

**Béla Nagy Jr: PLATELET ACTIVATION MARKERS: IN VITRO
EXPERIMENTS AND STUDIES IN PROTHROMBOTIC DISORDERS**

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SUMMARY

Cardiovascular and cerebrovascular diseases are still the leading cause of death in the developed world. Activated platelets are fundamentally involved in the pathomechanism of thrombotic complications in these diseases. That is why the investigation of activated platelets has become more obvious in the daily routine. Our primary goal was to detect increased platelet activation in time that occurs often in acute or chronic vascular disorders or induced by invasive therapeutic intervention in such states.

In this present study, we found that in type 2 DM and obese patients, platelet and soluble P-selectin levels were significantly elevated compared to the findings in healthy controls, but the most studied P-selectin gene polymorphism (Thr715Pro) did not affect plasma soluble P-selectin values in the patient groups. In DM patients divided into different subgroups according to several demographical variables, the levels of soluble P-selectin still did not vary notably according to their genotype for this polymorphism.

In patients with stable angina, significantly increased levels of PMPs, platelet P-selectin and platelet-monocyte aggregates were measured compared to angina patients underwent catheterization alone. However, soluble P-selectin levels did not show marked difference between the two study groups. Thus, the measurement of PMP levels can be considered as an early sensitive activation marker to detect platelet activity right after invasive cardiological interventions.

In vitro, FXIII-A₂ was not expressed from its intracellular localization on washed platelets activated by TRAP. Thus, surface-bound FXIII on stimulated whole blood platelets is of plasma origin. The presence of $\gamma A/\gamma'$ fibrinogen significantly potentiated

the binding of purified FXIII-A₂B₂ on stimulated washed platelets, but no FXIII-A positivity was seen without this type of fibrinogen or with fibrinogen having γ A-chain only. Accordingly, plasma FXIII is unable to bind directly to the activated platelet surface, and significant binding of non-active FXIII occurs only when GPIIb/IIIa receptor-bound fibrinogen with γ '-chain is present. Analysis of FXIII binding to platelets may be an additional sensitive activation marker in the future.

KEYWORDS

Platelet activation

P-selectin

Flow cytometry

Thr715Pro P-selectin polymorphism

Type 2 diabetes mellitus

Factor XIII

Fibrinogen

TRAP

Stenting

Microparticle

TÁRGYSZAVAK

Vérlemezke aktiváció

P-szelektin

Áramlási citometria

Thr715Pro P-szelektin polimorfizmus

2-es típusú diabetes mellitus

XIII-as faktor

Fibrinogén

TRAP

Sztentelés

Mikropartikula