Cardiac Manifestations Following Electrical Injury: An Overview

***I Gusti AIH Pratiwi[1], I Gusti AP Purwanthi[2]**
[1]Faculty of Medicine, Udayana University, Bali, Indonesia.
[2]Division of Plastic, Reconstructive and Aesthetic Surgery, Department of Surgery, Faculty of Medicine, Udayana University/Sanglah General Hospital, Bali, Indonesia.

***Correspondence:** intenheny@gmail.com

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ABSTRACT
Cardiac manifestations are the effects of an electrical injury that could be life-threatening. Although it is rare, arrhythmias and myocardial tissue injuries are the two major cardiac complications that can happen after electrical injury. Lack of recommendation and review makes it confusing to physicians in managing electrical injury patients with cardiovascular complications. This article provides an overview of electrical injuries, the cardiac manifestations that can occur, and management of electrical injuries that focus on cardiac effects.

KEYWORDS: Electrical Injury; Arrhythmia; Myocardial Injuries; Cardiac Monitoring.

1. INTRODUCTION

Electrical injury is one of the mechanical traumas, resulting from lightning and low to high voltage injury. It can create a wide variety of clinical manifestations, starting from skin burns until cardiorespiratory arrest. Cardiac manifestation itself is one of the rare but serious clinical manifestations. It has been known that electrical injury causes 0.54 deaths per 100,000 people per year, and the mortality rates range from 3% to 15%, resulting from cardiac arrest and arrhythmias. The universal mortality rate of electrical injuries ranges from 1% to 9.1%. There are about 1000 deaths per year in the United States caused by an electrical injury. Four hundred of them are due to high voltage injury, and 50 to 300 were caused by lightning. There are another 30,000 shock incidents per year that are not fatal. This injury usually happens in an occupational setting, and it becomes the fourth-leading cause of workplace-related traumatic death. This review aims to explain the clinical cardiac manifestations of electrical injury and its management [1-3].

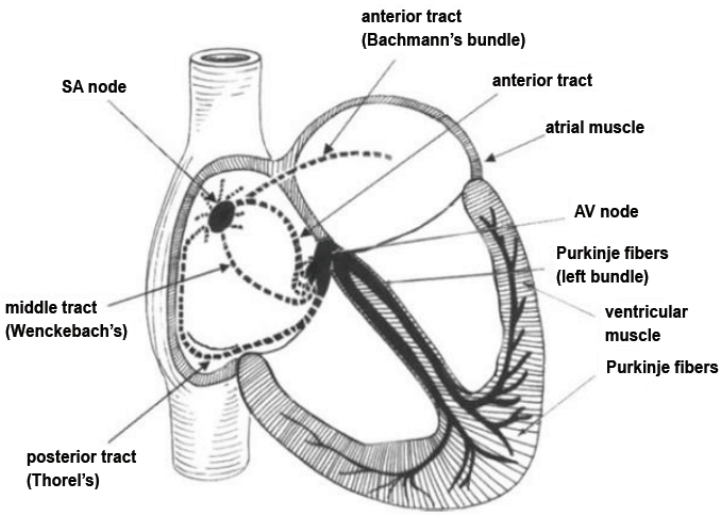
2. CARDIAC CONDUCTION SYSTEM

The cardiac conduction system is a group of specialized cardiac cells that generate electrical impulse responsible for heart contraction. It consists of sinoatrial (SA) node as the heart pacemakers, followed by atrioventricular (AV) node, bundle of His, the branches, and Purkinje fibers (Figure 1). The depolarization and repolarization of these cellular levels are caused by influx and outflow of calcium, potassium, and sodium across the membrane. The cells contain more concentration of potassium while at rest. Depolarization occurs with the rapid influx of sodium and calcium ions, transmitting the movement of an action potential across myocardium, from epicardium down to the endocardium. The restoration of resting potential or repolarization happens from endocardium to epicardium, where potassium and sodium outflows from the cell to maintain the negativity of the interior cell wall. The impulse of normal heart contraction was initiated by SA node, located at posterior wall of right atrium. SA node establishes 70 to 100 beats per minute of sinus rhythm. The electrical impulse travels down to AV node located near the interatrial septum, through the bundle of His, down the bundle branches, and through the Purkinje fibers, maintaining the synchronization in contraction of the ventricles. This electrical impulse activity can be seen from electrocardiography [4, 5].

