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ORIGINAL PAPER

Arrhythmias and laboratory abnormalities after an electrical accident: a single-center, retrospective study of 333 cases

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Abstract

Background Even though electrical injuries are common in the emergency room, guidelines, consensus, and general recommendations for the management of these patients do not exist in Europe. Documented cases of delayed arrhythmias are rare and their connection with electrical injury has not been fully confirmed. We also use cardio-specific markers for the risk stratification of myocardial injury, but there is no significant study referring to their utility in this clinical situation. These reasons led us to retrospectively analyze all cases of electrical injuries over 23 years to determine the prevalence of cardiac arrhythmias (mainly malignant arrhythmias and delayed arrhythmias).

Methods We retrospectively searched all patients admitted to the University Hospital in Pilsen, CZ, with a diagnosis of electric injury (ICD diagnostic code T754) from 1997 to 2020. The hospital´s information system was used to research the injury; data were drawn from patient medical records.

Results We identified 333 cases of electrical injury in our hospital. Men accounted for about two-thirds, and women one-third. Children accounted for about one-third of cases. Most were low-voltage injuries (< 1000 V, 91.6%). All participants had an initial ECG, and 77.5% of patients had continuous ECG monitoring, usually lasting 24 h. Cardiac arrhythmias were noticed in 39 patients (11.7%). The most frequent arrhythmias were: ventricular fibrillation, sinus tachycardia, bradycardia and arrhythmia, atrial fibrillation, and supraventricular tachycardia. The ECG showed cardiac conduction abnormalities in 28 patients (8.1%), and ten patients (3%) had supraventricular or ventricular extrasystoles. In ten cases (3%), we found changes in ST segments and T waves on the initial ECG. Thirty-one patients (9.3%) suffered a loss of consciousness and 50 patients (15.02%) reported paresthesia. The most frequent ion disbalances were hypokalemia (18%) and hypocalcemia (3.3%). Patients with an ion disbalance had significantly more arrhythmias and newly diagnosed cardiac conduction abnormalities. Troponin levels (cTnI or hs-cTnT) were measured in 258 cases (77.48%) and found to be elevated above the 99th percentile in 19 cases (5.7%). Almost one-third of patients had burns of various degrees of seriousness, and 41 patients (12.3%) had concomitant traumatic injuries. Eleven patients underwent pre-hospital resuscitation, three died in the hospital, and another died as result of intracranial hemorrhage.

Conclusion All malignant arrhythmias occurred immediately after the electrical injury, delayed life-threatening arrhythmias were not observed, and no predictive factors of malignant arrhythmias were found. While elevations of cardiac troponins were observed sporadically, they did not appear helpful for risk stratification. In patients with arrhythmias, ion disbalance may be more critical. We concluded that asymptomatic, uninjured adult and pediatric patients with normal initial ECG findings do

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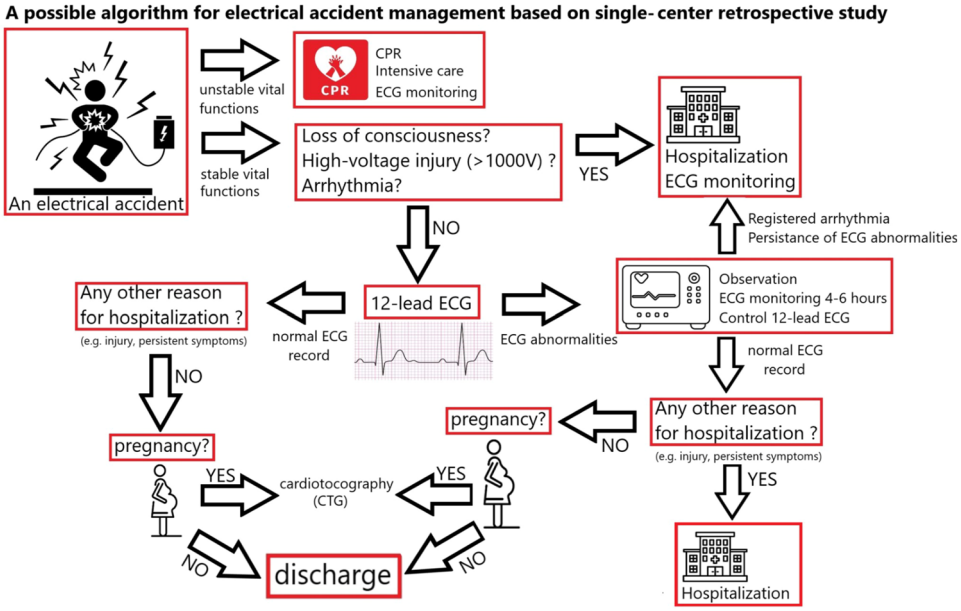
not need continuous ECG monitoring and may be discharged home. Recommendations for high-risk patients and patients with mild ECG abnormalities at admission are less obvious.

Graphical abstract



Keywords Electrical accident · Arrhythmia · Cardiac monitoring · Troponins · Ion disbalance

Introduction

Electrical injuries are very heterogeneous, ranging from minor skin burns to life-threatening injuries to internal organs. [1] The severity of injuries depends on the intensity of the electrical current (determined by the voltage of the source and resistance of the skin), type of current (direct or alternating), the pathway of the electrical current through the body, and duration of contact with the source of current. [2, 3]

Ventricular fibrillation is the most common cause of immediate death after an electrical injury. It can happen when the current reaches the heart within its vulnerable period. [3, 4] Another cause of death is asystole or respiratory arrest (secondary to paralysis of the central respiratory control system or due to paralysis of the respiratory muscles). [2] We can also observe non-lethal arrhythmias (e.g., sinus tachycardia, sinus bradycardia), isolated premature atrial or ventricular beats, bundle branch blocks, sinoatrial blocks, and various degrees of atrioventricular blocks after the electrical accident. [3]

The incidence of delayed malignant arrhythmias is extremely rare. Few documented cases can be found in the medical literature, but only two were reported with initial ECGs. [6, 7] However, fear of delayed arrhythmias is still a reason why patients are admitted to monitoring units, even if they have no risk factors, symptoms, or ECG abnormalities.

