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On the Degree of Platelet, Coagulation and Fibrinolysis Activation after Cerebral Infarction and Cerebral Haemorrhage and the Clinical Outcome

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Summary: Thrombocytic, haemostatic and fibrinolytic quantities were investigated in 47 patients with cerebral infarction and 34 patients with cerebral haemorrhage. Sixteen of the infarction patients and ten patients of the haemorrhage group were on acetylsalicylic acid medication. Of the remaining 55 patients without acetylsalicylic medication $21/31 = 67.7\%$ of the patients in the infarction group and $9/24 = 37.5\%$ of the patients in the bleeding group had unphysiologically enhanced ADP-induced platelet aggregation. With regard to the coagulation and fibrinolysis markers no significant differences were found between the two groups. In both groups, coagulation activity markers (fibrin monomer and thrombin-antithrombin III), as well as D-dimers were significantly higher than in controls in a high proportion of cases. In 5/47 of the infarction patients and in 3/34 of the haemorrhage patients the fibrin monomer levels were elevated to such an extent, that it can be considered as low grade disseminated intravascular coagulation.

In the cerebral haemorrhage group, 80.3% of the patients who subsequently died showed a significantly enhanced fibrin monomer concentration, compared with 28.6% of those who survived. The corresponding frequencies for D-dimer were 100% compared with 66.7%.

In the cerebral infarction group, the only analytical quantity showing a significant difference between patients with a fatal outcome and those with a non-fatal outcome was ADP (2 $\mu\text{mol/l}$) induced platelet aggregation (83.3% in the fatal group, 40.0% in the non-fatal group).

Introduction

Cerebral infarction and cerebral haemorrhage are events which may be accompanied by coagulation activation and (reactive) fibrinolysis. The clinical significance and relevance of coagulation and platelet abnormalities (1, 2) on the one hand and of coagulation and fibrinolysis factors (3) on the other hand have been studied earlier. None of the factors studied so far has shown a significant difference from normal in cases of cerebral infarction and cerebral bleeding.

In recent years a number of new, more sensitive quantities have become available for testing for clotting and fibrinolysis: fibrin monomers (4), thrombin-antithrombin-III complexes (5) and D-dimers (6). These quantities may be useful for sensitive monitoring of the course of the disease, for the better follow-up of the therapy and the possible estimation of the prognosis.

Aim of this study was to get more insight in the extent of platelet coagulation and fibrinolysis activation after recent cerebral bleeding and infarction.