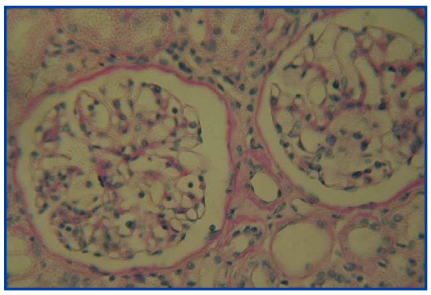

Figure 1. Renal biopsy of transplanted organ showing two glomerules with intact structure and a significant level of tubular atrophy (haematoxyline-eosin stain).

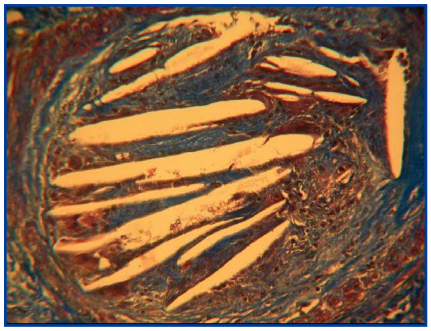

Figure 2. Renal biopsy of transplanted organ with a transversal slice of arcuate artery where we observe the impact of multiple cholesterol crystals (Masson's trichrome stain).

Complementary tests:

Analytical tests: plasma creatinine 5.5mg/dl, urea 131mg/dl, leukocytosis, normochromic normocytic anaemia, metabolic acidosis and elevated systemic inflammation parameters.

Doppler ultrasound: 12cm kidney, thin cortex, normal resistance indexes.

Renal biopsy: five glomerules with structure intact, without cellular alterations. Permeable capillaries. Tubular atrophy. In arcuate artery 100 micra in diameter, multiple cholesterol crystals occluding the vessel lumen. In the surrounding area, giant multinuclear cells and histiocytes.

Given with the irreversibility of the lesions and the persistence of creatinine clearance under 10ml/min, the patient resumed PHD.

Discussion

The cause was a spontaneous cholesterol atheroembolism in a patient with multiple CRFs and a previous episode of atheroembolism after an emergency treatment procedure. The literature states that in both transplanted and native kidneys, when there is no clear trigger factor and no symptoms on other levels, cholesterol atheroembolism is an occasional finding of a renal biopsy.[1-5]

Much data shows atheroembolism to be a cause of acute and sub-acute renal failure, and it is associated with a poor vital prognosis in affected patients.[1,2,5] It is still an under-diagnosed condition, and in many cases can present as an asymptomatic decrease in kidney function. In some series, it is found in 12% of all autopsies of patients with severe atherosclerosis.[2] There is little published data on atheroembolism in transplanted kidneys.[3,4]

The reviews by Ripple and Takats give a poorer prognosis to patients with early presentation of the atheroembolic event, just after the transplant or in the first year following it; most of these patients lose the transplanted organ.[3,4] For many of these patients, the source of the embolism is considered to be the donor, most of whom are older and have associated CRFs, and often receive multiple organ donations. Late cases are associated with a better prognosis, and frequently part of the renal function is recovered. However, our patient suffered a severe initial damage and lost the transplanted organ.

Cholesterol atheroembolism should be considered in the differential diagnosis of asymptomatic decrease in the function of a transplanted kidney.

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Sudden death in patient with cholesterol atheroembolism

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Dear Editor:
Cholesterol Embolism (CE) is a serious complication of invasive intravascular processes and anti-coagulation treatments in patients with arteriosclerosis and ulcerated aortic plaques. In addition to affecting the kidney, cholesterol crystals may also affect small-diameter arteries in other areas, such as the central nervous system, the coronary arteries, the mesentery, and the pancreas, damage to which is one of the principal causes of mortality in these patients.[1]

A male patient aged 78 years had a personal history of chronic kidney disease that was probably secondary to arterial hypertension and diabetes mellitus (baseline serum creatinine 1.5mg/dl), was a former smoker and had hypercholesterolaemia. In February 2007 he was diagnosed with ischaemic cardiopathy with a severely diseased vessel, and an angioplasty was performed

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.
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Antiphospholipid syndrome and thrombotic microangiopathy

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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.