**1100-60 Platelet Dysfunction Following a 300 mg Clopidogrel Loading Dose in Type 2 Diabetes Undergoing Coronary Stenting**

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**Background:** A 300 mg clopidogrel loading dose (LD) is currently recommended to reduce the early risk of stent thrombosis (ST). However, ST still occurs and most frequently in patients with type 2 diabetes (D). Aim of this study was to compare platelet aggregation (PA) in D with that of non-diabetic (ND) patients undergoing coronary stenting (CS) receiving a 300 mg clopidogrel LD. **Methods:** We studied a total number of 48 pts undergoing CS receiving a 300 mg clopidogrel LD at intervention time. All pts were on aspirin (100mg/d) and treated with clopidogrel (75 mg/d) for one month. Patients receiving GP IIb/IIIa blockers were not included. Diabetic status was defined according to WHO criteria. PA was assessed by light transmittance following ADP-stimuli (6µM) at baseline (B), and 4 hours and 24 hours following CS. **Results** (means±SD): In the overall study population, 13/48 (27%) were D and 35/48 (73%) were ND. D had a higher degree of PA at B (63±22 vs 52±15; p=0.05) and a lower response to clopidogrel LD at 4 (51±18 vs 37±14; p=0.007) and 24 (44±22 vs 30±17; p=0.05) hours. Furthermore, PA was significantly higher (p=0.004) in D during the overall study time course by MANOVA analysis (Figure). **Conclusion:** D have a higher degree of PA when only on aspirin treatment (confirming data from previous studies) and a lower response to a standard 300 mg clopidogrel LD. This may explain the increased early risk of ST in D and suggest the use of a higher clopidogrel LD in these patients.

**1100-61 Effect of a 600 mg Clopidogrel Loading Dose on Platelet Function in Patients Undergoing Coronary Stenting**

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**Background:** A 300 mg clopidogrel loading dose (LD) is currently used to reduce the early risk of stent thrombosis. Since a variable individual response to such regimen has been described, a higher LD is suggested to optimize antplatelet effects. The aim of the study was to compare platelet function in pts undergoing coronary stenting (CS) and receiving a 300 mg or 600 mg clopidogrel LD. **Methods:** We studied 40 pts receiving a 300 mg (LD-300, n=20) or 600 mg clopidogrel LD (LD-600, n=20) at intervention time. All pts were on aspirin (100mg/d) and treated with clopidogrel (75 mg/d) for 1 month. Platelet aggregation (PA) was assessed by light transmittance aggregometry in ADP (5µM) stimulating platelets, and platelet GPIIb/IIIa activation was assessed by whole blood flow cytometry using anti-fibrinogen (AFB) polyclonal antibodies following ADP (2 µM) stimulus at baseline (B), 4 hrs, 24 hrs and 48 hrs following CS. **Results** (means±SD): Although no differences were found in ADP-induced PA between 300-LD and 600-LD pts at any time point (table), an earlier and a greater inhibition on ADP-induced GPIIb/IIIa activation was achieved in 600-LD pts during overall study time course (p=0.001; MANOVA). There were no differences in bleeding complications. **Conclusion:** Although a 600 mg clopidogrel LD does not increase inhibition of ADP-induced PA compared to a 300 mg LD, it confers a more rapid and extensive inhibition of GP IIb/IIIa early after CS sug- gesting a more effective protection against early thrombotic risk.

**1101 Intravascular Ultrasound and Drug-Eluting Stents**

**Monday, March 08, 2004, 3:00 p.m.-5:00 p.m.**

Morial Convention Center, Hall G

**Presentation Hour: 3:00 p.m.-4:00 p.m.**

**1101-41 Intravascular Ultrasound Assessment of Lesions With Sirolimus-Eluting Stent Failure**

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**Background:** Little intravascular ultrasound (IVUS) information is available in patients with sirolimus-eluting stent (SES) failure. **Methods and Results:** IVUS was performed in 33 lesions of 23 patients with SES failure (30% diabetic patients and 26% brachytherapy failure). (1) There were 2 subacute and 1 late thromboses that resulted in myocardial infarction (n=2) and death (n=1); stent under-expansion was identified in 2 patients, residual edge dissection in 1, and residual edge stenosis in 1. (2) In 1 lesion, follow-up IVUS revealed no stent. In this case post-stenting IVUS showed significant stent protrusion into the aorta; we speculate that stent embolization might have occurred during disengagement of the guiding catheter. (3) In 3 patients, there was a new stenosis within the adjacent reference segment, but not at the edge. (4) SES-in-stent restenosis (ISR) was observed in 25 lesions, including 5 edge restenoses. Pre-SES lesion morphology included 5 bifurcations, 2 ostial lesions and 1 total occlusion. A gap between stents was shown in 4 ISR lesions (16%); and stent underexpansion was identified in 50% (especially, in 4/5 bifurcation lesions (Fig.A)). Both negative remodeling (Fig.B) and intimal hyperplasia (Fig.C) were observed in 5 stent edge ISR. **Conclusions:** The most common IVUS finding in SES failure is stent underexpansion. However, other findings are also seen including gaps between multiple stents, new stenosis formation, and negative remodeling and intimal hyperplasia at edge restenosis.