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Organic heart damage in electrical injury

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Abstract. Electrical injuries represent one of the contemporary problems in healthcare, often associated with high morbidity and mortality. Myocardial morphological damage can be caused by electrical injury and is found in 1.2% of victims. **Evidence Collection.** Literature sources were included in the study if they: 1) were published in Ukrainian, English, or Spanish; 2) reported cardiac arrhythmias associated with electrical injury; 3) provided information on the prevalence of organic myocardial cardiac lesions in electrical injury; 4) used an observational design (cohort or cross-sectional). **Evidence Synthesis.** A number of mechanisms of myocardial damage in electrical injury have been described. These include direct thermal damage, induction of coronary artery spasm, ischemia secondary to arrhythmic hypotension, acute hypertension due to chemoreceptor stimulation, catecholamine-mediated damage, and ischemia in the coronary artery basin as a component of generalized injury. The degree of external skin damage should not be used to determine the extent of internal injury, as the current flows through the body along various pathways depending on tissue resistance, contact surface area, and the volume of affected tissue. Patients with electrical injury are prone to developing myocardial infarction, considering the damage caused by the current at the level of the vascular intima and the occurrence of thrombosis. Myocardial infarction can be caused by spasm of the coronary arteries or their occlusion by blood clots. Acute tissue ischemia resulting from spasm of the smooth vascular muscles plays a significant role in the mechanism of these conditions. Electricity can lead to focal or diffuse heart damage and often causes necrosis involving the myocardium, nodal tissue, conduction pathways, and coronary arteries. A 24-hour observation period under continuous cardiac monitoring is currently recommended, even for asymptomatic patients without prior predisposing conditions. An elevated troponin level for diagnosing electrical cardiac injury mostly lacks fundamental clinical significance. **Conclusions.** Electrical damage to the cardiovascular system is accompanied by various life-threatening complications, including the possibility of long-term ones. Cardiac monitoring in victims of electric shock should be performed for at least 24 hours.

Keywords: electrical injury, organic myocardial damage, myocardial infarction, myocardial necrosis, cardiac monitoring

Introduction

Electrical injuries represent one of the contemporary problems in healthcare, often associated with high morbidity and mortality. In surviving patients, cardiac damage and the risk of delayed cardiovascular complications are of particular concern. Currently, there is no universally accepted protocol for the diagnosis and management of such patients [1]. As a result, physicians often feel uncomfortable managing these patients [1]. Electric shock can lead to various clinical conditions, from simple burns to fatal arrhythmias [2]. Myocardial morphological damage can be caused by electrical injury [3–5] and is found in 1.2% of victims [6].

Evidence Collection

Literature sources were included in the study if they: 1) were published in Ukrainian, English, or Spanish; 2) reported cardiac arrhythmias associated with electrical injury; 3) provided information on the prevalence of organic myocardial cardiac lesions in electrical injury; 4) used an observational design (cohort or cross-sectional). A retrospective search was performed using a spatial vector descriptive model, supplemented by a manual search of selected articles. Forty-four literary sources were selected, of which 97.8% were from the last 10 years.

Evidence Synthesis

Electrical damage to the cardiovascular system is a rare but threatening complication of both high-voltage and low-voltage electric shock [7]. The degree of myocardial damage is influenced by factors such as pre-existing damage, the amount of electrical energy, current type (direct or alternating), duration of contact with the electrical source, and the path of electricity through the body [8]. Cardiac injuries caused by electric shock can be divided into arrhythmias, conduction disorders, and myocardial damage, regardless of whether it was a direct effect of the electric current, secondary damage due to hypotension and circulatory hypoxia, or coronary artery spasm [9].

Patients with a normal ECG were more frequently exposed to low voltage; however, victims of high-voltage trauma were hospitalized more often [10, 11].

A number of mechanisms of myocardial damage in electrical injury have been described. These include direct thermal damage, induction of coronary artery spasm, ischemia secondary to arrhythmic hypotension, acute hypertension due to chemoreceptor stimulation, catecholamine-mediated damage, and ischemia in the coronary artery basin as a component of generalized injury [4, 12].