We analyzed data from all patients examined in the emergency room after accidental electrocution (AE) to determine the prevalence of cardiac arrhythmias (mainly malignant arrhythmias and delayed arrhythmias) and ECG abnormalities. Furthermore, we searched for significant laboratory abnormalities.

Subjects and methods

Study design and population

We retrospectively searched for all patients admitted to the University Hospital Pilsen, CZ, between 1997 and 2020, with ICD diagnostic code T754 (effects of electric current). We used the hospital information system to research and extract required data from patient's medical records. We did not apply any age limitations. We focused on these patient characteristics: age and sex of the patients, circumstances of the AE, type of current, source voltage, medical history and presenting symptoms (e.g., loss of consciousness, tingling or numbness of extremities, pain in the extremities, chest pain, palpitations, shortness of breath, dizziness, and others).

We looked for cardiac arrest, arrhythmias, ECG abnormalities, loss of consciousness, burns (and their severity), concomitant traumatic injuries, rhabdomyolysis, acute kidney injury, and other consequences of electrical injury. We also examined laboratory results, i.e., serum sodium, potassium, magnesium, calcium, phosphate, creatinine, cardiac troponin (cTnI or hs-cTnT), creatine kinase (CK), and myoglobin. Before 2015, cardiac Troponin I (cTnI) was used as a cardiospecific marker at the University Hospital Pilsen. Starting in 2015, this laboratory marker was replaced by high-sensitive cardiac Troponin T (hs-cTnT). This is why some subjects had data for cTnI and others for hs-cTnT.

Statistical analysis

All relevant data were recorded and edited in Microsoft Excel 2010. MedCalc software, version 19.4.6. (MedCalc, Ostend, Belgium) was used for statistical analysis. The frequencies of categorical variables are provided as absolute numbers and percentages, while continuous variables are presented by means, standard deviations or medians. The difference between the categorical variables was tested using the Chi-square test. The significance level (α) was set at 0.05.

Cardiac troponin I from samples between 2001 and 2015 was measured using an Abbott AxSym and Beckmann Coulter AccuTnI contemporary assay, and cardiac troponin T from samples between 2015 and 2020 was determined using a high-sensitive assay (fifth generation) on a Roche Cobas e602 analyzer.

Serum potassium levels < 3.8 or > 5.2 mmol/l were considered hypokalemia and hyperkalemia, respectively; serum calcium levels < 2.2 or > 2.6 mmol/l were considered hypocalcemia and hypercalcemia, respectively; serum magnesium levels < 0.8 or > 1.1 mmol/l were considered hypomagnesemia and hypermagnesemia, respectively. A normal myoglobin level was set < 86 ug/l for male and < 68 ug/l for female. A normal level of CK was set 0.3–3.3 ukat/l for male and 0.3–3.3 ukat/l for female.

Results

Patient characteristics

During the study period (23 years), 333 patients were admitted to the emergency room with the ICD T75.4 diagnosis (effects of electric current). Two-thirds were adults (155/74 males/females). The mean adult age was 34.84 years (range 19–85). There were 104 children (31.23%) in our sample, most were 1–5 years or 10–15 years. The main characteristics and medical history of patients are presented in Table 1.

Low-voltage injuries were by far the most common (< 1000 V, $n = 305$; 91.59%). Only 16 patients (4.80%) had high-voltage injuries. In 12 cases (3.60%), the source voltage was unknown. Direct current caused six injuries (1.80%), and lightning was suspected in one case. Forty-nine cases (14.71%) occurred at the workplace, Table 2.

Less than half of patients ($n = 143$; 42.94%) presented with symptoms at admission. The most common complaints were tingling and pain in the extremities, chest pain or pressure, palpitations, dizziness, headache, and weakness. All complaints are presented in Table 3.

Cardiopulmonary resuscitation (CPR)

Eleven patients (3.30%) had to be resuscitated immediately after AE. In 8 cases (2.40%), ventricular fibrillation was the cause of the cardiac arrest, and these patients were defibrillated with subsequent restoration of circulation. In one patient, defibrillation was followed by asystole required the use of a mechanical CPR device (LUCAS), after which blood circulation was restored. In two cases, CPR was performed by a bystander; on the first ECG recorded by EMTs, the injured were in sinus rhythm, and no other resuscitation was needed. In the last case, CPR was also performed by a bystander; on arrival, EMTs found the patient in sinus rhythm; however, the patient was dyspneic and had to be intubated and ventilated.

Four patients requiring CPR were associated with a high-voltage injury, and one was associated with a low-voltage injury. In the remaining cases requiring CPR, source voltage information was missing. Based on case circumstances, three appeared to be low-voltage sources. In summary, there were four high-voltage injuries, six low-voltage injuries, and one was unknown.

Three resuscitated patients died during hospitalization (one died on day 19, the second on day 5, and the third on day 74). The cause of death was post-hypoxic encephalopathy and brain edema. In all cases, patients were male and had likely suffered low-voltage injuries.

