Haemostasis in Blood Donors with Hepatitis C Virus

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In volume 22, No. 6, of Haemostasis, it was described by Kloczko et al. [1] that among several parameters tested, decline in protein C (PC) level was the earliest detectable in the coagulation system in an individual subgroup of alcoholic liver disease. In this paper we describe coagulation parameters in 41 asymptomatic blood donors (3 female and 38 male, aged 20-53 years) diagnosed as hepatitis C virus (HCV), by serologic test Elisa, 2nd generation (Abbott). The patients were subdivided into three groups. Twenty-one patients with chronic active hepatitis (CAH), 6 with chronic persistent hepatitis and 14 virus C carriers. The diagnosis was based on clinical, laboratory and histological data. All CAH patients were classified as mild chronic hepatitis according to International Group [2]. The control group consisted of 22 normal subjects, sex and age matched.

Previous investigations have found mild or no alteration in pro-thrombin time (PT), vitamin K independent factors and factor V in CAH patients, and the results were related to the degree of liver damage [3]. In our patients, the synthe-

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min in HCV patients corroborate this hypothesis, since in liver disease, a fairly good correlation between its serum concentration and measured synthesis often exists [4].

sis liver function seems to be preserved since PT, factor V and factor VII are within the normal range and not different from the control group. Normal values of prealbumin

Table 1. Hemostatic parameters in HCV patients and controls (mean $\pm$ SD)

Control VCC CPH CAH

VCC = Virus C carrier; CPH = chronic persistent hepatitis; APTT = activated partial thromboplastin time; PS = protein S; ATIII = antithrombin III; PA = prealbumin.

Plasma prekallikrein (PK) can be decreased in patients with chronic liver disease, and the entity of liver damage is the most important factor influencing the behavior of this protein [5]. Our results suggest no alteration of contact phase in HCV patients, since plasmatric PK, factor XII, factor XI and C1 inhibitor were not different from the control group (table 1).

CAH patients showed an increase in α_2 -macroglobulin and factor VIII ($p < 0.05$). These factors are acute-phase response proteins, and can be elevated in various clinical conditions, including chronic hepatitis and cirrhosis [3, 5].

It has been reported that the antithrombin III plasma level is not altered in CAH [6-8], and our results corroborate these findings.

We detected a decrease in immunologic PC ($p < 0.01$) in CAH patients. PC concentration correlated with one-stage PT and factor VII ($p < 0.05$). It is quite surprising and interesting that although different etiologic agents are involved, similar findings were found in individual subgroups of alcoholic liver disease by Kloczko et al. [1]. Impaired liver synthetic rate could be the major factor contributing to these low PC levels, but we cannot rule out the possibility of increased turnover, as has previously been found in chronic liver disease for other hemostatic components. In conclusion, our results confirm

that most of the blood coagulation abnormalities observed in patients with liver disease depend on the severity of the disease [8]. In our study, CAH patients were all asymptomatic blood donors in an initial disease stage and classified as mild-degree chronic hepatitis, by histological biopsy. Like in alcoholic liver disease, PC should be considered as another marker of liver cell function in HCV, since it was decreased before there was other evidence of liver damage.

However, functional methods must be employed for a better evaluation.

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