Direct myocardial damage, although rare, can occur due to electrothermal injury (conversion of electrical energy to thermal energy), electroporation (formation of pores in cell membranes due to electric current), myocardial contusion due to the explosive force or secondary blunt trauma, which more closely resembles traumatic injury and is less similar to the ischemic type of damage leading to myocardial infarction. Myocardial contusion is the most common pathological cardiac finding, while myocardial infarction occurs more rarely [2, 13, 14]. In high-voltage electrical injury, irreversible electrothermal damage can occur [13]. Electrical injury can lead to damage of various parts of the heart tissue — the myocardium, cardiac valves, coronary arteries, and the conduction system [6, 15].

Myocardial tissue damage can be caused by high- and low-voltage electricity and occurs very quickly after the electrical injury [14, 16, 17]. Electrical injuries can also directly

damage cardiac myocytes [1, 16]. Electrical burns to the anterior chest wall can cause direct thermal damage to the myocardium [19].

The danger of electrical injuries compared to burns of other etiologies lies in the potential for hidden damage to internal tissues and organs, especially the heart [20]. Cardiac dysfunction can also occur due to the burn mechanism itself [8]. The average percentage of total body surface area burned was higher in patients with a normal ECG than in patients with arrhythmia and ischemia [14]. Ventricular fibrillation can cause cardiac arrest [21]. Arrhythmogenic ventricular cardiomyopathy is a common background for abnormal cardiac electrical impulse conduction, leading to irreversible myocardial damage [22].

The degree of external skin damage should not be used to determine the extent of internal injury, as the current flows through the body along various pathways depending on tissue resistance, contact surface area, and the volume of affected tissue [11]. Deep tissues can be seriously damaged by electric current, even if they appear superficially intact [23]. Domestic and foreign clinicians suggest that systemic damage in electrical injury with extensive burns has a milder course than without burns. This is explained by the coagulative necrosis of the skin, which limits the deep passage of electric current through the body [24, 25]. In a regression model, burn depth significantly correlated with arrhythmic and ischemic disorders ($p<0.04$) [14]. The pathogenesis of electrical injury can involve the development of acute cardiovascular failure, hemorrhagic pericarditis, and acute hypertension with peripheral vasospasm [26, 27].

The anterior location of the right coronary artery is recognized as making it more vulnerable to electrical damage [13]. Blood vessel walls through which current passed for a sufficiently long time can undergo necrosis [4, 12]. Progressive myocardial remodeling and ventricular dysfunction can also involve microvascular damage with impaired coronary microcirculation [28]. Vascular causes of myocardial infarction with vascular obliteration due to electrical exposure have been reported [29]. Due to its high water content, the vascular system is an excellent conductor. Damage is mostly associated with high-voltage electric shock, with predominant damage to small vessels, which are prone to medial necrosis with aneurysm formation and subsequent rupture [1]. Patients with electrical injury are prone to developing myocardial infarction, considering the damage caused by the current at the level of the vascular intima and the occurrence of thrombosis [30]. Sometimes, predominantly after high-voltage accidents, myocardial infarction can be caused by spasm of the coronary arteries or their occlusion by blood clots [31]. Acute tissue ischemia resulting from spasm of the smooth vascular muscles plays a significant role in the mechanism of these conditions [32, 33]. A fairly common complication that can lead to acute cardiac death is thrombosis of the coronary arteries with the formation of acute coronary insufficiency [13, 33]. If victims have pre-existing atherosclerotic vascular changes, the development of myocardial infarction with subsequent ventricular fibrillation is not excluded after electrical injury [34, 35].

Myocardial trauma can occur as a result of reduced blood supply (ischemia) or direct tissue death (necrosis) [2, 31]. Electrocardiographic results sometimes show signs of diffuse coronary ischemia. Electrical injury often correlates poorly with standard patterns of cardiac ischemia, as the electric current does not necessarily pass directly through the coronary vessels. However, ischemic signs in electrical injury most often characterize damage to the posterior myocardial wall.