3. PRINCIPLES AND DETERMINANTS OF ELECTRICAL INJURY

Current, measured in Ampere (I), is the flow of electrons through a potential gradient between two physical points. The differences of potentials, the gradients from high to low concentration, are stated in voltage (V). Resistance is an obstacle in the flow of electrons, measured in ohms (R). Resistance in the human body is divided into low resistance and high resistance. Blood vessels, neurons, and muscles are excellent conductors, while bone, fat, and skin are poor conductors of electricity. The higher the resistance, the greater damage it will get as a result of electrical injury. Ohm's law described the relationships of current,

Figure 1: Conduction systems of the heart [5].



voltage, and resistance as follows: I = V/R. Impact of electric current on the human body creates a set of pathophysiological events, called electric shock. Lightning is the excessive form of electrical injury, whereas electrocution is the term used when the electrical injury results in death. The injury can also occur by direct and indirect mechanisms, where the direct injuries are caused by close contact of the victim with the electrical energy and indirect injuries are linked to secondary trauma due to flash or flame. No current travels into the skin in flash injury, while current may or may not pass the skin in flame injury [2, 6].

Some factors that affect the severity of this trauma are voltage, resistance to the current flow, type of current, the pathway, and duration of contact. Types of current are divided into alternating (AC) and direct current (DC), where AC is found to be more dangerous as it can cause tetanic muscle contraction as the victim cannot release the energy source. The current intensities are linked to both voltage and resistance. Voltage is one of the markers used in categorizing the electrical trauma, where less than 1000 V is categorized as low voltage injury and more than 1000 V is categorized as high voltage injury. The higher voltage is more dangerous. Resistance also has an important role, as it can result in fatal complications if the resistance is low. Longer duration leads to worse injury. The path of entry is usually found in a hand, and the most common ground is usually a foot. Organ that usually gets affected by the common entry and ground is the heart, which may result in fatal arrhythmias. The example of entry and exit pathway of electrical injury is shown in Figures 2 and 3. Central nervous system can be damaged if the current passes through head. Electrical injury can cause damage to many organ systems other than central nervous systems, such as skin burns, musculoskeletal effects, respiratory arrest, kidney failure, and other cardiac manifestations that are presented in Table 1 [2, 6–8].

Figure 2: Entry wound of electrical injury [9].

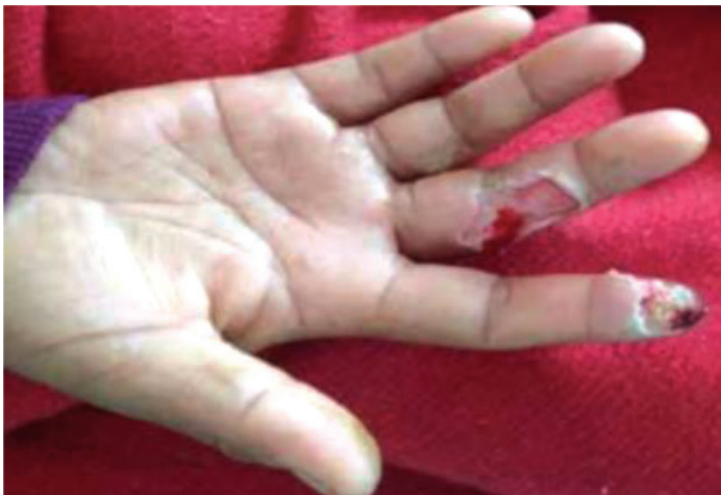


Figure 3: Exit wound of electrical injury [9].

