Hypodipsia-hyponatraemia syndrome is characterised by persistent hypernatraemia that cannot be explained by loss of volume, absence of thirst, partial diabetes insipidus, and normal renal response to vasopresin.[2] Causes related to this syndrome are listed in Table 2. Its physiopathology seems to be related to an osmoreceptor disorder that makes these patients dilute and concentrate urine at inappropriately high levels of plasma osmolality.[3]

The most frequent type of dysnatraemia in patients with CRF and type 2 DM is pseudohyponatraemia secondary to hyperglycaemia or hypertriglyceridaemia; in CFR patients, hyponatraemia is usually much more common than hypernatraemia.[4-5] For that reason, in a patient with CRF and type 2 DM the presence of hypernatraemia, even on a single occasion, requires assessment and a differential diagnosis. The absence of polydipsia made us suspect the presence of hypodipsia-hypernatraemia syndrome, and we were able to identify and correct its cause.

Conflicts of interest

The authors affirm that they have no conflicts of interest related to the content of this article.

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Renal artery rupture during complicated recovery from angioplasty to treat renal stenosis

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To the Editor,

Renovascular hypertension is defined as high blood pressure (HBP) caused by renal hypoperfusion, and it results from renal ischaemia due to stenosis or occlusion of one or both renal arteries. One requirement for establishing this diagnosis is that reperfusion of the kidney reduces hypertension. This is one of the most frequent causes of secondary HBP, which is present in 1% of the general population and 30% of the population group with clinical characteristics suggesting this disease.

Another consequence of renal hypoperfusion is ischaemic nephropathy, which leads to renal atrophy and loss of nephrons, increasing the risk of progressing to end-stage renal disease, which causes chronic renal failure in 11%-18% of dialysis patients.

Atherosclerotic renal vascular disease may lead to ischaemic nephropathy, which is accompanied by severe bilateral dysfunction or overall renal ischaemia leading to treatment-resistant HBP. Atherosclerosis is a common and underreported cause of hypertension and renal failure. The risk of kidney atrophy depends on the degree of arterial stenosis and the disease progresses more rapidly in severe cases of stenosis.

Renal stenosis is the most common cause of secondary hypertension, with a prevalence of between 3% and 5% in patients with high blood pressure. Percutaneous transluminal angioplasty and revascularisation surgery are the two main options for treatment. The re-stenosis rate observed for renal stents is quite variable, ranging from 0% to 38% in recent studies.

Revascularisation surgery is the best treatment option for re-stenosis, but it is more complex.

We present the following case study in which we evaluate the management of renal stent surgery and its possible complications.

CASE STUDY

Female patient aged 67 years with multiple risk factors: HBP, hypercholesterolaemia, dysglycaemia, obesity, acute coronary syndrome, etc.

Patient with a solitary left kidney and stenosis due to stage 3 chronic kidney disease (KDOQI Guidelines) secondary to ischaemic renal disease with preserved diuresis.

An angiography revealed critical stenosis of the left renal artery and uncontrolled hypertension despite triple drug therapy (angiotensin converting enzyme inhibitors, beta blockers, calcium channel blockers, diuretics and alpha blockers). Preliminary tests revealed creatinine levels of 1.9-2.4mg/dl and intractable HBP.

Anaesthesia risk was moderate, and the patient underwent scheduled surgery to place a stent in the left renal artery under locoregional anaesthesia, with blood pressure under constant monitoring. During surgery, the patient remained haemodynamically stable with preserved diuresis.

During the postoperative period, she experienced sudden intense pain in the left renal fossa, accompanied by hypotension, vertigo and weakness and rapid-onset oligoanuria. She then experienced chest pain radiating to the back with no alterations on the electrocardiogram and no elevated cardiac markers. In any case, treatment for ischaemic heart disease was initiated. As stent thrombosis was suspected, angiography was performed via the left femoral artery, confirming thrombosis in the renal artery (doctors attempted thrombolysis without success) and also confirming migration of the stent, which could not be returned to its location. The patient was haemodynamically unstable during the procedure and required vasoactive drugs and a blood transfusion. She also experienced anuria, increased urea and creatinine levels, so a dialysis catheter was placed. She required high doses of vasoactive drugs to control the HBP, haemodiafiltration for the anuria, with increasing levels of urea and creatinine, and echocardiography to rule out cardiogenic causes. She presented blood clotting disorders with no signs of active haemorrhage, and rapid deterioration of general health with respiratory failure that required use of mechanical ventilation. An abdominal CT revealed a retroperitoneal haematoma and urgent laparotomy was performed. The patient's condition was complicated by the onset of distributive shock refractory to vasoactive drugs, systemic inflammatory response syndrome and anuric renal failure. It resulted in multiple organ dysfunction syndrome secondary to complications from the vascular surgery to correct renal ischaemia in a patient with a single kidney. The outcome was death, directly caused by shock refractory to treatment.

DISCUSSION

The strategy for treating atherosclerotic renal vascular disease has undergone significant changes in recent years. Initially, renal revascularisation procedures were intended to control HBP resistant to pharmacological treatment. Evidence suggesting that the progression of vascular stenoses of renal arteries could lead to progressive loss of renal parenchyma and parenchymal function changed this approach to treating the disease. At present, the main goal is to protect or improve renal function, whether by surgical or percutaneous methods of revascularisation.

