20×10^6 IU/m^2. Before rIL-2 therapy the patient did not receive any treatment except levothyroxine. Metastatic lesions were found in lung and liver but no renal abnormality was detected in scannography examination. All laboratory tests showed normal values at the onset of rIL-2 treatment. Proteinuria, not detected during the first course, appeared on day 2 of the second course (3.6 g per 24 h) increasing to 5.9 g per 24 h on day 4. A moderate renal impairment (serum creatinine 150 μmol/l, urea nitrogen 16.8 mmol/l and creatinine clearance 58 ml/min) with oliguria and weight gain (5 kg) were noticed. Simultaneously, pruritic erythroderma with necrotic lesions on the neck was observed. Skin biopsy disclosed only a microvasculitis. Urinalysis showed neither haematuria, nor leucocytes and the urine was sterile. White blood cell count was 19700/mm^3 with 20% eosinophils; C3 and C4 levels were normal and antinuclear antibodies were undetectable. Following the discontinuation of rIL-2, the proteinuria disappeared in 3 days while renal function and weight returned to normal. Skin lesions completely resolved with desquamation 2 weeks later.

According to drug monitoring criteria [4], the reversible proteinuria was associated with rIL-2 therapy, starting after commencement of the treatment and resolving 48 h after discontinuation. There was no sign of other associated factors. Drugs prescribed during rIL-2 therapy included: levothyroxine, propacetamol, metoclopramide, ranitidine, loperamide and colloids solution. Thus, the patient did not receive non-steroidal anti-inflammatory drugs, or other nephrotoxic treatment. No history of cyclic idiopathic oedema was noticed and HIV was negative. The high level of proteinuria and the speed of reversibility suggest a glomerular disorder such as glomerular minimal change but no kidney biopsy was done. Cutaneous necrosis could suggest renal vasculitis. However the clinical presentation and the course of the nephropathy do not argue for diffuse vasculitis, although cutaneous vasculitis had been reported previously [5]. rIL-2 increases vascular permeability inducing a dose-dependent vascular-leak syndrome [6]. In the present case, such a toxic reaction was observed and related in part to the high dose used. As previously suggested, rIL-2 therapy might stimulate the release of other cytokines resulting in changes of vascular permeability [7]. In addition, T-cell cytokines have been incriminated in the pathogenesis of minimal change nephrotic syndrome [8]. This suggests a direct or indirect role of rIL-2 in the occurrence of increased glomerular permeability. However, while rIL-2 therapy is widely used, proteinuria has been described only once [3]. In the light of this reference routine screening for proteinuria is recommended in patients treated with high dose rIL-2.

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High-dose Interferon-beta in Treatment of Spindle-cell Sarcoma of Breast

R.P. de Dycker, T.Schumacher and R.L.A. Neumann

IN NOVEMBER 1985, a biopsy of the right breast was done on a 17-year-old girl. Histological examination revealed a spindle-cell sarcoma grade III. A total mastectomy with axillary dissection was done. The 2 year recurrence-free survival decreases from 85% at grade I to 17% at grade III.

After the operation the patient received adjuvant chemotherapy according to the Gottlieb scheme. After the first local recurrence in May 1986, treated with extensive excision with an abdominothoracic sliding flap, the patient received high-dose irradiation of the right thoracic wall up to 70 Gy. After recurrence, mortality rate within 6 months is 85%.

Because of this bad prognosis and the fact that a severe Epstein–Barr viral infection had been diagnosed 4 months before the spindle-cell sarcoma occurred, we administered, from November 1986, 4 million IU interferon-beta 24 times. In March 1987, a new local recurrence was diagnosed. We repeated the extensive excision of the tumour. In July 1987, we again started 24 administrations of 4 million IU interferon-beta for 8 weeks every 6 months until March 1989.

To date the patient has remained free from tumour symptoms. This result supports our opinion that acute viral infections may influence the individual immunity to such an extent that there will be tumour induction in cases with appropriate genetic disposition.

Correspondence to R.P. de Dycker.
The authors are at the Department of Gynecology, Acad Marienhospital, University of Essen, Hospitalstr. 24, D-4300 Essen 12, F.R.G.

Correction

The growth of metastatic non-seminomatous germ cell testicular tumours measured by marker production doubling time. — In these two articles by P. Price *et al.* (Vol. 26, pp. 450–453 and 453–457), some figures were incorrectly presented. Figure 1 on p. 455 should have been repeated on p. 451. Also, Figs 1 and 2 on p. 451 should have appeared as a two-part Fig. 2 with "and AFP" inserted at the end of the legend.