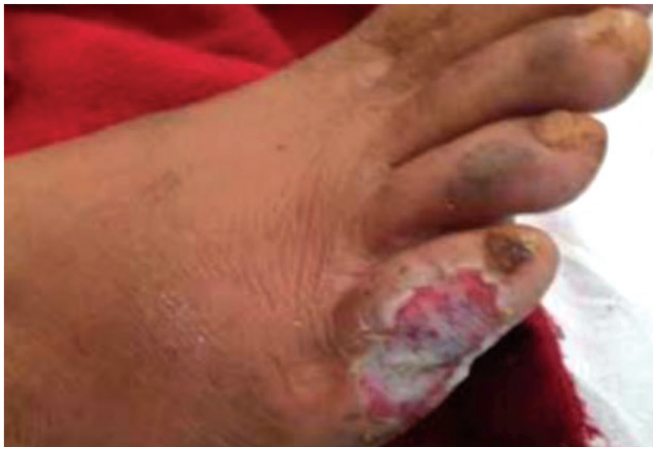


Table 1: Manifestations of electrical injury based on organ damage [8].

Systems	Manifestation
Skin	Cutaneous burn
Cardiac	Arrhythmias, cardiac arrest
Respiratory	Respiratory arrest due to central nervous system or muscle tetany
Vascular	Aneurysm formation, tissue ischemia
Neurologic	Loss of consciousness, transient paralysis or paresthesia, peripheral neuropathy, or spinal cord injury
Musculoskeletal	Fractures or dislocations secondary to muscle spasms or falls, muscle necrosis, or compartment syndrome
Renal	Myoglobinuria, renal failure
Other	Cataracts, neuropsychological effects

4. CARDIAC MANIFESTATIONS OF ELECTRICAL INJURY

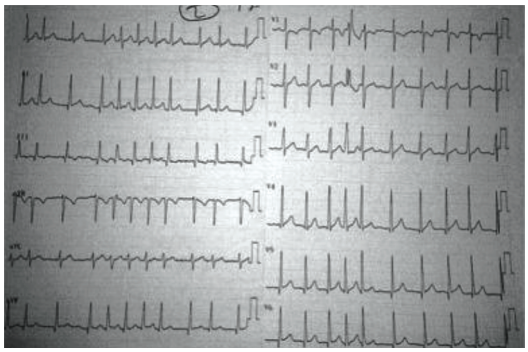
Cardiac manifestations usually occur if the current travels from hand to hand or hand to foot. It can also occur because the currents travel through the lowest resistance part of the body, such as nerves and blood vessels [7]. An exposure to low voltage AC is the most probable cause of cardiac consequences after electrical injury. Cardiac arrest, arrhythmias, and myocardial injury are the most frequent cardiac manifestations that occur after an electrical injury. Vascular injury, hemorrhagic pericarditis, transient autonomic dysfunction, and transient arterial hypertension are also some other cardiac manifestations after the electrical trauma. Cardiac arrest is frequently seen in high voltage electrical injury and associated with arrhythmia, particularly ventricular fibrillation [3, 6, 8, 10, 11].

4.1. ARRHYTHMIAS

Disruption of heart rhythm might be caused by relatively low current. The mechanism of how arrhythmia occurred still remains unclear and is believed to be multifactorial. The alterations of electrolyte concentration, necrosis, and change of permeability of myocyte membranes are some of the possible mechanisms that can lead to arrhythmias in electrical injury. Biopsies reported that arrhythmogenic foci in patchy myocardial fibrosis were found with the increasing Na^+ and K^+ pumps. It is believed to be associated with transient and localized alterations in potassium and sodium transport and also concentrations with resultant changes in membrane potential. This heterogeneity of repolarization areas triggered arrhythmias, with possible abnormal enhanced automaticity or after depolarization, and triggered activity several hours after the injury [6, 12].

Arrhythmias usually occur immediately after electrical injury. Ventricular fibrillation occurs if the current reaches the heart in vulnerable timing, which is the most frequent cause of death after electrical injury. High voltage electric injuries cause asystole, and low voltage was found to cause ventricular fibrillation. Some of the arrhythmias may be caused by physiological reaction to pain or anxiety such as sinus tachycardia. Other arrhythmias reported are sinus bradycardia, premature atrial contraction (PAC), and premature ventricular contractions (PVC). Conduction dysfunctions, such as atrioventricular blocks and bundle branch

Figure 4: Atrial fibrillation after electrical injury [17].



blocks, are also common. Atrial fibrillation is also reported. Delayed arrhythmias frequently occur in patients who had a rhythm disturbance on presentation [3, 12–14].

