Cardiac troponin I after external electrical cardioversion for atrial fibrillation as a marker of myocardial injury – a preliminary report

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Abstract

Background: It is uncertain whether external electrical cardioversion (CV) of atrial fibrillation (AF) can cause myocardial injury identifiable by troponin I (cTnI).

Aim: To examine whether external CV of AF can cause cTnI rise as measured with high-sensitivity assay, and to identify factors determining this elevation.

Methods: Patients with non-valvular AF selected for CV were included. Exclusion criteria were myocardial ischaemia, elevated D-dimer, heart and renal failure. Patients underwent monophasic or biphasic CV. Troponin I was measured before, and 6 and 12 hours after the procedure with TNI-ADV assay; NT-proBNP was measured before CV. Echocardiography was performed in all patients.

Results: Twenty-two patients were examined. Troponin I 6 and 12 hours after CV [0.04 ng/ml (0.00-0.30), 0.04 ng/ml (0.00-0.13)] was significantly higher than before [0.017 pg/ml (0.00-0.08)] (p=0.01, p=0.02). Only in one patient did cTnI exceed the cut-off for myocardial infarction after 6 hours (>0.16 ng/ml) with subsequent normalisation after 12 hours. Left ventricular end-diastolic dimension (LVEDD) was significantly higher and ejection fraction lower in the group with cTnI rise in comparison with the group with no cTnI elevation (54,2±6,3 vs. 47,6±5,7 mm, p=0,02; 56,2±8,9 vs. 63,2±7,1%, p=0,05). LVEDD=53 mm had 75% sensitivity and 72% specificity for predicting cTnI elevation after CV. Age, gender, AF duration, type of CV, energy, left atrial dimension, baseline cTnI and NT-proBNP were not predictive of cTnI increase.

Conclusions: Cardioversion can lead to mild but significant cTnI rise as measured with a high-sensitivity assay. The influence of CV on cTnI elevation appears to be more pronounced in patients with relatively large LVEDD.

Key words: cardiac troponin I, cardioversion, atrial fibrillation

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Introduction

Markers of myocardial necrosis are useful in the diagnosis of myocardial infarction (MI). The introduction of cardiac troponin (cTn) assays into routine clinical and laboratory practice brought about MI redefinition [1], a pivotal part of which is the elevation of cardiac specific troponin I or T in the patient's serum. It is known that markers with limited cardiac specificity such as creatine kinase (CK), its cardiac isoenzyme MB (CKMB) or myoglobin may be increased in skeletal muscle injury, including electrical injury sustained during cardioversion (CV) [2, 3].

In the past, when troponin assays were not available, increased CK or CKMB could obscure the diagnosis of MI coexisting with arrhythmia requiring CV. This could lead to false negative (CK/CKMB increase attributed to CV in the case of a true MI) or false positive results (CK/CKMB increase attributed to MI in the case of skeletal muscle electrical injury). On the other hand the levels of cardiac-specific troponins should not increase in response to CV if it does not cause myocardial injury. However, over 30 years ago animal model trials already revealed that repeated external electrical shocks can cause myocardial injury seen on autopsy and histological examination [4].

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Since that time much research has been performed in order to establish whether CV can lead to myocardial injury resulting in the release of detectable amounts of troponins and, if so, whether these amounts can reach the values used for the diagnosis of MI [5, 6]. The results published in the literature are equivocal or even contradictory. According to some authors, CV has no effects on troponin concentration but others claim that troponin elevation in response to CV is substantial [7, 8]. One of the reasons for these discrepancies could be differences in analytical sensitivity of troponin assays and their various diagnostic cut-off values for MI. Because the issue of troponin rise after CV may be of potential clinical importance, especially in the context of MI diagnosis, and it has not been resolved unequivocally, we decided to measure troponin concentration in patients undergoing CV of atrial fibrillation (AF).

The aim of our study was to determine whether external electrical CV of AF can cause significant troponin I elevation as measured with a high-sensitivity assay and to identify clinical, demographic, echocardiographic and biochemical predictors of troponin I release.

