Evidence against DIC in liver cirrhosis

EVIDENCE AGAINST DIC IN LIVER CIRRHOSIS. G. Flori, G. Mambelli, A. Vascelli, P.M. Sturza, Odempede La Cañeta, Lourou, and Department of Medicine, University of Milan, Switzerland.

To evaluate the hypothesis of disseminated intravascular coagulation (DIC) in liver cirrhosis (LC), we measured the plasma levels of fibrinopeptide A (FPA) and of fibrinogen-fibrin degradation fragment (FgF) in 58 patients with LC, and compared the results with those measured in 32 healthy subjects as well as in 42 patients with coning fibrin formation and lysis related to acute thromboembolism (TE, n = 33) and overt DIC (n = 9).

Results: Mean plasma FPA in LC was 2.4 ng/ml compared to 1.8 ng/ml in normals (p < 0.05) and 12.2 ng/ml in patients with TE or DIC (p < 0.0001). Mean plasma FgF in LC was 108 ng/ml compared to 31.2 ng/ml in normals (p < 0.0001) and to 616 ng/ml in patients with TE or DIC (p < 0.0001). 15 patients with LC (26%) had hypofibrinogenemia (fibrinogen < 1.7 g/l). Among these patients, plasma FPA and FgE were 2.3 and 134 ng/ml, as compared to 22.1 and 1310 ng/ml in patients with hypofibrinogenemia related to DIC (p < 0.0001). Intravenous heparin (60 IU/kg) resulted in a prompt decrease of plasma FPA in 14 patients with TE or DIC from 11.8 to 4.3 ng/ml (p < 0.005), but did not significantly change the FPA level in 15 patients with LC as well as in the healthy subjects.

Discussion: The present data indicate that thrombin mediated proteolysis of fibrinogen is only marginally increased in LC regardless of whether hypofibrinogenemia is present or not. The data are strongly against the hypothesis of DIC as a frequent and quantitatively important complication of LC and substantiates the conclusion that a "consumption coagulopathy" is not a major determinant of impaired hemostasis in liver cirrhosis.

Fibrinopeptide B 15-42 in liver cirrhosis: a sensitive indicator of mild fibrinolytic activity

Fibrinopeptide B15-42 in liver cirrhosis: a sensitive indicator of mild fibrinolytic activity. F. Marongiu, A.C. Acca, G. Mulass, M. Conti, G. Soriano, and A. Balestrieri. Institute of Internal Medicine, University of Cagliari, Italy.

In order to detect even minimal fibrinolytic activation in liver cirrhosis and to investigate whether an increased plasmin activity is related to a mild blood coagulation activation, we measured fibrinopeptide A (FPA) (Mallinkrodt) and fibrinopeptide B15-42 (BB 15-42) (IMCO and SORIN Biomedica) in 26 patients (16 men and 10 women, mean age 55.8 ± 13.1 years) with histologically proven liver cirrhosis. Mann-Whitney test, Student's t test and correlation coefficient r were employed for statistical analysis when appropriate. FPA and BB 15-42 were not normal distributed and thus their levels were expressed as median and range. FPA values were significantly different in cirrhotic patients (3.9, 0.5-24.2 ng/ml) from those of the controls (2.5, 0.5-3.9 ng/ml) (p < 0.01). BB 15-42 levels were significantly higher in cirrhotic patients (19.4, 7.1-103.1 ng/ml) than in controls (10.4, 5.1-15.4 ng/ml) (p < 0.01). Post mortem the patients were divided in two subgroups according to whether their FPA levels were high (subgroup 1, n = 10, FPA > 4.0 ng/ml) or normal (subgroup 2, n = 16, FPA ≤ 4.0 ng/ml). In patients with high FPA levels we found higher levels of BB 15-42 (22.2, 9.3-103.1 ng/ml) than in patients with normal FPA (13.6, 7.1-30.7 ng/ml). This difference was significant (p < 0.02). There was no relationship between FPA and BB 15-42. Our data indicate that in liver cirrhosis a mild fibrinolysis activation may occur. The role of a chronic intravascular coagulation appears to be significant in this regard. However the impaired clearance of plasminogen activators, the decreased synthesis of fibrinolysis inhibitors and the increased level of plasminogen-activator-inhibitor or glycoprotein may be also involved in determining fibrinolysis activation as suggested by the lack of correlation between FPA and BB 15-42.