Angioplasty has a high incidence rate of technical failure, and much effort has been spent in improving stent placement results. According to the most recently published studies, results seem to be better with 94%-100% of cases having improved renal function. In the studies we reviewed, survival rates after renal stent placement were high, blood pressure and renal function were better up to 5 years after surgery.

The indication of percutaneous renal angioplasty in renal vascular disease is a matter for debate, as it is known that creatinine levels above 3mg/dl are associated with low kidney survival rates.

In patients with a single kidney, the indication for revascularisation is even more controversial; some studies advocating medical treatment. The studies that we reviewed reported no significant differences in morbidity and mortality among patients with solitary kidneys and those with only one working kidney out of the two. We found differences in long term survival, with higher survival rates among patients with a normal contralateral kidney. Predictors of a favourable long-term clinical outcome include GFR above 30ml/min/1.73m^2, kidney larger than 9cm and no immediate decrease in renal function following the endovascular process. Surgery in solitary kidney patients is a safe process which improves or stabilises renal function in 87% of patients. Its clinical benefits depend on preoperative eGFR, kidney size and renal failure following the procedure, provided that no complications occur.

Conflicts of interest

The authors affirm that they have no conflicts of interest related to the content of this article.

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Sub-acute renal failure in patient with fever of unknown origin

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To the Editor,

In a typical case of sarcoidosis, the patient will present with pulmonary lesions, be diagnosed, and later develop renal disorder. Nevertheless, in rare cases, renal symptoms may precede pulmonary symptoms.[1-3]

In this article, we present the case of a male patient aged 18 years, employed as an animal caregiver. He was admitted to the emergency department of his referral hospital due to a long-term fever, and while hospitalised, he developed sub-acute renal failure. He was subsequently referred to our department for evaluation; with plasma creatinine (PCr) values of 6mg/dl.

He had experienced a high fever during 2 months, which began a week after a suspicious tick bite. Fever was present daily following no particular schedule, and he also had night sweating. The local hospital had provided him with different empirical antibiotic treatments for a suspected case of boutonneuse fever or other infectious diseases. Upon admission, antibiotic treatment had been discontinued for 20 days and fever persisted. Results from the analysis upon admission are shown in Table 1. Other relevant data: circulating immune complexes, 54.9μg/ml; 1.25(OH)2D, 8.6pg/ml; 25(OH)D, 8.1pg/ml; ACE, 23IU/l. All results were negative for a wide-range of infections. Thoracic x-ray: normal upon admission; 1 month later, signs of small bibasilar pulmonary infiltrates with halo sign. Axial computed tomography was normal. Renal ultrasound was normal. The lung scintigraphy image is shown in Figure 1.

The decrease in renal function initially suggested a pre-renal +/- post-infectious origin, possibly in association with acute tubular necrosis. During the first days, renal function improved progressively before worsening once again, with values returning to those measured upon admission (Figure 2). Proteinuria, which was initially in the non-nephrotic range (1-2g/24h), later reached the nephrotic range (Figure 2). In light of the above data, doctors decided to perform a renal biopsy (26 August 2011). After receiving the results from the biopsy and the lung scintigraphy, treatment was initiated with prednisone 1mg/kg/day; the fever resolved, PCr improved (1.5mg/dl) and proteinuria also improved.

Our final diagnosis was stage II chronic kidney disease secondary to sarcoidosis with mild hypercalcaemia and granulomatous interstitial nephritis +/- associated with glomerular process, secondary hypoparathyroidism and distal tubular acidosis secondary to interstitial nephritis.

DISCUSSION

Hypercalcaemia is the most frequently detected abnormality in sarcoidosis, and is present in between 10% and 15% of cases. The mechanism leading to hypercalcaemia works due to activated macrophages in the lungs and lymph nodes being capable of increasing calcitriol production. In our patient, 1,25(OH) and 25(OH) vitamin D levels were low, possibly due to the long period of hospitalisation without exposure to sunlight. In addition, we detected severe suppression of parathyroid hormone (PTH) despite the fact that PTH levels should have been higher according to the patient's stage of chronic kidney disease. We concluded that the decrease in PTH levels was caused by hypercalcaemia, despite the fact that vitamin D values were not elevated.[4,5]

Approximately 20% of patients with sarcoidosis have a granulomatous renal disease. Granulomatous interstitial nephritis is common in sarcoidosis[6,7]; however, development of manifest clinical symptoms of renal failure is unusual. In

Table 1. Evolution of analysis results

	Admission 19/7/2011	At 6 days	At 16 days	At 29 days	Renal biopsy 29/8/2011	Start of treatment 9/9/2011	After 20 days	After 51 days
Haemoglobin (g/dl)	10	11.4	9.9	10.1	8	9.3	12	14.6
Diuresis (cc)	4000	5300	4500	3500	2200	2200	2200	4000
Urea (mg/dl)	70	44	70	54	41	58	49	47
Creatinine (mg/dl)	4.7	2.8	4.4.	4	3.5	2.3	1.5	1.6
Proteinuria (g/24h)		4.5	1.9	5.4	1.8			0.7
P-type calcium (mg/dl)	10.6	10.3	10.2	10.7	9.9	10	9.5	11.1
O-type calcium (mg/dl)		8			10			11.3
Parathyroid hormone (pg/ml)			10					25