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wall motion was quantified by both gated SPECT and echocardiography.

Results: At gated SPECT, SC implantation was scored as effective in terms of improvement of flow and function at rest in patients #1 and #4. In patients that improved, gated SPECT showed a reduction of scar area of 12% and 34% in terms of extension and severity, respectively. Patients that did not improve showed more extensive scar area and impairment of regional wall motion before surgery than patients that improved after SC. At QMCE, SC implantation was scored as effective in terms of flow improvement in patients #1,#2 and #4, whereas regional wall motion improved in patient #1 only. Frequent or complex ventricular arrhythmias were not observed at 24-h ECG Holter monitoring during follow-up.

Conclusion: In patients with previous myocardial infarction and dominance of scar, SC implantation may reverse hypoperfusion and regional wall motion abnormalities. However, extensive scar and severe akinesis seem to benefit less from this procedure.

1004-99

Remodeling of Radial Artery Anastomosed to Internal Thoracic Artery as an I-Composite Graft

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Background: Internal thoracic artery (ITA) remodels its diameter in response to the flow requirement. However, it is unclear whether radial artery (RA) has a capacity for remodeling, when anastomosed to ITA as an I-composite graft. The purpose of this study was to elucidate the remodeling of the RA used as an I-composite graft with ITA. Methods: We studied 16 consecutive patients who underwent coronary artery bypass grafting using RA anastomosed to ITA as an I-composite graft. The diameter of ITA and RA were evaluated before surgery and at 3 weeks and 1 year after surgery. Results: The RAs were significantly larger in diameter than ITAs (3.5±0.3 mm vs 2.2±0.3 mm, p<0.0001) and the ratio of RA to ITA was 1.6: 1 before surgery. Among 16 patients 2 patients had Icomposite grafts with left ITA and RA, while 14 patients had I-composite grafts with right ITA and RA. 15 RAs had two sequential anastomoses and 1 RA had a single anastomosis. 3 weeks after surgery all grafts were confirmed to be patent by angiography and there was no significant difference in diameter between RAs and ITAs (2.2±0.5 mm vs 2.1±0.5 mm). The ratio of RA to ITA significantly reduced to 1.0 : 1 (p=0.0008) as compared with preoperative ratio. 4 patients were followed a year after surgery. The diameter of ITAs and RAs were increased 11 % and 10 %, respectively in 3 patients without changing the ratio of RA to ITA. Conclusion: This study suggests that RA adjusts the diameter in response to the ITA flow, when anastomosed to ITA as an I-composite graft. These results may support the use of ITA-RA composite graft as an extended arterial graft with remodeling capacity.

POSTER SESSION

1022A-MP Moderated Poster Session...Hyperglycemia and Diabetes in Acute Coronary Syndromes I

Sunday, March 30, 2003, 9:00 a.m.-10:00 a.m. McCormick Place, Hall A

9:00 a.m.

1022A-MP-203 E-Selectin Gene Promoter Is a Predictor of Outcome in Unstable Angina

Maria M. Irurita, Florentino Sanchez, Angel Loma-Ossorio, Ana Canas, Angel Castaneda, Juncal Irurita, Liduvina Lopez, Candida Deniz, Pedro Saavedra, Ricardo Chirino, Fernando Arós, Miguel Iriarte, Dr Negrin Hospital, Las Palmas, Spain, Txagorritxu Hospital, Vitoria, Spain

Inflammation and genetic factors are attracting increasing interest as dynamic promoters of acute coronary syndromes. Searching for a permanent marker of risk we studied two inflammation genotypes tumor necrosis factor-alpha (TNF-a) and endothelial selectin (Eselectin) in a cohort of 462 patients following uncomplicated unstable angina.

For this study we compared 135 diabetics (cases 29.2%) here in after "D", with 327 nondiabetics "nD" (controls), with similar age and gender. Allelic frequencies for TNF-a gene promoter T1(G-308) and T2(A-308)(allele T1/T2, presence/absence of restriction fragment Ncol) were 0.892/0.108 "D" and 0.934/0.066 "nD". Allelic frequencies for CD62 gene (E-selectin) E1(A561) and E2(C561)(E1/E2, presence/absence of restriction fragment Pstl were 0.830/0.170 "D" and 0.888/0.112 "nD". Genotyping frequencies were in Hardy-Weinberg equilibrium.

Hypertension (67.4 "D" vs. 44.6% "nD"; p<0.0001); smoking (61.5 "D" vs. 48.9% "nD"; p<0.014) or dyslipidemia (55.6 "D" vs. 44.6% "nD"; p<0.04) and multiple vessel disease (44.2 "D" vs. 28.6% "nD"; p<0.005), were more prevalent in diabetics. Variant genotypes were significantly higher in diabetics TNF-a (30.4 "D" vs. 21.1% "nD"; p<0.041) and Eselectin (20 "D" vs 9.8% "nD"; p<0.005). Smoking (p<0.006) and variant E-selectin genotypes (p<0.0001) predicted 78.5% of recurrence (restenosis-angina and myocardial infarction) in a multivariate logistic regression model (including risk factors, genotypes, prematurity and gender).

