DXL (Figure 1B). This observation could be explained by the rapid half-life of DXL (about 4 min) causing no accumulation of the drug in the central compartment after repeated administration. So these differences in DXL plasma concentrations looked accidental. The observed transient increase of DXL plasma concentrations during HD is probably due to altered protein binding of the drug. To evaluate DXL toxicity, blood-count controls were taken in periodic intervals. Grade 2 leuco- and thrombocytopenia (National Cancer Institute Common Toxicity Criteria, NCI-CTC) were detected 10–20 days after the second cycle of chemotherapy (data not shown).

Restaging at day 42 revealed further regression of the central tumour and a decreased bronchial wall thickness. The mediastinal lymph nodes remained stable and a restaging mediastinoscopy showed necrosis but also residual tumour cells. Afterwards, sequential radiotherapy of the mediastinum and the primary completed the treatment.

In conclusion, a carboplatin/DXL combination might be a suitable regimen for NSCLC patients undergoing HD.

Conflict of interest statement. None declared.

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Fig. 1. Pharmacokinetic analysis of DXL. Plasma samples taken before and after the HD capillary were taken 1, 3 and 5 h after the end of DXL infusion. Samples were analysed by HPLC for the total plasma concentration of DXL. (A) DXL plasma levels in samples taken from the arterial (white) and venous line (black). (B) DXL plasma levels taken from the venous line during HD after the first (black) and second cycle of chemotherapy (white).

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Decreased serum fetuin-A levels after a single haemodialysis session

Sir,

Cardiovascular disease (CVD) is the leading cause of mortality in haemodialysis (HD) [1]. Elevated incidence of vascular calcification (VC) is observed in HD. Fetuin-A (AHSG, α2-Heremans–Schmid glycoprotein) is an important inhibitor of VC [2]. Reduced serum fetuin-A levels are associated with inflammation and increased CV mortality in HD [3]. In this study, we examined the effects of a single haemodialysis session on serum fetuin-A levels and other markers of inflammation, such as C-reactive protein (CRP), fibrinogen and albumin. We evaluated 20 clinically stable patients (40% males), aged 67 ± 12.8 years, with an HD vintage > 9 months, 50% in standard bicarbonate-HD and 50% in haemodiafiltration (HDF). Serum levels of fetuin-A, CRP, fibrinogen and albumin were measured at the beginning and at the end of a 4-h HD session. Table 1 shows that serum CRP, fibrinogen and albumin levels did not change during the HD session. In contrast, serum fetuin-A levels decreased significantly after 4 h of HD (P = 0.027). We did not observe significant differences in serum fetuin-A levels between patients treated with standard bicarbonate-HD and HDF. The present study confirms that HD patients have reduced serum fetuin-A levels compared with the normal population. Furthermore, serum levels of fetuin-A decreased during a single HD session. Inflammation is one of the most important non-traditional cardiovascular risk factors associated with VC in HD patients. The inflammatory processes induced by HD treatment might contribute to reduced fetuin-A levels and therefore to increased mortality, even if this process does not seem to be improved by convective treatment.

Table 1. Inflammatory markers during a single HD session

<table>
<thead>
<tr>
<th></th>
<th>Pre-HD</th>
<th>Post-HD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fetuin-A (g/l)</td>
<td>0.26 ± 0.06</td>
<td>0.21 ± 0.06*</td>
</tr>
<tr>
<td>CRP (mg/l)</td>
<td>9.5 ± 1.3</td>
<td>8.9 ± 1.2</td>
</tr>
<tr>
<td>Albumin (g/dl)</td>
<td>3.77 ± 0.48</td>
<td>3.91 ± 0.33</td>
</tr>
<tr>
<td>Fibrinogen (mg/dl)</td>
<td>351 ± 87</td>
<td>415 ± 93</td>
</tr>
</tbody>
</table>

*P = 0.027.
Conflict of interest statement. None declared.

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Anti-CD20 antibody treatment in refractory Class IV lupus nephritis

Sir,

Selected cases of class IV lupus nephritis patients have been treated with rituximab monotherapy [1,2] and in combination with cyclophosphamide [3]. Data on refractory patients who have failed multiple therapies are limited [4]. We have successfully treated four patients with rituximab, who have failed multiple conventional immunosuppressive regimens, including cyclophosphamide, azathioprine, mycophenolate and ciclosporin. Patient characteristics and response to rituximab are summarized in Tables 1 and 2, respectively.

Patient 1

A third episode of class IV nephritis failed to respond to four cycles of cyclophosphamide and rendered her neutropenic. Concurrently, she had evidence of cerebral and cutaneous lupus. She was treated with methylprednisolone and ciclosporin but her renal and cerebral disease failed to respond, and she received a single infusion of 500 mg rituximab. All clinical features began to improve within 10 days. Clinical relapse at 9 months was retreated with two weekly infusions of 500 mg rituximab. At 26 months, her B cell count rose from 20 to 200 cells/μl and there was evidence of renal and immunological relapse and she was retreated with four infusions of rituximab. The B cell count fell and she clinically improved. She remains stable at 36 months and is maintained on 10 mg prednisolone.

Patient 2

A fifth episode of class IV nephritis failed to respond to three cycles of cyclophosphamide. Due to bone marrow suppression, cyclophosphamide could not be continued. She received three 700 mg infusions of rituximab, and the parameters improved. At 19 months post initial infusion, she was retreated due to reappearance of active urinary sediment and a doubling of her proteinuria. At 3 months post second infusion, she has demonstrated serological and clinical improvement. Her current drug regimen is azathioprine 25 mg and prednisolone 10 mg.

Patient 3

A fourth episode of class IV lupus could not be treated with cyclophosphamide as he was suffering borderline neutropenia from maintenance therapy with azathioprine. The patient had also been treated with ciclosporin in the past and there was evidence of ciclosporin toxicity on biopsy. He received two weekly 600 mg infusions of rituximab. He is stable at 26 months and still has B cell depletion (24 cells/μl). His current drug regimen is 10 mg prednisolone and the ciclosporin dose was halved to 50 mg bd.

Patient 4

He presented with symptoms of nephrotic syndrome but was also anaemic and thrombocytopenic. Biopsy showed class IV/V lupus nephritis. He had received multiple courses of cyclophosphamide and because of concerns regarding bone marrow suppression, he was treated with three 500 mg

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Ethnicity</th>
<th>Duration of disease years</th>
<th>Previous cycles of cyclophosphamide</th>
<th>Previous therapeutic failures</th>
<th>Number of previous flares</th>
<th>Organ involvement at baseline</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>34</td>
<td>F</td>
<td>Asian</td>
<td>6</td>
<td>17</td>
<td>MMF  AZA</td>
<td>2</td>
<td>Renal</td>
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<td>29</td>
<td>F</td>
<td>Philippino</td>
<td>10</td>
<td>16</td>
<td>MMF  AZA  CSA</td>
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<td>Cutaneous Cerebral Renal</td>
</tr>
<tr>
<td>3</td>
<td>27</td>
<td>M</td>
<td>Caucasian</td>
<td>9</td>
<td>10</td>
<td>AZA  CSA  CSA</td>
<td>3</td>
<td>Renal Rheumatological Pleural/pericardial</td>
</tr>
<tr>
<td>4</td>
<td>28</td>
<td>M</td>
<td>Asian</td>
<td>7</td>
<td>9</td>
<td>AZA  AZA  AZA</td>
<td>3</td>
<td>Renal Haematological</td>
</tr>
</tbody>
</table>

AZA, azathioprine; MMF, mycophenolate; CSA, ciclosporin.