Pilecky *et al*. reported that sinus bradycardia is found in 10.4% of its sample, followed by sinus tachycardia (4.4%). One patient had newly diagnosed atrial fibrillation, and other was detected with arrhythmia (ventricular tachycardia) [13]. Akkaş *et al*. reported nine deaths, where almost all deaths were related to cardiac complications, particularly rhythm abnormalities. Asystole is observed in seven cases with high voltage injury, and only one ventricular fibrillation case was found with low voltage injury [10]. Another study showed a result of 28.7% cases of pediatric arrhythmias and 24.2% cases of adult arrhythmias. Sinus tachycardia, bradycardia, ventricular ectopic beats, isolated supraventricular, first-degree atrioventricular block, and incomplete right bundle branch block were reported [15]. Krämer *et al*. in a retrospective study reported that cardiac arrhythmia was found in 16 patients, where 14 of them were in contact with low voltage electricity [3].

First degree of atrioventricular block was described by Beton *et al*. after a low voltage (380 V) electrical injury. The input pathway is found at the left hand, and the gluteal became the exit pathway of the current. The history of previous arrhythmias or another cardiovascular disease is not known, ensuring that the electrical injury is the reason of electrocardiogram (ECG) changes [16]. Bilik *et al*. reported a case of atrial fibrillation as a result of low voltage electrical injury. The patient had skin burns on the extensor side of middle phalanx of the left hand as the entry pathway and another skin burn on the right arm as the exit. Any other previous cardiac disease of the patient is unknown. The ECG is shown in Figure 4 [17]. Another atrial fibrillation case, reported by Paudel *et al*., was of electrical injury with an entry and exit wound from hand to toe [9]. The cases are in sync with the theories of other authors that combination of pathway cardiac damages and low voltage injury more often leads to cardiac consequences.

4.2. MYOCARDIAL INJURY

Myocardial injury after electrical injuries can occur due to several mechanisms (Figure 5). The clinical features of myocardial injury are concordant with the mechanism itself. Coronary spasm or thrombosis leads to clinical symptoms of myocardial infarction. The severity of myocardial injury is mostly determined by the voltage and type of current. The manifestation will be more serious with higher voltage and if the type is AC. This injury of the heart can be confirmed by abnormal ECG echocardiographic finding, cardiac enzymes, coronary angiography, and magnetic resonance imaging (MRI). The frequent finding in ECG is

Figure 5: Mechanism of myocardial injury after electrical injuries [6].

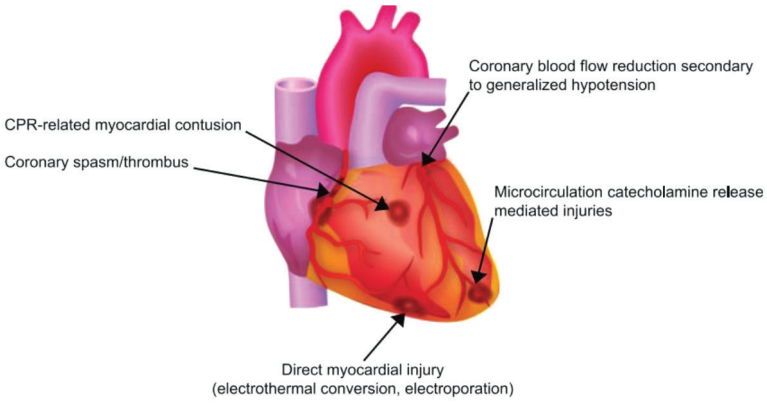
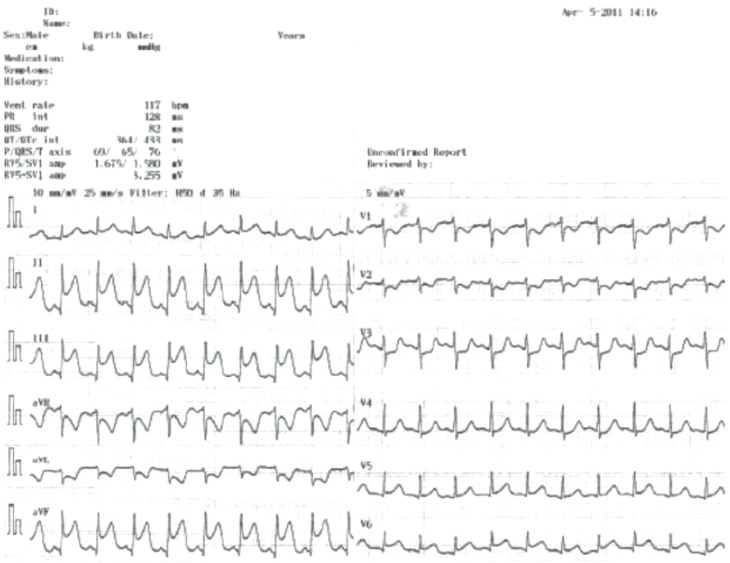


