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LETTER TO THE EDITORS-IN-CHIEF

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ERYTHROCYTE DEFORMABILITY IN PATIENTS WITH PRIMARY HYPERTRIGLYCERIDEMIA.

Amparo Vayá, Marcial Martínez, Victor Moreno and Justo Aznar
Department of Clinical Pathology Hospital "La Fe". Valencia.

In a previous study (1) we observed that patients suffering from primary hyperlipoproteinemia (PHLP) presented a decreased red blood cell deformability when compared with a normolipemic healthy control group. This increased rigidity could not be attributed to the increased cholesterol in the red blood cell membrane which these patients showed, because there was no statistically significant correlation between the two parameters. Moreover, the increased rigidity could not be attributed to either hematimetric or morphological changes in the erythrocytes. Among PHLP, primary hypertriglyceridemias (PHTG) frequently show alterations in the hydrocarbon metabolism.

Another of the causes of decreased red cell deformability is the increase in glycosylated hemoglobin (HbA_1c) (2). In order to find out whether the increased erythrocyte rigidity observed in PHTG is related to the HbA_1c level, we have studied the rigidity index (RI), using the Hanss Hemorheometre (3) and the HbA_1c level in 21 patients belonging to six families affected by PHTG.

Table I shows the values of serum lipids, glucose, HbA_1c, leucocyte count, remanent leucocyte count in the 8% RBC suspension and RI.

Table 1

Rigidity index (RI), plasmatic lipids (TG, CHOL), glucose, HbA_1c leucocytes (Leuc) and remanent leucocytes (Rem Leuc) in the 8% RBC suspension in primary hypertriglyceridemia (PHTG) and in the control group (CG).

	PHTG	CG
TG mg/dl	548 ± 775*	96 ± 32
CHOL mg/dl	204 ± 42 ns	215 ± 35
Glucose mg/dl	99 ± 12 ns	90 ± 18
HbA_1c %	4.95 ± 0.36 ns	4.80 ± 0.36
RI	9.16 ± 1.0*	8.23 ± 0.8
Leuc x 10^9 /l	7.0 ± 2.0 ns	6.5 ± 2.3
Rem Leuc x 10^9 /l	0.20 ± 0.05 ns	0.18 ± 0.2

ns: not significant; * $p<0.001$

We observed that RI is significantly higher ($p<0.001$) in PHTG (9.16 ± 1.0) than in the control group (8.23 ± 0.8). However, there are no statistically significant differences between patients and controls with respect to glucemia (99 ± 12 mg/dl vs 92 ± 17 mg/dl) or HbA_1c level ($4.95\% \pm 0.36$ vs $4.80\% \pm 0.36$). In addition, no correlation exists between RI and the above mentioned parameters.

On the other hand, the increased RI found in our PHTG patients can not be attributed to the presence of contaminant leucocytes in the RBC suspension, because there are no differences of this kind between the patients and control group. Moreover, in a previous study we showed that RI is not influenced by remanent leucocytes, when the leucocyte count is in the normal range (4).

Our results agree with those of other authors who used other methods to measure the red blood cell deformability in PHLP. Eijzenbach (5) using the ektacytometer and Müller (6) using fluorescence polarization found that PHTG patients showed less deformable red blood cells than normolipemic patients. The cause of the decreased red cell deformability in PHTG remains to be established but it does not seem according to our results to have to do, with the accompanying alterations of the hydrocarbon metabolism, concretely with the HbA_1c level.

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