Methods

Consecutive patients admitted to the department for CV for non-valvular persistent AF were included in the study. If arrhythmia lasted more than 48 hours, at least 5-week oral anticoagulation was mandatory before CV. Exclusion criteria were conditions that could potentially lead to troponin elevation: clinical or electrocardiographic signs of myocardial ischaemia, increased probability of pulmonary embolism (D-dimer concentration >0.5 µg/ml), renal insufficiency (creatinine concentration >1.5 mg/dl) or symptomatic heart failure (class C or D according to the new heart failure classification). Patients underwent monophasic or biphasic CV depending on the availability of a particular cardioverter at the time of the procedure. Cardioverter electrodes were placed in a standard fashion: at the apex and at the base of the heart, the latter one pointing with its long axis at the left acromyon. The Local Bioethics Committee approved the study.

Cardiac troponin I concentration (cTnI) was measured in the samples collected before CV (cTnI0), and 6 (cTnI6) and 12 hours (cTnI12) after the procedure. In samples drawn before CV, the N-terminal probrain natriuretic peptide (NT-proBNP) was also measured. The cTnI level was measured with a high-sensitivity Troponin I Advanced AxSYM assay (TNI ADV AxSYM, Abbott Laboratories, Chicago, Il). This is a microparticle immunoassay (MEIA) with antibodies directed against troponin I and C complex. Its analytical sensitivity, concentration at 99th percentile of an apparently healthy population, cut-off recommended by the assay manufacturer for MI diagnosis according to the year 2000 definition (imprecision expressed as coefficient of variation ≤10%, CV ≤10), and cut-off for MI according to WHO definition are as follows: 0.02 ng/ml, 0.04 ng/ml, 0.16 ng/ml and 0.4 ng/ml. The NT-proBNP concentration was measured on an Elecsys analyser (Roche). Echocardiography was performed with a Vivid 3 echocardiograph (General Electrics). Left atrial dimension (LA) and left ventricular end-diastolic dimension (LVEDD) were measured in M-mode according to Penn convention. Left ventricular ejection fraction (LVEF) was calculated using the Simpson method and LV volumes obtained in 2D-mode in apical views.

Statistical analysis

Differences between cTnI0, cTnI6 and cTnI12 were determined with the Wilcoxon test. Correlations between cTnI elevation and examined parameters were estimated with non-parametric Spearman test. Differences between groups were estimated with U Mann-Whitney test for data without normal distribution and Student's t-test for data with normal distribution. The cTnI elevation after CV was defined as an absolute rise in cTnI concentration to at least the level of analytical sensitivity. Logistic regression analysis was performed in order to identify independent predictors of cTnI elevation. Odds ratio (OR) for cTnI elevation was calculated. A ROC curve was constructed to quantitatively estimate the ability of analysed parameters to predict cTnI release after CV. A p value <0.05 was considered statistically significant.

Results

We examined 23 patients (12 males, 11 females), mean age 67±13 years. Atrial fibrillation lasting >48 hours was present in 9 patients, monophasic CV was performed in 14 patients, whereas the remaining 11 patients underwent biphasic CV. Detailed clinical, demographic, echocardiographic and biochemical characteristics are provided in Table I.

The cTnI values increased significantly after CV. Mean cTnI concentration 6 hours after CV [cTnI6 0.04 (0.00-0.30) pg/ml] and 12 hours after CV [cTnI12 0.04 (0.00-0.13) pg/ml] was significantly higher than before the procedure [cTnI0=0.017 (0.00-0.08) pg/ml]. Mean cTnI 6 and 12 hours after CV were not significantly different (Figure 1).

Only in one patient (patient 17) did cTnI exceed the cut-off recommended by the assay manufacturer for MI diagnosis according to the year 2000 definition. The cTnI concentration returned to normal 12 hours after CV. In no patient did cTnI rise above the cut-off value for MI diagnosis by WHO. Kinetics of cTnI concentrations in all patients is presented in Figure 2.

Table I. Demographic, clinical, echocardiographic and biochemical data of the studied patients

Variable	All patients (n=23)	Patients with cTnI increase (n=12)	Patients with no cTnI increase (n=11)	p
Age [years]	67.4±13.2	64.3±15.1	70.8±10.4	0.37
Cumulative energy [J]	416 (90-1220)	353 (90-860)	485 (90-1220)	0.55
LVEF [%]	59.5±9.2	56.2±8.9	63.2±7.1	0.05
LVEDD [mm]	51.2±7	54.2±6.3	47.6±5.7	0.02
LA [cm]	4.5±0.4	4.53±0.37	4.51±0.56	0.98
cTnI0 [ng/ml]	0.017 (0.00-0.08)	0.017 (0.00-0.06)	0.018 (0.00-0.08)	0.87
NT-proBNP [pg/ml]	1842 (176-4889)	2030 (176-4889)	1651 (401-3461)	0.67