Myocardial infarction resulting from accidental electric shock requires immediate attention and intervention [2]. This pathological condition, confirmed by cardiac enzyme studies, is a rare complication that mostly occurs immediately after high-voltage electric shock [3, 19].

Patients may experience thermal and electrical damage to the heart muscle, causing myocardial infarction, pericardial damage, and heart failure. ST-segment elevation myocardial infarction is well-described in the literature, often accompanied by normal coronary angiography results and is likely caused by coronary artery vasospasm [7].

Myocardial necrosis is among the most common cardiac complications caused by electric shock [8, 36]. Electricity can lead to focal or diffuse heart damage and often causes necrosis involving the myocardium, nodal tissues, conduction pathways, and coronary arteries [8]. Myocardial necrosis and changes in myocyte membrane permeability can lead to the formation of an arrhythmogenic focus [6, 16]. Rare cardiac complications include myocardial perforation/rupture due to coagulative necrosis [13, 14, 37]. Such a defect can sometimes be closed during cardiopulmonary bypass [19].

The histological picture of the heart in fatal electrical injury is characterized by diffuse focal necrosis of the myocardium (including specialized conductive tissue), necrosis of smooth muscle cells in the tunica media of the coronary arteries, and cytological changes of the nucleus. The bundle of His with its branches can also be affected. The nerve structures of the heart are mostly minimally damaged [22]. Myocardial cell membranes can be disrupted by electrical injury, leading to cellular edema, changes in electrical conductivity, and subsequent fibrotic transformations [38].

Histological examination of the myocardium reveals fragmentation of cardiomyocytes, loss of cross-striations, and foci of necrosis [31]. The literature describes a wide spectrum of cardiac changes in cases of electric shock, including cardiomyolysis, hemorrhagic areas, separation of myofibrils, and alternating hypercontracted-hypoextended myocytes [14].

A clear demarcation between damaged and healthy myocardium is most often observed along with the presence of erythrocytes in the interstitium and myoglobin denaturation detected in distal renal tubules (an indirect peripheral sign). Rupture of myocardial fibers with square nuclei may be observed as a possible diagnostic sign of malignant arrhythmia followed by circulatory arrest due to ventricular fibrillation, especially for distinguishing damage caused by high- and low-voltage electric current. In larger studies, the only differential finding was interstitial hemorrhagic infiltration of the myocardium. Myofibril rupture includes several histopathological patterns, such as bundles of stretched myocardial cells alternating with hypercontracted cells with expansion or segmentation of intercalated discs, square nuclei (rather than ovoid morphology), and non-eosinophilic bands of hypercontracted sarcomeres. These should not be considered artifacts secondary to histological processing, as they can also occur in sudden deaths caused by cardiac arrhythmias. However, all the described changes cannot be attributed exclusively to electric shock, as no specific electrically induced morphological cardiac signs have been definitively identified to date [14, 22, 38]. Histological findings in the myocardium in fatal electric shock must be carefully considered, as other pathological conditions can demonstrate similar defects.

Thickening and fragmentation of cardiomyocytes with anisonucleosis, myofibril rupture, or square nuclei in hypercontracted myocytes are also common markers of ventricular fibrillation and hypertrophy. Interstitial hemorrhagic infiltration and necro-

as can result from direct electrical injury and in cases of hypoxic damage. When electrical injury affects the cardiac conduction system, fatal cardiac arrhythmia often prevents the identification of specific histopathological aspects, especially when electrical marks are absent. In such cases, determining the cause of sudden death can be difficult [22].

Diagnosing myocardial damage in electrical injury is often quite challenging [13]. Sometimes, with a moderately satisfactory condition of the victim, disorders of cardiac function arise, manifested by muffled heart sounds, the appearance of a systolic murmur, weakened pulse, tachycardia, extrasystole, intraventricular blocks, and hypertension. Sometimes victims complain of pain in the heart area (anginal pain) [14].

