whether or not these polymorphisms directly correlate with the risk of coronary thrombosis. The functionality of the –402A is not yet defined, and the –402A allele is in strong linkage disequilibrium with the –670C and –630G alleles of two other FVII promoter polymorphisms, being part of the common haplotype associated with significantly higher risk of coronary events, as shown by Carew et al. (2). The mechanism by which this haplotype modulates FVII levels is not yet elucidated, and the haplotype may also be associated with other genetic polymorphisms or genetic traits contributing to the features related to risk for coronary thrombosis.

In summary, the results from our study supports the hypothesis of an association between the haplotype containing the –402G/A polymorphism and increased risk of myocardial infarction.

References

Gender and stable angina pectoris: Women have greater thrombin-evoked platelet activity but similar adenosine diphosphate-induced platelet responses

Dear Sir,

Previous studies have revealed that platelets from premenopausal women have enhanced reactivity to aggregating agents compared to platelets from males of a similar age (1–3). We have chosen to study middle-aged or older patients with stable angina pectoris with respect to gender differences of platelet reactivity. The local ethics committee approved the study. 21 females (age 64±10 (SD) years) and 72 males (age 61±8 (SD) years) with stable angina pectoris (Table 1A) were included. All had at least one significant flow limiting stenotic lesion in at least one major coronary artery. The upper age limit was 75 and patients having diabetes mellitus, rheumatoid arthritis or a myocardial infarction in the preceding three months were excluded. The Student’s t-test for unpaired data was employed for testing differences between means. Platelet counts and reactivity were analysed before elective angiography. A Cell-Dyn 4000 (Abbott Diagnostics, CA, USA) was used to determine platelet counts. Citrate anticoagulated whole blood was used for flow cytometry measurements. As a measure of platelet reactivity surface bound fibrinogen after stimulation (4, 5) was analysed with a Cytotron Absolute Flow Cytometer (Ortho Raritan, NJ, USA). Platelets were stimulated ex vivo with a thrombin-receptor activating peptide (TRAP-6) (57 mM and 74 mM) and ADP (1.7 µM and 8.5 µM). Platelets were identified using a phycoerythrin-conjugated monoclonal antibody against glycoprotein Ib (Dako AS, Denmark). A fluorescein isothiocyanate-conjugated chicken anti-human fibrinogen polyclonal antibody (Biopool AB, Sweden) recognised

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Table 1: A) Clinical data for the two study groups, B) Gender differences of platelet counts and platelet reactivity in stable angina pectoris.

|                      | Women (n=21) | Men (n=72) | p-value  
|----------------------|--------------|------------|---------  
| **A)**                |              |            | Student's t test |  
| Age                  | 64±10(SD)    | 61±8(SD)   |          |  
| Current smokers (%)  | 19           | 12         |          |  
| Hypertension requiring medical treatment (%) | 48            | 24         |          |  
| Previous myocardial infarction (%) | 24           | 24         |          |  
| Medication at admission |             |            |          |  
| Aspirin (%)          | 81           | 97         |          |  
| ACE-inhibitors (%)   | 9            | 18         |          |  
| Beta-blockers (%)    | 90           | 90         |          |  
| Ca²⁺ channel blockers (%) | 43          | 25         |          |  
| Lipid-lowering drugs (%) | 90          | 65         |          |  
| Prophylactic nitro-glycerine (%) | 48         | 64         |          |  
| **B)**                |              |            |          |  
| Number of diseased coronary arteries (1–3) | 1.5±0.7     | 1.8±0.8    |          |  
| Platelet counts (x10¹¹) | 273±60      | 230±55     | <0.01   |  
| 57 µM TRAP-6 (% activated cells) | 57±16       | 39±20      | <0.001  |  
| 74 µM TRAP-6 (% activated cells) | 59±19       | 46±17      | <0.01   |  
| 1.7 µMADP (% activated cells) | 37±19       | 30±15      | NS      |  
| 8.5 µMADP (% activated cells) | 63±14       | 57±17      | NS      |  

The data are expressed as mean±SD. NS = not significant; SD = standard deviation; TRAP-6 = thrombin-receptor activating peptide; # % fibrinogen-positive cells after stimulation = platelet reactivity

References


Does obesity constitute a risk factor for upper-extremity deep vein thrombosis?

Dear Sir,

Of the various risk factors associated with lower extremity deep vein thrombosis, obesity seems to constitute an independent risk factor whose magnitude ranges between 2 and 3 (1–5). At present, whether obesity constitutes a risk factor for other thrombotic locations has not been established. In particular, body mass index (BMI) has not been considered as a thrombotic risk factor for upper-extremity deep vein thrombosis (UEDVT) in the various papers dealing with inherited and acquired risk factors for this unusual thrombotic location (6–14). Therefore, we aimed to determine whether a high BMI constitutes an independent risk factor in patients with UEDVT. Sixty-two consecutive, unrelated, first well-documented UEDVT patients were included. Ten patients were excluded: seven because of malignancy and one each because of infectious, renal or hepatic disease. Diagnosis had been confirmed by venography, Doppler-ultrasonography or dynamic venography (for thoracic outlet syndrome diagnosis). They all had been referred to the Haemostasis and Thrombosis

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