In conclusion: our data indicate that diabetic patients with unstable angina have higher prevalence of major risk factors, variant inflammation genotypes and multiple vessel dis-

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ease. E-selectin, is an inexpensive (approximately \$ 40/patient) major independent predictor of recurrence and may contribute to characterize individual variability in coronary

9:15 a.m.

1022A-MP-204 Admission Glucose, Baseline Glucose Control, and Mortality After Acute Myocardial Infarction in Diabetic **Patients**

Jie J. Cao Michelle Jankowski, Michael Hudson, Frederick Whitehouse, W. Douglas Weaver, Henry Ford Hospital, Detroit, MI

Background: Clinical observations suggest that admission hyperglycemia is associated with increased mortality after acute myocardial infarction (MI) in patients with diabetes mellitus. We investigated the association of admission plasma glucose, baseline glucose control and in-hospital mortality among diabetic patients following acute MI. Methods: The study population included 827 diabetic patients consecutively admitted to our coronary care unit for acute MI from 1997 to 2001. Baseline glucose control was the average glycated hemoglobin (HgA1C) within 2 years prior to admission. Multivariate logistic regression was used to assess the association of admission glucose, average glucose control and in-hospital mortality. Results: 9% of the cohort died after acute MI. The mean admission glucose was 316± 143 mg/dL and 234± 117 mg/dL for those with and without mortality, respectively. In the multivariate analysis, after the conventional risk factors and severity of MI were taken into account, the odds ratios (OR) of admission glucose for the risk of subsequent mortality were 1.62 (0.65,4.1), 3.79 (1.78, 8.1) and 3.81 (1.60, 9.10) for glucose 200-250 mg/dL, 251-350 mg/dL and = 351 mg/dL compared to glucose < 200 mg/dL respectively. The average HgA1C did not differ significantly between the two groups: 8.1± 1.7% and 8.3± 1.8% (p=0.51) for groups with and without mortality. There was no independent association between average HgA1C and mortality, [OR of 0.93 (0.76, 1.15)]. The admission glucose and average HgA1C were not highly correlated (Pearson correlation coefficient: 0.22). Conclusion: The admission hyperglycemia was associated with increased mortality after acute MI in diabetic patients. Baseline glucose control did not correlate with admission glucose and was not associated with risk of mortality following acute MI.

1022A-MP-206 Increased Platelet Reactivity in Patients With Diabetes and Acute Coronary Syndromes: Direct Effects of Glucose Attributable to Its Osmolality

Friederike K. Keating Samer S. Kabbani, Erica L. Grayson, Burton E. Sobel, David J. Schneider, University of Vermont, Colchester, VT

Background: Recurrent cardiac events are prevalent in patients with diabetes mellitus who sustain an acute coronary syndrome (ACS). In those without diabetes increased platelet activation in response to stimuli (i.e. platelet reactivity) presages such events. In patients with diabetes improved glycemic control decreases subsequent cardiac events after myocardial infarction. Accordingly, we characterized platelet reactivity in patients with ACS with and without diabetes to determine whether high concentrations of glucose per se can increase platelet reactivity.

Methods: Blood was obtained from patients with ACS with (n=29) and without (n=28) diabetes. Platelet reactivity (surface expression of P-selectin in response to a low concentration [0.2 µM] of ADP) was quantified with the use of flow cytometry. Blood from healthy volunteers was incubated in the presence of glucose or isosmolar concentrations of mannitol and sorbitol (both metabolically inactive agents) for 60 minutes before assay of platelet reactivity.

Results: Platelet reactivity was greater in patients with diabetes (percentage of platelets expressing P-selectin after exposure to 0.2 μ M ADP =13±8% [SD] compared with 8±8% in patients without diabetes, p=0.02), Addition of glucose to whole blood from healthy volunteers led to increased platelet reactivity (fold induction with 27.5 mM [250 mg/dl] = 1.6 ± 0.2 , with 55 mM = 4.13 ± 0.9 [SEM], n=6, p=0.003). Isosmolar concentrations of glucose, sorbitol and mannitol exerted comparable effects (fold induction with 25 mM glucose = 1.7 ± 0.2 , n=10, sorbitol = 1.9 ± 0.3 , n=7, and mannitol = 1.8 ± 0.2 [SEM], n=8; p < 0.05 for comparison of each with control).

Conclusions: Platelet reactivity is increased in patients with ACS who have diabetes. The increased reactivity may contribute to cardiovascular risk. Osmotic effects of hyperglycemia can increase platelet reactivity directly.