

# Atrial fibrillation induced by low-voltage electrical injury

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## Summary

The clinical manifestations of electrical injuries are very heterogeneous, from small skin burns to sudden death due to cardiac or respiratory arrest. In patients surviving an electrical shock, any organ can potentially be damaged. Cardiac consequences are among the most common and serious, with arrhythmias and myocardial damage being the most frequent manifestations. Cardiac complications are usually reversible but, in rare cases, long-term cardiac damage can arise, emphasising the importance of individual follow-up. We report a rare case of self-limited atrial fibrillation induced by a low-voltage electrical injury in a domestic context in a healthy 33-year-old man without cardiac predisposing factors. We also briefly review the literature about the cardiac complications related to electrical injuries.

Key words: electrical injury; arrhythmias; atrial fibrillation; myocardial injury

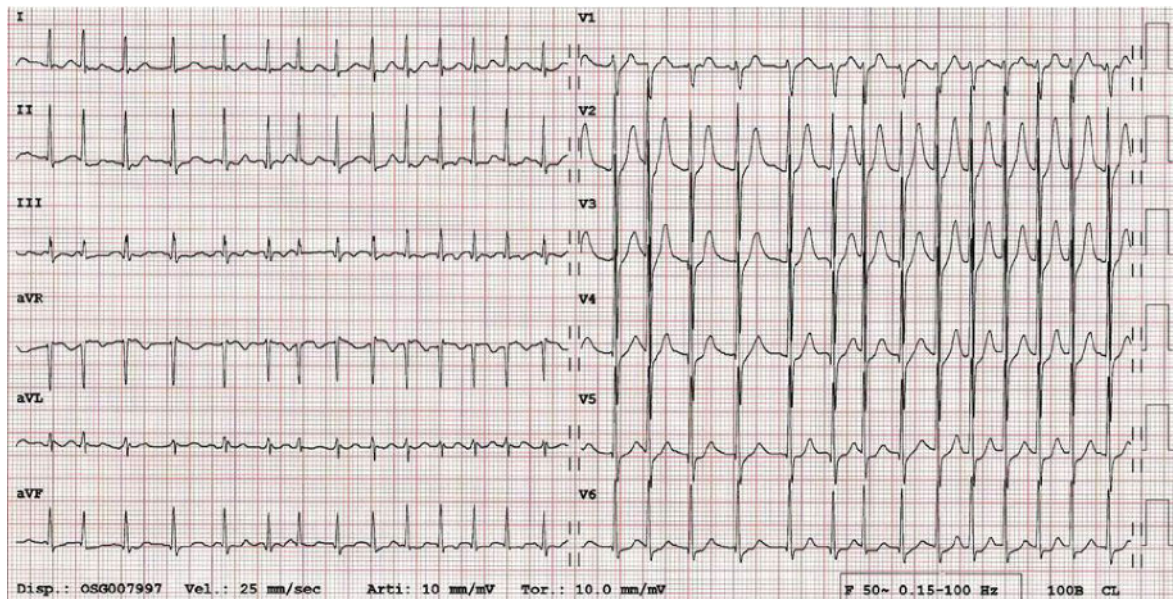


## Case report

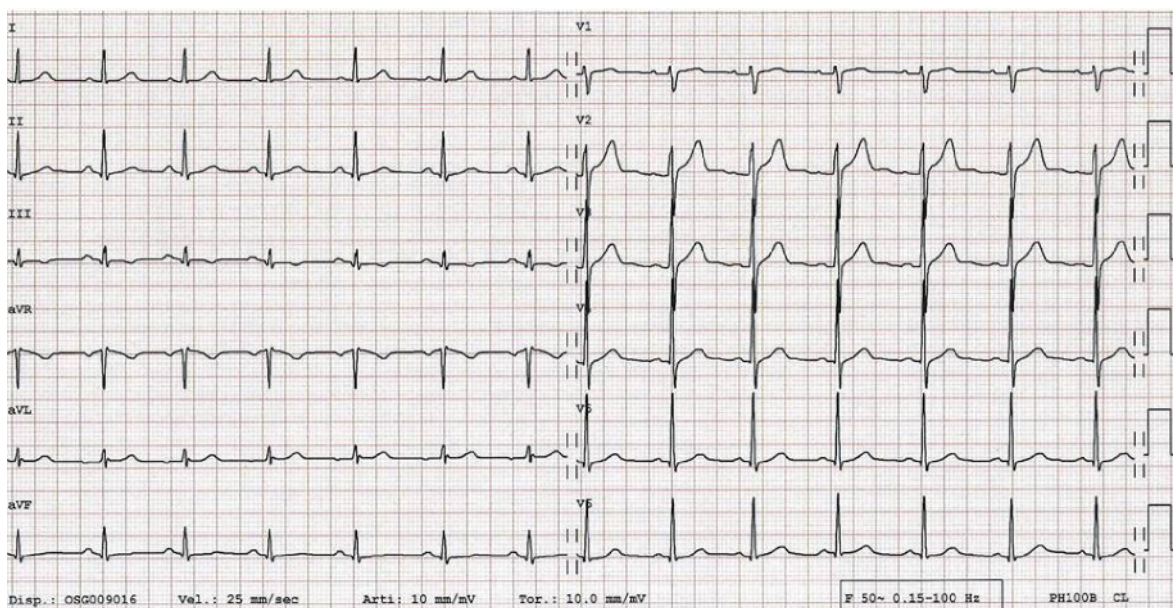
A 33-year-old young man arrived in the emergency room by his own means after being struck by a 230 V, 50 Hz alternating current electrical shock through an accident in a domestic context. While he was trying to adjust the temperature of an old industrial refrigerator by rotating the thermostat with his right hand with the help of a clamp, he suddenly received an electrical shock. The young man was on his knees in front of the appliance and during the shock he could not detach himself from the clamp, feeling a strong internal heat for several seconds. A cousin, who was in the same room, disconnected the valves responsible for supplying electricity. The patient experienced a short episode of malaise and dizziness and was driven to hospital. Upon arrival, about 30 minutes after the