and a stent implanted. A month after this procedure, the patient came to the Emergency Room due to frank haematuria related with an excess of oral anticoagulants. The patient was admitted due to finding high levels of serum creatinine at 4.5mg/dl. A physical examination was performed and an arrhythmia was detected by cardiac auscultation, and cyanosis was detected in the first, third and fifth toes on the right foot. The systematic urine analysis showed blood +++ and the 24-hour protein count was 1.2g/day. Other analytical alterations that we detected were as follows: haemoglobin 10.6g/dl, eosinophilia 7.6%, and a decrease in complement C3, 69mg/dl (NV: 79-152) and complement C4, 11mg/dl (NV:16-32). In the kidney ultrasound, the kidneys appeared with cortical cysts, but with no other morphological changes and no sign of enlargement. The echocardiogram showed a decreased EF (29%). Due to lack of improvement of renal function, it was necessary to begin haemodialysis as replacement therapy. During the seventh haemodialysis session, 15 minutes after having begun, the patient went into cardiac arrest and could not be revived.

Given that sudden death had arisen from an unclear cause, a necropsy was carried out (thoracic-abdominal study), which revealed a severe aortic atheromatosis and a digestive haemorrhage in the ileum as the most relevant macroscopic findings. The microscopic study revealed the presence of cholesterol crystals in small-diameter arteries in the kidneys, stomach, spleen, pancreas and prostate.

Although CE may occur spontaneously in patients with atheromatosis who suffer a breakage in the plaque,[2] in most cases it is derived from inadequate treatment in invasive procedures (angioplasty or vascular surgery) and, over the long term, from anti-coagulation treatment.[3] Depending on the location of the plaque, migration of cholesterol crystals to multiple organs has been described; these include the central nervous system, the retina, coronary arteries, the pancreas and the adrenal glands. In the case of the kidney, such damage can manifest itself as acute (during the first week following the procedure), sub-acute (weeks or months later), or it can be chronic.[1]

In our case, although there was a high probability that the acute renal failure (ARF) was due to a CE, the autopsy was what confirmed that the kidneys were affected by cholesterol crystals, and that crystals were also present in other organs. There is no cure for CE, so the treatment options are based on symptomatic and preventative measures;[1,4] its mortality rate is high.[5] As with other forms of ARF, the mortality of the patients with CE is not due to ARF (in our case, the patient had begun replacement therapy and was on dialysis when he went into cardiac arrest). Rather, it results from the concomitant visceral ischaemia.[1] In this case, the ultimate cause of the patient's death may have been the haemorrhage in the ileum shown in the macroscopic study, which we cannot confirm as the pathological examination of the brain has not been carried out.


1. Meyrier A. Cholesterol crystal embolism: diagnosis and treatment. Kidney Int 2006;69:1308-12.
2. Cross SS. How common is cholesterol embolism? J Clin Pathol 1991;44:859-61.
3. Thadhani R, Camargo C, Xavier R, et al. Atheroembolic renal failure after invasive procedures. Natural history based on 52 biopsy-proven cases. Medicine 1995;74:350-8.
4. Belefant X, Meyrier A, Jacquot C. Supportive treatment improves survival in multivisceral cholesterol crystal embolism. Am J Kidney Dis 1999;33:840-50.
5. Jucgla A, Moreso F, Muniesa C, Moreno A, Vidaller A. Cholesterol embolism: still an unrecognized entity with a high mortality rate. J Am Acad Dermatol 2006;55:786-93.



**M. Heras[1], S. Salinas Moreno[2], M.J. Fernández-Reyes[1], R. Sánchez[1]**

[1] Nephrology Department. [2] Anatomical pathology Department. General Hospital of Segovia. Segovia, Spain.

**Correspondence:** Manuel Heras Benito
Servicio de Nefrología.
Hospital General de Segovia. Segovia.
manuhebe@hotmail.com


## Antiphospholipid syndrome and thrombotic microangiopathy

Nefrología 2009;29**(3)**:276-277.

**Dear Editor:**

Primary Antiphospholipid Syndrome (primary APS) is an illness that is characterised by thrombotic phenomena, which are due to the presence of antiphospholipid antibodies. In 25% of cases the kidneys can be affected; this can present as progressive kidney failure, proteinuria, sediment changes, renal infarction,[1,2] and less commonly, acute renal failure.

We would like to present the following case: female patient aged 65 years diagnosed with primary APS 20 years ago. Her personal history included several miscarriages, bilateral deep vein thrombosis, recurrent thrombophlebitis and lacunar stroke. Before she was admitted, her creatinine level was 1mg/dl, platelet count was 166,000/mm$^3$ and she was being treated with acenocoumarol.

She was admitted for symptoms of acute cholecystitis, for which surgery was required, and during post-operative period she experienced a fever of 38º C, lumbar pain and oligoanuria.

An analysis was performed that recorded an Hb level of 9.3g/dl, Ht at 26%, platelets 66,000/mm$^3$, creatinine 8mg/dl, urea 112mg/dl, K 4.8mg/dl, Ca 7.8mg/dl, LDH 790 and CRP 280mg/dl.

The test showed positive for anti beta-2 glycoprotein antibodies (55u/ml), lupus anticoagulant (88s) and anticardiolipine IgG (66u/ml); the rest showed no changes.

Systematic urine analysis: proteinuria, 0.7-0.9g/day; microhaematuria; and leukocyturia with granular casts.

Obstructive pathology and renal vascular pathology was ruled out with an abdominal CT.

