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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 attended in emergency rooms. Prompted by this ‘epidemic’ pathalogy, 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). Haemodyalisis 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.

Conflict of interest statement. None declared.

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Sir,

A 64-year-old Austrian female patient presented to a Hungarian hospital with dyspnoea, sensory aphasia and disorientation. Apart from a pulmonary embolism years earlier, she had no apparent medical history. The laboratory findings were as follows: Na 142.2 mmol/l, K 4.8 mmol/l, Ca 2.38 mmol/l, GOT 23U/l, GPT 22U/l, ALP 130U/l, GGT 20U/l, Hb 10.2 g/dl, Hkt 49%, thrombocytes 217 000/l, creatinine 317 mmol/l, LDH 644U/l, pH 7.10, pCO2 9.8 mmHg, pO2 146.4 mmHg, O2 Sat 98.1%. No information on chloride levels and serum osmolality to differentiate the metabolic acidosis were provided. A computed tomography of the brain showed no pathologies, neither did an ultrasound of the abdomen. She was treated for her acute renal failure with intravenous fluid administration combined with forced diuresis and transferred to our hospital in Vienna, Austria, as soon as the neurological symptoms allowed transportation. In our hospital, a computed tomography, an EEG, a renal ultrasound and urine analysis, as well as the acid–base status, were normal. The patient’s history gave no hint of intoxication; the patient denied intake of non-steroidal anti-inflammatory drugs or any other nephrotoxic substances. A renal biopsy was performed when renal function did not improve after several days of adequate intravenous fluid administration.

The biopsy showed a severe acute dystrophy of the tubuli with loss of brush borders, shedding of tubular epithelial cells and intraluminal discrete amounts of birefringent crystalline deposits (Figure 1). The histological findings were highly suspicious for oxalosis. Primary oxalosis was ruled out by the age of the patient. Of the causes of secondary oxalosis, the only plausible cause for the acute tubular necrosis caused by calcium oxalate crystals was ingestion of ethylene glycol [1,2].

Intensive questioning of the patient revealed accidental intake of anti-freeze solution by brewing coffee with liquid falsely thought to be water from a container in the kitchen in her summer cottage in Hungary. The patient recovered from her neurological symptoms and her renal impairment without specific therapy. Her most recent creatinine was 1.1 mg/dl.

In most cases, the common causes of acute renal failure—pre-renal disease, acute tubular necrosis and urinary tract obstruction—can be diagnosed without renal biopsy. Prior to renal biopsy, laboratory findings, urine sediment analysis and ultrasound examination are used as non-invasive techniques to establish a diagnosis. In our case, the typical acid–base disorder [3] was no longer present at presentation at our hospital and the patient was completely oblivious to the poisoning. Biopsy is indicated in settings like our case, in which the cause of renal failure is still uncertain after all non-invasive diagnostic measurements [4], and can reveal the most unexpected diagnoses.

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Persistent proteinuria is an expression of renal damage caused by numerous factors, but it also represents an important cause of renal injury progression that can lead to chronic renal failure (CRF) [1]. Effectively, the presence of plasmatic proteins within tubular lumen is a source of further


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