accident, he reported profound asthenia associated with strong palpitations and lower left limb pain. Clinical evaluation revealed a conscious patient with pulse rate of about 140 bpm and irregular, blood pressure of 115/75 mm Hg and oxygen saturation 98% on ambient air. There was no evidence of injury to the right hand

(current entry point), but a skin burn was detected on the left knee, the likely exit point. In addition, proximal left lower limb paresis was observed. The ECG showed atrial fibrillation with a rapid ventricular rate of up to 200 bpm without further significant abnormalities (fig. 1). Chest radiography was normal. Blood examinations, including thyroid function tests, were normal. Intravenous hydration was prescribed and the patient was admitted to the intensive care unit for further evaluation and monitoring. The ventricular rate progressively decreased and after about 3 hours the patient spontaneously converted to sinus rhythm with a normal ECG, and the palpitations disappeared (fig. 2). Serial blood examinations showed an increase in creatine kinase (maximum 2014 U/l, reference <200 U/l) and creatine kinase MB fraction (maximum 132 U/l, reference <24 U/l), and a slight increase in high-sensitivity cardiac troponin I (maximum 43 ng/l, reference <40 ng/l). A transthoracic echocardiogram was entirely normal. The patient was monitored for 24 hours and no further arrhythmias were recorded. The increase in creatine kinase was attributed to rhabdomyolysis. A neurological evaluation highlighted a partial left lumbar plexus injury. After 4 days the patient was discharged home without medication. Of note, the patient had always enjoyed good health, he had never had palpitations or other cardiac symptoms, never taken medication or abused of alcohol or illicit drugs. He was sedentary and he did not practice sports activities. Family history for cardiovascular diseases was negative.

