and malignant hypertension. Intern Med 2004;43:496-502.
2. Arnaud L, Huart A, Plaiser E, et al. Anca-relatec crescentic glomerulonephritis in systemic sclerosis: revisiting the normotensive scleroderma renal crisis. Clin Nephrol 2007;68:165-70.
3. NanKe Y, Akama H, Terai C, et al. Rapidly progressive glomerulonephritis with D-penicillamine. Am J Med 2000;320:398-402.
4. García Porrua C, González Gay MA, Bouza P. D-penicillamine induced crescentic glomerulonephritis in a patient with scleroderma. Nephron 2000;84:101-2.
5. Arunachalam R, Thiraviam K, Tholappan R, et al. Scleroderma with crescentic glomerulonephritis: a case report. J Med Case Reports 2008;13:151-5.
6. Endo H. Scleroderma renal crisis: hypertensive and normotensive. Nihon Rinsho Meneki Gakkai Kaishi (Jpn J Clin Immunol) 2000;23:656-60.
7. Kamen DL, Wigley FM, Brown AN. Anti-neutrophil cytoplasmic antibody-positive crescentic glomerulonephritis in scleroderma-a different kind of renal crisis. J Rheumatol 2006;33:1886-8.

M. Polaina Rusillo, M.D. Sánchez Martos, G. Viedma Chamorro, I. Ruiz Ávila, V. Pérez Bañasco
Nephrology Department. Anatomical Pathology Department. Hospital Complex of Jaén.
Correspondence: Manuel Polaina Rusillo
Servicio de Nefrología. Servicio de Anatomía Patológica. Complejo Hospitalario de Jaén.
nefropolaina@yahoo.es

Not significant stenosis of renal artery in a single kidney does not counter-indicate placing an endovascular aortic prosthesis

Nefrología 2009;29**(3)**:282-283.

Dear Editor:

We present the case of a female patient aged 75 years who has been hypertensive for many years and has a urological history of recurring pyelonephritis, which in June 1992 required a nephrectomy of the right kidney. Since then, she has maintained stable renal function with a glomerular filtration rate between 35-40ml/min/1.73m^2 according to the simplified MDRD. In May 2002, a routine abdominal ultrasound examination detected an aneurism of the subrenal abdominal aorta 3.5cm in diameter that is checked periodically. An abdominal CT in July 2006 showed that the aneurism had grown to reach 5.6cm in diameter and that it was accompanied by stenosis of approximately 50% of the left renal artery at the point of origin and of the inferior polar renal artery that irrigated more than a third of the kidney (figure 1). Despite these technical difficulties, in February 2007 we proceeded to place an aortouniliac infrarenal endoprosthesis which left the exit of the polar artery and the left renal artery free (figure 2). From that moment, the patient has maintained a stable renal function with acceptable management of blood pressure.

Figure 1. Arteriography of the abdominal aorta and iliac arteries. We observe a single left kidney, the lumen of the aneurism on the infrarenal aorta, and a left renal artery with stenosis on its point of origin and a large-diameter inferior polar artery.

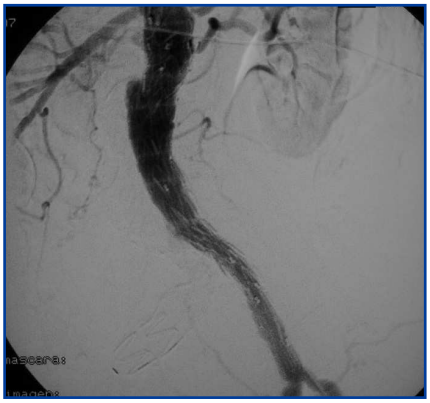

Figure 2. Arteriography of the abdominal aorta and iliac arteries with right aortouniliac infrarenal prosthesis.

Aneurisms of the abdominal aorta are a very prevalent condition, with an incidence that varies depending on age and sex. Aneurisms of the abdominal aorta are normally asymptomatic and are detected incidentally. On many occasions, the first sign is rupture (with a low risk for aneurisms ≤ 5.5cm in diameter, but high for larger diameters) with a mortality rate of 80% for those that break.

For years, elderly patients and those with multiple diseases were not treated surgically, but in 1991 endovascular prostheses arrived on the scene. This technique consists in the placement of a synthetic Y-shaped prosthesis with two arms, one for the abdominal aorta and an iliac artery, and the other for the contralateral iliac artery, which is inserted through the common femoral artery and guided using radioscopy. The prosthesis is located near the neck of the aneurism and the stents that are in place guarantee that it is in the right position, preventing it from shifting. There are also prostheses with windows for the renal arteries, for cases in which the aneurism affects them as well.

For the case in question, our priority was to preserve the inferior polar artery, which left a short aneurismatic pouch behind. For this reason, we used an aortouniliac prosthesis combined with a femoro-femoral bypass that guaranteed vascularisation of the left leg. At that time, we decided to do nothing about the stenosis of the renal artery, as it was not significant.

By placing endovascular prostheses, we now have less invasive treatment alternative; it has a low mortality rate

and provides an option to patients who are not approved for conventional surgery.

