size of five CAPD patients. Therefore, we used a random cross-over design and computed a paired $t$-test.

These pilot data suggest reduced inflammation and consequently an improved biocompatibility of GBF peritoneal fluids compared with IBF fluids. Certainly, further evaluation in larger studies is needed.

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1Department of Nephrology
KH Elisabethinen, Linz
2Department of Nephrology
Medical University of Vienna
3Emergentec Biodevelopment GmbH, Vienna
4Austrian Dialysis and Transplant Registry, Austria
E-mail: rainer.oberbauer@meduniwien.ac.at

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Crescentic glomerulonephritis in a patient with advanced lung cancer during erlotinib therapy

Sir,

Erlotinib (Tarceva®), an epidermal growth factor receptor (EGFR) tyrosine kinase inhibitor, has been shown to improve survival of previously treated non-small cell lung cancer (NSCLC) [1]. The common adverse effects of this agent include diarrhoea and anorexia [1], which may cause severe dehydration and renal failure, although their incidence has been low [2]. Here, we report a case of pauci-immune crescentic glomerulonephritis (CrGN) and acute renal failure in a patient with advanced NSCLC treated with erlotinib.

In May 2009, a 72-year-old man with advanced NSCLC was admitted to our department because of acute renal failure. In July 2007, he underwent a pulmonary lobectomy for NSCLC. Because of intrapulmonary recurrence, he received multiple chemotherapies between November 2007 and December 2008, including carboplatin, docetaxel, paclitaxel, irinotecan and gemcitabine. In February 2009, erlotinib (150 mg daily) was started due to the progression of intrapulmonary lesions.

When erlotinib was started, serum creatinine (sCr) was 88 $\mu$mol/L and urinalysis showed only slight proteinuria without haematuria. During erlotinib treatment, diarrhoea and acneiform eruptions were transiently observed. Six weeks later, microhaematuria and 2+ proteinuria were detected. Over 1 month, proteinuria progressed to 3+ and sCr rose to 141 $\mu$mol/L.

On admission, he presented with anorexia, diarrhoea and severe dehydration. Although he was almost anuric, urine test revealed 3+ proteinuria and microscopic haematuria. Marked renal dysfunction and metabolic acidosis were noted (Table 1). The onset of microhaematuria and progressive renal failure and proteinuria suggested the possibility of rapidly progressive glomerulonephritis. However, he was critically ill and a renal biopsy was considered dangerous. In addition, acute tubular necrosis following pre-renal azotemia was also probable. Thus, erlotinib was discontinued and supportive therapy with haemodialysis was started. Despite adequate fluid replacement, anuria persisted, and 1 month later, he died of pneumonia. By an autopsy, pauci-immune CrGN was diagnosed (Figure 1). No vasculitic lesion was found in other organs.

Discussion

Acute renal failure with nephritic urine sediment is an atypical manifestation during erlotinib therapy. As for

Table 1. Result of blood test on admission

| Test                          | Result                        |
|-------------------------------|-------------------------------|
| White blood count             | $11.6 \times 10^{9}$/L         |
| Haemoglobin                   | 8.4 g/dL                      |
| Platelets                     | $156 \times 10^{9}$/L         |
| Blood urea nitrogen           | 37.8 $\mu$mol/L               |
| Serum creatinine              | 1,228 $\mu$mol/L              |
| Serum protein, total          | 61 g/L                        |
| Serum albumin                 | 21 g/L                        |
| Glycosylated haemoglobin $A_{1c}$ | 5.4%                      |
| C-reactive protein            | 131 mg/L                      |
| Immunoglobulin (Ig)-G         | 12.8 g/L                      |
| IgA                           | 8.7 g/L                       |
| IgM                           | 0.5 g/L                       |
| C3                            | 1.0 g/L                       |
| C4                            | 0.4 g/L                       |
| Antinuclear antibody (ANA)    | $<1:40$                       |
| Cryoglobulin                  | Negative                      |
| MPO-ANCA                      | Negative                      |
| PR3-ANCA                      | Negative                      |
| Anti-GBM antibody             | Negative                      |
| pH                            | 7.18                          |
| PaCO$_2$                      | 30.8 mmHg                     |
| Bicarbonate                   | 11.3 mmol/L                   |

Case

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Discussion

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Microscopic renal findings of the postmortem examination. A quarter of glomeruli were globally sclerotic. Ninety percent of remaining glomeruli demonstrated crescents, mostly fibrocellular, without intracapillary proliferation. Immunostaining for IgG, IgA and IgM were negative. Focal interstitial lymphoplasmacytic infiltration was also noted (Periodic acid Schiff staining, original magnification $\times 200$).

renal complications, hepatorenal syndrome and acute renal failure have been reported, and both were considered to be associated with baseline hepatic impairment or severe dehydration [2]. Indeed, in the present case, pre-renal azotaemia due to dehydration was considered presumptively.

The mechanisms of CrGN are not clear. Microscopic polyangiitis and Wegener’s granulomatosis were excluded because of negative findings on blood tests and the lack of extra-renal symptoms. EGFR was detected in glomerular parietal epithelial cells [3] and in the connective tissue of fibrocellular crescents in primary glomerulonephritis and lupus nephritis [3]. In addition, there have been several cases of leucocytoclastic vasculitis during treatment with erlotinib [4] or gefitinib, another EGFR tyrosine kinase inhibitor [5]. Pauci-immune CrGN is often observed with leucocytoclastic vasculitis; thus, it is possible that CrGN was induced by erlotinib. In fact, microscopic haematuria and renal function deterioration commenced during erlotinib therapy and no case of CrGN was reported for the other anticancer agents used in this case. Interstitial lymphoplasmacytic infiltration might be associated with erlotinib, because two cases of tubulointerstitial nephritis with gefitinib have been reported [6, 7].

In summary, we describe a case of pauci-immune CrGN and acute renal failure, possibly induced by erlotinib. Further cases with renal complication during erlotinib therapy should be investigated.

Conflict of interest statement. None declared.

1 Department of Medicine
2 Department of Pathology
3 Department of Respiratory Surgery, Mitsui Memorial Hospital
Tokyo, Japan
E-mail: kurita-noriaki@mitsuihosp.or.jp

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