After 4 months he was completely asymptomatic and did not complain of any palpitations. The lower limb paresis had completely regressed. Clinical examination and resting ECG were normal. A 24-hour Holter ECG revealed constant sinus rhythm with average heart rate of 84 bpm (range 47–155 bpm) with only four atrial and three ventricular premature beats. An exercise stress test was completely normal. No further follow-up was planned, but we informed the patient that in the event of palpitations or other cardiac symptoms a prompt cardiac evaluation would be justified.



**Figure 1:** ECG at hospital admission. Atrial fibrillation with fast ventricular rate varying between 136 and 200 bpm with normal narrow QRS complexes and nonspecific repolarisation abnormalities.



**Figure 2:** ECG after spontaneous interruption of atrial fibrillation. Sinus rhythm at 81 bpm with normal ECG tracing.

## Discussion

The clinical presentation of accidental electrical injuries can vary from minor skin burns to sudden death. Their management is sometimes difficult because of their rarity, the limited literature and the absence of systematic recommendations [1, 2]. We report a rare case of a transient atrial fibrillation induced by low-voltage electrical shock and we briefly review the literature concerning the cardiac involvement in electrical injuries.

The consequences of an electrical injury depend on several factors: the voltage and the body resistance determining the current intensity, the contact duration with the source, the thermal energy delivered, the type of current (alternating or direct) and the current path through the body. Voltage is often the only known variable in an electrical accident. Electrical shock are classified in low-voltage (<1000 V) and high-voltage ( $\geq 1000$  V), with lightning being a particular form of very high-voltage shock ( $>10^6$  V), but lasting only some milliseconds. Alternating current causing tetanic mus-

cle contractions can lead to a more prolonged exposure with the source, as was the case in our patient. The domestic power supply in Europe carries a 220–230 V, 50–60 Hz alternating current, whereas high-voltage lines usually carry direct current [1].

The age distribution of patients suffering from electrical injuries is bimodal. The first peak occurs in children, mainly by low-voltage domestic accidents; the second peak in adults, more frequently involving high-voltage occupational accidents [1, 2]. There are no data about the incidence of electrical injuries but they are a rare cause of emergency department consultations, accounting for between 0.001% and 0.17% of all visits [3–5]. In Switzerland during the last 20 years, electrical accidents have been responsible for 2 to 10 deaths per year, more than 80% being of males between 20 and 60 years old [6]. More than 90% of these deaths arise immediately after the electrical shock [7]. The mechanisms are thought to be cardiac arrest due to ventricular fibrillation or asystole, or respiratory arrest due to injury of the respiratory centre or paralysis of the respiratory muscles [7]. Patients hospitalised after an electric shock have a mortality varying between 0 and 21%, which is higher after lightning strikes, intermediate after high-voltage accidents and lower following low-voltage injuries [4, 5, 8–11].

Electrical injuries can virtually affect all organs. Cardiac effects are among the most common. Electrical current easily follows blood vessels owing to their low resistance. Moreover, due to its central position in the thorax, the heart can be affected both by horizontal (hand to hand) and vertical (head or hand to foot), as in our patient, currents through the body. Other frequently involved organs are the skin, the central and peripheral nervous systems, the respiratory system, the peripheral blood vessels, the musculoskeletal system and the kidneys [1, 2]. Our patient had injuries to the heart (atrial fibrillation), the peripheral nervous system (lumbar plexus), the muscles (rhabdomyolysis) and the skin.

