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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Pilar Fraile-Gómez[1], Susana Domínguez-Quibén[2], Josepha Sebastía-Morant[1], Cristina Lucas-Álvarez[1], José M. Tabernero-Romo[1]

[1] Servicio de Nefrología. Hospital Universitario de Salamanca. (Spain).
[2] Unidad Docente Medicina Familiar y Comunitaria. Hospital Universitario de Salamanca. (Spain).

Correspondence: Pilar Fraile Gómez
Servicio de Nefrología.
Hospital Universitario de Salamanca.
Paseo de San Vicente 58-182. 37007 Salamanca. (Spain).
pilarfg@usal.es
pilarfg9@hotmail.com

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

nested polymerase chain reaction (PCR) and for cytomegalovirus DNA by real time PCR was negative. The wet mount microscopic examination with potassium hydroxide did not show intracellular yeasts or fungi compatible with histoplasma *capsulatum*. We did not find Koch bacilli or methicillin-resistant *Staphylococcus aureus*.

With a presumptive diagnosis of invasive pulmonary aspergillosis, voriconazole was introduced (200mg twice a day) and immunosuppression was discontinued. Two days later, enlarged thyroid gland and pain on compression were detected. An ultrasound revealed moderate goiter of left lobe prevalence with parenchymal hypoechogenicity and heterogeneous structure, changes in the left lobe: solid-cystic heterogeneous nodular lesion 27 x 16 x 23mm, thick peripheral hypoechoic halo without calcifications, peripheral vascularisation, solid hypoechoic lesions above nodule, relatively defined margins and heterogenous structure without cystic foci or calcifications, 14.8 x 10.8mm. Right lobe, 55 x 12 x 18mm, dispersed cystic foci, follicular colloid appearance, the largest 3.9mm. Left lobe 59 x 23 x 25mm, isthmus 6.1mm. The patient had no history of thyroid disease. The thyroid enlargement was sudden. Thyroid profile: thyrotropin 0.45µIU/ml, ultrasensitive peroxidase 7.6IU/ml, anti-thyroglobulin 10IU/ml.

The gland was punctured and purulent exudate was extracted with fungal filaments identified as *Aspergillus flavus*. After 48 hours, the fever and pain disappeared. Voriconazole treatment lasted 20 weeks. The patient made good progress and 45 days after diagnosis, an ultrasound of the gland revealed normal shape, size and structure. Left lobe, 48 x 17.5 x 16mm cystic nodule with peripheral halo of 21 x 14 x 13mm, denser, without microcalcifications. Right lobe, 52 x 15 x 14mm, colloid cysts, the largest 4mm. Isthmus 2.4mm. The galactomannan assay was repeated 60 days after the first assay with a result of 0.10 units. Plasma creatinine stabilised at 1.02mg/dl.

CONCLUSION

The galactomannan assay can detect aspergillosis before symptoms appear, but sensitivity and specificity in solid organ transplant patients are lower than in haematological patients.[8,9] With the results of this test, we suspended immunosuppression and administered voriconazole, since it is recommended to start treatment when symptoms appear.[9]

Voriconazole, the most used and most effective drug for treating invasive aspergillosis,[2,8] inhibits the activity of cytochrome P450-3A4; as such, the tacrolimus dose should be adjusted to prevent nephrotoxicity.[4] We used the recommended dose,[10] which in adults increases exposure by a factor of 2.5, the area under the concentration-time curve.[2]

Differential diagnosis becomes more complicated because the pain may be due to viral infection, but at the same time, the gland is resistant to infection.[5,6] In a similar case, it was thought that a patient had fungal pneumonia and subacute thyroiditis and she was treated with prednisolone unsuccessfully; aspergillosis was confirmed by puncture and voriconazole was administered with a positive result.[5] Another patient with acute myeloid leukaemia was diagnosed with *Aspergillus flavus* in the thyroid gland in the autopsy.[6] The diagnosis is only confirmed by aspiration[5] and potential false positives should be considered in the galactomannans assay.[3,8,9] In our case, the treatment lasted 20 weeks, the disease was resolved and this allowed renal function to improve.

Conflicts of interest

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

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Federico Cicora[1], Fernando Mos[1], Marta Paz[1], Javier Roberti[2]

[1] Servicio de Trasplante renal. Hospital Alemán de Buenos Aires (Argentina).

[2] Fundación para la Investigación y la Asistencia de la Enfermedad Renal (FINAER). Buenos Aires (Argentina).

Correspondence: Javier Roberti
Fundación para la Investigación y la Asistencia de la Enfermedad Renal (FINAER).
Austria 2381, 5. 1425 Buenos Aires, Argentina.
javierroberti@gmail.com
fcicora5@gmail.com