

Figure 1. Abdominal CT scan: coronal with complicated cyst in upper third right kidney and air in the urinary tract.

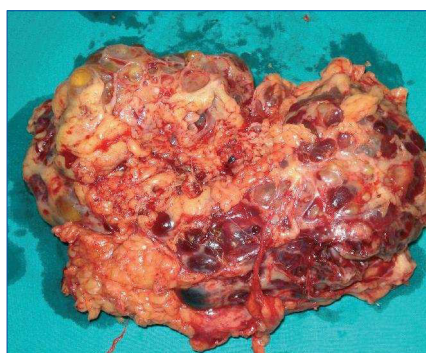


Figure 2. Surgical specimen: polycystic right kidney 20x14cm, 4kg, with multiple complicated cysts with pus.

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K. Salas Gama, C. Neyra Pérez, V. Esteve Simó, M. Ramírez de Arellano Serna

Nephrology Department. Terrassa Hospital, health Consortium of Terrassa, Barcelona, Spain.

Correspondence: Karla Salas Gama

Servicio de Nefrología. Hospital de Terrassa. Consorci Sanitari de Terrassa. Spain. ksalasgama@gmail.com

A case of IgD lambda multiple myeloma with acute kidney injury

Nefrología 2010;30(1):141-2

Dear Editor,

Monoclonal gammopathies include multiple clinical entities characterised by clonal proliferation of plasma cells that produce a homogeneous monoclonal paraprotein.¹ To identify this protein serum immunoelectrophoresis or immunofixation need to be used.¹ These monoclonal gammopathies can be classified as malignant, uncertain, idiopathic or transitory (the latter can occur in patients undergoing kidney transplantation due to immunosuppressive therapy, especially in older age).¹ Multiple myeloma or Kahler's disease is the prototype of malignant monoclonal gammopathy and is the most frequent neoplasm in plasma cells.¹ It occurs mainly in elderly males (60-65 years) and its clinical features include bone, kidney, neurological, and, of course, haematological disease. Kidney function is impaired in 50% of patients at the time of diagnosis.² It may be seen with tubular and/or vascular glomerular injury.² Kidney involvement is the single most influential factor in poor prognosis and in 50% of cases is triggered by hypercalcaemia.¹ The second mechanism of kidney injury is due to excessive production of immunoglobulins and/or light chains (Bence-Jones proteinuria) which are filtered by the glomeruli and saturate the kidney's ability to absorb by the proximal tubule and catabolise in the lysosomes.²

We report the case of a man aged 84 who attended emergency due to poor health, anorexia and weight loss during a month. His background revealed a history of hypertension and coronary artery disease. The physical examination showed a tendency to hypotension with discrete signs of dehydration without other relevant findings. The blood test gave: Hb 8.9g/dL, platelets 67,000/L, leukocytes 7,700 per μ L, creatinine 12mg/dL, urea

250mg/dL, K 6.7mEq/L, HCO_3 9mEq/L. Abdominal ultrasound displayed both kidneys of 12cm with good corticomedullary differentiation and without signs of obstructive uropathy. On one occasion, a proteinogram in blood and urine was requested in the ward, which detected an IgD lambda monoclonal peak in blood of 0.89g/dL and urine of 1.68g/24 h. 60-90% infiltration of plasma cells was detected in the bone marrow study. Chemotherapy with bortezomib, melphalan and steroids is prescribed after diagnosis of IgD myeloma with cast nephropathy (or myeloma kidney). Inclusion in a haemodialysis programme was requested urgently. After 6 months, there was no objective improvement of kidney function.

This case is interesting not so much for pathology, but for one of its subclasses. IgD monoclonal gammopathy represents 1% of all myelomas, compared with IgG and IgA myelomas, which constitute 53 and 28% respectively of the total.¹ The IgD myeloma takes a characteristically more aggressive course, and is associated with increased kidney disease rate (virtually 100% of the cases have Bence-Jones proteinuria).^{3,5} Myeloma clinical suspicion must always be present in the differential diagnosis of any cases of acute kidney injury, especially in elderly patients with a history of constitutional syndrome.

