Dear Sir,

Changes in plasma antithrombin III (AT III) during hemodialysis is of current interest. Turney et al. [1, 2] reported an expected decrease in plasma AT III after intravenous bolus injection of heparin in hemodialysis patients. This decrease was ascribed to sequestration or accelerated consumption of heparin-AT III complexes. Subsequent hemodialysis was, however, associated with an unexpected increase in plasma AT III reaching 150% of predialysis values. The rise at AT III during hemodialysis was proposed to reflect release of AT III from contact activated platelets and/or release from vascular endothelial cells damaged by infusion into the patient of platelets, coagulation factors or complement activated in the extracorporeal circuit. The reports by Turney et al. [1, 2] prompted the present authors to study the effect of various dialyzer membranes on AT III during hemodialysis. Groups of stable patients on regular hemodialysis treatment (n = 12–14) were studied during a routine hemodialysis using hollow-fiber capillary kidneys with membranes based on cellulose acetate, regenerated cellulose and saponified cellulose ester (Cordis Dow Corp. Company, Miami, Fla., USA) and parallel-plates with cuprophan and polycarbonate membranes (ab Gambro, Lund, Sweden).

Of the five membranes tested both immunological AT III and AT III activity were unchanged apart from a significant, but negligible, increase in AT III activity during hemodialysis with membranes based on saponified cellulose ester [3–5]. Jørgensen et al. [6] recently reported a small, but significant, increase in circulating AT III during hemodialysis. However, the increase in AT III corresponded to the increase in plasma albumin, and therefore most probably was a result of hemoconcentration.

Woo et al. [7] similarly found a minor increase in AT III activity during hemodialysis and suggested that increased liver synthesis, increased synthesis and release from endothelial cells and platelets, and damage to platelets and blood vessels incurred by the extracorporeal circulation were causative. Apparently the effect of ultrafiltration and consequent hemoconcentration were neglected. The results of the present authors have two implications. Firstly, circulating AT III levels are not an indicator of dialyzer biocompatibility. Secondly, AT III levels remain essentially unchanged during hemodialysis. The conflicting results regarding AT III levels during hemodialysis are most probably due to changes in plasma volume caused by ultrafiltration. If changes in AT III occur during hemodialysis these are probably of such a magnitude that one would doubt any clinical significance.

References


