Effects of a Normalized Protein Catabolism Rate on Platelet Functions in Non-Dialyzed Uraemic Patients

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Dear Sir,

It is accepted that urea [1], guanidinosuccinic acid [2], phenols and phenolic acids [1] are uraemic toxins impairing platelet adhesion and aggregation. These substances are enzymatic breakdown products of protein and amino acids [3]. The aim of our study was to elicit the effects of a normalized protein catabolism rate (NPCR) on platelet functions.

This study included 48 non-dialyzed patients (28 males, 20 females) with chronic renal failure. The mean age of patients was 46 ± 16 years (range 13-70). They were not using any drug affecting platelet functions. Dietary protein intake was 0.5 g/kg body weight/day.

The patients were divided into three groups according to body mass index (BMI, kg/m²; < 20 underweight, 20-25 normal, > 25 overweight). Findings were statistically interpreted by Student's t test, variance and regression analysis. The patients with low BMI had mild acidosis and infectious diseases (genito-urinary, gastro-intestinal), and their blood urea nitrogen levels (91.2 ± 26.7 mmol/l) were higher than those of the other groups (71 ± 22.6 , 58.9 ± 24.5 mmol/l; $p < 0.05$, $p < 0.01$). Serum albumin levels (25 ± 1.2 g/l) of the group with low BMI were lower than those of the other groups (38 ± 1.7 , 30 ± 3 g/l; $p < 0.0001$). Platelet counts (204 ± 55 , 219 ± 30 , $202 \pm 57 \times 10^9/l$) and hematocrit levels (0.25 ± 0.02 , 0.23 ± 0.04 , 0.24 ± 0.04 volume fraction) were not different among the groups ($p > 0.05$). Other results are shown in table 1.

When the results in table 1 are statistically investigated, glomerular filtration rates of each group are not different from the others ($p > 0.05$). The patients in the low-BMI group had a higher NPCR ($p < 0.001$, $p < 0.001$), lower platelet adhesion rates ($p < 0.001$, $p < 0.001$) and a more prolonged bleeding time ($p < 0.05$, $p < 0.01$) than the other groups. The patients in the high-BMI group had a lower NPCR than the normal-BMI group ($p < 0.01$), and their ADP-, epi-nephrine, ristocetin- and collagen-stimulated mean percent platelet aggregation values were higher than those of the low-BMI group ($p < 0.01$, $p < 0.0001$, $p < 0.01$, $p < 0.01$, respectively) and than those of the normal-BMI group for only epinephrine, ristocetin and collagen ($p < 0.001$, $p < 0.05$, $p < 0.05$, respectively). In low-BMI patients, ADP- and epinephrine-stimulated percent aggregation values were lower than those of the normal-

BMI group ($p < 0.02$, $p < 0.05$, respectively). There were inverse linear relations of NPCR to platelet adhesion rates and ADP- and epinephrine-stimulated percent platelet aggregation values in all the patients ($r = -0.701$, $r = -0.732$, $r \approx -0.703$, $p < 0.0001$, respectively).

In the present study, although the patients had the same degrees of renal failure, it

Table 1. NPCR and platelet functions in non-dialyzed uraemic patients (means $\pm$ SD)

epinephrine ristocetin collagen

Groups

GFR Bleeding Platelet Platelet aggregation,

ADP

ml/min time adhesion

1.73 ml/min %

NPCR

g/kg/day

Low BMI

(17.6 ± 1.4 , $n = 9$)

Normal BMI

(22.4 ± 1.7 , $n = 30$)

High BMI

(29.6 ± 3.1 , $n = 9$)

15.5 ± 8.8 5.1 ± 2.1 13.8 ± 6.2 35.2 ± 26.7 40 ± 11.9 59 ± 25.2 55.7 ± 29.2 1.41 ± 0.37

11.7 ± 6.3 3.6 ± 1.3 25.6 ± 8.1 59.3 ± 26.4 59.6 ± 25.9 70.5 ± 22.7 61.6 ± 37.1 0.91 ± 0.03

14.3 ± 8.1 3.3 ± 0.4 25.6 ± 5.2 71.7 ± 22.1 89.3 ± 12.8 88.5 ± 7.1 89.5 ± 16.2 0.86 ± 0.09

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was determined that platelet functions changed depending upon the NPCR and that increased NPCR values might cause platelet dysfunctions.

Klahr et al. [4] noted that a decrease in protein intake may prevent abnormalities in coagulation associated with other benefits. Bergström and Lindholm [5] reported that morbidity and mortality might be reduced if patients are well nourished.

It may be said that low protein and adequate energy intake may normalize the NPCR and platelet functions. To obtain clearer knowledge, detailed studies should be done.

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