Figure 6: Myocardial infarction after electrical injury [18].



abnormality in ST segment and QT intervals, and some cases reported pathological Q waves just like myocardial infarction pattern. Troponin and creatine kinase-MB (CK-MB) are the cardiac enzymes that predict injury or necrosis, but the significance still remains unknown due to lack of evaluations of troponin's specificity in electrical injury setting, and CK-MB is often associated with skeletal muscle necrosis too. Coronary angiography is performed to confirm the possible underlying coronary artery disease [6, 12].

Behçet *et al*. described a case of 43-year-old worker with accidental contact with high voltage electricity. Both of the palms of the patient became the entry point, while the soles were the exit point of the current. The ECG revealed ST segment elevation in inferior leads and depression in anterior leads as shown in Figure 6, indicating myocardial infarction. Normal systolic function and hypokinesia of left ventricular wall was found after echocardiography, and the cardiac enzymes were elevated. CK-MB level was 2177 when the normal range is 0–25 U/L, and the troponin I level was 0.366 (0–0.01 ng/mL). Coronary angiography was performed, and there was no occlusion within the right and left coronary arteries [18]. A case reported by Hung *et al*. suggested that the cardiac biomarkers' examination in detecting myocardial injury of electrical injury patients is important, while other author stated that there is insufficient evidence about cardiac biomarkers being the predictive factors of myocardial injury [13, 19].

Some myocardial injuries can be present with arrhythmias altogether. Krämer *et al*. reported two cases of combined ECG changes (ST segment changes and sinus tachycardia) from a low voltage electrical injury patient [3]. Gursul *et al*. reported a case of a 50-year-old male patient admitted with an AC electrical injury. The patient said that he had loss of consciousness with radiating pain from the chest to left arm. Initial ECG shows sinus tachycardia, increasing T wave, and premature ventricular contraction that changed to atrial fibrillation after 10 minutes. The atrial fibrillation was followed by ST segment elevation in inferior leads and ST segment depression in leads V1–V3. Coronary angiography of the patient revealed normal coronary blood flow, indicating that the changes of ST segment were caused by arterial vasospasm. Ejection fraction (EF) of the patient was estimated as 58% after the echocardiography, and there was no segmental motion defect [20].

5. MANAGEMENT OF ELECTRICAL INJURY

5.1. GENERAL MANAGEMENT OF ELECTRICAL INJURY

The first and most important management of electrical injury is to separate the victim from the current source before further damages can happen. The electrical source needs to be turned off first before rescuing the victim since he or she can be the conductor of the current itself. The management requires basic principle of cardiopulmonary resuscitation (CPR) and acute multiple trauma care, just like any other traumas. In case of cardiorespiratory arrest, resuscitation can be done according to Advanced Cardiac Life Support (ACLS) and Advanced Trauma Life Support (ATLS) algorithms [8]. Complete evaluation of the victim must be done. It is important to find the pathway of the electricity in clinical examination to consider potentially damaged organs. The other examination is done to find another injury, such as spinal cord injury, blunt trauma of abdomen, or thorax cavity, in addition to the laboratory examination of liver, pancreatic, and renal function. The laboratory examination aims to determine traumatic or ischemic injury in a case of cardiorespiratory arrest. The radiology imaging can be done to exclude spinal cord injury, blunt trauma, and fractures [6, 8, 12].