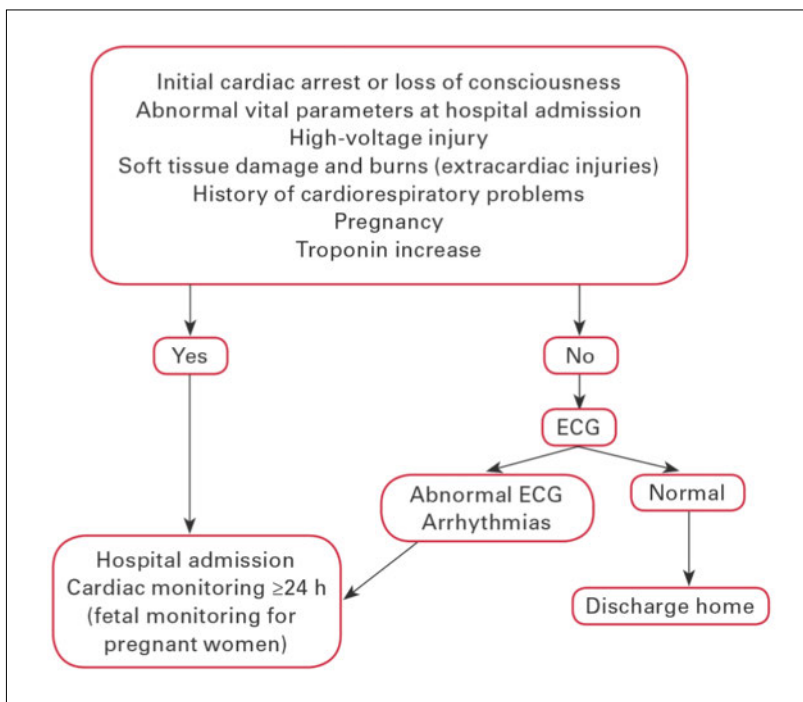
Arrhythmias and myocardial injuries are the two principal cardiac complications of electrical shock [1, 2]. Several mechanisms seem to be involved: cell depolarisation, myocardial necrosis through electroporation or electrothermal conversion, massive catecholamine release, coronary spasm or thrombus formation, anoxia, coronary hypoperfusion due to severe arrhythmia-induced hypotension and traumatic myocardial contusion [2].

Arrhythmias almost always occur immediately after the shock. Ventricular fibrillation and asystole are responsible for sudden death after the accident. Ventricular fibrillation is more often caused by alternating current with an impulse falling in the vulnerable period of

repolarisation, whereas asystole is more often associated with high-voltage direct current [2]. A wide variety of ECG abnormalities have been observed after an electric shock, with rates varying from 3 to 47% [3–5, 9–16]. However, most of these ECG abnormalities are minor, transient and nonspecific, such as sinus tachycardia or sinus bradycardia, atrial or ventricular premature beats, axis deviation, bundle-branch block or repolarisation abnormalities. They can result from the electrical injury but they also could be pre-existing abnormalities. Their significance should therefore be determined on an individual basis. Serious arrhythmias are rare and mostly reported as isolated cases. In several series, atrial fibrillation occurred in only 6 out of 1530 patients (0.4%) having an ECG after the electrical shock [3–5, 9–16]. Cases of atrial fibrillation after electrical injury have been published, but at present no consensus exists concerning its management [17–25]. Similarly to other cases of acute-onset atrial fibrillation [26], the arrhythmia is usually self-limiting in a few hours, as in our patient. Atrial fibrillation has sometimes been pharmacologically cardioverted [19, 22] or, because of haemodynamic instability, electrically cardioverted [21, 23]. It is interesting to note that 3 patients with atrial fibrillation and Wolff-Parkinson-White syndrome have been described, resulting in a rapid pre-excited ventricular rate [21–23]. Patients sustaining an electrical shock are usually young and healthy, as was the case with our patient, who had no characteristics suggesting a predisposition to atrial fibrillation. In general, no cases of relapse of atrial fibrillation have been described, but two cases of persistent atrial fibrillation following electrical shock have been reported [24, 25], raising the possibility of persistent atrial injury. Our patient remained asymptomatic after 4 months of follow-up without evidence of atrial excitability. In the case of suggestive symptoms, particularly if there is a predisposing condition, one should have a high index of suspicion for the resumption of atrial fibrillation.

Sick sinus syndrome and various degrees of atrioventricular block have been described [2]. They are usually transient, but in exceptional cases they require pacemaker implantation, reflecting a probable persistent injury to the sinus or atrioventricular nodes [27, 28]. Transient Brugada ECG patterns have also been observed [29, 30].

