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Subspeciality session: Cardiovascular medicine

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Atrial fibrillation is a strong predictor of death and cardiac events in angiographied coronary patients

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Background: No data on the prognostic impact of atrial fibrillation in angiographied coronary patients are available.

Materials and methods: In a consecutive series of 622 patients (418 men and 204 women) who underwent coronary angiography for the evaluation of established or suspected coronary artery disease we evaluated electrocardiograms according to the Minnesota Code. Prospectively, all-cause mortality, cardiac death (including fatal myocardial infarction, sudden cardiac death, and death from congestive heart failure due to coronary artery disease) and major coronary events (including death due to coronary heart disease and non fatal myocardial infarction) were recorded over 4 years.

Results: From our patients, 37 (5·9%) at baseline had atrial fibrillation, 576 (92·7%) exhibited sinus rhythm, and 4 (0·6%) had persistent supraventricular rhythms, and 5 (0·8%) pacemaker rhythms. Presence of atrial fibrillation was associated with a lower prevalence of significant coronary stenoses with narrowing of at least ≥50% at the baseline angiography [adjusted odds ratio = 0·121 (95% CI 0·051–0·289), $P < 0·001$). Prospectively, however, patients with atrial fibrillation were at a strongly increased risk of all-cause mortality [adjusted hazard ratio (HR) = 4·529 (2·070–9·911), $P < 0·001$], cardiac death [HR = 7·239 (2·569–20·399), $P < 0·001$], and major coronary events [HR = 4·529 (2·070–9·911), $P < 0·001$].

Conclusions: Atrial fibrillation is inversely associated with the presence of angiographically diagnosed coronary artery disease, but is a strong predictor of death and cardiac events in angiographied coronary patients.

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The resting heart rate is a powerful predictor of future vascular events among women undergoing coronary angiography

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Background: Epidemiological data suggest that a high heart rate is associated with an increased risk of cardiovascular events. A potential gender difference in the impact of the heart rate on vascular risk has not been investigated yet.

Materials and methods: We obtained the resting heart rate from the electrocardiograms of a consecutive series of 622 patients (418 men and 204 women) who underwent coronary angiography for the evaluation of suspected or established stable coronary artery disease. Prospectively, we recorded vascular events over 4 years.

Results: The incidence of vascular events was 10·3% among women and 21·1% among men. The resting heart rate in the total study population proved significantly predictive of vascular events [standardized adjusted hazard ratio (HR) = 1·253 (95% CI 1·028–4·487); $P = 0·025$]. In subgroup analyses with respect to gender, the resting heart rate was a strong predictor of vascular events among women [HR = 1·790 (1·119–2·864); $P = 0·015$], whereas it was not significantly associated with the incidence of vascular events in men [HR = 1·110 (0·884–1·392); $P = 0·369$]. An interaction term gender x resting heart rate was significant ($P = 0·044$), indicating a significantly stronger impact of the heart rate on the incidence of vascular events among women than among men.

Conclusions: The resting heart rate is a powerful predictor of future vascular events among women undergoing coronary angiography that should be considered in cardiovascular risk stratification.

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A 12-week aerobic training program improved proinflammatory biomarkers in young adults with metabolic syndrome

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Background: It is widely accepted individuals with metabolic syndrome presented a proinflammatory state that may finally result in an increased cardiovascular risk profile. A growing body of evidence implicates adipose tissue as a key regulator of this inflammation by expressing proinflammatory molecules. The present study was designed to determine the influence of regular exercise on both fat mass percentage and proinflammatory biomarkers in adults with metabolic syndrome

Materials and methods: Sixty male adults with metabolic syndrome according to the criteria reported by the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP-III) volunteered for this study. Forty five were randomly included in experimental group to perform a 12-week aerobic training program, 3 days/week, consisting of warm up (10 min), main part (20–35 min [increasing 5 min each 3 weeks]) at a work intensity of 60–75% of peak heart rate (increasing 5% each 3 weeks) and cool-down (10 min). Control group included 15 age, sex and BMI-matched young adults with metabolic syndrome that did not performed any training program. Blood samples were collected from antecubital vein 72 h before starting the program and after its ending. Cytokine levels [tumor necrosis

factor-alpha (TNF-α) and interleukin (IL)-6) were determined by ELISA (Immunotech, Coulter Corp., Westbrook, MA, USA). Fat mass percentage was determined by bioelectric impendence 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 ± 2·8% vs. 29·7 ± 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 fining 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 Fcg receptors after percutaneous coronary intervention

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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 angiolasty (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 Fcγ receptors, FcγRI, FcRIIa, FcγRIIb, and FcγRIII 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 FcγRI, FcγRIIa, and FcγRIII ($P < 0·005$ in all cases) and, a decreased expression of PBM FcgRIIb ($P < 0·001$). 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 FcgRs expression (enhanced FcγRI, FcγRIIa, and FcγRIII expression and decreased FcgRIIb 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 FcγRI, FcγRIIa, and FcγRIII, as well as the decremental peak of the PBM FcgRIIb 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

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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 (NSAIAs: 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 NSAIA 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 NSAIA (ldASA + NSAIA) on a chronic basis (a mean of >3 days/week).

Results: Patients taking ldASA + NSAIA 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 + NSAIA, 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 NSAIA have an increased morbidity.

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

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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 form 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.