Abbreviations: LVEF – left ventricular ejection fraction, LVEDD – left ventricle end-diastolic dimension, LA – left atrial dimension, cTnI0 – cardiac troponin I concentration measured before cardioversion, NT-proBNP – N-terminal probrain natriuretic peptide

A significant correlation was found between cTnI before and 6 hours after CV (r=0.53; p=0.009) and between cTnI 6 hours after CV and LVEDD (r=0.63; p=0.001). Furthermore, cTnI before CV also correlated with LVEDD (r=0.46, p=0.03). A negative correlation was observed between cTnI 6 hours after CV and EF (r=–0.45; p=0.03) and a trend towards negative correlation between cTnI before CV and EF (r=–0.39, p=0.06) was also observed.

Among available demographic, clinical, echocardiographic and biochemical data (baseline cTnI and NT-proBNP), only LVEDD correlated with cTnI elevation (r=0.41, p <0.05). When we compared all available baseline data in patients with troponin elevation after CV and those in whom cTnI concentration did not rise, it turned out that LVEDD was significantly higher and EF lower (borderline significance) in the group with cTnI elevation. Other parameters were not related to the cTnI release in response to CV (Table I). We found no relationship between cTnI increase after CV and the type of CV (monophasic vs. biphasic, p=0.48), gender (p=0.71) or duration of AF (>48 vs. <48 hours, p=0.73).

A stepwise logistic regression analysis confirmed that LVEDD was the most useful in predicting cTnI release after CV (χ^2=7.22, p <0.02). The ROC curve was constructed in order to evaluate the ability of LVEDD to predict cTnI elevation. The optimal cut-off value of LVEDD for predicting significant cTnI release was determined to be 53 mm. At this point, LVEDD had 75% sensitivity and 72% specificity in predicting cTnI elevation in response to CV (Figure 3).

Odds ratio (OR) for cTnI release was determined to be 5.3 for LVEDD over 53 mm vs. LVEDD of less than 53 mm. This translated into OR 0.19 (confidence interval, CI: 0.03-1.25) for cTnI release for 1 mm increase in LVEDD.

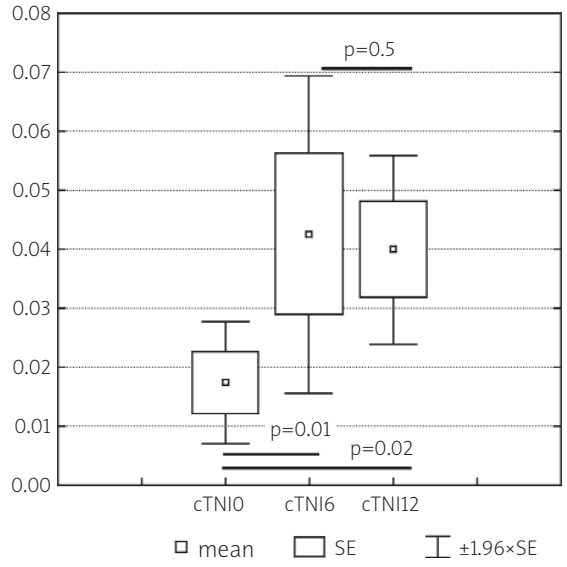

Figure 1. Troponin I before, 6 hours after, and 12 hours after external cardioversion for atrial fibrillation

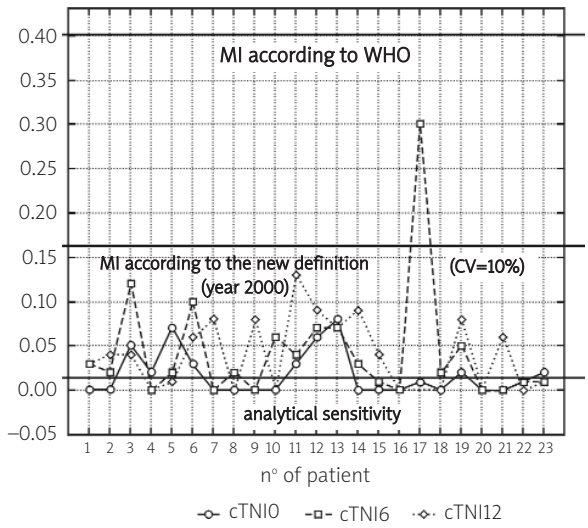

Figure 2. Changes in troponin I concentration: values before external electrical cardioversion for atrial fibrillation and 6 and 12 hours after in 23 examined patients

Broad 95% CI implies large variation of the variable, which may be attributed to the small number of patients.