Treatment of electrical injury victim is carried out symptomatically. In case of extensive burns, the victim must be administered to specialized burn unit. Wound care with silver sulfadiazine is recommended because it is less likely to cause imbalance of electrolyte. Fluid resuscitation is also important to compensate the fluid extravasation and to prevent acute renal failure by myoglobinuria. Surgical decompression such as fasciotomy can only be performed if there is any progressive neurological symptom such as vascular compromise, elevated compartment pressure, or suspected myonecrosis [8].

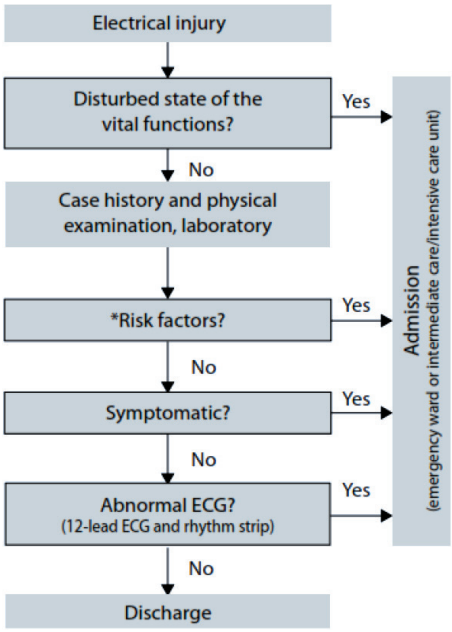
5.2. CARDIAC MONITORING

Cardiac monitoring is still controversial in management of electrical injuries. Cardiac monitoring is conducted by ECG and cardiac biomarkers' examination. All patients with electrical injury should get an initial ECG regardless of the voltage involved. The duration of monitoring is not well established or mentioned in any guideline, but 24-hour monitoring is recommended by most experts. It is aimed to identify delayed arrhythmia or myocardial injury after electrical trauma. It is suggested that the severe cardiac manifestations happened at the time of event, and it is rare for a patient to develop delayed serious arrhythmias [10, 21]. Some researchers believed that it is important to perform cardiac monitoring of a patient who has risk factors such as unwitnessed event, high voltage exposure, prior heart disease, and abnormal initial ECG. Cardiac biomarkers are still controversial as they lack specificity as mentioned above [6, 12, 22, 23].

A study about ECG monitoring necessity was conducted by Pilecky *et al*. The authors excluded patient with normal ECG and monitored 182 patients for 12.7 ± 7.1 hours. Symptomatic regular supraventricular tachycardia was found in one patient, and atrial fibrillation was documented in another patient. However, the two patients had history of cardiac manifestations even before the monitoring, where the first patient suffered palpitations before the supraventricular tachycardia rolled out and another patient had a history of paroxysmal atrial fibrillation [13]. Searle *et al*. described some new follow-up ECG changes after cardiac monitoring in their study. The follow-up of electrical injury patient with initial abnormal ECG was done. The monitoring revealed an incomplete RBBB with isolated supraventricular ectopic beats and ventricular couplet in pediatric patient with underlying complex heart defect, and the ECG change into sinus bradycardia during cardiac monitoring was also traced. None of the arrhythmias found requires treatment [15].

Krämer *et al*. conducted a retrospective study to find out whether cardiac monitoring id really necessary or not. The authors conclude that asymptomatic patients without any risk factors need no cardiac monitoring, while the patients of electrical injury with risk factor should be admitted to hospital or the intensive care unit for cardiac monitoring. The risk factors mentioned are loss of consciousness, high voltage injury, pregnancy, history of cardiovascular diseases, abnormal laboratory result, concomitant injuries, transthoracic current, and soft-tissue burns and damage. The authors recommended a management plan as shown in Figure 7 [3]. Another initial management protocol was stated by Blackwell and Hayllar, where several sources were used to create the protocol. Six-hour cardiac monitoring of patients with any abnormal initial ECG or relevant clinical symptoms should be done. The monitoring should continue if the patient still develops symptoms or there is no improvement in

Figure 7: Management of electrical injury patient [3].



ECG. Patient with no symptoms and normal ECG can be discharged [22]. Long-term follow-up is needed for some individuals depending on initial findings after electrical injury [6].

