hospitalised due to recent fever, pain and erythema at the subcutaneous tunnel, with mild draining at the catheter exit site. Doctors decided to provide systemic empirical antibiotic coverage with vancomycin and gentamicin.

The patient experienced pain in the left eye 2 days later, accompanied by redness of the conjunctiva and loss of visual acuity. Ophthalmological examination revealed an intraocular lens, hypopyon, Tyndall phenomenon +++, normal intraocular pressure, vitreous infiltration, macular thickening, vitreous abscess and detached choroid in the left eye. She was diagnosed with endogenous endophthalmitis based on a positive blood test for methicillin-resistant *Staphylococcus aureus*.

Given the poor anatomical and functional state, doctors prescribed vitrectomy with intravitreal vancomycin and ceftazidime injections in association with moxifloxacin, dexamethasone and cycloplegic agent eye drops. Vitreous humour cultures tested positive for *Staphylococcus aureus*, which confirmed the diagnosis. Treatment was maintained during 1 month; total detachment of the retina with proliferative vitreoretinopathy occurred as a complication.

Endogenous bacterial endophthalmitis is a devastating complication of septicaemia, which constitutes 2% to 8% of all cases of endophthalimitis.[1] It occurs when bacteria reach the eye through the bloodstream, by crossing the blood-retinal barrier. This is a severe disease which is often diagnosed late; its prognosis is very poor and many patients become blind.

Blood cultures may be positive in as much as 71% of patients with endogenous endophthalmitis, while vitreous humour and other aqueous cultures may test positive in 61% to 70% of cases.[2] These cultures are crucial for providing a diagnosis, but all other possible infection sites must be cultured, including catheters and draining lesions. It is not uncommon for eye cultures to be negative. The most common gram-positive bacteria are *Staphylococcus aureus*, Group B *Streptococcus, Streptococcus pneumoniae* and *Listeria monocytogenes*. Among gram-negative bacteria, the most common are *Klebsiella sp., Escherichia coli, Pseudomonas aeruginosa* and *Neisseria meningitides*.

Treatments for endogenous endophthalmitis include systemic and intravitreous antibiotics. Treatment duration should be determined based on the need to treat the underlying cause of bacteraemia. Systemic antibiotics alone will not be sufficient, which is why intravitreous injection of antibiotics is also necessary. Vitrectomy with intravitreous injection of antibiotics is indicated in most cases. Vitrectomy provides better visual results in severe cases of endophthalmitis.[3]

Endophthalmitis is a rare complication of septicaemia related to haemodialysis catheters. We have only found 5 articles on this entity, reporting a total of 8 cases.[4-7] In a case of suspected endophthalmitis, emergency ophthalmological assessment and antibiotic treatment are needed to reduce the risk of losing vision in the affected eye.

Conflicts of interest

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

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Transitory hypernatraemia and hypodipsia in a renal failure patient

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To the Editor,

We present the case of a female patient aged 71 years who was referred to our diabetic nephropathy department in April 2009. Her relevant medical history included high blood pressure and type 2 diabetes mellitus (DM2) for

Table 1. Evolution of patient's laboratory data

	First visit, April 2009	Second visit, August 2009	Third visit, November 2009. Ordered MRI	Preoperative analytical tests, February 2010	First postoperative February 2011	Analysis, results, August 2011
Urea (mg/dl)	100	105	110	111	91	106
Creatinine (mg/dl)	1.29	1.71	1.42	1.43	1.21	1.26
Uric acid (mg/dl)	8	5.6	6	5.6	6.2	5.5
Sodium (mmol/l)	138	138	147	140	140	139
Potassium (mmol/l)	4.9	5.2	5.3	4.4	4.8	4.9
HbA1C (%)	6.8	7.7	6.0	7	6.5	6.8
24 hour urine	3500	4000	4150	-	2900	3100
Urine protein (g/24h)	0.14	0.2	0.17	-	0.15	0.18
Microalbuminuria in 24h urine sample	Negative	Negative	Negative	-	Negative	-
Urine creatinine clearance (ml/min)	42.5	40.2	39.8	-	38.34	54.8

HbA_{1c}: glycosylated haemoglobin; MRI: magnetic resonance imaging

more than 20 years, with various diabetes-related complications (diabetic retinopathy); hyperuricaemia, dyslipidaemia, history of occasional treatment with non-steroidal anti-inflammatory agents, 7 pregnancies with 7 deliveries and no gestosis. Clinical measurements only revealed single-episode nocturia and significant loss of visual acuity in the right eye. Physical examination was normal: blood pressure 120/60mm Hg, weight 72.3kg and height 1.57m. The patient was being treated with alprazolam, furosemide, carvedilol, valsartan, calcium dobesilate and pravastatin. The patient provided an echocardiogram reading that showed moderate left ventricular hypertrophy, ejection fraction of 80% and grade I diastolic dysfunction. She also provided results from an analysis performed by her health centre (Table 1).

On her second visit in August 2009, she only provided analysis results from June 2009 (Table 1). In November 2009 on her third visit, the patient provided a more recent analytical test (Table 1) and an abdominal ultrasound, which showed a right kidney measuring 9.7cm and a left kidney measuring 9.4cm. Both presented good cortical/medullary differentiation. The patient stated that she had not brought the entire 24 hour urine output sample but that she did not have polydipsia. Since the patient presented hypernatraemia and polyuria, we requested a cerebral MRI without gadolinium. This test, evaluated in January 2010, showed a rounded expanding mass measuring 4cm compatible with parasagittal parietal meningioma in the right convexity, with no perilesional oedema and preserved meningeal grooves. The patient was referred to the neurosurgery division and underwent a medial craniotomy in April 2010 to remove bone infiltrated by tumour cells and have it replaced with a titanium mesh. Diagnosis based on histological results was grade I fibrous meningioma with focal bone infiltration. After being referred to the neurosurgery division, the patient did not make follow-up visits until she came in for an appointment in February 2011 in good general health and with improved visual acuity in the right eye. Her most recent evaluation was in August 2011; the patient remained asymptomatic, with no dysnatraemia and a slightly improved glomerular filtration rate (Table 1).

To resume, the patient had stage 3 chronic kidney disease (CKD), probably secondary to nephroangiosclerosis. On a single occasion during her follow-up period she presented with hypernatraemia, polyuria and euvolaemia, which led to a diagnosis of meningioma, and the tumour was subsequently resected. The differential diagnosis focused on the presence of hypernatraemia with normal total sodium levels. This would also include differential diagnosis for central diabetes insipidus or partial diabetes insipidus with hypodipsia.[1] The absence of polydipsia almost ruled out the diagnoses of diabetes insipidus or primary polydipsia.[1]

Table 2. Causes of hypernatraemia syndrome with hypodipsia

Meningioma
Germinoma
Craniopharyngioma
Teratoma
Hypophyseal adenoma
Metastatic bronchial carcinoma
Eosinophilic granuloma
Schüller-Christian disease
Sarcoidosis
Granulomatous tumour
Psychosis
Histiocytosis
Hypothalamic neuronal degeneration
Subarachnoid haemorrhage
Hydrocephalus
Cranial trauma
After surgical correction of cerebral aneurysm
Congenital hydrocephalus
Human chorionic gonadotropin-secreting tumour

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.