ORIGINAL ARTICLE

Prognostic importance of serum troponin concentration in patients with an implanted cardioverter-defibrillator admitted to the emergency department due to electric shock

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KEY WORDS
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ABSTRACT

BACKGROUND High-energy therapy with an implantable cardioverter-defibrillator (ICD) may increase serum cardiac troponin I (cTnI) concentrations.

AIMS We aimed to assess the effect of cTnI concentrations after ICD therapy on mortality.

METHODS We assessed 150 patients (mean [SD] age, 64.2 [12.8] years) admitted to emergency departments due to at least one electric shock during the last 24 hours, with known serum cTnI concentrations on admission. Age, sex, comorbidities, the number of shocks, therapy appropriateness, serum creatinine concentrations, and left ventricular ejection fraction (LVEF) were documented for a retrospective analysis. The personal identification numbers (PESEL) of patients were used to obtain survival data. The follow-up was defined as the time between the admission date and November 14, 2018, or until death had occurred or a period of 3 years had elapsed (1057 days).

RESULTS The cTnI concentration was increased in 92 patients (61.3%). The mortality rate was related to age (hazard ratio [HR], 1.04; 95% confidence interval [CI], 1.01–1.08; $P = 0.03$); increased cTnI concentrations (HR, 2.88, 95% CI, 1.30–6.37; $P = 0.01$); diabetes (HR, 2.19; 95% CI, 1.09–4.39; $P = 0.03$); ischemic heart disease (HR, 2.96; 95% CI, 1.11–7.87; $P = 0.03$); serum creatinine concentrations (HR, 2.17; 95% CI, 1.18–4.00; $P = 0.01$); LVEF (HR, 0.95; 95% CI, 0.91–0.99; $P = 0.009$), and previous or current coronary artery bypass grafting or percutaneous coronary intervention (HR, 0.38; 95% CI, 0.15–0.96; $P = 0.04$ and HR, 0.29; 95% CI, 0.13–0.65; $P = 0.003$; respectively).

CONCLUSIONS The reasons for increased mortality rate in patients with ICD shocks are multifactorial. An increased cTnI concentration on admission, but not the number of shocks, is an independent predictor of higher long-term mortality.

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INTRODUCTION Implantable cardioverter-defibrillators (ICDs) are used to prevent sudden cardiac death in patients at risk of fatal ventricular arrhythmias.[1] The number of patients admitted to the emergency departments (EDs) due to electric shock is constantly increasing.[2-6] It is expected that up to 50% of ICD recipients will present at the ED within a 4-year period, and half of the visits will be related to electric shocks.[7]

The mortality of patients who have suffered an ICD shock in the acute phase is relatively low, estimated at less than 1%.[8] However,

WHAT'S NEW?

Increased serum troponin concentrations after implantable cardioverter--defibrillator (ICD) discharge due to spontaneous cardiac arrhythmia is a well--known phenomenon. The present study shows the unfavorable effect of increased troponin concentration on prognosis, independently of known prognostic factors. Our results indicate the importance of a thorough diagnostic workup and appropriate treatment in this group of patients. The elevated serum troponin concentration may be provoked by myocardial ischemia, renal insufficiency, decompensated heart failure, and electric shock from an ICD. These factors may coexist. Distinguishing whether the source of this increase is an electric shock or another factor is not always possible. The response of the myocardium to a delivered electric current varies widely between individuals. In a 2-year follow-up, it was shown that an increased serum troponin concentration at admission to the emergency department due to electric shock increased the risk of death more than 2-fold compared with patients with a serum troponin concentration within the reference range.

the mid- and long-term mortality rates are significantly higher. Therefore, it is important to define factors associated with increased long--term mortality. The use of high-energy therapy with an ICD is higher than the expected rate of sudden cardiac arrests in a given population. Accordingly, it could be presumed that the population of patients with electric shocks is not homogenous, and special attention should be paid to those who are at the highest risk. Both adequate and inadequate electric shocks increase the risk of death; therefore, measures to prevent unnecessary shocks are taken, for example, programming with a prolonged detection window leading to delayed ICD therapy, or avoiding defibrillation threshold testing when possible.[3]

