shorter mean haemodialysis (HD) duration, while in our study, adult patients, many of them diabetic and on HD for much longer, were included; therefore, in the second case, the patients had a significantly greater oxidative and inflammatory burden, although a direct comparison between the respective levels of oxidative and inflammatory markers is not feasible.

In conclusion, despite the transient fluctuations in the above parameters caused by the extracorporeal circuit by the various membranes, it seems promising that there is solid evidence regarding the beneficial effects from the long-term use of vitamin E-coated membranes (VEMs) in the adult patient population on HD. The continuous use of VEM dialysers seems necessary for these effects to be sustained in the long term, as long as they are rather reversible after discontinuation of the membrane use. However, we agree that large-scale prospective randomized studies are needed to confirm our findings and to establish the beneficial effect of long-term use of vitamin-E coated dialysers in specific patient populations.

Conflict of interest statement. None declared.

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doi:10.1093/ndt/gfr230

Advance Access publication 28 April 2011

Outbreak of pneumocystis pneumonia occurring in late post-transplantation period

Sir,

We have read with interest the manuscript of Eitner et al. [1] concerning the impact of immunosuppressive treatment on the occurrence of pneumocystis pneumonia (PCP) in renal transplant recipients (RTR). In contrast with these authors, we have used post-transplantation PCP prophylaxis since 1992 (trimethoprim–sulphamethoxazole (TMP–SMX) or aerosolized pentamidine) and we observed only one sporadic PCP case in our renal transplant unit up until 2008. Between September 2008 and August 2009, a PCP outbreak of 17 cases occurred. The median delay between transplantation and PCP was 66 months (range: 11–143). Most patients manifested fever, cough and dyspnoea. Bilateral interstitial pneumonia was documented on chest X-ray in all cases. PCP was confirmed by the demonstration of Pneumocystis jiroveci (Pj) cysts (10 cases) or Pj DNA using real-time polymerase chain reaction (PCR) assay (7 cases) in bronchoalveolar lavage fluid (BALF). Three patients died. All outbreak cases received mycopHENolate moFetil (MMF) combined with calcineurin inhibitor (15 patients) or sirolimus (2 patients). In addition, nine patients received prednisolone. The total lymphocyte count (TLC) was <800 cells/mm³ during the 3 months preceding PCP in 10 patients and none had a TLC >1500 cells/mm³ at PCP diagnosis.

Typing of pneumocystis strains based on the internal transcribed spacer sequence analysis [2] indicated that eight of the nine characterized strains shared the same genotype. In addition, one PCP case was clearly nosocomial in origin. This patient was hospitalized for a community-acquired pneumonia with a first BALF examination performed at D1 showing negative PCR for Pj. Aggravation of pneumonia led to a second BALF examination at D15 that showed the presence of Pj cysts. Finally, Pj DNA was found in three air samples collected in the waiting room of transplantation outpatients.

Prophylaxis recommendations were progressively upgraded and completed in July 2009. Respiratory isolation of patients with PCP and use of surgical masks for outpatient visits were provided. PCP prophylaxis was reintroduced for patients with TLC <800 cells/mm³ (15% of our RTR population in 2011). TMP–SMX was administered during each new hospital admission. Only one PCP was documented since August 2009 suggesting the efficiency of our prophylaxis regimen.

In conclusion, PCP is not limited to the early post-transplantation period and airborne transmission must be considered to determine optimal prophylaxis.

Conflict of interest statement. None declared.

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doi:10.1093/ndt/gfr159