Nine patients had baseline cTnI higher than or equal to the assay's analytical sensitivity. Patients that were troponin-positive at baseline had significantly higher LVEDD and lower EF than troponin-negative patients (LVEDD 54.8±7.4 vs. 48.7±5.3 mm, p=0.03, EF 54.3 vs. 62.9%, p=0.02).

Discussion

Our results show that CV can cause detectable and significant cTnI elevation. This finding is in line with results reported by others [5, 8, 9].

In spite of a significant cTnI elevation, this phenomenon was of limited clinical importance because only in one patient did cTnI concentration exceed the cut-off recommended by the assay manufacturer for MI diagnosis according to the year 2000 definition. This occurred 6 hours after CV and cTnI returned to normal 12 hours after the procedure. In no patient did cTnI exceed the cut-off for MI diagnosis according to WHO. Thus, it appears that clinically significant cTnI rise after cardioversion is infrequent and if it occurs, subsequent measurements of cTnI in combination with clinical and electrocadiographic evaluation should allow MI to be excluded.

Another important observation made in our study is that cTnI elevation after CV significantly correlates with LVDD and LV systolic function. This adds new information to our current knowledge in this respect. In the study by Schulter et al. [10] internal CV by an implantable cardioverter-defibrillator led to the release of substantial amounts of troponin in patients with LV systolic dysfunction. In the study by Lund et al. [5] external CV caused significant troponin release only in one patient with heart and renal failure. Our data imply that even echocardiographically mild and clinically asymptomatic LV dilatation and dysfunction can predispose to electrical myocardial injury detectable by high-sensitivity cTnI assay. In conjunction with the results of studies with more numerous patient groups in which cTnI increased in response to monophasic CV only (higher cumulative energy) [8, 9] our data may incline physicians to choose a less traumatic, lower-energy biphasic CV especially in patients with LVEDD >53 mm and/or LV systolic dysfunction, although sensitivity and specificity of LVEDD in the range of 72-75% in predicting significant troponin release may still be too low for practical purposes.

Our study showed no correlation between the type of CV and its cumulative energy with cTnI elevation; however, this may be due to the very limited number of patients examined. The number of patients studied by Kosior et al. [8, 9] who showed an association between CV type and troponin elevation was substantially greater.

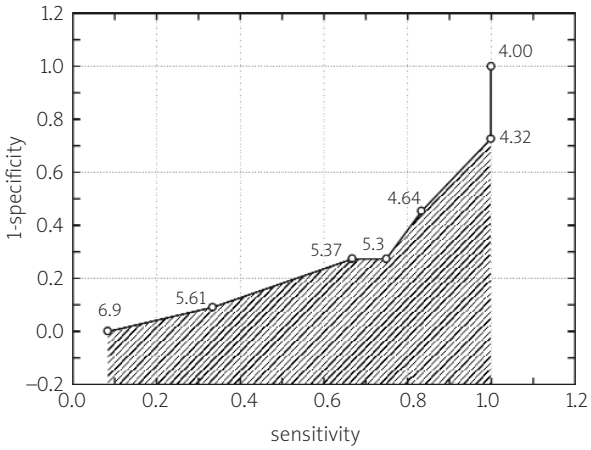

Figure 3. Different values of left ventricular end-diastolic diameter in predicting troponin I release after cardioversion

No other clinical, demographic, echocardiographic or biochemical parameters (baseline cTnI, NT-proBNP) predicted cTnI rise after CV. Baseline cTnI determined cTnI concentration 6 hours after CV, but not its elevation. Likewise, baseline NT-proBNP did not predict cTnI elevation. This is unique information since we did not find any reports on a potential relationship between NT-proBNP and cTnI after CV. The question arises why NT-proBNP, a biochemical marker of LV dysfunction, is not related to cTnI concentration or its elevation after CV, despite a relationship between echocardiographic markers of LV function and cTnI elevation. This could be explained by the fact that AF in itself can cause substantial NT-proBNP elevation, which was confirmed by the high baseline NT-proBNP values in our study and also that of Corell et al. [11].

