CASE REPORT
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Electrical shock-induced atrial fibrillation

Fibrilacija pretkomora uzrokovana strujnim udarom

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Abstract

Introduction. An electrical injury can cause various cardiac arrhythmias, such as asystole, ventricular fibrillation, sinus tachycardia, and heart blocks. However, it rarely causes atrial fibrillation (AF). **Case report.** The 47-year-old patient was admitted to the Emergency Department after receiving an electric shock (< 600 V). He subsequently lost consciousness, fell, and sustained back and head injuries. During the examination, the heart rate was irregular but with no heart murmurs. There was an entry wound on the front of the left thigh and an exit wound on the front of the neck. An electrocardiogram showed newly appearing AF. The laboratory tests showed no pathological deviation, and focus cardiac ultrasound showed that contractile force was preserved with no wall-motion abnormalities and normal left atrium dimensions. The patient was administered low-molecular-weight heparin subcutaneously and propafenone (600 mg) orally. At follow-up after 24 hrs, the electrocardiogram showed normal sinus rhythm. **Conclusion.** We reported a rare case of an electricity shock-induced AF, which was converted to sinus rhythm with the help of drug therapy. Although most cases of electricity shock-induced AF represent benign conditions that are self-limited, cardiac monitoring as a routine measure should be considered.

Key words:
anti-arrhythmia agents; atrial fibrillation; drug therapy; electric injuries; electrocardiography.

Apstrakt

Uvod. Strujni udar može dovesti do različitih poremećaja srčanog ritma poput asistolije, ventrikularne fibrilacije, sinusne tahikardije i srčanih blokova. Ipak, nastanak atrijalne fibrilacije (AF) kao posledice strujnog udara, dešava se retko. **Prikaz bolesnika.** Bolesnik, star 47 godina, primljen je u ambulantu nakon strujnog udara (< 600 V) koji je dobio. Nakon strujnog udara je izgubio svest, pao i zadobio povrede leđa i poglavine. Tokom pregleda, detektovana je aritmična akcija srca, ali bez šumova. Na levoj butini je registrovana ulazna, a na vratu izlazna rana. Elektrokardiogramom je registrovana novonastala AF. U urađenim laboratorijskim analizama nije bilo patološkog odstupanja, dok je ehokardiografskim pregledom registrovana očuvana kontraktilna snaga srca, bez poremećaja segmentne kinetike, uz normalne dimenzije leve pretkomore. Ordiniran je niskomolekularni heparin supkutano i 600 mg propafenona oralno. Nakon 24 h na elektrokardiogramu je registrovan normalni sinusni ritam. **Zaključak.** Prikazali smo redak slučaj AF uzrokovane električnom strujom koja je konvertovana u sinusni ritam primenom antiaritmika. Iako većina ovako nastalih epizoda AF predstavlja prolazni poremećaj srčanog ritma sa spontanom konverzijom, neophodno je rutinsko praćenje ovih bolesnika.

Ključne reči:
antiaritmici; fibrilacija pretkomora; lečenje lekovima; povrede električnom strujom; elektrokardiografija.

Introduction

Atrial fibrillation (AF) is the most common sustained arrhythmia at discharge from hospital [1]. The etiopathogenesis of AF is rather complex and usually multifactorial [2]. Arterial hypertension, valvular heart disease, and heart failure are listed as the most common causes of AF. An electrical injury can cause various cardiac arrhythmias, such as asystole, ventricular fibrillation, sinus tachycardia, and heart blocks. However, it rarely causes AF [3]. We present a case of a patient with electrical injury-induced AF as a consequence of an occupational accident.

Case report

The 47-year-old patient was admitted to the Emergency Department of the Institute for Treatment and Rehabilitation "Niška Banja" because he was feeling palpitations, dizziness,

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and instability when standing and walking. The patient stated that he had come into direct contact with an exposed wire while using a pool cleaning machine, thus receiving an electric shock (< 600 V). He subsequently lost consciousness, fell, and sustained back and head injuries. He was unaware of how long he had been unconscious, but he felt irregular heart rate, dizziness, and exhaustion immediately after regaining consciousness. The patient confirmed that he was not suffering from any disease and had not been prior hospitalized or clinically examined. In addition, he denied the consumption of alcohol and the intake of medications and psychoactive substances.

During the examination, the patient was conscious, oriented, eupnoeic, and had normal skin color. There was an entry wound on the front of the left thigh and an exit wound on the front of the neck (Figure 1). There were no pathological findings in the lungs. His heart rate was irregular but with no heart murmurs. Vital parameters were as follows: blood pressure 120/80 mm Hg, heart rate 80 bpm, SaO_2 98%, body temperature 36.6 °C, and respiration rate 12 per min.

An electrocardiogram (ECG) showed previously nonexisting AF (Figure 2). The laboratory tests, which included cardiac-specific enzymes, troponin, electrolytes, complete blood analysis, and thyroid hormones, showed no pathological deviation. Focus cardiac ultrasound showed that contractile force was preserved with no wall-motion abnormalities and normal left atrium dimensions.

The patient was administered low-molecular-weight heparin (enoxaparin) subcutaneously and propafenone (600 mg) orally (pocket therapy). Wounds were treated, and an antibiotic was administered. At follow-up after 24 hrs, an ECG showed normal sinus rhythm (Figure 3). Once again, laboratory tests confirmed normal values. The patient was discharged in a stable state. A decision was made not to continue antiarrhythmic and anticoagulant therapy (CHA2DS2VASc score 0, HAS-BLED score 0).

