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Non invasive monitoring of levosimendan infusion in patients with decompensated heart failure.
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Levosimendan has been proposed as an alternative to inotropic drugs treatment in patients with decompensated heart failure. Data from haemodynamic monitoring support the favourable effects of the drug. However there are no data regarding non invasive monitoring. We tested the hypothesis that non invasive monitoring of these patients treated with levosimendan can be equally successful.

Methods: Fifteen patients(3 dilated and 12 ischemic cardiomyopathy), with decompensated heart failure, treated with levosimendan, were studied. All of them had a Swan-Ganz catheter for hemodynamic monitoring and blood pressure was measured with sphygmomanometer. Levosimendan was given intravenously as a bolus (3 µg/kg) and infusion for 24 hours (0.1 µg/kg/min). At baseline and at the end of infusion a thorough Echocardiographic study was performed. Left ventricular (LV) dimensions and volumes were measured and ejection fraction was calculated. Mitral inflow E & A waves and E wave deceleration time, mitral regurgitation jet area, and aortic flow velocity and velocity-time integral were also measured and cardiac output (from LV outflow) was calculated. In 10 pts blood samples were collected at baseline, at 24 and 72 hours for pro BNP measurement.

Results: There were no complications from levosimendan infusion. Mean blood pressure decreased (81±11 to 74±8 mmHg, p<0.002) without change in heart rate. Pulmonary wedge pressure decreased (27.8±5 to 23.7±7 mmHg, p<0.01), and cardiac index (CI) increased (2.02±0.52 to 2.26±0.42 l/min/kg, p<0.04), while right atrial pressure had a decrease of marginal significance (13±6 to 11±5 mmHg, p<0.06). From Echocardiographic study: LV systolic diameter decreased (62±8 to 58±8 mm, p<0.002), LV ejection fraction increased (19±5 to 22±6%, p<0.001), CI increased (1.7±0.3 to 2.0±0.4 l/min/kg, p<0.005) and inferior vena cava diameter decreased (24±4 to 22±5 mm, p=0.03). Mitral regurgitation jet area, E wave deceleration time and mitral E/A ratio did not change significantly. Two patients without improvement of hemodynamic parameters were successfully detected by Echo. Pro BNP levels decreased significantly following therapy (150±229 fmol/ml to 130±271 fmol/ml at 24 h and 1045±217 fmol/ml at 72 h, p<0.006)

Conclusions: Levosimendan can be a valuable additional index of LV contractile reserve in those patients, treated with levosimendan, can be successfully assessed non invasively, making Swan-Ganz catheter optional in patient’s monitoring.

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Dose-dependent effects of sildenafil on endothelial function of forearm vessels in heart failure patients: correlation with peak VO2 and exercise blood flow redistribution.
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Background: Sildenafil is a new challenge in the pharmacotherapy of CHF patients. Its use is recommended in patients with compromised heart failure, treated with levosimendan, were studied. All of them had a Swan-Ganz catheter for hemodynamic monitoring and blood pressure was measured with sphygmomanometer. Levosimendan was given intravenously as a bolus (3 µg/kg) and infusion for 24 hours (0.1 µg/kg/min). At baseline and at the end of infusion a thorough Echocardiographic study was performed. Left ventricular (LV) dimensions and volumes were measured and ejection fraction was calculated. Mitral inflow E & A waves and E wave deceleration time, mitral regurgitation jet area, and aortic flow velocity and velocity-time integral were also measured and cardiac output (from LV outflow) was calculated. In 10 pts blood samples were collected at baseline, at 24 and 72 hours for pro BNP measurement.

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Conclusions: Levosimendan can be a valuable additional index of LV contractile reserve in those patients, treated with levosimendan, can be successfully assessed non invasively, making Swan-Ganz catheter optional in patient’s monitoring.

