Letters

Cyclosporin A-induced remission of primary membranous glomerulonephritis in a child

Sir, A 4-year-old boy presented with oedema and weight gain of 5 kg. There had been no preceding infections, vaccinations or medication. The family history includes the mothers mesangioproliferative glomerulonephritis. Laboratory results showed decreased albumin (13 g/l) concentration. Calculated glomerular filtration rate was 125 ml/min/1.73 m² and the U-albumin/U-creatinine ratio was 300 mg/mmol. The Na/K exchange after van de Walle [1] \( \frac{[U-K]}{[U-K + U-Na]} \) was 61%, indicating an underfill that changed to an overfill (15%) spontaneously. Hepatitis titres, ANAs, anti-DNS or other signs of infectious/auto-immunological diseases were not found. A malignancy was excluded. After diagnoses of nephrotic syndrome, treatment with prednisolone 60 mg/m²/day was started. Two days later, the Na/K-exchange represented an overfill (20%). Therefore, furosemide was administered. No remission was observed. A renal biopsy showed a membranous glomerulonephritis, stage I–II. Because of the family history, the treatment had been expanded to intravenous prednisolone 300 mg/m² every 48 h plus oral prednisolone 40 mg/ml before the result of the biopsy was known. A partial remission was achieved within 1 week. The U-albumin/U-creatinine ratio decreased to 50 mg/mmol, S-albumin was 37 g/l. Prednisolone was stopped 2 months later because of remission. After 12 months, no proteinuria was present under a maintenance therapy of CsA, which was discontinued at this timepoint. Two weeks later, nephrotic proteinuria started again. This time, CsA therapy (150 mg/m²) was initiated without steroids with trough levels between 85 and 117 ng/ml before the result of the biopsy was known. A partial remission was achieved within 1 week. The U-albumin/U-creatinine ratio decreased to 50 mg/mmol, S-albumin was 37 g/l. Prednisolone was stopped 2 months later because of remission. After 12 months, no proteinuria was present under a maintenance therapy of CsA, which was discontinued at this timepoint. Two weeks later, nephrotic proteinuria started again. This time, CsA therapy (150 mg/m²) was initiated without steroids with trough levels between 85 and 122 ng/ml. A partial remission was achieved within 1 week and a complete remission after 2 weeks. The patient has now been asymptomatic for another year.

Membranous glomerulonephritis is predominantly a disease of mid-adult life. It is a rare disorder in children [2]. Contrary to in adults, relapsing courses are more frequent. The idiopathic disease is the most common subtype. An association with malignancies, rheumatoid arthritis, infections or drugs can be found in only few cases. As a development of renal failure in children is the exception, the natural course of the disease is usually with spontaneous remission [3]. If proteinuria or a deterioration of renal function occur, treatment with corticosteroids is the standard. If this is ineffective, a more aggressive approach, such as the method of Ponticelli et al. [4], might be used in adults. It remains unclear what is the best model for children.

Two studies show positive effects of CsA in membranous glomerulonephritis [5,6]. Treatment with CsA has been successful in children with a steroid-resistant nephrotic syndrome [7], but data about membranous glomerulonephritis have not been published yet.

CsA therapy might be an additional approach in steroid-resistant membranous glomerulonephritis. Due to the low numbers of patients, policy cannot be derived from evidence of randomized trials.

Conflict of interest statement. None declared.

3. Lewis EJ. Idiopathic membranous nephropathy—to treat or not to treat? N. Engl J Med 1993; 329: 127–129

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Timing of acute renal failure in multiple myeloma: two distinct outcomes?

Sir, It is known that renal failure carries a poor prognosis to patients with multiple myeloma (MM), reducing the survival from 36 to 18 months [1]. The prognosis is even worse in patients without renal function recovery [2,3]. Most studies addressed patients with renal failure and newly diagnosed MM. To our knowledge, there is no information on the outcome of patients who develop acute renal failure during MM chemotherapy.

We retrospectively studied 26 patients with MM and renal failure, admitted to a university hospital, between 1998 and 2003. Patients with known chronic renal failure or those who were submitted to bone marrow transplantation were excluded. We compared patients who had renal failure at the time of MM diagnosis and had not received any MM treatment (group 1, \( n=15 \)) with patients who developed renal failure after MM diagnosis and were already under treatment (group 2, \( n=11 \)). Statistical analysis was performed using unpaired \( t \)-test, \( \chi^2 \) test and Kaplan–Meier survival curve.

Conflict of interest statement. None declared.

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