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EVALUATION OF SERUM ELECTROLYTE AND MYOCARDIAL ENZYME LEVELS DURING MONITORED STRESS TEST (MST)

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Aim of this study was to investigate the possible risks of pharmacological stress tests, of these are widely used for evaluation of myocardial ischemia and performance. For that purpose serum Ca^{2+} , Ca^{2+}/Mg^{2+} ratio, sodium, potassium, total and ionized calcium, magnesium and phosphorus levels of 30 ischemic heart patients (10 of whom admitted as control group) were determined before, during and after dobutamine infusion, which is one of the mostly utilized pharmacological stress test.

Serum sodium and ionized calcium levels were found to decrease in both MST and control groups at the 6th minute of exercise. Potassium, magnesium and total calcium levels elevated in dobutamine group in contrast to a fall in control group. Heartline phosphorus levels elevated in MST group while decreasing in control group. However all these alterations were within the reference ranges and were all statistically insignificant.

On the other hand, serum levels of Ca^{2+} increased to the values of 9.64 ± 1.00 (SEEM) and 12.01 ± 1.02 (SEEM) from the prior to test levels of 6.59 ± 0.59 (SEEM) and 8.8 ± 0.80 (SEEM) by the 16th hours in MST and control groups, respectively.

Ca^{2+}/Mg^{2+} ratios were determined as 21.55 ± 1.54 (SEEM) percent and 23.28 ± 1.70 (SEEM) percent in MST group before and after test, respectively ($p < 0.05$); same values estimated to be 13.5 ± 1.93 (SEEM) percent before and 44.30 ± 7.04 percent at 16th hours of tests in control group ($p < 0.001$).

As we found that serum myocardial enzyme and serum electrolyte levels did not alter significantly during MSTs, dobutamine induced pharmacological stress tests were concluded to be safe enough for applying to patients even in high infusion rates. (40 $\mu\text{g/kg/min}$)

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MYOCARDIAL TISSUE CONCENTRATIONS OF Cu, Zn, Mg AFTER AMI
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There is increasing interest for the role metal ions exert in cardiovascular disease. Studies carried indicating the decline in these elements after AMI (Acute myocardial infarction) prove that they play a role in the integrity of the myocardium. While there are many investigations carried in sera related to elemental variations the elemental status of the necrotic myocardium has not been fully elucidated. Thus our aim complementing our previous research carried in the sera of AMI patients has been to determine the elemental status of the necrotic myocardium. Thus, the elemental levels of the necrotic tissue were compared both to the intact myocardium of the same cases, as well as to the myocardial autopsy samples of other cases, where AMI was excluded as the cause of death.

For the extraction of Cu, Zn and Mg a technique utilizing $HNO_3/HClO_4(1/1)$ was applied, the incubation period being 72 h. Cu and Zn were determined by Atomic Absorption Spectrometry and Mg by a spectrophotometric method based on the formation of xylydyl blue-Mg complex.

Cu, Zn and Mg levels were 0.89 ± 0.86 , 7.94 ± 0.58 , 64.6 ± 28 in the necrotic tissue 1.25 ± 0.19 , 9.85 ± 1.18 , 108.43 ± 31.2 in the intact myocardium of the same cases 2.28 ± 0.58 , 9.82 ± 1.46 , 117.77 ± 13.37 in tissue samples of other cases where AMI had been excluded, respectively. (Results were given by $\mu\text{g/gr}$)

Briefly evaluating the results obtained, we conclude that there is a significant decline in Cu and Mg in the necrotic myocardium. Although a decline related to zinc is also noted, this is not significant. Our findings indicate that Cu, Zn and Mg are essential for the structural and functional maintenance of the myocardium.

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PLASMA MAGNESIUM LEVELS EVOLUTION DURING POST ACUTE MYOCARDIAL INFARCTION.

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Magnesium deficit is a frequently forgotten alteration in myocardial function. Hypomagnesaemia has been associated with arrhythmia, increased coronary vasospasm, and myocardial ischemic vulnerability. Similarly, an inverse relation between magnesium content in drinking water and cardiovascular disease has been described. Recent studies demonstrated the usefulness of Magnesium therapy for Acute Myocardial Infarction (AMI) treatment. Other studies found a pre-AMI Magnesium levels lower than normal, that continues during the first days post AMI.

The aim of this study was to assess plasma Magnesium status during AMI, prior to use Magnesium supplementation in this patients.

We studied 23 AMI diagnosed patients received at the Emergency room of our Hospital, and blood samples were drawn at the entrance, and 1, 3, 6, 9, 12, 18, 24, 36, 48 hours after and on days 3, 4, 5 and 6 post I AM. After clotting blood was immediately centrifuged and serum separated and sent frozen to laboratory for analysis. Magnesium levels were measured by Atomic Absorption Spectrophotometry (Perkin-Elmer 1100-B). No patients received Mg therapy during the study period.

Hypomagnesaemia (considered as serum Mg < 0.7 mmol/L) was detected in 5 patients (22%). Mean plasma Magnesium concentration at entry of this 23 patients were 0.84 mmol/L, table shows the evolution of Magnesium levels during the days studied.

Time h.	0	1	3	6	12	18	24	48	96
Mg mmol/L	0.84	0.77	0.80	0.81	0.84	0.82	0.84	0.88	0.98
s.d.	0.15	0.21	0.13	0.14	0.14	0.15	0.15	0.12	0.12

The high incidence of hypomagnesaemia suggest that it could be of interest to assess magnesium levels in this patients and Magnesium supplements could be an interesting strategy.

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CHANGES IN MUSCULAR PROTEINS DURING SIMULATED MICROGRAVITY

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We report on changes in muscle protein pattern in plasma of 5 volunteers staying 5 days in dry water immersion (foil covered water bath), to simulate microgravity. Additionally they were exposed to a standardized isometric load (40 contractions with max. force 5 sec with 10 sec rest in between) of quadriceps femoris muscle 2 hours after and 14 days before immobilisation. Plasma levels of slow twitch skeletal (cardiac β type) myosin heavy chain (MHC), myoglobin, CK and CK-MB-mass were measured before and daily during immobilisation and 6 hours after isometric exercises and a daily follow up was conducted for 5 days. Following muscle load 2 hours after immobilisation muscle protein levels increased dramatically (MHC 25 fold, peak 72 hours after load, Myoglobin 4 fold, CK 8 fold, CK-MB 8 fold, peak with 16 hours delay). Even twice as high isometric loads 14 days before immobilisation showed a significant lower response to loading (MHC 5 fold, Myoglobin 2 fold, CK 2 fold, CK-MB 2 fold). Measurements of c-GMP and cardiac specific troponin-I demonstrated no alteration of cardiac muscle cells. Since MHC is increasing, we conclude that totally conducted immobilisation is leading to hidden and diffuse lesions of slow twitch fibres mostly occurring in antigravitational muscles. This process is detectable and can be discriminated only following muscle load. We suggest these findings as a consequence of a functional adaptional process rather indicating regeneration than damage of slow skeletal muscle fibres.

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