5.3. TREATMENT OF CARDIAC MANIFESTATIONS OF ELECTRICAL INJURY

The treatment of cardiac manifestations follows the general management of electrical injury according to ACLS and ATLS principles. Cardiorespiratory arrests require CPR [8]. Evaluation of air, breathing, and circulation is necessary. In case of ventricular fibrillation, immediate defibrillation must be performed. Medication that can be used is amiodarone, lidocaine, or magnesium sulfate [24]. While treatment of other arrhythmias depends on their types. Bilik *et al*. recommend an administration of 5 mg metoprolol IV for ventricular rate control and enoxaparin (8000 IU/0.8 mL) for preventing thromboembolic complication in atrial fibrillation case after electrical injury. It is observed in some cases that after oral administration of metoprolol and enoxaparin in intensive care unit (ICU), the ECG came back normal on the first day of hospitalization [17]. There is another case of the return of sinus rhythm in atrial fibrillation after intravenous diltiazem administration, and some cases require cardioversion [25, 26].

Paudel *et al*. reported a complex case of a female patient with atrial fibrillation 6 hours after injury. She was administered metoprolol intravenously, but it failed to revert the sinus rhythm. Hence, cardioversion and administration of amiodarone were done after, and it is only 12 to 13 hours after the cardioversion that the ECG finally reverted to sinus rhythm [9]. One case of atrial fibrillation and spontaneous return of sinus rhythm and another case of supraventricular tachycardia that came back to sinus rhythm after vagal maneuvers were reported by Pilecky *et al*. [13]. Beton *et al*. reported a spontaneous return of sinus rhythm after atrioventricular block case in electrical injury, while other author reported a requirement of permanent pacemaker for atrioventricular block caused by electrical injury [16, 27].

Myocardial injury case reported by Behçet *et al*. documented an administration of anti-ischemic drugs, such as acetylsalicylic acid, angiotensin-converting enzyme inhibitor, and beta blockers, to the patient with ST segment changes after injury. The ST segment change was believed to be due to arterial spasm [18]. Celebi *et al*. stated that it is also challenging to treat the myocardial injury because there is no exact guideline for managing ST elevation after electric shock. The authors recommended that coronary angiography followed by percutaneous coronary intervention is a better initial reperfusion strategy than the fibrinolysis, and that coronary angiography is an important aspect to predict the treatment needed [28].

6. CONCLUSION

Cardiac manifestations of electrical injury are some rare incidents that need to be examined thoroughly as they can lead to life-threatening state. The most common manifestations are arrhythmia and myocardial injury [6]. Cardiorespiratory arrest is the most frequent cause of death after electrical injury, while arrhythmia usually presents acutely after incident and can be diagnosed by ECG examination. Myocardial injury is the less frequent manifestations, but it is more challenging to diagnose because of the lack of data or study about the role of cardiac biomarkers and other diagnostic tool to exclude other cause of myocardial injury. The management of these cardiac manifestations requires basic principles of ACLS and ATLS. Cardiac monitoring is not necessarily needed for each patient with cardiac manifestations, and the risk factors should be evaluated [3].

CONFLICT OF INTEREST

There is no conflict of interest.

REFERENCES

1. Zemaitis MR, Foris LA, Lopez RA, Huecker MR. Electrical injuries [Internet]. Treasure Island, FL: StatPearls Publishing; 2020. Available from: https://www.ncbi.nlm.nih.gov/books/NBK448087/. Accessed October 29th, 2020.
2. Choi JH, Han D, Kang S, Yoon C, Cho JR, Kym D. Retrospective study of prognosis and relating factors of cardiac complications associated with electrical injuries at a single centre in Korea. BMJ Open. 2019;9:e028741.
3. Krämer C, Pfister R, Boekels T, Michels G. Cardiac monitoring always required after electrical injuries? Med Klin Intensivmed Notfmed. 2016;111(8):708-14.
4. Daniel J, Jennifer H, Todd C. The cardiac conduction system: a mini review. COJ Tech Sci Res. 2018;1(2):1-3.
5. Laske TG, Iaizzo PA. The cardiac conduction system. In: Iaizzo PA, editor. Handbook of cardiac anatomy, physiology, and devices. New York, NY: Humana Press America; 2005. p. 123-6.
6. Waldmann V, Narayanan K, Combes N, Jost D, Jouven X, Marijon E. Electrical cardiac injuries: current concepts and management. Eur Heart J. 2018;39(16):1459-65.
7. Waldmann V, Narayanan K, Combes N, Marijon E. Electrical injury: clinical updates. BMJ. 2017;357:j1418.
8. Czuczman AD, Zane RD. Electrical injuries: a review for the emergency clinician. Emerg Med Pract. 2009;11(10).
9. Paudel N, Alurkar VM, Kafle RC, Sapkota S. Atrial fibrillation following electric shock requiring cardioversion. NHJ. 2016;13(1):31-4.
10. Akkaş M, Hocagil H, Ay D, Erbil B, Kunt MM, Ozmen MM. Cardiac monitoring in patients with electrocution injury. Ulus Travma Acil Cerrahi Derg. 2012;18(4):301-5.
11. American Heart Association. Electric shock and lightning strikes. Circulation. 2005;112:IV-154-5.

