Institutional report - Cardiac general

Effect of topic defibrillation on serum markers of myocardial damage

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Abstract

Serum levels of cardiac enzymes and troponins after external cardioversion (ECV) for atrial defibrillation and atrial flutter, and after endocardial cardioverter defibrillation by implantable cardioverter defibrillator (ICD), have been well investigated. The aim of this study was to assess the effects of topic defibrillation (TD), after cardiac surgery, on cardiac enzymes in patients with uncomplicated clinical course. Biochemical markers were analyzed prospectively for 20 patients after TD (group A) and for 20 patients that were not defibrillated (Control group). We obtained serum concentrations of cardiac Troponin I (cTnI), total creatine–kinase (CK), CK MB isoenzyme (CK-MB), Myoglobin (Myo) in both groups. The difference in cTnI plasma level and curve of raise was not statistically significant between the two groups, but there was a difference in the CK-MB and Myoglobin curve of raise between the two groups. Topic defibrillation does not influence the increase of cTnI, so a high cTnI should be correlated to myocardial damage and not to TD. In patients that received TD, it would be preferable to use cTnI as a marker of myocardial disease than CK-MB which is influenced by the TD.

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1. Introduction

Reperfusion ventricular fibrillation and atrial fibrillation during cardiac surgery is common and cardiac electric shocks are often needed to terminate it [1]. cTnI, a 24-kDa protein, is the inhibitory subunit of troponin, part of the thin filament cardiac contractile apparatus, troponin-tropomyosin complex. The troponin portion of this complex consists of three subunits: cTnI, which binds to actin and inhibits actin-myosin interactions; cardiac troponin T (cTnT), which binds to tropomyosin and facilitates contraction; and troponin C (TnC), which binds to calcium ions. cTnI and cTnT are the preferred markers for the diagnosis of myocardial injury [2–4]. cTnI is not highly specific and sensible while there are many diseases that can improve its plasmatic concentration. It is elevated in patients with renal failure or muscle trauma [5]. Some studies evaluated plasma levels of cTnI, CK, CK-MB, Myo following conventional ECV [6] and the extent of myocardial damage detectable after the delivery of endocardiac shocks by ICD [7]. The aim of this study was to assess the extent of myocardial injury detectable after repeated TD by the release of a cardiospecific marker cTnI in relation to other, not fully cardiospecific, markers of cardiac ischemia, CK, CK-MB and Myo.

2. Materials and methods

A total of 40 patients (mean 63±8.5 S.D. years, range 38–78) were enrolled into the study, group A (n=20) that received Topic Defibrillation and Control group (n=20) that did not receive Topic Defibrillation. Patients were included if they had ejection fraction (EF%) ≥50%, no chronic pulmonary disease (COPD), creatinine level <1.5 mg/dl, stable angina, uncomplicated clinical course (no difficulties in weaning from bypass, no ECG changes and no inotropic medication). Patients were excluded if they had had a pre-operative myocardial infarction, RCF, rheumatoid arthritis, IABP, pacemaker. The age of patients was 65±7.3 years (range 47–78 years) in group A; and 60±10 years (range 38–73 years) in Control group. The two groups were composed each one by: 9 coronary artery bypass (BAC), 4 aortic valve replacement (RVA), 4 mitral valve replacement (RVM), and 3 BAC + valve surgery (Table 1). TD was performed with paddles positioned on the right atrium and on the lateral wall of the left ventricle. Routinely, the initial energy used was 10 J; 20 J of delivered energy was used for subsequent cardioversion or defibrillation attempts when multiple shocks were required for termination of arrhythmia. Blood samples were drawn 10 min after aortic clamping, 10 min after declamping, at arrival in ICU, 8 h, 12 h and 24 h postoperatively and were analyzed for cTnI, CK, CK-MB, Myo. Total CK activity was quantified by Abbott AxSym (MEIA) system (Abbott Park, IL) with an upper normal limit of 174 IU/l. Total serum CK-
MB activity was quantified with Elecsys 2010 (Roche) with an upper limit of 3.77 ng/ml. Myoglobin was determined by Elecsys 2010 (Roche) with an upper normal limit level of 58 ng/ml. cTnI was assayed by Abbott AxSym (MEIA) system (Abbott Park, IL), which has an upper normal limit of 0.04 ng/ml.