Arrhythmias and cardiac electrical injuries

Thirty-nine cases of cardiac arrhythmias/dysrhythmias, 10 cases of premature complexes, and 28 cases of cardiac conduction disorders were noticed. The most alarming were the 8 cases of ventricular fibrillation (Table 4). However, in one case, uncertainties remain about the cause of ventricular fibrillation. The patient was a 58-year-old man who suffered cardiac arrest while repairing a washing machine, but pathological Q and ST elevations were present on an initial ECG (the inferolateral location). According to echocardiography, left ventricular akinesia was present in the corresponding areas. Additionally, coronary angiography

Table 1 Characteristics of population

	All patients ($n=333$)	Adult patients ($n=229$)	Pediatric patients ($n=104$)
Age (years, mean ± SD)	25.96 ± 17.61	34.87 ± 13.5	6.33 ± 5.55
Age groups			
15–18 years	–	–	13 (12.50%)
10–15 years	–	–	22 (21.15%)
5–10 years	–	–	8 (7.69%)
1–5 years	–	–	58 (55.77%)
< 1 year	–	–	3 (2.88%)
Male/female	222/111 (66.67/33.33%)	155/74 (67.69/32.31%)	67/37 (64.42/35.58%)
Actual pregnancy	5 (1.50%)	5 (1.50%)	–
Medical history			
Structural heart disease	9 (2.70%)	6 (2.62%)	3 (2.88%)
Coronary heart disease	6 (1.80%)	6 (2.62%)	–
Heart failure	3 (0.90%)	3 (1.31%)	–
Cardiac surgery	3 (0.90%)	2 (0.87%)	1 (0.96%)
Arrhythmias	11 (3.30%)	10 (4.37%)	1 (0.96%)
Bundle branch blocks	8 (2.40%)	6 (2.62%)	2 (1.92%)
Atrioventricular blocks	3 (0.90%)	3 (1.31%)	–
History of palpitations	3 (0.90%)	3 (1.31%)	–
Pacemakers, ICDs	5 (1.50%)	5 (2.18%)	–
Arterial hypertension	28 (8.41%)	28 (12.23%)	–
Diabetes mellitus	7 (2.10%)	7 (3.06%)	–
Hyperlipidemia	29 (8.71%)	29 (12.66%)	–
Kidney disease	6 (1.80%)	3 (1.31%)	3 (2.88%)
Bronchial asthma/COPD	13 (3.90%)	10 (4.37%)	3 (2.88%)
Thyroid gland disease	8 (2.40%)	7 (3.06%)	1 (0.96%)
Malignancy	5 (1.50%)	5 (2.18%)	–
Stroke	1 (0.30%)	1 (0.44%)	–

SD standard deviation, *ICDs* implantable cardioverter-defibrillator, *COPD* chronic obstructive pulmonary disease

Table 2 Descriptions of electrical injuries

	All patients ($n=333$)	Adult patients ($n=229$)	Pediatric patients ($n=104$)
Voltage of the source			
Small (< 50 V)	2 (0.60%)	1 (0.44%)	1 (0.96%)
Low (50–1000 V)	303 (90.99%)	203 (88.65%)	100 (96.15%)
High (1000–52 000 V)	16 (4.80%)	13 (5.68%)	3 (2.88%)
Unknown	12 (3.60%)	12 (5.24%)	–
Lightning	1 (0.30%)	1 (0.44%)	–
Direct current	6 (1.80%)	6 (2.62%)	–
Suicide attempt	2 (0.60%)	2 (0.87%)	–
Workplace accident	49 (14.71%)	48 (20.96%)	1 (0.96%)

found multi-vessel disease, but no culprit lesion was found. Dynamic elevation of cardio-specific markers can be explained by acute coronary syndrome and previous resuscitation with a myocardial contusion. In the end, electricity-related marks on the right hand indicate an electrical injury. This was probably a coincidence between a subacute myocardial infarction and the AE.

Other observed arrhythmias were sinus tachycardia, sinus bradycardia, sinus arrhythmia, atrial fibrillation,

Table 3 Complaints at admission

	All patients ($n=333$)	Adult patients ($n=229$)	Pediatric patients ($n=104$)
Tingling in the extremities	50 (15.02%)	40 (17.47%)	10 (9.62%)
Chest pain/pressure	33 (9.91%)	27 (11.79%)	6 (5.77%)
Pain/burning in the extremities	23 (6.91%)	17 (7.42%)	6 (5.77%)
Palpitations	19 (5.71%)	19 (8.30%)	–
Dizziness	13 (3.90%)	11 (4.80%)	2 (1.92%)
Amnesia	13 (3.90%)	12 (5.24%)	1 (0.96%)
Headache	11 (3.30%)	7 (3.06%)	4 (3.85%)
Weakness, fatigue, faintness	10 (3.00%)	8 (3.49%)	2 (1.92%)
Nauseous	8 (2.40%)	5 (2.18%)	3 (2.88%)
Muscle cramp	5 (1.50%)	5 (2.18%)	–
Vomiting	4 (1.20%)	3 (1.31%)	1 (0.96%)
Dyspnea/shortness of breath	4 (1.20%)	2 (0.87%)	2 (1.92%)
Abdominal pain	3 (0.90%)	2 (0.87%)	1 (0.96%)
Cyanosis	2 (0.60%)	2 (0.87%)	–
Backache	2 (0.60%)	2 (0.87%)	–
Blurred vision	2 (0.60%)	1 (0.44%)	1 (0.96%)
Muscle twitching	2 (0.60%)	1 (0.44%)	1 (0.96%)
Weakness of the extremities	2 (0.60%)	–	2 (1.92%)
Immobility of the extremities	1 (0.30%)	–	1 (0.96%)
Numbness of the extremities	1 (0.30%)	–	1 (0.96%)
Speech disorder	1 (0.30%)	1 (0.44%)	–
Confusion	1 (0.30%)	1 (0.44%)	–

Table 4 Malignant arrhythmias

Age	Sex	Voltage	Year	Medical history	Trauma	Burns	Death
16	M	Low	2002	–	No	No	Yes
24	M	Unknown	2004	–	No	No	Yes
39	M	High	2000	paroxysmal atrial fibrillation, tick-borne encephalitis at age 38	Yes	3°	No
43	F	Low	2002	–	No	No	No
44	M	High	2005	rib fractures at age 19	No	3°	No
47	M	Low	2011	–	No	3°	No
50	M	Low	2012	–	No	No	No
58	M	Low	2013	concomitant subacute MI	No	No	Yes

M male, *F* female, *MI* myocardial infarction

supraventricular tachycardia that were terminated by verapamil, and an unspecified tachycardia terminated by amiodarone. In ten cases we found changes in the ST segment and T wave on the initial ECG. One patient with atrial fibrillation (of unknown duration) showed signs of heart failure at the time of admission. Echocardiography confirmed left ventricular systolic dysfunction (LVEF 15–20%). In four other patients, mild left ventricular systolic dysfunction (LVEF 40–50%) was also newly diagnosed. (Table 5).