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T. Monzón Vázquez, A. Rodríguez Moreno, P. Delgado Conde, J. Delgado Domínguez, A. Gomis Couto, A. Barrientos Guzmán

Nephrology Department,
San Carlos Hospital Clinic, Madrid, Spain.

Correspondence: Tania Monzón Vázquez
Servicio de Nefrología.
Hospital Clínico San Carlos. Madrid. Spain.
taniarmv@msn.com

Supernumerary kidney

Nefrología 2010;30(1):142

Dear Editor,

We report the case of a 40 year-old patient with no urological history, who was admitted to our department because of pain in left kidney fossa, microhaematuria and pyuria. An examination showed tenderness in the left kidney fossa. The simple radiograph showed a radiopaque image in the left ureteral tract, compatible with calculi. With the clinical, laboratory and imaging data a left renoureteral crisis was diagnosed. In the antegrade pyelography, which confirmed the diagnosis, we observed a

smaller kidney with its own collection system in the cranial region of the left kidney, independent of the ipsilateral one (Figure 1).

The supernumerary kidney is the rarest of kidney malformations. It is always small and usually located caudal to the ipsilateral one, independent from it by its capsule and with its own irrigation and collecting system.^{1,2} It does not usually cause any symptoms until adulthood when it can generate pain, hypertension,

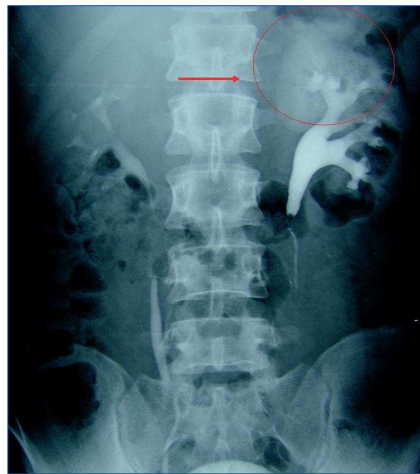


Figure 1. IV Urography

etc. It can occur in association with other diseases such as VATER syndrome³ or cardiological anomalies.⁴ The treatment will be the corresponding for the accompanying processes.

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M.A. Arrabal Polo, A. Jiménez Pacheco, M. Arrabal Martín

Urology Department. San Cecilio University Hospital, Granada, Spain.

Correspondence:

Miguel Ángel Arrabal Polo
Camino de Ronda, 143, 4.º F. 18003
Granada. Spain.
arrabal@ono.com

ERRATA

In the special edition, volume 29 number 5 “**Actualizaciones en Nefrología 2009**”, the authors included are incorrect. The correct authors are the following:

- In the Table of Contents, under the heading: **Manejo del paciente hipertenso anciano y muy anciano a la luz de las evidencias actuales**, the authors mentioned are: G. Fernández Fresnedo, M. Gago Fraile, They should be named: G. Fernández Fresnedo, J.M. Galcerán, M. Gorostidi, M. Gago Fraile.
- In chapter: “**Manejo del paciente hipertenso anciano y muy anciano a la luz de las evidencias actuales**” the authors mentioned are: G. Fernández Fresnedo, M. Gago Fraile, They should be named: G. Fernández Fresnedo, J.M. Galcerán, M. Gorostidi, M. Gago Fraile.
- In the Table of Contents, under the heading: **¿Hasta dónde con el bloqueo del sistema renina-angiotensina-aldosterona? ¿Qué aportan los nuevos fármacos y las nuevas combinaciones entre ellos en el tratamiento de la hipertensión arterial?**, the authors mentioned are: J.M. Galcerán, They should be named: J.M. Galcerán, M. Gorostidi, G. Fernández Fresnedo.
- In chapter: “**¿Hasta dónde con el bloqueo del sistema renina-angiotensina-aldosterona? ¿Qué aportan los nuevos fármacos y las nuevas combinaciones entre ellos en el tratamiento de la hipertensión arterial?**”, the authors mentioned are: J.M. Galcerán, They should be named: J.M. Galcerán, M. Gorostidi, G. Fernández Fresnedo.

We apologize to the journal's authors and readers affected.