3. Statistics

Data were expressed as mean ± S.D. The comparisons between the TD and the No-TD were made by the independent-samples t test. Statistical significance was considered for P value <0.05.

4. Results

The procedure proved to be effective, with the restoration of stable sinus rhythm in all the patients. The number of shocks delivered in all the patients was 1.75 ± 0.85. The maximal amount of energy delivered in a single patient was 22 ± 13.9 J. There was no statistically significant difference for cTnI plasma level between patients that received Topic Defibrillation and patients that did not receive TD. TD did not influence the curve of raise of cTnI, it was not different between the two groups (Fig. 1). CK plasma level was not influenced by the TD, in fact there was no difference in serum level and curve of raise between group A and control group (Fig. 2). CK-MB, Myo were influenced by topic defibrillation and the difference between group A and control group was in the curve of raise of these two cardiac enzymes. The increase of cTnI started at 10 min after aortic clamping and a peak was reached 8 h post-op in both groups. CK increased 10 min after declamping and reached a peak value on the 12th h post-op in group A and in control group. CK-MB in group A started to increase 10 min after declamping similar to control group but the peak was reached 8 h post-op while in the control group peak was reached on the first post operative day (Fig. 3). Myo increased 10 min after declamping and the peak was reached at arrival in ICU in group A and at 8 h post-op in control group (Fig. 4). We found a difference in the curve of raise of CK-MB and Myoglobin between the two groups. There was no statistically significant difference in the mean peak value of CK-MB and myoglobin between patients that received TD and the control group. Mean peak values were 8.44 ± 5.14 ng/ml for cTnI in group A and 12.5 ± 12 ng/ml for cTnI in Control group; 652 ± 331 U/l for CK in group A and 643 ± 436.9 ng/ml for CK in Control group; 43.9 ± 21.2 ng/ml for CK-MB in group A and 36.83 ± 31.4 ng/ml for CK-MB in Control group; 771 ± 522 ng/ml for Myo in group A and 807 ± 992 ng/ml for Myo in Control group (Table 2). The difference between cardiac markers was in the time of raise and not in the peak values.

5. Comment

The application of discharges of direct current can cause reversible damage of subcellular structures involved in oxidative phosphorylation and originate a variable release of non-specific proteins of the striate cardiac muscle, including CK, CK-MB, and Myo, which makes the diagnosis of a previous acute myocardial lesion difficult [6]. However, CK, CK-MB and Myo concentrations fail to reveal minimal myocardial lesions, especially if simultaneous skeletal muscle damage also exists. The use of cardiospecific markers like cTnI, which are not expressed in skeletal muscle, has made possible a more effective discrimination of the skeletal or myocardial origin of muscle lesions [2]. Troponins are regulatory proteins located in the striated muscle. They regulate actomyosine interactions. These proteins have a small cytosol distribution [8]. Plasma concentrations are low in healthy patients and have a cut-off of 18 ng/ml in patients who have a cardiac operation [5, 9]. cTnI is highly specific for myocardial injury [10]. During cardiac surgery there is an increase of cardiac markers, and to show
Comparison of cardiac markers level between Group A and Group B

If TD is able to influence cTnI plasma level 20 patients who received TD and 20 patients who did not receive TD were enrolled in the study. The way the energy is delivered to the heart is an important factor that influences the release of cardiac markers into circulation 4. Increasing CK and CK-MB concentrations after transthoracic and/or ICD shocks have been described [2,3,8,11,12]. It has been found that most of CK released after transthoracic cardioversion derives from chest wall skeletal muscles, because cardiospecific markers such as cTnI show no or minimal increase after transthoracic shock [8]. We found a similar increase of all markers between the two groups after cardiac surgery. It is remarkable that the time of increase of CK-MB and Myo between the two groups is different than the mean peak value for these cardiac enzymes.

In patients that receive TD it should be better to use cTnI for the diagnosis of cardiac damage [8–15]. An increase of cTnI should not be linked to TD. Future studies on the effect of TD on cardiac enzymes plasma level would be necessary to improve our knowledge in this field.

References