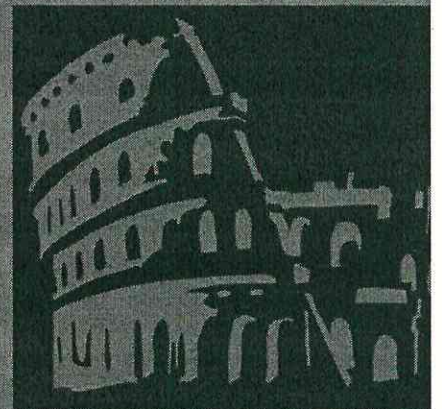


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included into antracycline family approved for treatment of secondary progressive and rapidly worsening relapsing-remitting MS. MTX may have cardiac toxicity.

Purpose. Aim of our study is to recognize early signs of cardiac dysfunction in MS pts treated with MTX in a period of 12 months

Methods. 20 pts were evaluated before starting MTX therapy and after any administration. By echocardiography Vivid 7 ultrasound System-GE with TVI function, atrial and ventricular diameters, volumes, ejection fraction, velocity of transmitral flow (E, A, E/A), tissue myocardial velocity of the mitral annulus (Sm, Em, Am) and the ratio E/Em, were measured. By 2D acquisition we analysed ventricular and atrial longitudinal peak systolic regional strain (2D-S) in apical 4 chambers views at basal LV septum (IVSb), lateral wall (LWb) and at left atrial wall near the roof (IAS, LAW). All data was exposed as media (m) and standard deviation (SD) at time before therapy (T0), after five (T5) and ten months (T10) therapy.

Results. At T5 decreased LVEF (64.27 ± 6.02 vs 68.42 ± 3.5 , $p < 0.05$; 1 pt LVEF reduced $>20\%$ and an other of 13%) and LVFS (35.4 ± 4.49 vs 38.37 ± 2.9 , $p < 0.05$). At T10 the LVEF increase (67.00 ± 3.44 vs 64.27 ± 6.2 , $p < 0.05$) also in pts with reduction at T5; Sm decreased (0.06 ± 0.01 vs 0.07 ± 0.02 , $p < 0.05$). The LVEF amelioration is possible with the reduction of the MTX therapy, but Sm may be to follow-up.

Conclusions. Echocardiography monitoring of cardiac function is very important for treatment of MS pts.

Prevenzione cardiovascolare e farmacologia

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IPERFOSFATEMIA E RISCHIO CARDIOVASCOLARE IN PAZIENTI IN INSUFFICIENZA RENALE CRONICA END-STAGE

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Background. Elevate concentrazioni sieriche di fosforo, spesso rilevate in pazienti in insufficienza renale cronica, si associano ad aumentato rischio di eventi cardiovascolari, aumentata incidenza di morte improvvisa e presenza di estese calcificazioni vascolari. I meccanismi patogenetici responsabili di questa associazione sono tuttavia poco noti e sembrano essere distinti da quelli legati ai tradizionali fattori di rischio cardiovascolare.

Metodi. Abbiamo analizzato retrospettivamente una popolazione di pazienti con insufficienza renale cronica sottoposti a trattamento dialitico (età media 59 anni, filtrato glomerulare <30 ml/min, età dialitica media 9 anni, 66% maschi) suddivisa in due gruppi in base alla fosfatemia (gruppo A fosfatemia ≥ 5 mg/dl; gruppo B fosfatemia <5 mg/dl). I pazienti sono stati sottoposti a esame obiettivo, raccolta dell'anamnesi cardiologica, elettrocardiogramma, ecocardiogramma color/Doppler.

Risultati. Non sono state evidenziate differenze statisticamente significative tra i due gruppi per le caratteristiche demografiche (età, sesso), per l'incidenza dei fattori di rischio cardiovascolare (diabete mellito, ipertensione arteriosa, ipercolesterolemia, fumo, familiarità per cardiopatia ischemica), né per anamnesi positiva per precedenti eventi cardiovascolari. Abbiamo inoltre analizzato parametri ecocardiografici (diametri e spessori del ventricolo sinistro, frazione d'eiezione del ventricolo sinistro, presenza di valvulopatie) ed elettrocardiografici (intervallo QT corretto, presenza di turbe di conduzione intraventricolari, frequenza cardiaca, aritmie ventricolari) che correlano con aumentato rischio di morte improvvisa, non trovando differenze statisticamente significative tra i due gruppi.

Conclusioni. I risultati del nostro studio non mostrano alcuna correlazione tra l'iperfosfatemia ed i tradizionali fattori di rischio metabolici per lo sviluppo di cardiopatia ischemica o i parametri strumentali predittivi di morte improvvisa. Questo suggerisce che l'iperfosfatemia rappresenti un fattore di rischio indipendente per lo sviluppo di eventi cardiovascolari in pazienti in insufficienza renale cronica end-stage, verosimilmente attraverso meccanismi patogenetici specifici correlati a fenomeni di calcificazione vascolare.

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CHRONIC COLA DRINKING REPRODUCES METABOLIC SYNDROME AND INDUCES CARDIAC ALTERATIONS IN RATS

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The rising consumption of soft drinks has been linked to development of metabolic syndrome in humans. However, in spite of much experimental data with fructose-enriched diets, little is known on the effects of cola beverage, in man or in animal models.

