Haemoglobin and erythropoietin levels in polycystic kidney disease

Sir,

Artunc and Risler [1] recently proposed a nomogram allowing an easy interpretation of serum erythropoietin values (EPO) by plotting them against haemoglobin using percentiles. They found that EPO correlated inversely with haemoglobin and patients with chronic kidney disease (CKD) of various aetiologies preserved the feedback loop although at a lower level, with most patients below the 25th percentile.

Interestingly, patients with polycystic kidney disease (PKD) were excluded based on the assumption that this entity is associated with increased EPO concentration irrespective of renal function. Although there are several reports claiming that patients with PKD had higher levels of haemoglobin and EPO [2,3], our data only partially support this view. In fact, in a cohort of 259 patients with PKD, in several stages of CKD, haemoglobin levels were only significantly higher in PKD patients in stages 3 and 4, when compared to 87 patients with other causes of CKD (Figure 1). In addition, in our practice, many patients with PKD have to start an erythropoietic-stimulating agent (ESA) in order to treat renal anaemia. We performed a radio-immunoassay determination of serum EPO (DSL1100EPOkit) in 107 patients with PKD and in 35 patients with CKD of other aetiologies, at various stages of CKD; none was on ESA, nor had other causes for anaemia. In PKD patients in stage 2, there is a significant rise in serum EPO levels that is not accompanied by a similar elevation of haemoglobin. In later stages, there is a continuous fall in haemoglobin. In early stages of CKD of other aetiologies, there is a significant negative correlation between EPO and haemoglobin that is lost in stages 4 and 5, but no correlation was found, in any stage, in patients with PKD. By plotting EPO against haemoglobin (Figure 2), it is clear that most values are below the 25th percentile and that there is no difference between PKD and other causes of CKD.

It has been demonstrated that cystic fluid and interstitial cells produce erythropoietin independent to the oxygen...
content [4], and this has been the main argument for the opinion that PKD patients produce more EPO than others. Although this may be true in early stages of CKD with volume expansion [5], this effect may be offset in the later stages as a result of uremia. Therefore as PKD progresses, more cysts produce more EPO and may contribute to higher haemoglobin in stages 3 and 4. In stage 5, the inhibitory effect of uremia may block the response of bone marrow to EPO. Our data are limited because the number of patients in the control group is small. Besides this limitation, our results show the utility of the nomogram in a clinical setting and that PKD patients have higher haemoglobin only in early stages.

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Acute renal failure associated to renin-angiotensin system (RAS) inhibitors—its burden in a nephrology department

Sir,

The introduction of renin–angiotensin system (RAS) inhibitors has been a progress in the cardiovascular therapies. Despite their beneficial actions on peripheral resistances, on heart function and on vasculopathy, we should not forget their effects on renal haemodynamics.

As it is well-known, angiotensin II (AII) is an important actor in the auto-regulation of glomerular filtration rate (GFR). AII increases efferent glomerular arteriole resistance, having an important role in the control of the glomerular capillary hydrostatic pressure [1]. Its reduction by RAS inhibition could have a renoprotective effect in case of glomerular hypertension [2], but it has a potential harmful effect on GFR when glomerular perfusion is maintained in the normal range precisely by the constrictor effect of AII on the efferent arteriole set. Such a situation takes place when real or effective hypovolaemia occurs (i.e. dehydration, heart failure or renal artery stenosis) [3].

In fact, renal failure associated to the use of RAS inhibitors has been described in up to 19% of patients with hypertensive nephrosclerosis [4], and the decrease in GFR could be irreversible if severe atheromatous disease, renal asymmetries or pre-existent renal insufficiency are concurrent [5].

Furthermore, patients suffering from acute renal failure (ARF) during treatment with RAS inhibitors are frequently admitted in emergency rooms. Prompted by this ‘epidemic’ pathology, we have retrospectively analysed its prevalence during 2004 in our Nephrology Department. Forty-one patients suffering from ARF without any immunologic, septic or toxic-related cause were admitted in our centre. Associated RAS inhibitors therapy was documented in 20 of them (48.7%). Their mean age was 70.8 ± 24 years (46–94); 12 males and 8 females. Nineteen had hypertension, 10 diabetes, 10 renal echographic asymmetries higher than 15 mm, six chronic heart failure, five stroke and four symptomatic peripheral vasculopathy.

In addition to ACEIs or ARBs, furosemide, potassium supplements or spironolactone were associated in seven patients. Co-adjuvants to ARF were vomiting and/or diarrhoea in 14 patients, insufficient fluids supply in two patients and digestive haemorrhage in one patient.

Serum creatinine before ARF was 153 ± 104 μmol/l (84–293) and at admittance, 658 ± 330 μmol/l (300–1700).

Fourteen patients (70%) showed hyperkalaemia: 5.8 ± 1.5 mEq/l (3.4–8.6). Haemodialysis was assessed in 13 cases (65%). One patient died in this setting.

At discharge, mean serum creatinine level was 262 ± 23 μmol/l (98–700).

In our experience, RAS inhibitors could have a pathogenic role in almost 50% of patients with haemodynamic ARF admitted to our Department. Advanced age, vascular disease, previous renal failure and renal asymmetries appear to constitute main risk factors. In this subset of patients, this therapy must be carefully balanced in terms of risk/benefit. A correct hydration must be warranted and frequent control of renal function and serum potassium levels are mandatory.

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