Electrical injury

Altogether, 101 patients (30.33%) suffered burns of various degrees (second-degree burns were the most common). Concomitant traumatic injuries were found in 12.31% of cases. The most common injuries were fractures, traumatic brain injury and lacerated/incised wounds. We observed rhabdomyolysis in 5 severe cases and acute kidney injury in 4 cases.

Patients also showed neurological abnormalities (e.g., paresthesia, hyp/anesthesia muscle convulsion/spasm/fasciculation, autonomic dysfunction or speech disorder) and

Table 5 Arrhythmias and cardiac electrical injury

	All patients ($n=333$)	Adult patients ($n=229$)	Pediatric patients ($n=104$)
Arrhythmias/dysrhythmias	39 (11.71%)	36 (15.72%)	3 (2.88%)
Ventricular fibrillation	8 (2.40%)	7 (3.06%)	1 (0.96%)
Sinus tachycardia	14 (4.20%)	14 (6.11%)	–
Sinus bradycardia	11 (3.30%)	9 (3.93%)	2 (1.92%)
Sinus arrhythmia	2 (0.60%)	2 (0.87%)	–
Atrial fibrillation	4 (1.20%)	4 (1.75%)	–
SVT	1 (0.30%)	1 (0.44%)	–
Unspecified tachycardia	1 (0.30%)	1 (0.44%)	–
Premature complexes	10 (3.00%)	8 (3.49%)	2 (1.92%)
Supraventricular	4 (1.20%)	4 (1.75%)	–
Ventricular	5 (1.50%)	4 (1.75%)	1 (0.96%)
Unknown	1 (0.30%)	–	1 (0.96%)
Cardiac conduction abnormalities	26 (7.81%)	26 (11.35%)	–
LAH	2 (0.60%)	2 (0.87%)	–
IRBBB/ intermit. iRBBB	21 (6.31%)/2(0.60%)	21 (9.17%)/2 (0.87%)	normal finding
RBBB/ intermit. RBBB	1 (0.30%)/1(0.30%)	1 (0.44%)/1 (0.44%)	–
SAB (second degree)	1 (0.30%)	1 (0.44%)	–
AVB (first degree)	1 (0.30%)	1 (0.44%)	–
ECG changes of ST/T	10 (3.00%)	9 (3.93%)	1 (0.96%)
ST elevation	2 (0.60%)	1 (0.44%)	1 (0.96%)
ST depression	5 (1.50%)	5 (2.18%)	–
Negative T wave	3 (0.90%)	3 (1.31%)	normal finding
Cardiac decompensation	1 (0.30%)	1 (0.44%)	–
Newly dg. systolic dysfunction	5 (1.50%)	3 (1.31%)	2 (1.92%)

SVT supraventricular tachycardia, *iRBBB* incomplete right bundle branch block, *RBBB* complete right bundle branch block, *intermit.* intermittent, *SAB* sinoatrial block, *AVB* atrioventricular block, *dg.* diagnosed

31 patients reported a loss of consciousness. Three patients entered a vegetative state as a consequence of post-hypoxic encephalopathy or intracranial hemorrhage. In one case, the patient's neurological condition improved, and he returned home. Two other patients died within a year. (Table 6).

Clinical course and cardiac monitoring

Three hundred and thirty-three patients were examined in the emergency room after the AE, and all of them had an initial ECG. Five children were discharged home, two patients signed out against medical advice, one patient was hospitalized in another hospital, and the remaining 325 patients were admitted to various hospital departments. All pregnant women underwent cardiotocography (CTG) with normal findings.

Patients treated by EMTs at the scene of the accident had a 3-lead ECG record immediately and afterwards they had a 12-lead ECG record at the emergency room. Patients who came to the hospital themselves had a 12-lead ECG record at the emergency room. Afterwards, the patients were connected to a telemetry ECG monitoring system that records a 3-lead ECG. In some cases (especially incidents from earlier years), patients had repeated 12-lead ECGs during hospitalization.

The average length of hospital stays was 3.53 days, but this number is heavily influenced by long-term hospitalizations (patients with cardiac arrest, severe burns, or intracranial injuries). If we exclude hospitalizations longer than 30 days, the average hospital stay was 1.55 days. Ultimately, 316 patients were discharged home, and 13 were transferred to other hospitals (e.g., Prague Burn Center, intensive care units, and long-term treatment centers). The 30-day mortality was 0.60% and the 1-year mortality was 1.20%. (Table 7).

Biochemical analysis

Laboratory values were available for almost all patients. The average creatinine level was 91 µmol/l in adults and 52 µmol/l in children. Acute kidney injury was diagnosed in 4 cases (1.20%). Ion disbalance was found in 67 patients (20.12%), with hypokalemia being the most common ($n=60$; 18.02%). The frequencies of other ion abnormalities are shown in Table 8.