Our aim was to investigate in rats the effects of chronic drinking of sucrose-sweetened beverages on metabolic and echocardiographic parameters.

Methods. Male Wistar rats were divided in 3 groups ($n = 8$ each) allowed to drink ad libitum for 6 months, either: tap water (Control, C); Coca-cola (K), or light coke (L). Both commercially available drinks had the same amount of phosphoric acid, caramel, caffeine, and sodium; K contained also sucrose (11 g/100 ml), while L was sweetened with aspartame. At baseline and at weeks 4, 8, 12 and 24, blood samples were taken for determination of glucose, triglycerides, and HDL-cholesterol. Systolic blood pressure by tail cuff method (SBP), and left ventricular (LV) dimensions (M-mode echo) were simultaneously recorded while rats were awake.

Results. After 6 months, K weighed 685.9 ± 21 g vs 617.1 ± 49.9 (C) and 630.7 ± 42.6 (L) ($p < 0.05$) and showed SBP of 146.6 ± 10.6 mmHg vs 136.9 ± 7.3 and 135.1 ± 8.2 , respectively ($p < 0.05$). Plasma glucose was 149.4 ± 16.3 mg/dl (K) vs 119.3 ± 13.5 (C), and 115.0 ± 6.5 (L) ($p < 0.001$); triglycerides were 182.6 ± 86.6 mg/dl (K) vs 72.6 ± 26 (C), and 84.5 ± 34.2 (L) ($p < 0.01$); while plasma concentration of HDL-Cholesterol showed no differences among groups. LV diastolic diameter was 6.78 ± 0.35 mm (C) vs 7.46 ± 0.3 (K; $p < 0.05$), and vs 7.1 ± 0.55 (L; ns). LV wall thickness was 0.42 ± 0.03 ; 0.37 ± 0.03 ; and 0.38 ± 0.03 mm, respectively (C vs K and L, $p < 0.05$). LV diastolic volume (ml) was 0.27 ± 0.04 (C) vs 0.35 ± 0.04 (K; $p < 0.01$); and vs 0.3 ± 0.07 (L; ns). Cardiac output (ml/min) was 113.96 ± 22.8 in C vs 161.15 ± 28.87 in K ($p < 0.01$), and vs 129.85 ± 38.09 in L (ns).

Conclusions: In rats, chronic consumption of coke affects body weight, blood pressure, glucose, and triglycerides, thus reproducing most of the features of metabolic syndrome. Furthermore, these animals showed LV dilatation and remodeling. These deleterious effects on metabolism and cardiac geometry were not seen in animals drinking light coke, thus indicating that they were largely due to the high calorie intake from sucrose in regular drink.

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HYDROXYAMINE CHLORIDRATE REDUCES OXIDATIVE-STRESS DAMAGE SUBSEQUENT TO BALLOON-INJURY RAT MODEL

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There is accumulated scientific evidence showing that abnormal generation of reactive oxygen species play a relevant role in the proliferation of vascular smooth muscle cells subsequent to balloon vascular injury in rat model. Recently, several data suggests that novel antioxidant compounds such as superoxide dismutase mimetics, exert protective effects against vascular injury in rats, although the molecular mechanism is still unclear. Here, we have investigated on the protective effects of the peroxynitrite decomposition catalyst, Hydroxylamine Chloridrate ($\text{NH}_2\text{OH HCl}$), on smooth muscle cells proliferation generated in response to balloon injury of common carotid artery in rats. In animals undergoing balloon-injury a significant restenosis and neointima formation occurred. This effect was associated to an elevated production of peroxynitrite, reactive free radicals, and to significant modulation of tissue cNOS and eNOS (as a measure of NO activation), nitrotyrosine (the footprint of peroxynitrite generation) and malondialdehyde levels (the marker of peroxidative processes). Treatment of rats with Hydroxylamine Chloridrate ($10\text{-}20\text{-}40$ mg/kg/day, i.p.), dose-dependently reduced post-injury neointima formation, an effect accompanied by decreased peroxynitrite generation and MDA accumulation and increased of cNOS and eNOS. The effect of Hydroxylamine Chloridrate was also associated to decreased expression of NF-kB, an intracellular transduction mediator associated to activation of free radical sensitive genes leading to smooth muscle cell proliferation. These results suggest that novel peroxynitrite antagonist may reduce post-injury neointima formation via inhibition of NF-kB-related intracellular pathway.

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LIVELLI PLASMATICI DI INTERLEUCHINA-18 IN PAZIENTI IPERTESI DOPO CARICO ORALE DI GLUCOSIO

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Il diabete mellito di tipo 2 si associa ad un aumento dello stress ossidativo e/o della infiammazione, che possono giocare un ruolo rilevante nello sviluppo di complicanze cliniche. Lo scopo di questo studio è stato quello di indagare le modificazioni degli indici circolanti di infiammazione/stress ossidativo durante un test da carico orale di glucosio (OGTT) in pazienti ipertesi. Abbiamo incluso nella casistica 14 pazienti affetti da ipertensione arteriosa essenziale senza evidenza di diabete mellito. Tutti i pazienti sono stati sottoposti ad una OGTT standard (70 g di glucosio). I livelli plasmatici circolanti di Interleuchina 6, interleuchina 18, lipoperossidi, capacità antiossidante (PAO) ed il