1. Greenhalgh RM, Powell JT. Endovascular repair of abdominal aortic aneurysm. N Engl J Med 2008;358:494-501.
2. Schermerhorn ML, O'Malley AJ, Jhaveri A, Cotterill P, Pomposelli F, Landon BE. Endovascular versus open repair of abdominal aortic aneurysms in the medicare population. N Engl J Med 2008;358:464-74.

M. Gago Fraile[1], G. Fernández Fresnedo[1], I. García Martín[2], S. Sanz de Castro[1], M. Arias[1]

[1] Nephrology Department. Marqués de Valdecilla University Hospital. Santander, Spain. [2] Cardiovascular Department. Marqués de Valdecilla University Hospital. Santander, Spain.

Correspondence:
Gema Fernández Fresnedo
Servicio de Nefrología. Hospital Universitario Marqués de Valdecilla. Santander.
nefffg@humv.es

Plasmapheresis as a co-treatment in acute renal failure secondary to myeloma kidney

Nefrología 2009;29**(3)**:283-284.

Dear Editor:

Acute renal failure is a relatively frequent complication in patients with multiple myelomas (MM). This complication is associated with a poor prognosis and shortened survival time.

There are many factors in its aetiology, including reduced volume, hypercalcaemia, tubulointerstitial nephritis and more rarely, hyperviscosity syndrome. However, the main cause of kidney damage is the excess of monoclonal production of light chains that cause obstruction on the tubular level; this is known as myeloma kidney.[2]

The main treatment for these patients is chemotherapy, which aims to reduce production of light chains. In those cases where there is renal failure secondary to intratubular deposits, haemodialysis and plasmapheresis constitute an adjuvant treatment option. The objective of these techniques is to eliminate the greater part of the light chains circulating in the plasma.

In the literature, evidence for the benefits of treatment with plasma exchange techniques is limited, although several studies do exist that show that plasmapheresis is more effective than haemodialysis.[1]

We present a case of acute renal failure secondary to myeloma kidney, with a good response to plasmapheresis, which led to recovery of renal function.

Clinical case

Female patient aged 59 years, with a history of hypertension and type-2 diabetes mellitus, diagnosed with monoclonal IgG gammopathy with progression subsequent to IgG lambda multiple myeloma. From the moment of diagnosis, various chemotherapy treatment schemes were administered, with no response. Six years after diagnosis, we were consulted for acute renal failure with conserved diuresis. The patient was in stage III-B, with analysis showing Bence Jones proteinuria and plasma creatinine of 7mg/dl.

Replacement therapy with combined conventional haemodialysis and plasmapheresis was initiated on a schedule of three sessions per week for one month, and one session per week thereafter. Initially, a plasma exchange volume of 3L was used, which was later increased to 4L when the patient changed to weekly sessions. The substitution liquid used was 5% albumin. Treatment resulted in a reduction in plasma creatinine of more than 50% and stable values were maintained, which allowed us to discontinue replacement therapy with haemodialysis.

Plasma levels of lambda light chains at the moment when treatment began were higher than 1,750mg/dl. With the start of adjuvant treatment, reductions of more than 60% were obtained in each plasmapheresis session. As we observe from figure 1, a rebound effect occurs between sessions, but the pre-plasmapheresis values are lower each time.

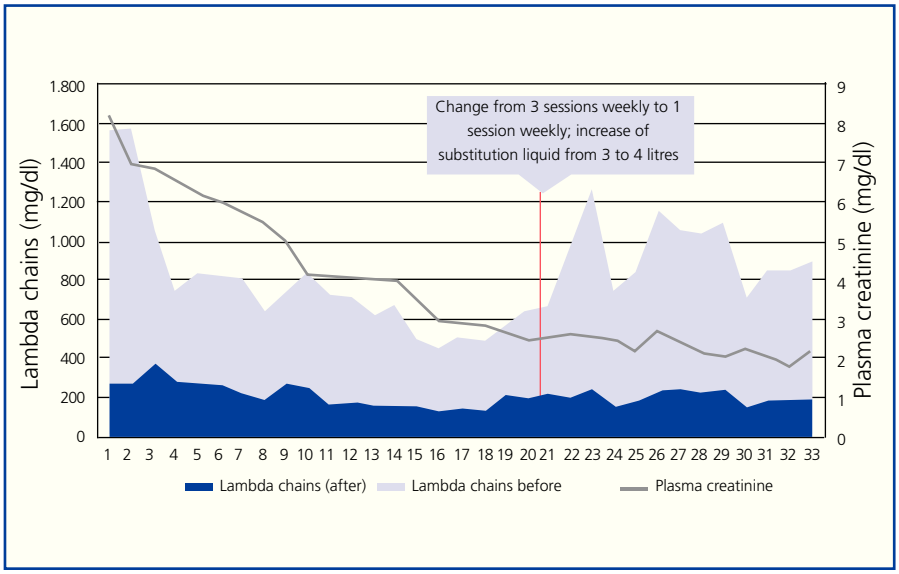

Figure 1. Evolution of plasma creatinine and levels of Lambda chains.