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Nitricrete peptides changes at stress-echocardiography predicts myocardial contractile reserve in patients with non-ischemic dilated cardiomyopathy.
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Background: Nitricrete peptides levels are increased, subject to the degree of systolic and diastolic left ventricular (LV) dysfunction in patients with chronic Heart Failure, while LV inotropic reserve has been proposed as a useful prognostic index in these patients. We assessed the relationship between LV inotrope reserve and nitricrete peptide changes during Dobutamine stress-echocardiography in pts with Non-Ischemic Dilated Cardiomyopathy (NIDC)

Methods: Twenty eight patients with angiographically proven NIDC, aged 55±6.1±4.9, NYHA functional class II-III and LV ejection fraction (EF) 32±2.9%, underwent to a low dose Dobutamine stress echocardiography (LDDE)(two-5 minutes stages with 5 and 10 µg/kg/min intravenous infusion of Dobutamine). N-Terminal pro-ANP (ANP) and B-Natriuretic peptides levels were measured 15 min before and 60 min after LDDE. LV was divided into 16 segments and the wall motion score index (WMSI) calculated at rest(0) and at peak stress(s).

Results: The mean WMSI was 2.13±0.24 while BNP and ANP plasma levels were 0.77±0.41 and 3.8±2.32 pmol/l respectively.

According to BNP changes (d) at LDDE, patients divided in those who decreased BNP (group I) and those who BNP levels remained stable or increased (group II).

There were no differences between two groups in age, NYHA functional class, LV dimensions, LVEF or WMSI.

Conclusion: Nitricrete peptide changes at LDDE showed a close relationship to LV inotrope reserve in pts with NIDC. Measurement of nitricrete peptides at stress may be a useful additional index of LV contractile reserve in those patients.

LEFT-VENTRICULAR FUNCTION

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An echocardiography-based management program for acute pericarditis.
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Background: Echocardiography can be very helpful in confirming acute pericarditis clinical suspicion disclosing even small effusions and to role out complications.

Method: From January 1998 we included all consecutive cases of acute pericarditis in our hospital. Diagnosis of pericardial effusion was made by clinical examination, the result of routine laboratory tests(blood cell count, sedimentation rate, acute phase reactants, creatin kinase, troponin I, serum creatinine) and transthoracic echocardiography to determine the amount of pericardial effusion and exclude cardiac tamponade. Patients without clinical negative predictors(fever<38°, Subacut onset, immun-odepression, trauma, oral anticoagulant therapy, myopericarditis, severe pericardial effusion, cardiac tamponade) were assumed to be idiopathic without a full echocardiographic search and considered low risk cases assigned to out of hospital treatment with high dose oral aspirin. In case of aspirin failure or with clinical negative predictors patients were considered high risk cases and hospitalized to perform a full diagnostic evaluation. A clinical and echocardiographic follow-up was performed at 48-72 hours, 1 month, 6 months and 1 year to detect pericardial effusion relapse and exclude constriction.

Results: We observed 350 cases of acute pericarditis(mean age 53.4±18.0 years, range 16-91 years; 226 males). 298 patients(85.1%) were considered low risk cases(group I). Initial treatment with ASA was effective in 265 cases(88.9%). 52 patients(14.9%) were considered high-risk patients and admitted to hospital(group II). Final diagnosis was idiopathic pericarditis in 287 cases(82.0%), a specific etiology was detected in 63 out of 350 cases(18.0%), but up to 41 out of 52 high risk patients(80.3%) showing the importance of patients stratification to start a full echocardiographic search. After a mean follow-up of 38 months no cases of cardiac tamponade were recorded in group I. A higher frequency of relapses and constriction was recorded in group II compared with group I(respectively 46.0% vs 10.4% for recurrences and 11.1% vs 0.4% for constriction; for all p<0.001). ASA failure alone was able to identify patients at higher risk of complications.

Conclusions: An echocardiography based management program for acute pericarditis stratification is efficacious to select low risk cases to be treated on an outpatient basis and to detect acute pericarditis complications.