limbs and hypocalcaemia were observed as the most common adverse effects. The latter appeared in 15% of participants, with only two patients requiring hospitalisation for intravenous treatment and in only one, hypocalcaemia was symptomatic.

The use of denosumab in patients with CRF is convenient, as it requires no dose adjustment and it is not nephrotoxic; as such, its use is expected to increase. In this group of patients, we should administer appropriate calcium and vitamin D supplements before beginning treatment with denosumab, in addition to monitoring these parameters in the laboratory after treatment begins, since a non-negligible number of patients will develop hypocalcaemia, which will be asymptomatic in the majority of cases.

### Conflicts of interest

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


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**Isabel M. Martín-Baez, Raquel Blanco-García, Mario Alonso-Suárez, Cynthia Cossio-Aranibar, Laura V. Beato-Coo, Francisco Fernández-Fleming.**
Servicio de Nefrología.
Complexo Hospitalario Universitario de Vigo.
Pontevedra. (Spain).
**Correspondence:** Raquel Blanco García
Servicio de Nefrología.
Complexo Hospitalario Universitario de Vigo.
Pontevedra. (Spain).
raquelblancog@hotmail.com


## Passenger lymphocyte syndrome: an uncommon form of anaemia in renal transplantation


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**To the Editor:**

Passenger lymphocyte syndrome (PLS) is an alloimmune haemolytic anaemia in solid organ transplantation (SOT) caused by alloantibodies derived from a donor B cell clone, that are transferred with the graft, resulting in a secondary immune response against the recipient's red blood cells in SOT with compatible but not identical blood groups.[1-5] It is an uncommon entity whose frequency has been related to the size (in terms of lymphoid content) of the transplanted organ and is much more common in heart/lung (70%) and liver (29%) than in renal transplantation (RT) (9%).[2,3] The antibodies are normally anti-ABO, uncommonly anti-D, and there are isolated cases of anti-C, anti-E, anti-Kell, anti-Jk and anti-Fy.[3]

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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**Verónica Pascual-Pérez[1], Nerea Gómez-Larrambe[2], Rocío Collantes-Mateos[1], Teresa Visus-Fernández de Manzanos[2], José M. Urbizu-Gallardo[2], Auxiliadora Mazuecos-Blanca[1]**

[1] Servicio de Nefrología. Hospital Universitario Puerta del Mar. Cádiz. (Spain).

[2] Servicio de Nefrología. Hospital Universitario de Cruces. Barakaldo, Vizcaya.

**Correspondence:** Auxiliadora Mazuecos Blanca
Servicio de Nefrología.
Hospital Universitario Puerta del Mar. Cádiz. (Spain).
mauxiliadora.mazuecos.sspa@juntadeandalucia.es


# 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