Cardiac symptoms, combining functional and morphological damage to the cardiovascular system, were present in 9% of victims during the first consultation for low-voltage injuries. These signs primarily manifested as temporary tachycardia and chest pain, which usually resolved within a few hours [16]. In the vast majority of cases, symptoms occur immediately after the accident, and only in rare cases are delayed manifestations observed. In some victims, electrical injury progresses to permanent scarring with late cardiac disorders in the form of arrhythmias [17].

The ECG upon hospitalization is the most prognostic element for cardiac complications [1]. The initial ECG is recommended for all patients if it was not performed prior to hospital admission. Diagnosing myocardial injury can be challenging because chest pain is often absent, and the damage may manifest only with non-specific ECG changes, such as ST-segment and T-wave abnormalities or tachycardia [2], interval prolongation, as well as other abnormal non-specific ECG changes, which often persist for several weeks after the injury. ST-segment and T-wave changes usually resolve without specific treatment [13, 19, 21]. On ECG, ST-segment abnormalities were the most common pathological finding (7.3%) in cardiac electrical injuries [10].

In cases of coronary artery damage or spasm, classic symptoms or ECG signs of myocardial infarction can be observed, including ST-segment elevation or the appearance of pathological Q waves [9, 16, 27, 31]. ST-segment changes and long QT syndrome can be considered potentially significant; however, the prognosis associated with them and electrical injuries is not well defined [17]. QT interval abnormalities are easily underestimated, so re-evaluation of the ECG is advisable [28]. In non-fatal electrical injuries, regardless of the current path, transient coronary insufficiency (formerly called intramyocardial contraction) is sometimes detected electrocardiographically [34].

Cardiac MRI should be used in acute cases to assess the extent of myocardial damage during the observation period to predict the risk of recurrent arrhythmias [21, 37]. Myocardial tissue damage can be caused by the direct effect of alternating current, coronary spasm, or thrombosis, which can be detected using imaging methods such as cardiac MRI. Attention is drawn to the progressive dysfunction of the left/right ventricle. The absence of myocardial edema on MRI suggests that the damage is not a result of persistent inflammation, but primarily of fibrotic remodeling, which is a consequence of the initial electrical injury [28].

Hemodynamic monitoring allows for direct measurement of intravascular volume and hemodynamic parameters. Many of these observations are continuous, enabling constant assessment of the effectiveness of therapeutic measures. Intravascular volume monitoring (central venous pressure, stroke volume variations) is combined with indicators of cardiovascular performance (pulse contour cardiac output, echocardiography, and thermodilution) to provide a continuous assessment of intravascular volume and cardiac activity [40].

According to current European Resuscitation Council (ERC) guidelines, ECG monitoring is recommended in electrical injury for patients with diagnosed cardiorespiratory injury or one several of the following risk factors: loss of consciousness, initial cardiac arrest, soft tissue damage and burns, an ECG abnormalities at the time of hospitalization [13].

Currently, a 24-hour observation period under continuous cardiac monitoring is proposed to prevent late cardiac complications, even for asymptomatic patients without prior predisposing conditions [16]. Patients should be monitored for more than 24 hours when suspicion of myocardial injury is high, as in cases of syncope, high-voltage exposure, transthoracic current path, or presence of ECG abnormalities. Physicians should pay attention to the current path and the presence of noticeable burns [13, 37]. Additional indications for prolonged monitoring include a history of cardiac disease, active chest pain, documented loss of consciousness, documented arrhythmia in the field or in the emergency department, any abnormalities on the initial ECG, post-resuscitation state, elevated troponin levels, and other signs of cardiac dysfunction (e.g., electrically induced rhabdomyopathy) [13].

The role of serum troponin measurement for assessing cardiac damage is uncertain and should be discussed with a cardiologist. Some experts believe that troponin concentration and echocardiography can detect myocardial damage after electric shock [38]. However, other researchers claim that troponin is not useful for predicting left ventricular dysfunction [13]. Although sensitive cardiac troponin I is a much more sensitive cardiac biomarker than creatine phosphokinase, creatine phosphokinase-MB, creatinine, and free myoglobin, it usually does not elevate after electrical injury. An increase in troponin level is observed only in some rare cases of myocardial ischemia and hypoxia, and for diagnosing electrical cardiac injury, it mostly lacks fundamental clinical significance [30, 41]. Nevertheless, most authors agree on the necessity of investigating cardiac enzymes and initiating heparin therapy to minimize these thromboembolic risks [39, 41].