Table 6 Consequences of electrocution (except arrhythmias and cardiac electrical injuries)

	All patients ($n=333$)	Adult population ($n=229$)	Pediatric population ($n=104$)
Death	4 (1.20%)	3 (1.31%)	1 (0.96%)
Death due to electrocution	3 (0.90%)	2 (0.87%)	1 (0.96%)
Cardiopulmonary resuscitation	11 (3.30%)	10 (4.37%)	1 (0.96%)
Respiratory arrest/disorder	2 (0.60%)	1 (0.44%)	1 (0.96%)
Loss of consciousness	31 (9.31%)	26 (11.35%)	5 (4.81%)
Skin burns	101 (30.33%)	49 (21.40%)	52 (50.00%)
1st degree	11 (3.30%)	4 (1.75%)	7 (6.73%)
1st–2nd degree	6 (1.80%)	3 (1.31%)	3 (2.88%)
2nd degree	46 (13.81%)	16 (6.99%)	30 (28.85%)
2nd–3rd degree	6 (1.80%)	2 (0.87%)	4 (3.85%)
3rd degree	20 (6.01%)	14 (6.11%)	6 (5.77%)
4th degree	1 (0.30%)	1 (0.44%)	–
Unknown	8 (2.40%)	6 (2.62%)	2 (1.92%)
Small electricity-related marks	50 (15.02%)	27 (11.79%)	23 (22.12%)
Corneal burns	2 (0.60%)	2 (0.87%)	–
Respiratory tract burns	1 (0.30%)	1 (0.44%)	–
Concomitant traumatic injuries	41 (12.31%)	28 (12.23%)	13 (12.50%)
Fractures	14 (4.20%)	10 (4.37%)	4 (3.85%)
(Sub)luxations	3 (0.90%)	2 (0.87%)	1 (0.96%)
Lacerated/incised wounds	6 (1.80%)	5 (2.18%)	1 (0.96%)
Traumatic brain injury	11 (3.30%)	7 (3.06%)	4 (3.85%)
Lung contusion	4 (1.20%)	2 (0.87%)	2 (1.92%)
Pneumothorax	1 (0.30%)	1 (0.44%)	–
Kidney fissure	1 (0.30%)	1 (0.44%)	–
Spleen damage	1 (0.30%)	–	1 (0.96%)
Rhabdomyolysis	5 (1.50%)	4 (1.75%)	1 (0.96%)
Acute kidney injury	4 (1.20%)	3 (1.31%)	1 (0.96%)
Neurological abnormalities			
Paresthesia	50 (15.02%)	40 (17.47%)	10 (9.62%)
Hypoesthesia/anesthesia	2 (0.60%)	1 (0.44%)	1 (0.96%)
Convulsion/spasm/fasciculation	9 (2.70%)	8 (3.49%)	1 (0.96%)
Autonomic dysfunction	1 (0.30%)	–	1 (0.96%)
Speech disorder	1 (0.30%)	1 (0.44%)	–
Post-hypoxic encephalopathy Brain edema	3 (0.90%)	2 (0.87%)	1 (0.96%)
Vegetative state	3 (0.90%)	2 (0.87%)	1 (0.96%)

The level of creatine kinase (CK) was determined in 252 patients (75.68%) and was elevated in 106 cases (31.83%). The average level of CK was 5.6 ukat/l. Myoglobin level was measured in 233 patients (69.97%) and was elevated in 95 cases (28.53%). The average level of myoglobin was 316.64 ug/l. Cardiac troponin was measured in 258 cases (77.48%; cTnI in 171 cases and hs-cTnT in 87 cases), it was measured once in 184 patients, twice in 45 patients, and three or more times in 19 cases. Increased troponin levels above the 99th percentile were detected in 19 cases (5.71%; cTnI in 16 cases and hs-cTnT in 3 cases). The average level of TnI was 0.02 ug/l and the average level of TnT was 8.13 ng/l (in the first measurement).

We registered that 27.1% patients with ion disbalance and only 7.6% patients without ion disbalance had arrhythmia after AE (Fig. 1). This means that patients with ion disbalance (most often hypokalemia) had significantly more arrhythmias (significance level $P<0.0001$). The same situation applies to relationship between the incidence of ion disbalance and newly diagnosed cardiac conduction abnormalities (significance level $P<0.0232$). Newly diagnosed cardiac conduction abnormalities were

Table 7 Clinical course and cardiac monitoring

	All patients ($n=333$)	Adult population ($n=229$)	Pediatric population ($n=104$)
Hospitalization	326 (97.90%)	227 (99.13%)	99 (95.19%)
Refusal of hospitalization	4 (1.20%)	4 (1.75%)	–
Continuous ECG monitoring	258 (77.48%)	196 (85.59%)	62 (59.62%)
Duration of hospitalization (days. mean)	3.53	3.74	2.95
Without those longer than 30 days	1.55	1.64	1.34
Without those longer than 7 days	1.14	1.10	1.21
Discharge	310 (93.09%)	215 (93.89%)	95 (91.35%)
Transfer to another department	16 (4.80%)	14 (6.11%)	2 (1.92%)
Transfer to another hospital	13 (3.90%)	10 (4.37%)	3 (2.88%)
30-day mortality	2 (0.60%)	1 (0.44%)	1 (0.96%)
1-year mortality	4 (1.20%)	3 (1.31%)	1 (0.96%)

Table 8 Ion disbalances

	All patients ($n=333$)	Adult population ($n=229$)	Pediatric population ($n=104$)
Total	67 (20.12%)	58 (25.33%)	9 (8.65%)
Hypokalemia	60 (18.02%)	55 (24.02%)	5 (4.81%)
Hypocalcemia	11 (3.30%)	8 (3.49%)	3 (2.88%)
Hypomagnesaemia	8 (2.40%)	6 (2.62%)	2 (1.92%)
Hypophosphatemia	5 (1.50%)	5 (2.18%)	–
Hyponatremia	4 (1.20%)	4 (1.75%)	–
Hyperkalemia	4 (1.20%)	3 (1.31%)	1 (0.96%)
Hypercalcemia	3 (0.90%)	2 (0.87%)	1 (0.96%)
Hypermagnesemia	1 (0.30%)	1 (0.44%)	–
Hyperphosphatemia	1 (0.30%)	1 (0.44%)	–

