Looking backward: a review of the treatment of systemic lupus erythematosus in end-stage renal disease after a quarter of century

Sir,

It has now been nearly a quarter of a century since we first reported that the activity of systemic lupus erythematosus (SLE) often becomes quiescent after progression to end-stage renal disease (ESRD) and that the survival rates of those patients were nearly identical to ESRD patients without SLE [1]. I suppose that is why it is so disheartening to read that, despite the great technological advances since our first report, Siu et al. [2] report a mortality rate of 4.3 times the rate of ESRD patients with chronic GN. How can this be? It may have been the demographics. Our patients were younger (28.1 vs 40.8 years), more likely to be women (28/5 vs 13/5), Caucasian, received more transplants (43 vs only 28%) and most notably only 2/28 of our patients underwent continuous ambulatory peritoneal dialysis (CAPD), which was just beginning to be recognized as a viable treatment for ESRD. Yet our patients were more anaemic since erythropoietin had not been discovered when we first published our work.

Furthermore, since there were no real differences in survival between CAPD, transplantation and haemodialysis in our data (Figure 1), the poor survival of Dr Siu’s patients becomes even more striking. I cannot help wondering if the prolonged use of immunosuppression may have played a role. We were very aggressive in withdrawal from immunosuppression and indeed we found that only a small minority of patients (3/28) required any immunosuppression. That appears to be in stark contrast to the 16/18 patients who continued on immunosuppression in the report of Dr Siu, despite the fact that he reports that only half (9/18) were interpreted to show any disease activity. One of the purposes of our original report was to encourage the aggressive withdrawal of immunosuppressants and I am puzzled by the rationale of continuing such immunosuppression in the absence of clinical disease. Although only one of their death cases was of an infectious nature (fungal peritonitis), the remainder were due to vascular complications, yet we now know that the quality and quantity of immunosuppression result in endothelial dysfunction [3], which promotes vascular disease and which may be responsible for the high incidence of graft coronary artery disease in transplants [4].

While we have achieved many advances in the care and treatment of patients with SLE over the last quarter of the previous century, I would think that the conclusions of our original work remain as cogent today as ever. ‘The most prudent course of management would appear to be... reduction (of immunosuppressants) to the lowest dosage that nonrenal manifestations will allow result in a reduced risk of death,’ remains important as a message now as when we first published our work.

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