An ICD therapy may cause cardiac injury and systemic inflammation, which may result in increased cardiac troponin I (cTnI) and C-reactive protein concentrations.[9,10] Furthermore, some of the potential triggers of electric shocks, such as decompensated heart failure, diarrhea, hypokalemia, infection, cardiac ischemia, and psychological stress, are also related to an increase in cTnI concentrations.[9,11,12] Moreover, it has been reported that an increased cTnI concentration at baseline increases all-cause mortality in patients with ICD shocks.[13,14]

The aim of the study was to assess whether increased cTnI concentrations in patients admitted to the ED due to an ICD shock are related to long-term prognosis.

METHODS A retrospective analysis was performed on the medical records of patients admitted to 2 EDs between 2013 and August 2018 after experiencing an electric ICD shock. The following data were collected: age, sex, number of electric shocks, therapy appropriateness (assessed as either adequate or inadequate), comorbidities, left ventricular ejection fraction (LVEF), and serum creatinine concentrations. A total of 150 patients with known cTnI concentrations on admission were included in the analysis.

The personal identification numbers (PESEL) of patients were used to obtain survival data on November 14, 2018. The follow-up was defined as the time between the admission date and November 14, 2018, or until death had occurred or a period of 3 years had elapsed (1057 days).

The study was approved by the local Bioethics Committee of Wroclaw Medical University.

Statistical analysis Data were presented using means (SDs), medians (interquartile ranges [IQRs]), or numbers (percentages). The data were compared with the *t* test, Mann–Whitney test, or χ^2 test as appropriate. The cTnI concentration was assessed with different methods; therefore, to enable the statistical analysis, the raw data were dichotomized into those within normal limits and those exceeding the upper limit of normal. A logistic regression analysis was performed to find factors related to increased cTnI concentrations. The survival rate between patients with normal and increased cTnI concentrations and with up to 2 shocks and at least 3 shocks were assessed using the Kaplan–Meier method and a log-rank test. The multivariate analysis of survival with the use of Cox proportional hazard stepwise regression was performed. A *P* value of less than 0.05 was considered significant.

RESULTS **Patient characteristics** The study group consisted of 150 patients at a mean (SD) age of 64.2 (12.8) years (range, 22–89 years), with the majority being male (127 [84.7%]). In 90 patients (60%), ICDs were implanted as primary prevention, and in 60 patients (40%), as secondary prevention. The underlying diseases were ischemic heart disease in 94 patients (62.7%), nonischemic cardiomyopathy in 43 (28.7%), hypertrophic cardiomyopathy in 9 (6%), arrhythmogenic right ventricular disease in 2 (1.3%), Brugada syndrome in 1 (0.7%), and preexcitation syndrome and sudden cardiac arrest during exercise in 1 patient (0.7%).

During a median follow-up of 467 days (IQR, 261–730 days; range, 1–1057 days), 37 patients (24.7%) died. Increased cTnI concentrations were found in 92 patients (61.3%). The logistic regression analysis revealed that increased troponin levels were related to adequate shock delivery (odds ratio [OR], 4.92; 95% CI, 1.75–13.82; *P* <0.003), at least 3 shocks (OR, 5.80; 95% CI, 2.44–13.81; *P* <0.001), and secondary prevention (OR, 0.35; 95% CI, 0.16–0.78, *P* <0.01), but not to serum creatinine concentrations (OR, 0.17; 95% CI, 0.003–8.989; *P* <0.37).

The comparison of patients with increased cTnI concentrations with those with normal cTnI concentrations is presented in TABLE 1.

