

2 Subspecialty session: Cardiovascular medicine

factor- α (TNF- α) and interleukin (IL)-6) were determined by ELISA (Immunotech, Coulter Corp., Westbrook, MA, USA). Fat mass percentage was determined by bioelectric impedance method. Written informed consent was obtained from all participants. This protocol was approved by a local Ethics Committee.

Results: At baseline, TNF- α and IL-6 contents were 9.1 ± 2.6 (6.2–12.0) pg/mL and 6.9 ± 1.4 (5.1–8.7) pg/mL respectively in experimental group. After being exercised TNF- α and IL-6 concentrations were 7.8 ± 1.1 (6.4–9.3) pg/mL and 5.3 ± 1.2 (4.0–6.6) pg/mL respectively. Consequently, when compared to baseline, concentrations of both TNF- α (9.1 ± 2.6 vs. 7.8 ± 1.1 pg/mL; $P > 0.05$) and IL-6 (6.9 ± 1.4 vs. 5.3 ± 1.2 pg/mL; $P > 0.05$) were reduced significantly. Similarly, fat mass percentage also decreased significantly ($31.0 \pm 2.8\%$ vs. $29.7 \pm 2.1\%$; $P < 0.05$) in experimental group. On the contrary, no significant differences were found for any tested parameter in control group ($P > 0.05$).

Conclusions: It was concluded a 12-week aerobic training program improved significantly serum proinflammatory biomarkers in young adults with metabolic syndrome what may be explained at least in part by a reduction of fat mass percentage. This finding was of particular interest since it only lasted 12 weeks, being shorter than previously published training programs. Further studies on this topic are required.

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Expression of monocyte Fc γ receptors after percutaneous coronary intervention

F. Briceño, P. Ruiz, C. Rivera, J. Oneto & F. Gomez

Department of Medicine, Hospital Universitario Puerto Real, University of Cadiz, School of Medicine, Spain

Background: Monocytes play a central role in restenosis after coronary intervention (CI).

Objective: To assess the relationship between peripheral blood monocyte (PBM) activation and coronary restenosis after balloon angioplasty (BA) or stent implantation (SI).

Methods: PBM were studied in 157 patients undergoing CI for one lesion (immediately before, and after the CI: 48 h, 7 days, 1 month, and then every 3 months during a 1-year follow-up). We determined the PBM count, circulating IL-6 levels and, the PBM surface expression of Fc γ receptors, Fc γ RI, FcRIIa, Fc γ RIIb, and Fc γ RIII by flow cytometry. The same patient before the CI and, 58 patients undergoing angiography without CI served as controls.

Results: Patients undergoing BA or SI have an enhancement of their PBM count, levels of IL-6 and, expression of PBM Fc γ RI, Fc γ RIIa, and Fc γ RIII ($P < 0.005$ in all cases) and, a decreased expression of PBM Fc γ RIIb ($P < 0.001$). Angiographic restenosis (percent diameter stenosis $>50\%$), was observed in 41 patients (26%) at the end of follow-up. These patients showed a significantly larger maximum PBM count, maximum IL-6 level and maximum alteration of PBM Fc γ Rs expression (enhanced Fc γ RI, Fc γ RIIa, and Fc γ RIII expression and decreased Fc γ RIIb expression) ($P < 0.01$ in all cases). Changes in the total white blood cell count, the absolute neutrophil count or, the absolute lymphocyte count were not significantly related with the development of restenosis. These findings were similarly observed in both patients undergoing BA or SI.

Conclusions: These results suggest that the peak enhancement of the monocyte count, IL-6 levels and, the expression of PBM Fc γ RI, Fc γ RIIa, and Fc γ RIII, as well as the decremental peak of the PBM Fc γ RIIb expression are associated with coronary restenosis after BA or SI.

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Nonsteroidal anti-inflammatory drugs limit the cardiovascular preventive effects of aspirin

M. A. Escobar, A. A. García-Egido, F. J. Fernandez-Delgado,

F. M. Gomez-Soto, C. Asencio, J. L. Puerto, J. L. Andrey & F. Gomez
Department of Medicine, Hospital Universitario Puerto Real, School of Medicine, University of Cadiz, Spain

Background: Previous studies suggest that treatment with ibuprofen might limit the cardioprotective effects of acetyl salicylic acid (ASA), while treatment with others non-steroidal anti-inflammatory agents (NSAIDs: Diclofenac or naproxen) does not seem to alter the cardioprotection of aspirin.

Objectives: To assess whether patients with known cardiovascular disease who take low dose ASA (ldASA <326 mg/day) plus other NSAID have increased cardiovascular mortality or morbidity.

Methods: Patients (20,173) discharged after first admission for cardiovascular disease between January 1, 1992 and December 31, 2001, that were prescribed ldASA and survived for at least 1 month were studied. Mortality and morbidity of patients taking ldASA alone was compared with that of patients taking ldASA plus any NSAID (ldASA + NSAID) on a chronic basis (a mean of >3 days/week).

Results: Patients taking ldASA + NSAID did not have an increased of all-cause mortality (adjusted hazard ratio 1.13, 95% CI 0.77–1.48, $P = 0.0892$), cardiovascular mortality (1.26, 0.79–1.72, $P = 0.0817$) or cardiovascular morbidity (1.47, 0.81–2.13, $P = 0.0897$). Nevertheless, patients taking ibuprofen plus ldASA had an increased risk of all-cause mortality (1.99, 1.53–2.42, $P = 0.0012$) and cardiovascular mortality (1.92, 1.63–2.21, $P = 0.0183$), while patients taking diclofenac or naproxen had a decreased risk of all-cause and cardiovascular mortality (< 0.88 and $P < 0.0298$, respectively). Morbidity was increased in patients taking ldASA + NSAID, all-cause (1.77, 0.58–1.96, $P = 0.0052$) or overt gastrointestinal bleeding (3.55, 2.76–4.34, $P = 0.006$).

Conclusions: These results suggest that ibuprofen may limit the secondary cardioprotective effects of ldASA, while diclofenac or naproxen do not decrease secondary cardiovascular prevention by ldASA. Patients taking low dose ASA plus NSAID have an increased morbidity.

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Distribution of traditional and new risk markers among subjects with familial history of premature heart attack

H. G. Carstensen*, J. F. Hansen[†] & A. Sajadieh*

*Department of Cardiology, Copenhagen University Hospital of Amager, Denmark, [†]Department of Cardiology, Bispebjerg Hospital, Copenhagen, Denmark

Background: Association between familial predisposition to premature heart attack and the new risk markers of coronary artery disease is poorly studied.

Methods: A total of 643 middle-aged and elderly subjects with no apparent heart disease were selected from community and studied by interview, clinical and laboratory examination and 24-h Holter monitoring. A subject was regarded as familial predisposed if father, mother or any of the siblings had a heart attack or suffered from sudden death below age of 60. Time domain measures heart rate variability (HRV) were evaluated for 24-h, day-time and night-time separately.

Results: A total of 90 subjects (14%) had a familial predisposition. More women reported a familial predisposition ($P = 0.001$). Among the traditional risk factors only triglyceride level were slightly higher in subjects with predisposition (1.8 + 2.40 vs.