A victim of electrical injury, even if feeling satisfactory, should not be left without supervision and is subject to hospitalization for at least three days, as they belong to the category of severely ill patients [34, 42]. In cases of transthoracic current path and high-voltage, prolonged current exposure, or abnormal skin conditions (loss of integrity, contamination with conductive substances, significant moisture), it is advisable to extend the victim's stay in the ICU [43]. Long-term outpatient follow-up may be useful for determining the clinical spectrum of signs of electrical myocardial damage. The average hospitalization time for patients with electrically induced ischemic ECG changes should be longer than in other cases [14].

Conclusions

1. Electrical damage to the cardiovascular system is a relatively rare occurrence but is accompanied by serious life-threatening complications.

2. Remote cardiac complications cannot be excluded after electric shocks.

3. Cardiac monitoring in victims of electric shock should be performed for at least 24 hours.

4. Organic myocardial injuries in electrical trauma are currently insufficiently studied and require further research.

Conflicts of interests

Authors declare the absence of any conflicts of interests and own financial interest that might be construed to influence the results or interpretation of the manuscript.

Authors' contributions

Ivanenko O.V. — conceptualization, original draft;
Yakhnitsa V.O. — data analysis and interpretation, writing the article;
Sadomirov V.A., Stasiuk D.M. — review & editing;
Makhnik I.Z. — translation.

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Органічні ураження серця при електротравмі

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Анотація. Електричні травми є однією з суттєвих проблем охорони здоров'я, яка часто пов'язана з високою захворюваністю та смертністю. При електричній травмі можуть спостерігатися морфологічні ушкодження міокарда, які відрізняють у 3,2% потерпілих. **Мета дослідження.** Літературні джерела були включені до дослідження, якщо вони: 1) опубліковані українською, англійською або німецькою мовами; 2) містили інформацію про «ядерні» ознаки, що пов'язані з електричною травмою; 3) інформували про поширеність специфічних органічних уражень міокарда при електричній травмі. **Висновки.** Незважаючи обговорюваний дизайн (експертний або перевірений). **Ключові слова:** Перші описані випадки виявлення ушкодження міокарда при електротравмі. До них належать прямі первинні ушкодження, індуковані струмом коронарних артерій, вторинна до аритмічної гіпотензії ішемія, гострі гіпертензивні епізоди спазмуючі міокардіоцитів, опосередковані катехоламінами ушкодження та ішемія в басейні коронарних артерій як складова генералізованого ушкодження. Ступінь клінічного ушкодження серця не прямо пропорційно залежить від опору тканин, площі контактної поверхні та об'єму уражених тканин. Пацієнти з електричною травмою схильні до розвитку інфаркту міокарда, навіть якщо ураження, що спричинюються струмом на рівні типових стінок судин, та виникнення тромбозу. Інфаркт міокарда може бути спричинений спазмом коронарних артерій або їх оторацією вторинним тромбом. Великі зміни в механізмі розвитку цих станів має пастка тканин у результаті спазму гладких судинних м'язів. Електрична травма може призвести до вогнищевого або дифузного ураження серця і часто спричиняє напруг із залученням міокарда, ішемією тканин, прогресуючої коронарною аритмії. Наразі затверджується 24-годинний період спостереження після безперервного кардіологічного моніторингу для безсимптомних пацієнтів без попередніх серцевих дефектів. Зростання рівня тропоніну для діагностики електричного кардіоміопатичного ураження здебільшого не має принципового клінічного значення. **Висновки.** Поширеними електричними травмами судинної системи супроводжується сприятливим життєвим наслідком і ускладнення не виключаються в подальші. Кардіомоніторинг у постраждалих від впливу електричного струму тривання здійснювати протягом 24 год.

Ключові слова: електричні травми, органічні ушкодження міокарда, інфаркт міокарда, некроз міокарда, кардіоміопатія.

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