argued that the reduction of proteinuria only reflected alterations of GFR (89 ± 29 versus 78 ± 28 ml/min, mean ± SD, P = n.s.). To objectify our findings a fractional protein clearance (proteinuria/creatinine clearance ratio) was calculated which significantly decreased during the treatment period (0.08 ± 0.05 versus 0.05 ± 0.04 mg/ml/min, P < 0.04). This indicates that the amelioration of urinary protein excretion was not directly related to changes of GFR. It could also be argued that the decrease of proteinuria may be due to a better diuretic-induced control of blood pressure or reduction of extracellular fluid volume. However, in our patients we found no correlation between the fall of proteinuria and decline of blood pressure (R = 0.3, P = n.s.) or body weight (R = 0.09, P = n.s.). It is also unlikely that reduction of proteinuria might be caused by a partial recovery of the glomerular disease since renal disease had been controlled for more than 1 year in the majority of patients and mainly nephropathies with a low incidence of spontaneous recovery were included. Furthermore we wish to state that no changes, even in dosage, were made in the immunosuppressive drug regime of kidney-graft recipients, that none of the other patients received any immunosuppressive agents and that angiotensin-converting-enzyme inhibitors were not administered. A possible antiproteinuric effect due to calcium-channel blockers is also unlikely because therapy was neither initiated nor altered in dosage in pretreated patients.

Although we cannot offer an explanation for the observed decrease of proteinuria, our data would confirm the findings of a previous clinical study which showed that proteinuria was reduced in 42% of nephrotic patients treated with torasemide as monotherapy [2].

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Severe acute renal failure after administration of contrast media in a patient treated with cisplatin

Sir

Cisplatin is an alkylating agent frequently used because of its wide range of activity in the treatment of solid tumours. Its main side-effect is nephrotoxicity; this toxicity is dose-limiting with a cumulative action and can lead to irreversible renal dysfunction [1]. Preventive measures are now well known and consist mainly in expansion of the extracellular fluid volume and suppression of concomitant therapy with other nephrotoxins (especially aminoglycosides and non-steroidal anti-inflammatory drugs) [2]. Very few data are available on the association of cisplatin and iodiinated radiocontrast agents.

We report here the case of a man, treated with parenteral cisplatin for lung cancer, who developed a severe acute renal failure after administration of radiocontrast agents despite adequate preventive measures.

A 66-year-old man was admitted to our unit because of oligoanuria, drowsiness, and dyspnoea. His history included arterial hypertension, ischaemic cardiopathy with myocardial infarction, and an episode of renal colic.

The exploration of left thoracic pain 2 months previously led to diagnosis of a small-cell lung epithelioma. At the time of diagnosis there were already metastases in the liver and bone marrow.

Chemotherapy was started with cisplatin (70 mg/day for 2 days), cyclophosphamide, etoposide, and corticosteroids in association with the administration of 2000 ml of 0.9% saline per day. At the same time, two computerized tomographies with intravenous injection of contrast media were performed on day 1 and day 4 to complete metastatic evaluation. Serum creatinine level before chemotherapy was 1.2 mg/dl and calculated creatinine clearance 72 ml/min. From day 3, renal function started to deteriorate and oligoanuria appeared. Chemotherapy was stopped.

At his admission to our unit on day 7, blood pressure was 90/45 mmHg. Physical examination revealed drowsiness dyspnoea with left pleural effusion, and oligoanuria (250 ml/day) despite intravenous administration of frusemide (250 mg/day).

Laboratory studies disclosed the following values: serum urea nitrogen 2.6 g/l, creatinine 5.5 mg/dl, potassium 3.5 mmol/l, bicarbonate 37 mmol/l, calcium 1.81 mmol/l with total protein 63 g/l, phosphorus 3.2 mmol/l and haemoglobin 10.7 g/dl. Abdominal ultrasonographic study excluded urinary obstruction.

An internal jugular catheter was inserted and the patient was haemodialysed on the day following his admission. Haemodialysis was continued (the patient was haemodialysed 6 times during his 19 days of hospitalization) until the progressive amelioration of renal function and urine output. When he was discharged, laboratory values were: serum urea nitrogen 1.3 g/l and creatinine 3.1 mg/dl. The patient died one week later from acute respiratory deficiency.

Recently Oymak [3] reported the induction of an irreversible acute renal failure following intraperitoneal cisplatin chemotherapy and contrast media injection in a woman treated for ovarian cancer.

The pathophysiology of cisplatin’s nephrotoxicity remains unclear and disputed, but the main mechanism is a direct tubular toxicity affecting the distal and collecting tubules in human [4]. Transient renal ischaemia and direct renal tubular toxicity have been suggested as possible mediators in the development of contrast media nephrotoxicity [5]. Therefore both cisplatin and contrast media may enhance the nephrotoxic potential of each other.

In conclusion, we suggest that radiographic procedures with intravascular contrast material should be delayed for at least 2 weeks in patients receiving chemotherapy with cisplatin.

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Correlation of whole blood activated partial thromboplastin time and plasma activated partial thromboplastin time in haemodialysis patients

Sir,

Heparinization during haemodialysis remains a challenging problem. Under-anticoagulation may result in the clotting of vascular access sites and in the extracorporeal circuit. Over-heparinization exposes the patient to risks of prolonged bleeding, aggravating the bleeding tendency common in uraemic patients.

Figure 1 demonstrates 21 sets of data obtained from the five haemodialysis patients. There was good linear correlation between WBAPPT and plasma aPPT ratio \( r^2 = 0.62 \). The endpoint has been used to evaluate the ability of a heparin dosing protocol to maintain WBAPPTs within a desired range [4].

In summary, there is good in vivo correlation between plasma aPPT and WBAPPT in haemodialysis patients using the endpoint. It provides an alternative tool which is useful for monitoring heparin therapy during haemodialysis.

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4. Low CL, Bailie GR, Morgan S, Eisele G. Effect of a sliding scale protocol for heparin on the ability to maintain whole blood activated partial thromboplastin times within a desired range in hemodialysis patients. Clin Nephrol 1996; 45: 120–124

Transcatheter intravascular embolotherapy of renal arteriovenous fistula—method of choice

Sir,

The occurrence of an arteriovenous (AV) fistula in native kidneys is still a well-recognized complication of percutaneous biopsy, with an incidence of up to 16% [1,2]. A 13-year-old, previously healthy boy experienced a severe Henoch–Schönlein purpura with life-threatening gastrointes-