TABLE 1 Comparison of patients with increased and normal cardiac troponin I concentration

Parameter	Patients with raised cTnI concentration (n = 92)	Patients with cTnI within normal range (n = 58)	*P* value
Age, y, mean (SD)	63.4 (12.5)	65.4 (13.2)	0.34
Male sex, n (%)	76 (82.6)	51 (87.9)	0.36
Creatinine, mg/dl, mean (SD)	1.22 (0.82)	1.22 (0.35)	0.27
Secondary prevention, n (%)	32 (34.8)	28 (48.2)	0.15
Adequate shock delivery, n (%)	75 (81.5)	42 (72.4)	0.19
Number of shocks, median (IQR)	3.5 (1.5–6.5)	2 (1.0–4.0)	0.002
At least 3 shocks, n (%)	55 (59.8)	19 (32.8)	0.001
LVEF, %, mean (SD)	33.3 (13.1)	32.6 (13.3)	0.74
Diabetes, n (%)	28 (30.4)	16 (27.6)	0.71
CABG, n (%)	16 (17.4)	14 (24.1)	0.32
PCI, n (%)	38 (41.3)	28 (48.3)	0.41
Myocardial infarction, n (%)	47 (51.1)	31 (53.5)	0.78
Nonischemic cardiomyopathy, n (%)	27 (29.4)	16 (27.6)	0.82
Ischemic heart disease, n (%)	56 (60.9)	38 (65.5)	0.57
Others ICD indications, n (%)	9 (9.8)	4 (6.9)	0.54

Abbreviations: CABG, coronary artery bypass grafting; cTnI, cardiac troponin I; ICD, implantable cardioverter-defibrillator; IQR, interquartile range; LVEF, left ventricular ejection fraction; PCI, percutaneous coronary intervention

Long-term survival The Kaplan-Meier survival rates after ED admission due to electric shock in patients with elevated and normal cTnI concentrations are presented in FIGURE 1. The difference between groups was observed shortly after admission. Long-term survival rates did not differ between patients admitted to the ED due to 1 or 2 shocks and those with at least 3 shocks (P = 0.89; FIGURE 2).

Multivariate analysis The mortality rate after ED admission due to ICD shock(s) was related to age (HR, 1.04; 95% CI, 1.01–1.08; P = 0.03), increased cTnI concentrations (HR, 2.88; 95% CI, 1.30–6.37; P = 0.009), diabetes (HR, 2.19; 95% CI, 1.09–4.39; P = 0.03); ischemic heart disease (HR, 2.96; 95% CI, 1.11–7.87; P = 0.03), serum creatinine concentrations (HR, 2.17; 95% CI, 1.18–4.00; P = 0.01), LVEF (HR, 0.95; 95% CI, 0.91–0.99; P = 0.009), and previous or current coronary artery bypass grafting or percutaneous coronary intervention (HR, 0.38; 95% CI, 0.15–0.96; P = 0.04 and HR, 0.29; 95% CI, 0.13–0.65; P = 0.003; respectively).

DISCUSSION

The main finding of the study is that an increased cTnI concentration after admission to the ED due to ICD shock(s) increases the risk of overall long-term mortality. In order to draw conclusions from this finding, the relationship between the following 3 elements should be considered: electric shocks, cTnI concentration, and mortality rate.

Implantable cardioverter-defibrillator shocks and mortality External electrocardioversion does not increase the long-term mortality rate.[15,16] On the other hand, ICD shock delivery, both adequate and inadequate, worsen the long-term prognosis.[17] It was reported that an ICD shock is related to a 2- to 5-fold increase in mortality.[18] However, the cause is not well established. The presence of ICD shocks may be a marker of underlying disease progression or may cause and worsen myocardial damage.[19] Therefore, shocks may indicate the exacerbation of heart disease, and it is not surprising that it is also related to higher mortality.[19] In the present study, all patients received high-energy electric therapy. The analysis of survival after occurrence of up to 2 shocks and at least 3 shocks did not reveal any significant difference. No association was also found in the multivariate analysis. However, the higher number of shocks was related to higher cTnI concentrations; therefore, the association between the number of shocks and survival could be altered when cTnI was included in the multivariate analysis.

