Does echocardiographic stress test induced release of hsTnT and TnI II?

Le Goff C.1, Laurent T.1, Garweg C.3, Kaux J-F.2, Deroyer C.1, Fillet M.1, Lancellotti P.3, Pierard L.3, Chapelle J-P.1

1 Department of Clinical Chemistry, University Hospital of Liege, Belgium
2 Department of Cardiology, University Hospital of Liege, Belgium
3 Department of Clinical Sciences, University of Liege, Belgium

**Background:**
Cardiac troponins (cTn) are considered as the best biomarkers for detection of myocardial cell injury. In this study, cTnT and cTnI were measured by new commercially available high-sensitive methods in patients undergoing brief exercise- or pharmacologic-induced stress. Our aim was to compare cTnT and cTnI levels before and after the stress tests, in the patients with or without reversible ischemia.

**Materials and Methods:**
- Fifty patients (28 men and 22 women) underwent an echographic stress test (ST) for suspected ischemic heart disease.
- Of these 50 patients:
  - 28 received pharmacological ST (dobutamine injection)
  - 22 dynamic ST (bicycle exercise)
- The patients were subdivided into two groups according to the presence or absence of documented transient reversible ischemia:
  - 14 with reversible ischemia (RI; mean age: 67.71±9.66 y)
  - 36 without ischemia (NRI; mean age: 63.17±11.72 y)
- In all patients, cTnT and cTnI concentrations were measured by high sensitive methods (hsTnT, Roche Diagnostics and TnI II, Abbott Diagnostics) on heparin plasma immediately before (T0) and after ST (T1). The lower detection limit of these assays was 0.005μg/L for hsTnT and 0.01μg/L for TnI II.
- The protocol was approved by the ethics committee of the University of Liège (Belgium). All patients gave informed consent. All statistical analyses were performed using Medcalc version 8.1 for Windows. P value <0.05 was regarded as statistically significant.

**Results:**
There was no significant difference between hsTnT concentrations at T0 and T1, neither in the whole patient group, nor in the subgroups of subjects who received pharmacological ST or dynamic ST. The same was true for TnI II.

Although there was no change in hsTnT levels during test in ischemic and in non ischemic patients, the latter tend to demonstrated higher median T0 levels (25th, 75th percentiles): 0.011 (0.007, 0.029) vs 0.007 (0.0047, 0.1125) ng/ml, p=0.09 and median T1 levels: 0.014 (0.065, 0.03) vs 0.007 (0.003, 0.0102) ng/ml, p=0.08 (Figure 1).

Higher TnI II levels were also recorded in ischemic patients as compared to non ischemic patients at T0: 0.014 (0.0072; 0.0265) vs 0.005 (0.003; 0.01) ng/ml, p=0.08 and T1: 0.013 (0.0085-0.03) vs 0.006 (0.0035-0.008) ng/ml, p=0.08 (Figure2). Also, TnI II levels did not change during test in both subgroups.

**Legend:**
These figures are the result of stress echocardiography inducing reversible or no myocardial ischemia.

**Conclusions:**
Measurement of cardiac troponins by high sensitive methods did not allow to detect significant release of biomarkers from the heart during exercise- or pharmacological-induced, even in patients who demonstrated reversible myocardial ischemia. The type of test – pharmacological or dynamic - was without effect. The patients with induced transient ischemia had however higher troponin T and I levels at baseline, this difference remaining during test.

**Acknowledgement:** This experimentation was partially financed by “Standard de Liège 2009” grants (Leon Fredericq Funds).