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The effects of continuous venovenous hemofiltration on coagulation activation

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Abstract

Introduction The mechanism of coagulation activation during continuous venovenous hemofiltration (CVVH) has not yet been elucidated. Insight into the mechanism(s) of hemostatic activation within the extracorporeal circuit could result in a more rational approach to anticoagulation. The aim of the present study was to investigate whether CVVH using cellulose triacetate filters causes activation of the contact factor pathway or of the tissue factor pathway of coagulation. In contrast to previous studies, CVVH was performed without anticoagulation.

Methods Ten critically ill patients were studied prior to the start of CVVH and at 5, 15 and 30 minutes and 1, 2, 3 and 6 hours thereafter, for measurement of prothrombin fragment F1+2, soluble tissue factor, activated factor VII, tissue factor pathway inhibitor, kallikrein-C1-inhibitor and activated factor XII-C1-inhibitor complexes, tissue-type plasminogen activator, plasminogen activator inhibitor type I, plasmin-antiplasmin complexes, protein C and antithrombin.

Results During the study period the prothrombin fragment F1+2 levels increased significantly in four patients (defined as group A) and did not change in six patients (defined as group B). Group A also showed a rapid increase in transmembrane pressure, indicating clotting within the filter. At baseline, the activated partial thromboplastin time, the prothrombin time and

the kallikrein-C1-inhibitor complex and activated factor XII-C1-inhibitor complex levels were significantly higher in group B, whereas the platelet count was significantly lower in group B. For the other studied markers the differences between group A and group B at baseline were not statistically significant. During CVVH the difference in the time course between group A and group B was not statistically significant for the markers of the tissue factor system (soluble tissue factor, activated factor VII and tissue factor pathway inhibitor), for the markers of the contact system (kallikrein-C1-inhibitor and activated factor XII-C1-inhibitor complexes) and for the markers of the fibrinolytic system (plasmin-antiplasmin complexes, tissue-type plasminogen activator and plasminogen activator inhibitor type I).

Conclusion Early thrombin generation was detected in a minority of intensive care patients receiving CVVH without anticoagulation. Systemic concentrations of markers of the tissue factor system and of the contact system did not change during CVVH. To elucidate the mechanism of clot formation during CVVH we suggest that future studies are needed that investigate the activation of coagulation directly at the site of the filter. Early coagulation during CVVH may be related to lower baseline levels of markers of contact activation.

CRRT = continuous renal replacement therapy; CVVH = continuous venovenous hemofiltration; ELISA = enzyme-linked immunosorbent assay; F1+2 = prothrombin fragment F1+2; FVIIa = activated factor VII; FVIIIa = activated factor XII; IL = interleukin; PAP = plasmin-antiplasmin; TFPI = tissue factor pathway inhibitor; t-PA = tissue type plasminogen activator.

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