The present findings are in line with the results of the study carried out by Grene et al.[19] Contrary to this finding, many reports recognize electrical storm as a predictor of higher mortality rates.[20-22]

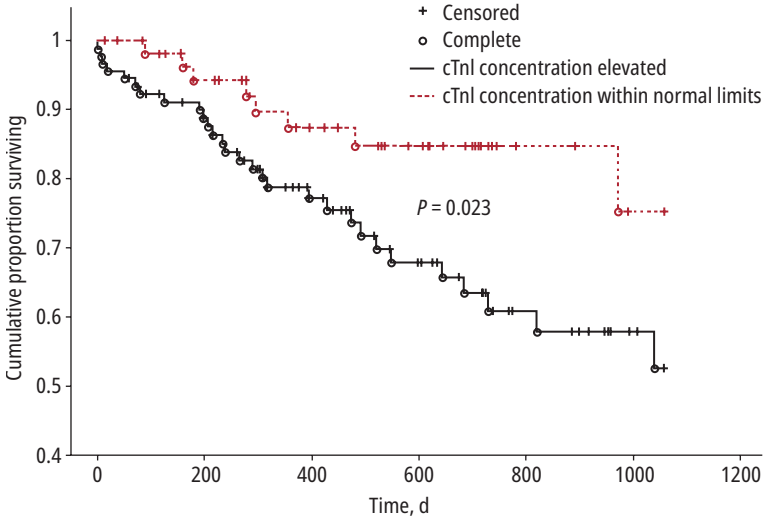

FIGURE 1 Long-term survival rates after emergency department admission due to electric shock in patients with normal and elevated cardiac troponin I (cTnI) levels

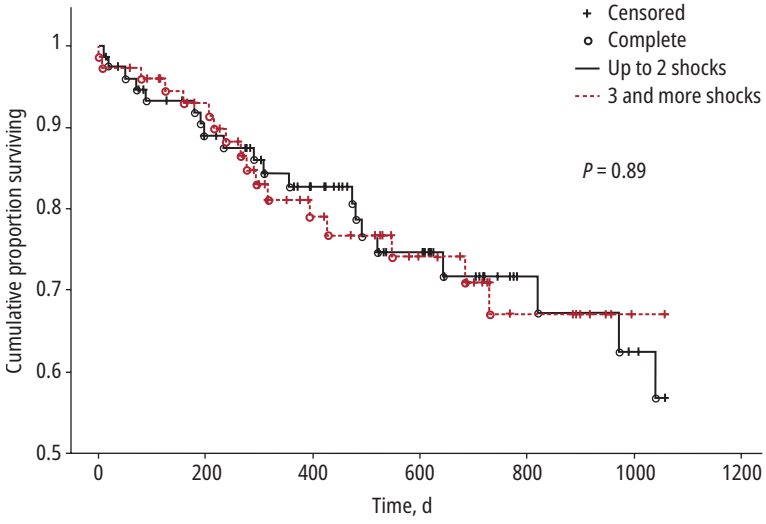

FIGURE 2 Long-term survival rate after emergency department admission due to 1 or 2 electric shocks and at least 3 shocks

Our findings are consistent with the previous report that the damage related to ICD shock is less dependent on the number of shocks than on the underlying pathology.[3] The most probable hypothesis is that the reasons for an increased mortality rate in patients with ICD shocks are multifactorial, including the progression of the underlying disease and the influence of concomitant diseases, and the number of ICD discharges is merely a marker of a higher mortality risk.

However, we did not analyze the number of antitachycardia pacing events. Therefore, the number of electric shocks of up to 2 does not exclude the criteria for electrical storm being fulfilled.