Thus, LVEDD, and to a lesser extent EF, determined the risk of cTnI release after CV. The reason for this could be the altered haemodynamics of an even mildly dilated LV Increased intraventricular pressure and myocardial stretch leading to disturbed synthesis of both contractile elements and energy storing compounds could make myocardium less resistant to electrical trauma. Future trials should establish whether it is clinically reasonable to attempt some kind of specific or non-specific myocardial protection before CV in patients with organic heart disease and/or increased LVEDD (or LVEDD in the upper part of the reference range).

The correlation between LVEDD, EF and troponin release from myocardium in response to CV found in our study contradicts the results of Cemin et al., who examined a much greater number of patients (n=193) and found no association between depressed LV function and the risk of troponin elevation [12]. These discrepant results may be explained by the fact that in that study troponin I was measured 18-20 hours after cardioversion when its concentration can normalise.

Another issue that has to be considered is the cTnI assay sensitivity and the choice of cut-off value for MI diagnosis. These two factors are manufacturer-dependent as there is no standardisation of cTnI tests. Studies conducted at the beginning of the 21st century, in which no cTnI elevation was detected after CV, used cTnI assays with lower analytical sensitivity than the assay we used [13, 14]. Studies from the same or earlier period, in which cardiac troponin T (cTnT) was measured, used first or second generation tests. Their cardiac specificity and analytical precision are presently considered inadequate. One more recent study [3], which included a greater number of patients than our study (n=74), failed to show significant cTnI elevation after CV. This may be explained by the difference in the cTnI assays used. They used DPC Immulite with five-fold lower analytical sensitivity than the TNI ADV AxSYM assay used in our study. In that study [3], cTnI elevation was observed only in two patients with elevated baseline concentration of this marker, which suggests cTnI fluctuation within the range where analytical precision is inadequate rather than a true release of an additional pool of the marker from the myocardium after CV. It appears that the high sensitivity of our assay could contribute to the detection of cTnI elevation despite the very limited number of patients in our study.

In our study, 9 patients had elevated (detectable) cTnI at baseline (cTnI equal to or over the cut-off corresponding to analytical sensitivity). We could not find an unequivocal explanation for this observation, since patients with potential causes of cTnI elevation were excluded from the study. Some authors suggest that AF or other types of tachycardia can by itself bring about troponin release, especially in patients with organic heart disease, but data supporting this hypothesis are sparse [15]. In fact, our patients that were troponin-positive at baseline had a significantly higher LVEDD and lower EF, which could imply that even mild and clinically silent LV systolic dysfunction could lead to troponin release in the presence of arrhythmia, even before CV is performed.

Study limitations

The most important limitation of our study is the small number of patients. This reduced the number of patients who accidentally had lower EF to an even smaller number. The latter is also a result of exclusion criteria – only patients without clinical signs or symptoms of overt heart failure were included as decompensated circulatory insufficiency is a recognised cause of troponin elevation. Therefore, one should be careful not to extrapolate our results onto patients with more severe LV dysfunction. Because of the limited number of patients, our findings need to be confirmed in a larger study.

Conclusions

1. Electrical external cardioversion of AF can cause significant troponin I elevation detectable by a high-sensitivity assay, although the clinical relevance of this phenomenon in the context of a MI diagnosis appears to be moderate.
2. The influence of external cardioversion on troponin I elevation appears to be more pronounced in patients with relatively large LVEDD.

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Troponina I oznaczana metodą o wysokiej czułości po zewnętrznej kardiowersji elektrycznej migotania przedsionków – doniesienie wstępne

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Streszczenie

Wstęp: Wiele dotychczas opublikowanych danych wskazuje na brak wzrostu stężenia troponin po zewnętrznej kardiowersji elektrycznej migotania przedsionków (AF). Niektórzy autorzy donoszą jednak o możliwości nieznacznego wzrostu stężenia tych markerów w odpowiedzi na zewnętrzną kardiowersję elektryczną u chorych z AF. W ostatnim czasie pojawiają się nowe komercyjne zestawy do oznaczeń troponin o coraz wyższej czułości, których dynamika po zabiegach potencjalnie nieuszkadzających miokardium, w tym po zewnętrznej kardiowersji, nie jest znana. Nie wiadomo też, czy istnieje związek między stężeniami troponiny po kardiowersji i stężeniami peptydów natriuretycznych.