12. Koumbourlis AC. Electrical injuries. Crit Care Med. 2002;30(11 Suppl):S424-30.
13. Pilecky D, Vamos M, Bogyi P, Muk B, Stauder D, Racz H, *et al*. Risk of cardiac arrhythmias after electrical accident: a single-center study of 480 patients. Clin Res Cardiol. 2019;108:901-8.
14. Bailey B, Gaudreault P, Thivierge RL. Cardiac monitoring of high-risk patients after an electrical injury: a prospective multicentre study. Emerg Med J. 2007;24(5):348-52.
15. Searle J, Slagman A, Maass W, Mockel M. Cardiac monitoring in patients with electrical injuries. An analysis of 268 patients at the Charite Hospital. Dtsch Arztebl Int. 2013;110(50):847-53.
16. Beton O, Efe TH, Kaya H, Bilgin M, Asarcikli LD, Yilmaz MB. Electrical injury-induced complete atrioventricular block: is permanent pacemaker required? Case Rep Cardiol 2015;2015:158948.
17. Bilik MZ, Akyuz A, Akil MA, Inci U, Acet H, Toprak N. Case report: unexpected guest: atrial fibrillation due to shock. Int J Cardiovasc Acad 2016;2:40-1.
18. Behçet AL, Yarbil P, Özer H, Aslan S, Yıldırım C, Davutoğlu V. A rare complication of electric shock: myocardial infarction. J Emerg Med Case Rep. 2012;3(1):4-8.
19. Hung L, Tien T. Myocardial injury after electric accident: dynamic change of cardiac biomarkers was the important key for diagnosis of this serious complication. J Biosci Med. 2015;3:78-81.
20. Gursul E, Bayata S, Aksit E, Ugurlu B. Development of ST elevation myocardial infarction and atrial fibrillation after an electrical Injury. Case Rep Emerg Med. 2015;2015:953102.
21. Fish RM. Electrical injury: part III: cardiac monitoring indications, the pregnant patient, and lightning. J Emerg Med. 2000;18:181–7.
22. Blackwell N, Hayllar J. A three year prospective audit of 212 presentations to the emergency department after electrical injury with a management protocol. Postgrad Med J. 2002;78:283-5.
23. Koren O, Paz E, Rozner E, Mahamid M, Turgeman Y. Late myocardial sequelae of electrical injury. Clin Case Rep. 2020; 8(12):3408-11.
24. Rita A, Camera GL, Francesco AD, Chiarenza F, Calaveri M, Navarria DV, *et al*. Electric Shock and Ventricular Fibrillation. Acta Medica Mediterranea. 2011;27:99.
25. Butler ED, Gant TD. Electrical injuries, with special reference to the upper extremities. Am J Surg. 1977;134(1):95-101.
26. Gozlukaya A, Serinken M. Elektrik carpmasina bagli gelisen atriyal fibrilasyon. JAEM. 2010;1:27-9.
27. Hyun DW, Yoon HJ, Kwon TG, Kim KY, Bae JH. Electrical injury-induced high-degree atrioventricular block requiring a permanent pacemaker. Korean Circ J. 2006;36(11):767-70.
28. Celebi A, Gulel O, Cicekcioglu H, Gokaslan S, Kututcularoglu G, Ulusoy V. Myocardial infarction after an electric shock: a rare complication. Cardiol J. 2009;16:362-4.