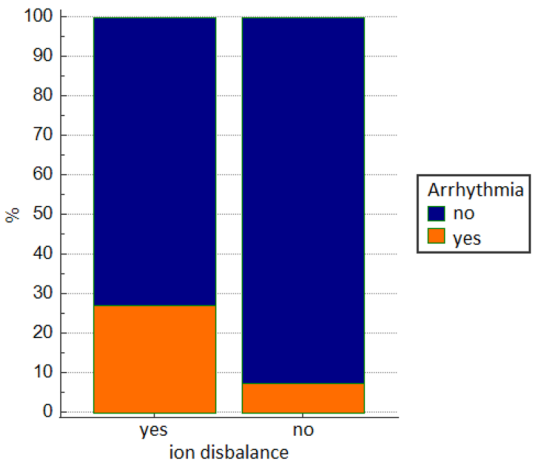


Fig. 1 relationship between the incidence of ion disbalance and arrhythmia

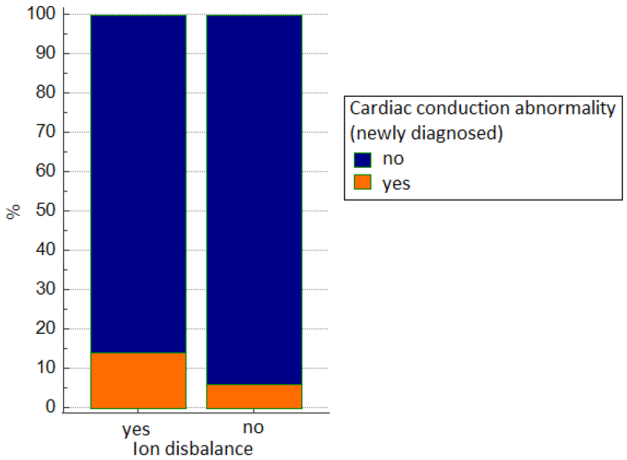


Fig. 2 relationship between the incidence of ion disbalance and newly diagnosed cardiac conduction abnormalities

seen in 14.3% of patients with ion disbalances but only 6.1% of patients without ion disbalances (Fig. 2).

To clarify these figures, the contingency tables that were the basis for creating the graphs are provided below.

	Patients with ion disbalances	Patients without ion disbalances
Patients with arrhythmia	19 (27.14%)	20 (7.60%)
Patients without arrhythmia	51 (72.86%)	243 (92.40%)
Patients with cardiac conduction abnormality	10 (14.29%)	16 (6.08%)
Patients without cardiac conduction abnormality	60 (85.71%)	247 (93.92%)

Pediatric subpopulation

Approximately one-third of subjects were under 18 years ($n=104$; 31.23%), the average age was 6.33 years. The distribution of particular age categories is shown in Table 1. The ratio of boys to girls was approximately the same among pediatric cases as in adult cases. High-voltage injuries ($n=3$; 2.88%) were rare in children, as was any history of serious diseases. The most common medical problems were: bronchial asthma, kidney disorders, and structural heart diseases. Fewer children reported symptoms at admission than adults ($n=24$; 23.08% vs. adult population $n=119$; 51.97%). This may be explained by the limited abilities of young children to express themselves. The most common complaints were tingling and pain in the extremities. Chest pain was less frequent in children ($n=6$; 5.77% vs. adults 11.79%) and no children reported palpitations. Only one child required cardiopulmonary resuscitation. Unfortunately, the child died on the fifth day of hospitalization. Loss of consciousness was also less frequent in pediatric cases ($n=5$; 4.81% vs. adults 11.35%). The incidence of the concomitant traumatic injuries was approximately same in children ($n=13$; 12.50% vs. adults 12.23%), but the incidence of burns was more than double ($n=52$; 50% vs. adults 21.40%). The number of detected arrhythmias was significantly lower in children ($n=3$; 2.88% vs. adults 15.72%). There was one case of ventricular fibrillation, two cases of sinus bradycardia, and two cases of premature complexes. No cardiac conduction abnormality was registered (vs. adults population 12.23%). A 15-year-old male with severe, extensive burns, spleen damage (with hemoperitoneum and the need for splenectomy), lung contusion, craniocerebral trauma, and a pelvic fracture had sinus bradycardia and diffuse ST elevations on the second day of hospitalization (at the time the potassium was 6.2 mmol/l). Ion disbalances were less common in children ($n=9$; 8.65% vs. adults 25.33%), the most common disbalance was hypokalemia ($n=5$; 4.81%). After the outpatient examination five children were discharged home, and 99 children were admitted to the hospital. Less than two-thirds of children had continuous ECG monitoring ($n=62$; 59.62% vs. adults 85.59%). Of the hospitalized children, 95 were discharged home, three were transferred to another hospital, and one died during hospitalization as a result of post-hypoxic encephalopathy and irreversible coma.

Discussion

Arrhythmias and ECG monitoring after electrical injury

We retrospectively analyzed 333 cases of accidental electrocution (AE). We observed that all life-threatening, malignant arrhythmias occurred immediately after the electrical injury. Other clinically relevant arrhythmias were rare, and the initial ECG was sufficient to diagnose them. Moreover, not all ECG findings should necessarily be evaluated as pathological (e.g., sinus tachycardia as a physiological response to pain/anxiety, incomplete RBBB in young patients, sporadic premature beats or non-specific ST/T changes). In addition, some ECG findings (e.g., sinus tachy/bradycardia, supraventricular tachycardia, premature complexes, cardiac conduction abnormalities or ST/T changes) may have been present before the electrical accident and they do not have to be the result of it. During ECG monitoring, we registered only one case of arrhythmia. It was paroxysm of atrial fibrillation that could be explained as a reaction to pain and severe burns (the same as sinus tachycardia present since admission) and did not require any treatment. No delayed malignant arrhythmia was observed.

According to the guidelines of the European Resuscitation Council (2015, Sect. 4), ECG monitoring is recommended after AE in all patients if they have a history of cardiorespiratory problems or have experienced a loss of consciousness, cardiac arrest, ECG abnormalities, or soft tissue damage or burns. It follows that ECG monitoring is not required when the patient is asymptomatic and the initial ECG is normal [16]. This recommendation is consistent with the results of our study (none of these patients had an arrhythmia during ECG monitoring).