Implantable cardioverter-defibrillator shocks and troponin levels The percentage of patients with an increased cTnI concentration on admission to the ED was 61.3%, similar to the results by Miranda et al,[17] who reported elevated cTnI concentrations in 62% of patients with more than 3 electric shocks around 12 hours after the last shock.[17] We found that patients with increased cTnI concentrations had a higher number of shocks. This finding is concordant with the report of Hasdemir et al.[23] The increase in cTnI concentrations is not a specific finding in a patient admitted to the ED after an ICD shock. The increased cTnI concentration may be chronic and might have been present before ICD discharge due to various conditions such as heart or kidney failure, but might not have been known or assessed previously.[13] It may also be related to tachyarrhythmia, which can trigger ICD shocks. Finally, it may be caused by electrical injury of the myocardium. The effects of electric shock without lead deployment or tachyarrhythmia were presented in the setting of lead fracture and confirmed that electric shock leads to elevation of cTnI levels.[24,25] Normal cTnI concentrations may be present in the early period of myocardial injury; therefore, a lack of increase in cTnI concentrations may occur when admission occurs immediately after the shock. Increased cTnI concentrations may be a marker of cardiac injury caused by an electric current. The relationship between electric current and serum troponin concentrations depends on the shock energy and the method of its delivery. The myocardial vulnerability in diseased myocardium is higher than in a healthy individual. Tenma et al[26] reported a relationship between high shock energy accumulation and overall mortality in patients with reduced LVEF and atrial fibrillation. On the one hand, the importance of energy delivery is confirmed by findings showing that cTnI concentrations are within normal limits after external cardioversion of hemodynamically stable patients with supraventricular tachycardia or atrial fibrillation that was assessed by serial measurements.[27-29] Elevation of CTnI levels was smaller after subcutaneous 80-J discharge than after intracardiac 35-J discharge in experimental conditions, and this was not found in a human study.[30,31] On the other hand, electrical injury in a young and otherwise healthy person in an occupational setting may cause cardiac injury that presents with an increased cTnI level.[32] This injury is considered to be caused by coronary artery spasm, a thermal effect on the myocardium, a thrombogenic effect on coronary arteries, or generalized vascular injury.[32] The cTnI concentration after ICD implantation without defibrillation testing was related to the number of screw-in lead deployments.[33,34] The group with defibrillation testing showed a higher increase in cTnI concentrations. However, the cTnI concentration in all cases did not exceed 50-fold of the upper limit.[33] Brewster et al[33] reported that higher cTnI concentrations were related to exposure to higher total shock energy, lower ventricular

fibrillation (VF) cycle length, and longer VF duration. These authors concluded that the oxygen supply–demand mismatch was higher in the case of a faster VF rate and in the case of a longer VF duration. Contrary to this finding, Semmler et al[34] reported that cTnI concentrations depend on the shocks but not on the ventricular fibrillation provoked before shocks.[34]

Cardiac troponin I concentration and mortality Increased cTnI level was a risk factor of mortality in stable patients in a low-risk outpatient population presenting for cardiovascular disease prevention.[12] Elevation of cTnI levels in chronic heart failure both with reduced and preserved ejection fraction indicates poor prognosis.[35] Cheng et al[12] reported that increased cTnI concentrations in patients with an implanted ICD for primary prevention predicted all-cause mortality but not electrical discharge. The increased concentration of cTnI on admission to ED in the present study may depend on several factors, including myocardial injury caused by electric shocks, heart failure decompensation, and cardiac ischemia. Our analysis does not allow an unequivocal statement of whether an increased cTnI level is a marker of the severity of the disease and is chronically increased or if it is related to electrical injury of the myocardium. Similar results were reported by Blendea et al.[11] However, in their group, less than 50% of patients had spontaneous ICD shock.[11]

Limitations Clinical studies regarding ICD discharges are difficult to perform in clinical settings. The discharges are unpredictable, and many potentially confounding factors should be considered. These problems can limit such studies, but some of them can be omitted when planning future research. The first limitation of our study is that the timing of the blood testing to measure cTnI concentrations in relation to the shock(s) was not assessed. Moreover, patients had multiple shocks during different periods. Regarding further studies, we recommend analyzing cTnI concentrations after the last shock and assessing the time between the first and last shocks. Hemodynamic disturbances related to a prolonged event of ventricular tachycardia or ventricular fibrillation may have a greater impact on myocardial injury than multiple shocks. Finally, comorbidities such as acute coronary syndrome, acute myocarditis, or acute severe heart failure decompensation at the time of admission were not analyzed.

Conclusions Increased troponin concentration occurs in two-thirds of patients admitted to EDs after high-energy therapy with an ICD, and it is a predictor of long-term mortality. The reasons for an increased mortality rate after ICD shocks are multifactorial, including the progression of the underlying disease, influence of concomitant diseases, and reaction of the diseased myocardium to shocks. The number of shocks itself is not a predictor of survival. Further studies are warranted to assess the cause and effect relationship between those factors and long-term mortality.

ARTICLE INFORMATION

CONFLICT OF INTEREST None declared.

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