Table 1. Mean estimated baseline glomerular filtration rates in the study group broken down by sex

	Male	Female	*P*
Group 1 (MDRD (ml/min)	78.01 (7)	60.85 (8)	0.000
Group 1 (Cockcroft-Gault) (ml/min)	58.78 (11)	45.63 (9)	0.007
Group 2 (MDRD (ml/min)	43.60 (15)	37.30 (8)	NS
Group 2 (Cockcroft-Gault) (ml/min)	31.86 (16)	29.69 (6)	NS

Group 1: sCr ≤1.1mg/dl (6 males, 32 females). Group 2: sCr >1.1mg/dl (19 males, 23 females). NS: not significant.

In our study, we also found that eGFR (estimated using the two methods listed here) in women with sCr within the normal range was significantly lower than in men. However, these differences are not as pronounced in the patient group with altered sCr (Group 2). Our data therefore confirm a higher prevalence rate of CKD in women if they are evaluated by eGFR; this statement is especially true for the subjects in Group 1.

The differences in GFR between the sexes and study groups may lie within the mathematical formulae used to estimate GFR. The mathematical formulae used in our study are based on sCr, which involves the patient's muscle mass and nutritional state. Therefore, these significant differences in GFR between the sexes among patients with a normal sCr are more likely to show women's smaller muscle mass rather than their true GFR. It is also important to note that the MDRD formula was designed in patients with altered renal function and not validated in a healthy population: applying the MDRD formula to estimate GFR in individuals with a normal sCr may underestimate true GFR by up to 50%.[6] On the other hand, when we use the MDRD formula in patients with altered renal function, the differences between males and females are less pronounced. This means that the resulting eGFR may successfully show the presence of kidney disease rather than the patient's nutritional state and/or muscle mass.

In conclusion, sex is a factor to consider when checking for chronic kidney disease in the elderly. The systematic use of formulae based on creatinine levels can lead to healthy elderly women being considered carriers of occult renal disease.

Conflicts of interest

The authors declare they have no potential conflicts of interest related to the contents of this article.

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C) BRIEF CASE REPORTS

Adrenal myelolipoma associated with primary hyperaldosteronism

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To the Editor,

We present the case of a kidney recipient who recently underwent laparoscopic surgery for an adrenal myelolipoma associated with primary hyperaldosteronism. Myelolipomas are rare tumours; they are benign, grow slowly, and vary in size. They are made up of adipose and haematopoietic tissue. These tumours are typically non-functional and if they reach a large size, they can cause pain, pressure on adjacent organs and acute intratumoural or retroperitoneal bleeding.

The patient was a male aged 54 years, obese and a smoker, with long-standing hypertension (HT) and chronic kidney disease (CKD) secondary to malignant nephroangiosclerosis that was diagnosed by kidney biopsy in 2000. He started peritoneal dialysis in 2006 and underwent a deceased donor transplant in 2008. Previous x-ray studies already showed a right adrenal mass compatible with a myelolipoma; in 2005, it measured 5*5.4cm in

diameter. When we were monitoring the patient's CKD in our department, the patient also presented persistent hypokalaemia due to hyperreninemic hyperaldosteronism secondary to the underlying disease (malignant HT), which ruled out the possibility of a functional adrenal mass. However, as part of the pre-transplant study in 2008, the patient underwent a CT-guided needle biopsy of the adrenal mass, and the results from the histological study suggested a myelolipoma, thus confirming the initial diagnosis. As the tumour was benign, it did not contraindicate kidney transplantation.

During the two-year outpatient monitoring period following the transplant, the patient presented refractory HT requiring six different hypotensive drugs to achieve rather poor blood pressure control. His renal function deteriorated slowly over this time, and he presented proteinuria and microhaematuria. The persistent hypokalaemia reappeared and doctors ordered a new hormonal study. This time, the study found high plasma aldosterone (1098pg/ml) and suppressed plasma renin activity (0.13ng/ml/h). The patient was then diagnosed with primary hyperaldosteronism and the CT and MRI scans were repeated; the adrenal mass had reached 12*5cm in diameter along the cranio-caudal plane and 10cm in diameter along the transversal plane. It contained mainly fatty tissue with dense soft tissue areas. The patient was referred to the General Surgery Department, and in March 2010, underwent laparoscopic right adrenalectomy with excellent and prompt recovery.

The histological study showed an adrenal myelolipoma with hyperplasia of the adrenal cortex (zona glomerulosa) secondary to the pressure exerted by the large size of the myelolipoma. This explained the patient's primary hyperaldosteronism, even though the tumour was benign and non-functional.

We could reduce the hypotensive drugs by half in the post-operative phase. The patient now has excellent control over his hypertension with the aid of two hypotensive drugs and blood potassium levels are normal, which suggests that the renal hyperplasia was not bilateral and was clearly associated with the myelolipoma.

We found cases of myelolipomas associated with arterial HT in the literature, but the tumours have never been shown to be functional. Arterial HT was rather explained by renovascular causes, due to pressure exerted by the tumour, or associated with obesity or endocrine conditions such as Cushing's syndrome or Conn's syndrome. This case is exceptional as primary hyperaldosteronism was caused by a myelolipoma, which could possibly be explained by the pressure exerted on the adrenal gland by the large tumour.

Conflicts of interest

The authors declare they have no potential conflicts of interest related to the contents of this article.

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Reactive haemophagocytic syndrome associated with parvovirus B9 in a kidney-pancreas transplant patient

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To the Editor,

Reactive haemophagocytic syndrome or secondary haemophagocytic lymphohistiocytosis (HLH) is a disorder of the mononuclear phagocyte system characterised by generalised, ineffective and uncontrolled histiocytic proliferation that leads to cell damage and multiple organ dysfunction with haemophagocytosis. The first description of secondary forms of this disease was by Risdall et al,[1] who in 1979 described a syndrome characterised by a proliferation of histiocytes with haemophagocytic activity, associated with a viral infection. This syndrome was later described in association with infections of all types and with non-infectious diseases such as rheumatoid arthritis, lupus, leukaemia, lymphomas, myelodysplastic syndromes and carcinomas.

Its pathogenesis is still unclear, although there are several hypotheses. The development of this syndrome is likely to be due to an immunological disorder that results in uncontrolled T-lymphocyte activation,[2] causing hypercytokinaemia, and consequently, excessive macrophage activation.