Significantly, high-risk patients (with high-voltage injury, loss of consciousness, ECG abnormalities, soft tissue damage/burns, and a history of cardiovascular disease) were included in our study. Additionally, there was no new-onset arrhythmia during ECG monitoring (except for the previously described case of atrial fibrillation). Bailey et al. reached the same conclusion in prospective study published in 2017. One hundred and thirty-four patients with at least one risk factor (transthoracic current, tetany, loss of consciousness or voltage source ≥ 1000 V) were included in the study and none developed a potentially lethal late arrhythmia during the 24 h of cardiac monitoring. [9] On the other hand, our study registered a higher proportion of high-voltage electrical injuries in the group of patients with arrhythmias compared to those without arrhythmias. Post-treatment for these patients remains unclear, and high voltage should still be considered a risk factor.

Predictive factors of malignant arrhythmias

We observed eight cases of ventricular fibrillation in 7 men (87.5%) and 1 woman (12.5%). The percentage of men was slightly higher than the male–female ratio in the whole cohort. In 5 cases (62.5%), arrhythmias were induced by low-voltage and in 2 cases (25%) by high-voltage current. This was similar to the percentage of low-voltage injuries

vs. the percentage of high-voltage injuries in the overall cohort. The mean age of ventricular fibrillation patients was 40 years (range 16–58). Only one patient had a history of arrhythmia (paroxysmal atrial fibrillation). Medical histories of the other patients were entirely unremarkable (Table 4). We did not find any obvious predisposing factor for malignant arrhythmias, and the number of cases was insufficient for this purpose.

Delayed arrhythmias

Cases of delayed malignant arrhythmia are extremely rare. We found only six published cases in the medical literature. Moreover, some of them lack important information (e.g., an initial ECG). No delayed malignant arrhythmia has been reported in our study or in other larger recent studies. [3, 5, 8, 9, 15, 17, 20]

Few cases of delayed malignant arrhythmias have been published, and only two included an initial ECG. In 1987 Jensen et al. described three patients with delayed ventricular arrhythmias after electrical injury (onset 8–12 h after the accident). One patient developed recurrent ventricular fibrillation, another ventricular tachycardia, and one experienced numerous ventricular extrasystoles. [10] None had an initially recorded ECG after the electrical accident—if they had, it is unlikely that the ECG would have been normal.

In 1990, Sharma et al. published the case of a patient who, after a low-voltage injury, had a first-degree AV block and low voltage in the ECG at the time of admission. During cardiac monitoring, the AV block progressed to higher degrees over 2 h, ending to ventricular fibrillation requiring defibrillation. [6]

In 2001, Bailey et al. described a 16-year-old woman who was found dead 10 h after discharge from the hospital, where she had been treated for painful burns. No ECG was available, and the circumstances of her death were unclear, but we can suppose that a malignant arrhythmia was one possible cause. [11]

In 2015, Karataş et al. reported on a patient admitted to the hospital with a prolonged QTc interval (500 ms) and fragmented QRS complex. He developed pulseless ventricular tachycardia within 24 h. Follow-up ECG showed that the abnormalities had normalized within one month. [7]

Predictive value of the cardiac-specific biomarkers and the role of the ion disbalance

The predictive value of cardiac-specific biomarkers in risk stratification of myocardial injury after AE is still unclear. In 2010 Orak et al. analyzed the relationship between serum pro-brain natriuretic peptide (NT-proBNP), myoglobin, CK levels, cTnI, and morbidity and mortality after high-voltage electrical injuries. They found that NT-proBNP levels were higher in patients with arrhythmias, but CK-MB and cTnI levels were not. [12] Other studies also concluded that CK-MB was not a helpful marker for risk stratification of cardiac complications since it can be elevated due to muscle and soft tissue damage associated with the AE [13, 14]. Attention was then focused on the cardiac troponins, which are cardiac-specific and more sensitive. However, increased troponin levels were not usually noticed after an AE [8, 9, 15]. The most extensive retrospective study of AE, carried out by Pilecky et al., found CK elevation to be common, especially in patients with high-voltage injuries, but without any increase in CK-MB%. Significant elevation of cTnI was only detected in one patient, who had been resuscitated after ventricular fibrillation. [3]

We detected an elevation of CK in 31.83% of patients, myoglobin elevation in 28.53% of patients, and troponin elevation in 5.71% of patients. An elevation of CK and myoglobin was 5–6 times more common than troponin elevation, which can be explained by the extent of soft tissue damage. On the other hand, mild chronic elevation of troponin levels can be explained by the patient's medical history (arterial hypertension with possible myocardial hypertrophy, coronary artery disease, chronic heart failure, renal failure, etc.). Any dynamic increases are mainly consequence of the electrical injury (myocardial injury, AE-associated CPR with myocardial contusion, shock with myocardial hypoperfusion, etc.).

Patients with an ion disbalance (most often hypokalemia) had significantly more arrhythmias and newly diagnosed cardiac conduction abnormalities. This finding led us to ask whether an ion disbalance could be a risk factor for the development of arrhythmia following AE. Hypokalemia should be looked for in all patients with arrhythmias, premature complexes or cardiac conduction abnormalities and any ion disbalance should be normalized.

Pediatric subpopulation

There was a significantly lower incidence of arrhythmias and ST/T changes on initial ECGs in children compared to adults. Furthermore, no cardiac conduction abnormalities were registered. Only one case of malignant arrhythmia was reported and no delayed arrhythmias were detected. It follows that there is no need for a special pediatric approach to AEs compared to adults. In 2013, an evidence-based approach to electrical injuries in children was published. In cases involving stable, asymptomatic patients without concomitant traumatic injuries or burns outpatient management was recommended. For all others, inpatient management with laboratory examination, ECG and intravenous fluids was recommended [18]. The results and conclusion of our study agree with these recommendations.

Results of previous studies

The results of our study are consistent with previously published clinical studies. Only the incidence of ventricular fibrillation/tachycardia was higher than usually reported, but it is still a very small percentage of patients (2, 4%). We have no specific explanation for this fact.

