

Aterotrombosi e Tromboembolismo arterioso: aspetti clinici

PLATELET ACTIVATION AND THROMBOSIS IN PATIENTS WITH FACTOR V LEIDEN MUTATION: A CROSS SECTIONAL STUDY

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Background. Factor V Leiden (FVL) is the most common inherited thrombophilia and a well-established risk factor for venous thromboembolism (VTE). Beyond its role in venous thrombosis, emerging evidence suggests that FVL may contribute to arterial thrombotic events through enhanced thrombin generation and platelet activation. We aimed to characterize the prothrombotic profile of FVL carriers by assessing biomarkers of platelet and coagulation pathway activation.

Methods. We conducted a preliminary cross-sectional study including 50 patients with genetically confirmed heterozygous or homozygous FVL mutation and previous VTE, compared with 50 age- and sex-matched individuals with prior VTE but no detectable inherited thrombophilic mutations. Blood samples were collected for measurement of soluble P-selectin (sP-selectin), soluble CD40 ligand (sCD40L), and prothrombin fragment 1+2 (F1+2) as markers of platelet activation and thrombin generation. Clinical and laboratory characteristics were recorded for all participants.

Results. Preliminary analyses showed that patients carrying FVL exhibited significantly higher levels of platelet activation markers, including sP-selectin and sCD40L, compared with the reference group. Similarly, F1+2 levels were increased in FVL carriers, indicating enhanced thrombin generation. These findings suggest that FVL is associated with a persistent prothrombotic state characterized by concomitant platelet hyperreactivity and activation of the coagulation cascade, despite ongoing anticoagulant therapy in a proportion of patients.

Conclusions. FVL mutation is associated with enhanced platelet activation and hypercoagulability, supporting a broader thrombotic phenotype beyond activated protein C resistance. These findings may contribute to improved thrombotic risk stratification in FVL carriers.