portrays the typical variations in cell light scattering properties of a HUCB sample from time 0 (just before culturing) to 72 hours after culture commencement. Table 1 gives the results obtained, clearly showing that there were no differences in PHA's capacity to stimulate T-lymphocytes between the HUCB and PB samples tested, as evaluated in terms of $CD3^+CD25^+$ and $CD3^+HLA\text{-}DR^+$ co-expression and in terms of percentage of cell number in S-phase. Finally, Figure 1B shows the pattern of change in HLA-DR and CD25 co-expression by $CD3^+$ T-cells and in the S-phase of the cell cycle at time 0 and throughout the 72 hours of culturing.

As shown, no differences were revealed between HUCB and adult PB functional assays, suggesting that HUCB lymphocyte functional immaturity could reside in other immunologic mechanisms, thus needing further investigation.

Key words

Human umbilical cord blood, lymphocyte blastogenesis, flow cytometry.

Correspondence

Giovanni D'Arena, M.D., Division of Hematology, IRCCS "Casa Sollievo della Sofferenza" Hospital, 71013 San Giovanni Rotondo, Italy. Phone: international +39-0882-410539 • Fax: international +39-0882-411705

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A patient homozygous for mutation 20210 A in the prothrombin gene with venous thrombosis and transient ischemic attacks of thrombotic origin

ANGEL JOSÉ GONZÁLEZ ORDÓÑEZ,* JESÚS MARÍA MEDINA RODRIGUEZ,* CARMEN ROSA FERNÁNDEZ ALVAREZ,* MARÍA DOLORES MACIAS ROBLES,# ELIECER COTO GARCÍA°

**Hematology and #Internal Medicine, S. Agustin Hospital, Avilés, Spain; °Molecular Genetics, Asturias Central Hospital, Oviedo, Spain*

It is well established that genetic disorders interact with environmental factors to cause thrombotic diseases.[1] Therefore, antithrombin, protein C, protein S deficiencies and the more recently described factor V Leiden and prothrombin mutations are currently been investigated to explain some thrombophilic states. We report the case of a 63-year-old man who developed two transient ischemic attacks and two years later an extensive femoro-iliac venous thrombosis. He was genotyped as FV R506Q negative and FII G20210A positive in homozygous state (FII 20210AA).

A polymorphism in the 3'-UTR (untranslated region) of the prothrombin gene, due to a G to A transition at nucleotide 20210 has been recently described.[2] This anomaly was associated with elevated plasma prothrombin (factor II) levels and with an increased risk of venous thrombosis.[2] This situation is not uncommon. The variant (A allele) was identified in 2.3% of healthy controls and in 6.2% of unselected patients with venous thrombosis, in the original paper which describes the Dutch population. It was established that there was a 2.8-fold increased risk of venous thrombosis in the carriers.

Subsequently, other reports described different populations with similar frequencies: 2.6% in healthy donors and 5.0% in patients with venous thromboembolism, in the Cambridge report[3] and 1.8% in the control group versus 7.1% in patients from Sweden.[4]

The first homozygous patient was mentioned in the original paper.[2] Specifically, the first report was made on a young Mexican male (24 years old) who had a myocardial infarction (MI) and subsequently iliofemoral venous thrombosis and massive pulmonary embolus. Although the patient was found to be a factor V Leiden carrier,[5] an association between the arterial thrombotic events and the prothrombin muta-

tion can be suspected. Rosendaal *et al.* have recently demonstrated the relation between 20210 GA heterozygosity and MI.[6] They studied young women with MI and reported an odds ratio of 4.0 in comparison with healthy age-matched women. Another study from Austria also suggests an association with coronary heart diseases.[7] The possible relation with arterial thrombotic events in the Mediterranean area, however, has not been well established. This mutation was not associated with cerebral ischemia in a retrospective Italian case-controlled study[8] nor in another Spanish prospective one.[9] This latter study was not able to show an effect in coronary heart disease either, because the result (4% versus 2%) did not reach statistical significance.[9] No homozygous patients (20210 AA) were found in any of these series.

We report the case of a 65-year-old man who developed an extensive femoro-iliac venous thrombosis the seventh day after a transurethral resection for benign prostatic hyperplasia. He was heparinized with standard LMWH doses but, eight days afterwards, during coumarin introduction, he developed a new thrombosis in the same territory (left limb). In reviewing his previous clinical history two further episodes were brought out: transient global amnesias suggesting transient ischemic attacks of thrombotic origin when the patient was 63 years old. The electroencephalographic record was normal. The electrocardiographic and echocardiographic studies ruled out an emboligenic heart disease. The supra-aortic echo-Doppler ruled out vascular stenosis and the CT scan showed multiple hypodense ischemic areas. He was diagnosed as having cerebrovascular disease involving small vessels. He was analyzed for DNA mutations in factor V and factor II by PCR, a homozygous state for the FII: G20210A (FII 20210 AA) being found. The amplified DNA digested by HindIII showed a single 322-bp band in electrophoresis versus a 345-bp band found in normal (20210 GG) subjects and both bands (345 and 322-bp) in heterozygotes. One month after the acute event, oral anticoagulant therapy was stopped and a subtherapeutic dose of nadroparine was started to measure prothrombin level. It was 142%, similar to the 146%, previously reported.[5]

As occurs with factor V:Q506, homozygous subjects will have a higher risk of thromboembolic events. However, in these cases, the relative risk will be established with more dificulty given the rareness of homozygotes. Considering a similar allelic frequency to the 1.2% originally described[2] or to other published ones,[3,4,6-9] the probability of the homozygous state in a Hardy-Weinberg equilibrium is low (0.0002-0.00007), but not exceptionally so.

Key words

Prothrombin gene, 3'-UTR, factor II, homozygous, thrombosis

Correspondence

Angel J. González Ordoñez, M.D., Servicio de Hematologia, Hospital de S. Agustin, 33400 Avilés, Spain. Fax: international +34.985.123012 •E-mail: jagonzalez@medinet.es

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Role of autologous bone marrow transplantation as consolidation chemotherapy in acute promyelocytic leukemia patients in complete remission

Emanuela Ottaviani, Giovanni Martinelli, Nicoletta Testoni, Giuseppe Visani, Monica Tani, Sante Tura

Institute of Hematology and Medical Oncology "Seràgnoli", University of Bologna, Italy

Autologous bone marrow transplantation (ABMT), which consents a low mortality rate, has been proposed as an alternative approach to maintenance chemotherapy in patients with acute promyelocytic leukemia (APL) in first complete remission irrespective of the patients' molecular status. Sixteen patients with acute APL in complete remission were submitted to ABMT and were analyzed for the presence of the