In 2002, Blackwell et al. presented a study that differs from others in its prospective character. In the study, 212 patients after low-voltage AE were included in a 4-year follow-up. A new management protocol was developed and evaluated. It reduced the number of hospitalizations without any negative impact on mortality and morbidity [17].

In 2013 Searle et al. presented a retrospective study with 268 children and adults who were usually monitored for more than 12 h. On admission 66 patients had mild cardiac arrhythmias (sinus tachycardia or bradycardia, isolated extrasystoles), and none developed an arrhythmia requiring intervention. They also presented a standard protocol flowchart for patients with electrical injuries. Hospital-based monitoring was not recommended in asymptomatic, uninjured patients without ECG changes. [8] Three other studies (each involving more than 100 patients) were published with the same conclusions: asymptomatic patients without any risk factors and with a normal 12-lead ECG need no cardiac monitoring after an electrical injury. [5, 19, 20]

In 2016, Pawlik et al. retrospectively identified 240 patients who suffered an AE. They were monitored for an average of 4.25 h, no malignant arrhythmias were detected and the 90-day mortality was 0% [15]. Three years later, Pilecky et al. published the largest study on the risk of cardiac arrhythmias after AE. It was a retrospective, single-center study that included 480 patients. They registered 80 cases of arrhythmia (most often sinus tachycardia and bradycardia, with only one case of ventricular fibrillation before admission) and 92 ECG abnormalities (most often non-specific ST-T changes). According to the authors, routine ECG monitoring appears unnecessary, the 30-day mortality was 0%, and no late-onset malignant arrhythmias were observed [3].

Studies with high-risk patients were conducted by Bailey et al., who observed 134 patients with at least one risk factor (a transthoracic current, voltage source $\geq$ 1000 V, tetany > 1 s, any loss of consciousness). Although 11% of patients had mild ECG changes on admission, none required treatment, and no malignant arrhythmias occurred during 24-h monitoring period. Moreover, none of the patients had cardiac complications during the 1-year follow-up [9]. In 2018, Gille et al. studied 162 patients admitted to burn intensive care units. Arrhythmias were observed in 23 patients, including seven patients who required CPR (3 for ventricular fibrillation and 4 for asystole). Four patients developed self-limiting arrhythmias during hospitalization (one lasted more than 24 h after admission). [21]

Finally, there are studies focused on pediatric patients, and their results do not differ from those of adults in any meaningful way [22, 23]. In 1995, Bailey et al. described 151 cases of children with household AE. In 113 patients, cardiac monitoring was performed for 4 h (median), and no arrhythmias were observed. In conclusion, ECG monitoring did not appear necessary for children without risk factors after household electrical injuries [22]. Fifteen years later, Claudet et al. arrived at the same result. They analyzed 48 cases of children under 15 years following an AE, ten had risk factors (e.g., mainly wet skin or thoracic pain) and eight showed non-serious ECG abnormalities on admission. No delayed arrhythmias occurred. [23]

Limitations

The major limitations of our study are (1) the retrospective character of data collection, (2) inconsistent patient management and missing data, (3) unequal ratio of the low-voltage to high-voltage injuries, and (4) a change, during the study period, in the type troponin used to monitor cardiac injury and the method of its determination.

Data analyzed in the study were not primarily intended for research but represented routine medical documentation. Therefore, the quantity and quality of the information were not the same in all cases. For example, source voltage, circumstances surrounding the AE, biomarkers levels, and duration of ECG monitoring were unknown in some cases. The characteristics of the studied population also lack information regarding medication, especially antiarrhythmics and beta-blockers. Since high-voltage injuries were underrepresented, overall study results cannot be unconditionally extrapolated.

Unfortunately, continuous ECG monitoring was not performed in all patients. In a minority of patients, ECG data took from of periodic 12-lead ECGs recorded during hospitalization. It cannot be excluded that some non-serious arrhythmias may have occurred between recordings. The same situation could have occurred between the AE and the first ECG examination because not all patients went to the hospital immediately after the incident. Also, cardiac troponins levels were unavailable for every patient, and times from AE to blood sampling were variable. Another issue associated with cardiac troponins was that in 2015, we changed the type measured troponin from Troponin I (cTnI) to high-sensitive Troponin T (hs-cTnT). In general, hs-cTnT has a slightly higher sensitivity but, in specific situations, a lower specificity for diagnosing acute myocardial ischemia [24]. Due to the different diagnostic properties of the two molecules, the 99th percentiles are not comparable. Similarly, serum levels are different and independent, i.e., one level cannot be inferred from the other.

Although the number of patients enrolled in the study is relatively large compared to other studies, this number was still insufficient for general conclusions and globally applicable recommendations. A meta-analysis of all available studies would be needed for this purpose.

Conclusion

Our study suggests that routine ECG monitoring of all patients after a low-voltage electrical accident is unnecessary. All malignant arrhythmias occurred immediately after the electrical accident and delayed life-threatening arrhythmias were not observed. Considering the results of all previously published studies, it is clear that asymptomatic, uninjured patients with normal initial ECG findings do not need continuous ECG monitoring and may be discharged home. Recommendations for high-risk patients and patients with mild ECG abnormalities at admission are less obvious. The conclusions of individual studies of these patients are not consistent. Although, based on our results, even in these cases, new-onset of clinically relevant arrhythmias is unlikely. We found that the pediatric patients did not differ from adults, and there is no reason for special management. No predictive factors for malignant arrhythmias were found in this study. Elevations of cardiac troponins were sporadically observed and we did not observe any statically significant association of arrhythmias and elevated troponin levels. Determination of cardiac troponins is not necessary for all patients and should be indicated individually. In patients with arrhythmias or cardiac conduction abnormalities, prudent testing for ion disbalances is called for and adjusted as needed.

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Data availability The datasets generated and analyzed during the current study are available from corresponding author on reasonable request.

Declarations

Conflict of interest All authors declare that they have no conflicts of interest.

Ethical approval This retrospective study was performed in conformity with the Helsinki Declaration.

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