Serum Angiotensin-Converting Enzyme during Haemodialysis: Effect of Membrane Biocompatibility

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Markets of dialysis membrane biocompatibility are of current interest. Nielson et al. [1] have reported that serum angiotensin-converting enzyme (ACE) increases during haemodialysis. This rise in serum ACE was thought to reflect pulmonary endothelial injury and it was suggested that serum ACE might prove to be a marker of dialysis membrane biocompatibility. We have tested this hypothesis by measuring serum ACE during haemodialysis with cuprophan (Gambro Lundia Plate) and polycarbonate membranes (Biogambrane, Gambro).

Seven patients on maintenance haemodialysis for 3 months to 13 years were studied during one routine haemodialysis session with each membrane. Serum ACE was measured at the start and after 240 min of haemodialysis. Predialysis levels of serum ACE were within the normal range (16–53 U/l) in all patients. There was no significant increase in serum ACE during dialysis either with the cuprophan (mean ± SD at 0 min = 43 ± 5 U/l and 43 ± 6 U/l at 240 min, p > 0.1) or with the polycarbonate membrane (41 ± 5 U/l at 0 min and 46 ± 11 U/l at 240 min, p > 0.1).

Haemodialysis with the cuprophan membrane was associated with a more severe neutropenia at 15 min (0.6 ± 0.5 vs. 2.2 ± 1.0 × 10^9/l, p < 0.01) and a more pronounced fall in arterial pO2 from predialysis levels after 30 min (-24.7 ± 11.6 vs. -16.8 ± 9.2 mm Hg, p > 0.1) than with the polycarbonate membrane.

ACE is predominantly located in the luminal surface of pulmonary endothelial cells and serum ACE increases with pulmonary damage induced by paraquat [2], thiourea [3] and oleic acid [4]. Serum ACE has, therefore, been used as a marker of pulmonary vascular endothelial injury. Haemodialysis induces a marked transient neutropenia due to pulmonary sequestration of neutrophil aggregates [5]. In animals, this is associated with pulmonary hypertension and oedema due to vascular endothelial damage [6]. If serum ACE reflected the degree of pulmonary leucosequestration and dysfunction during haemodialysis, a greater increase in serum ACE would be expected with the cuprophan than with the polycarbonate membrane. Our findings suggest that serum ACE is not a useful marker of dialysis membrane biocompatibility.

References