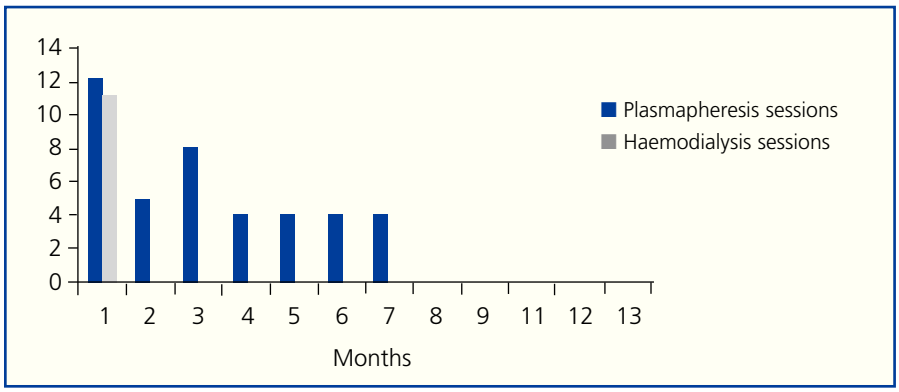


**Figure 1.** Number of haemodialysis and plasmapheresis sessions required during seven months (after three months, patient is re-admitted due to worsening profile).

Evolution: thrombotic microangiopathy was detected from the renal biopsy. Eight plasmapheresis sessions were administered in 17 days and the diuresis recovered progressively, although haemodialysis was still necessary during the first month. The patient was discharged and the plasmapheresis sessions became less frequent (figure 1).

Three months later, the patient was admitted again due to decreased renal function associated with abdominalgia, choroidal ischaemia and positive antiphospholipid antibody titres. The profile was interpreted as a relapse of the disease and plasmapheresis sessions were started again and administered during four months, which led to improvement in renal function and the ocular condition.

One year later, the platelets stabilised, the creatinine descended slowly (figure 2), and the antiphospholipid antibody titres were normal; there was no new thrombotic events.

The treatment of choice for primary APS is not well-defined[2] and varies according to the clinical presentation. Some experts recommend high doses of anticoagulants,[3] while others support the use of antiplatelet drugs or prophylactic anticoagulants in low doses. Other less successful treatments have used immunosuppressors and corticosteroids.[3] For patients on anticoagulants who suffer thrombotic events that severely affect the kidneys and/or other organs, plasmapheresis is an option,[4] as it was in our case.

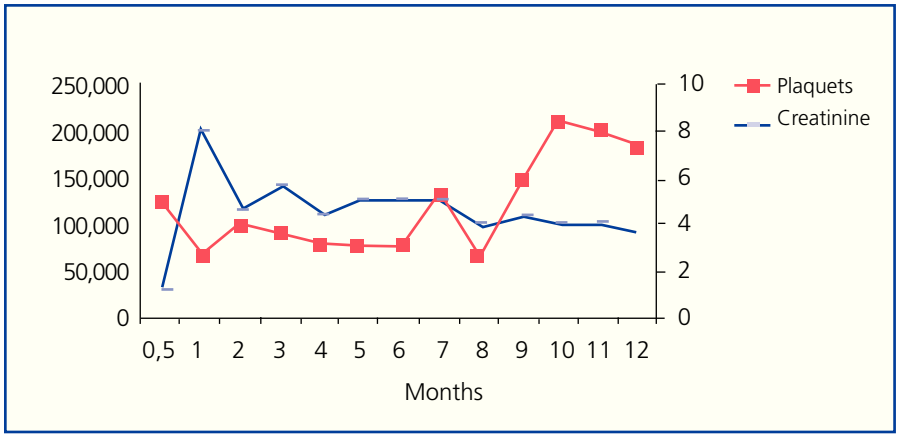


**Figure 2.** Development of the platelets and serum creatinine during the 12 months. A slow and progressive decrease in the creatinine coinciding with a platelet increase was observed.


1. UpToDate, Bonnie L Bermas, Doruk Erkan, Peter H Schur: Clinical manifestations of the antiphospholipid syndrome; En: UpToDate, Rose, BD (ed); vol 15.3, septiembre 2007, abril 2008. UpToDate, Waltham, Massachusetts, http://www.uptodate.com.
2. Joseph RE, Radhakrishnam J, Appel GB. Antiphospholipid antibody syndrome and renal disease. Curr Opin Nephrol Hypertens 2001;10:175-1.
3. Appel, Radhakrishnam, D'Agati. Síndrome por anticuerpos antifosfolípido, glomerulopatías secundarias. En: Barry M. Brenner, El Riñón. 7.ª ed. Madrid: Elsevier; 2005;1:1397-8.
4. Koschmieder S, Miesbach W, Fauth F, Bojunga J, Scharrer I, Brodt HR. Combined plasmapheresis and immunosuppression as rescue treatment of a patient with catastrophic antiphospholipid syndrome occurring despite anticoagulation: a case report. Blood Coagul Fibrinolysis 2003;14(4):395-9.



**O. Fikri Benbrahim, F. Levy, J.J. Santos Barajas, M.L. Carrasco Prado**
Nephrology Department. Healthcare Complex of Burgos. Burgos, Spain.
**Correspondence:** Oussamah Fikri Benbrahim
Servicio de Nefrología. Complejo asistencial de Burgos. Burgos.
fikrioussamah@yahoo.fr


## Pyelonephritis in crossed-fused renal ectopia

Nefrología 2009;29**(3)**:277-278.

**Dear Editor:**

Crossed-fused renal ectopia is the second most common variety of renal fusion, with an incidence of 0.01% in the general population. There are at least six varieties of crossed-fused renal ectopia, and it is thought to be produced by a change in the migration of the kidney due to a vascular obstacle, or due to genetic or teratogenic factors. It is generally associated with other alterations of the gastrointestinal and locomotor systems.