Cel: Ocena dynamiki stężenia sercowej troponiny I (cTnI) o wysokiej czułości (ang. *advanced troponin I*, cTnI ADV) po zewnętrznej kardiowersji elektrycznej AF. Ustalenie klinicznych, demograficznych, echokardiograficznych i biochemicznych predykatorów ewentualnego wzrostu cTnI po kardiowersji elektrycznej.

Metodyka: Do badania włączono kolejnych chorych kwalifikowanych do kardiowersji elektrycznej z powodu przetrwałego AF. Kryteriami wykluczenia były stany predysponujące do wzrostu stężenia troponin: podmiotowe lub elektrokardiograficzne cechy niedokrwienia mięśnia serca, objawowa niewydolność serca, podejrzenie zatorowości płucnej (D-dimer >0,5 ng/ml), niewydolność nerek (stężenie kreatyniny >1,5 mg/dl). U wszystkich chorych oznaczono cTnI (AxSYM TnI ADV, Abbott Lab.) i N-końcowy propeptyd natriuretyczny typu B (NT-proBNP) (Elecsys, Roche Diag.) przed kardiowersją. Oznaczenia cTnI powtarzano 6 i 12 godz. po zabiegu. U wszystkich chorych wykonywano badanie echokardiograficzne, w którym oceniano wymiar końcowo-rozkurczowy lewej komory (LVEDD), wymiar lewego przedsionka (LA) i frakcję wyrzutową lewej komory (LVEF). Kardiowersję zewnętrzną wykonywano kardiowerterem jedno- lub dwufazowym, zależnie od dostępności określonego typu sprzętu.

Wyniki: W badaniu wzięło udział 23 chorych (12 mężczyzn i 11 kobiet) w średnim wieku 67,4±13,22 lat. Kardiowersję elektryczną wykonano kardiowerterem jedno- (n=14) lub dwufazowym (n=9). Migotanie przedsionków trwało >48 godz. u 14 chorych, a <48 godz. u 9 chorych. Stężenie cTnI 6 godz. po kardiowersji było istotnie wyższe niż przed kardiowersją, odpowiednio: 0,04 (0,00–0,30) ng/ml, 0,017 (0,00–0,08) ng/ml; p=0,01. Stężenia cTnI po 12 godz. od kardiowersji były również istotnie wyższe niż przed zabiegiem, odpowiednio 0,04 ng/ml (0,00–0,13) ng/ml i 0,017 (0,00–0,08) ng/ml, p=0,02. Natomiast nie było istotnej różnicy między stężeniem cTnI w 6. i 12. godz. od zabiegu (p=0,5). W żadnym przypadku nie odnotowano wzrostu cTnI powyżej punktu odcięcia dla zawału wg definicji WHO (>0,4 ng/ml) i tylko w jednym stwierdzono wzrost stężenia cTnI >0,16 ng/ml (stężenie oznaczone z nieprecyzyjnością ≤10%, punkt odcięcia zalecany przez producenta do rozpoznania zawału wg definicji z roku 2000). Po 12 godz. u tego chorego doszło do normalizacji stężenia cTnI. Wymiar końcoworozkurczowy lewej komory był istotnie większy, a LVEF mniejsza w grupie ze wzrostem stężenia cTnI po kardiowersji elektrycznej w porównaniu z grupą bez wzrostu stężenia cTnI po zabiegu (odpowiednio: 54,2±6,3 *vs* 47,6±5,7 mm, p=0,02; 56,2±8,9 *vs* 63,2±7,1%, p=0,05). Wymiar końcoworozkurczowy lewej komory był jedynym niezależnym predykatorem wzrostu stężenia cTnI po kardiowersji elektrycznej. Wartość LVEDD=53 mm przewidywała wzrost stężenia TnI z czułością 75% i swoistością 72%. Płeć, wiek, czas trwania AF (powyżej lub poniżej 48 godz.), typ kardiowersji, zastosowana energia, wymiar LA, wyjściowe stężenie NT-proBNP i cTnI nie wiązały się ze wzrostem stężenia cTnI po kardiowersji elektrycznej.

Wnioski: Zewnętrzna kardiowersja elektryczna AF prowadzi do istotnego wzrostu stężenia cTnI oznaczanej czułym testem. Wpływ kardiowersji zewnętrznej na wzrost stężenia cTnI może być bardziej wyrażony u chorych z względnie większym LVEDD.

Słowa kluczowe: troponina I, kardiowersja, migotanie przedsionków

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