Myocardial damage is the other major cardiac complication of electrical injuries [1, 2]. The incidence is unknown and the diagnosis can be difficult. The ECG can manifest nonspecific repolarisation abnormalities, ST-segment depression or elevation, Q-wave formation and QT interval prolongation. Moreover, various forms of arrhythmia can accompany myocardial injury [1, 2].



**Figure 3:** Management of patients after an electrical injury concerning decision making on hospitalization and cardiac monitoring or safe discharge [from references 2, 3–5, 7, 11–14, 39].

Thus, the ECG abnormalities can mimic coronary ischaemia and coronary angiography should therefore be considered if an acute coronary syndrome is suspected. Creatine kinase MB elevation is not useful in the diagnosis of myocardial injury, since it lacks specificity as a result of the frequent concomitant rhabdomyolysis [1, 2]. The role of cardiac troponins (cTn) remains undetermined, their specificity being also possibly affected by rhabdomyolysis [31]. Indeed, in several series, the proportion of patients with cTn elevation, generally of mild amplitude, varied between 0 and 65% [3–5, 12, 32]. A small study of patients suffering from high-voltage electrical injuries revealed that minor elevations of cTnI (in 13 out of 20 patients) was not associated with impairment of left ventricular contractility, evaluated through speckle tracking echocardiography [32]. In cases of suspected myocardial damage, an echocardiogram or, in selected patients, cardiac magnetic resonance imaging (MRI) should be carried out. Our patient had a slight elevation of high-sensitivity cTnI that we considered nonspecific, and possibly related to rhabdomyolysis or fast atrial fibrillation, given the normal echocardiogram and absence of electrocardiographic signs of ischaemia. Several cases of myocardial dysfunction after an electrical injury have been reported [1, 2, 16, 33–36]. Myocardial dys-

function is usually reversible, but cardiac contraction abnormalities may sometimes persist [33–36], emphasising the importance of adequate follow-up of these patients.

A frequent concern is the need for hospital admission and cardiac monitoring after an electrical shock. The fear is of delayed life-threatening arrhythmias, as they have been reported in a small number of cases. One study described three patients with severe ventricular arrhythmias (ventricular fibrillation, ventricular tachycardia and ventricular parasystole) detected 8 to 12 hours after the electrical accident [37]. Another young patient with a history of palpitations was found dead 10 hours after an electrical shock [15]. Nevertheless, no ECG immediately after the accident was available for these four cases. Finally, a patient admitted after an electrical injury with first-degree atrioventricular block developed complete atrioventricular block followed, after 24 hours, by ventricular fibrillation [38]. On the other hand, in several series of patients admitted for cardiac monitoring with a normal initial ECG, no delayed arrhythmias were noticed [3–5, 7, 11–14]. Therefore, late arrhythmias with a normal initial ECG should be considered a very rare entity. The European Resuscitation Council guidelines recommend cardiac monitoring, usually for 24 hours, in the following situations: history of cardiorespiratory problems, cardiac arrest, loss of consciousness, abnormal ECG, arrhythmias, soft tissue damage and burns [39]. It also appears reasonable to monitor patients after high-voltage electrical injuries, which usually carry a higher morbidity, and patients with significant troponin elevation [2]. In pregnant women, electrical injuries involve the risk of complications to the fetus, therefore fetal cardiac monitoring has been suggested [2]. Conversely, patients without the above-mentioned risk factors and with a normal ECG can be safely discharged home [2, 3–5, 7, 11–14, 39] (fig. 3). In conclusion, patients surviving an electrical shock can develop cardiac complications, as our rare case of atrial fibrillation illustrates. These manifestations are usually reversible, but in rare cases long-term consequences are described emphasising the importance of an individual follow-up.

#### Disclosure statement

No financial support and no other potential conflict of interest relevant to this article was reported.

#### Authors' contribution

MD and MRJ contributed equally as first authors

#### References

The full list of references is included in the online version of the article at [www.cardiovascmed.ch](http://www.cardiovascmed.ch).

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