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VINDER AF 1. PRÆMIE

VED DSTH'S FOREDRAGSKONKURRENCE

1. PRÆMIE

ABSTRACT:

THE ANTIPLATELET EFFECT OF ASPIRIN DECLINES THROUGH 24 HOURS IN PATIENTS WITH PREVIOUS DEFINITE STENT THROMBOSIS

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BACKGROUND

■ Low-dose aspirin given once daily is considered to inhibit platelets for 24 hours. However, recent studies point toward an attenuation of platelet inhibition within this standard dosing interval. Patients with previous stent thrombosis (ST) have an increased risk of cardiovascular events, and we have previously shown that they have a reduced antiplatelet effect of aspirin.

AIM

■ We investigated if platelet inhibition by aspirin declines through the 24-hour dosing interval in patients with previous ST. Furthermore, we explored if an increased platelet turnover is associated with reduced platelet inhibition by aspirin.

METHODS

■ We included 50 patients with previous ST, 100 patients with stable coronary artery disease and 50 healthy volunteers. All participants were on 75 mg once-daily aspirin mono-therapy. Platelet aggregation induced by arachidonic acid (1.0 mM) and collagen (1.0 µg/ml) was measured 1 and 24 hours after aspirin intake using the Multiplate[®] Analyzer. Markers of COX-activity (thromboxane B₂), platelet turnover (immature platelets and mean platelet volume), platelet produc-

tion (thrombopoietin), and platelet activation (soluble P-selectin) were measured. Compliance was assessed by serum thromboxane B₂.

RESULTS

■ All patients were compliant with aspirin. Platelet aggregation increased significantly from 1 to 24 hours after aspirin intake ($p < 0.0001$) with corresponding increases in COX-1 activity ($p < 0.0001$) and platelet activation ($p < 0.0001$). Patients with previous ST had the highest level of platelet turnover ($p < 0.01$) and platelet production ($p < 0.0001$).

CONCLUSIONS

■ Platelet inhibition by aspirin declines significantly during the 24-hour dosing interval as reflected by increased levels of platelet aggregation, COX-1 activity and platelet activation. Patients with previous ST have a distinct prothrombotic phenotype involving increased platelet turnover and platelet production. Our findings suggest that not all patients derive optimal cardiovascular protection from once-daily aspirin. It should be investigated if a twice-daily treatment regimen provides incremental clinical benefit, particularly in patients with previous ST.

JULIE BROGAARD LARSEN

VINDER AF 2. PRÆMIE

VED DSTH'S FOREDRAGSKONKURRENCE

2. PRÆMIE

ABSTRACT:

COULD VON WILLEBRAND'S DISEASE BE OVERLOOKED IN WOMEN USING COMBINED ORAL CONTRACEPTIVES?

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BACKGROUND

■ Women of fertile age referred for diagnosis of von Willebrand's disease (vWD) are often treated with combined oral contraceptives (COCs) because of menorrhagia. Only a few previous studies have investigated the effect of COCs on von Willebrand factor (vWF), and the results of these are conflicting, but COCs could possibly cause an increase in vWF levels and thereby mask the disease in women taking COCs.

AIMS

■ The aim of the study was to investigate the influence of COCs on vWF in healthy women.

METHODS AND MATERIALS

■ We included two groups of healthy participants, 1) a group of women starting COC treatment and 2) a control group who did not receive COC treatment during the study. Blood samples were obtained at the time of inclusion and after 3 months in both groups. The COC group began treatment after the 1st sampling. Analysis of vWF antigen (vWF:Ag), ristocetin cofactor (vWF:RCO) and collagen binding (vWF:CB), factor VIII clot (FVIII:C) and C-reactive protein (CRP) were performed in both groups, and ABO blood group was determined.

RESULTS

■ Our preliminary data showed significant increases in median levels of vWF:RCO, vWF:CB, FVIII:C and CRP in the COC group. The increase was also significantly higher than the increase found in the control, except for vWF:CB. Median vWF:Ag levels did not change from baseline to 3 months in either group.

CONCLUSION

■ The present study indicates that COCs do have an influence on vWF in healthy women. This may impair the diagnosis of vWD in women taking COCs.

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