Three months later, a 24 hrs ECG Holter monitoring showed normal sinus rhythm. Moreover, a follow-up cardiac ultrasound showed preserved ejection fraction, with neither valvular heart disease nor segmental wall-motion abnormalities, with the left atrium of 36 mm. A submaximal exercise stress test did not show signs of myocardial ischemia or arrhythmias.

Discussion

An electric shock can lead to myocardial necrosis, left ventricular dysfunction, arrhythmia, and conduction disorders [4]. The most common arrhythmias are sinus tachycardia, sinus bradycardia, ventricular fibrillation, and asystole, whereas the most prevalent conduction disorders are branch blocks, AV blocks of different degrees, and QT interval prolongation. On the other hand, it is quite rare for an electric shock to cause AF [5].

The mechanism of AF occurrence as a consequence of an electric shock is complex and only briefly examined.

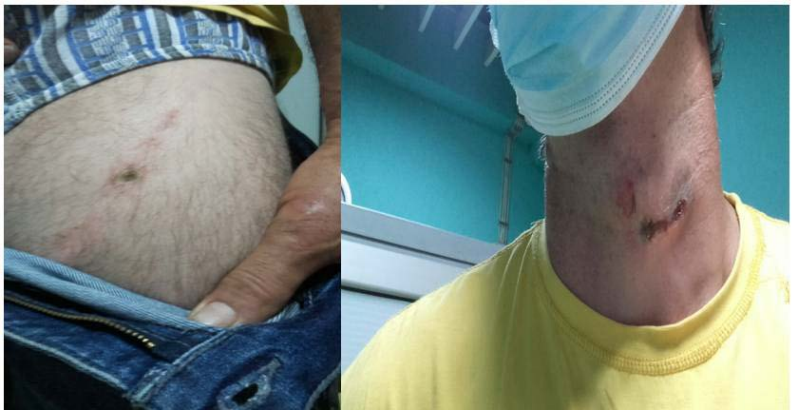

Fig. 1 – The entry wound on the left thigh and exit wound on the neck.

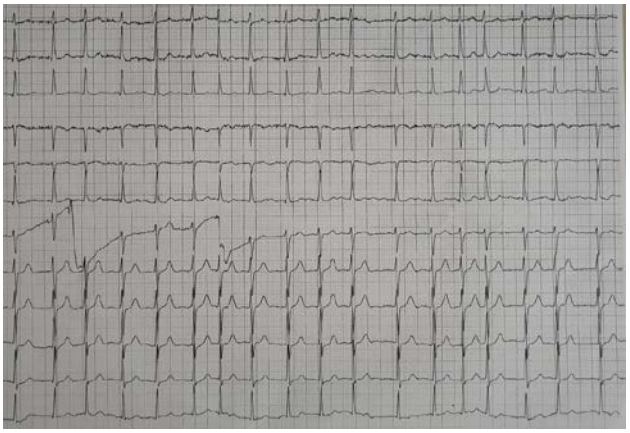

Fig. 2 – Electrocardiogram after the electrical injury.

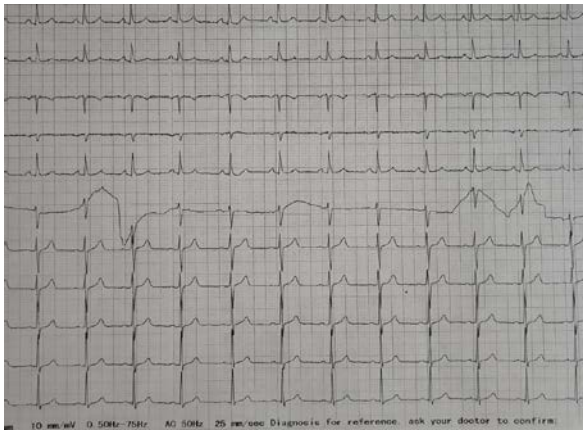

Fig. 3 – Electrocardiogram 24 hrs after the electrical injury.

Blood is an excellent conductor of electricity, and the heart is one of the most vulnerable organs when it comes to the impact of electric current. An electrical injury can lead to myocardial necrosis with subsequent fibrosis that may become a chronic arrhythmogenic focus [6]. Furthermore, an electric shock can disrupt the sodium-potassium pump with a subsequent increase in the concentration of potassium, which can potentially lead to a change in the permeability of cardiomyocytes and, therefore, to myocardial depolarization [7].

Other pathogenic mechanisms, such as coronary spasm, catecholamine release, and coronary hypoperfusion due to arrhythmia-induced hypotension are uncommon in the basis of the pathogenesis of electric shock-induced AF [8].

Which type of arrhythmia or conduction disorder exactly occurs as a consequence of an electric shock depends on the intensity of the electric current and its type (direct and alternating), the surface area of the body coming into contact with the electric current, the duration of the contact, and the state of the patient [9]. The changes caused by high voltage currents are usually complex and pronounced, whereas those caused by low voltage currents (< 600 V) are likely benign and transitory, which was the case with our patient. Most AFs after electric shocks are self-limiting [10], especially when caused by low voltage current [3]. Moreover, no AF relapse has been reported so far. However, Boggild et al. [6] reported a case of electric shock that caused AF for over 20 years, supporting the notion of chronic damage to the left atrium. That makes cardiac monitoring a necessary routine measure.

Various treatment methods have been developed to deal with electrical shock-induced AF but with a lack of proper guidance and protocols. These include DC cardioversion, pharmacological reversion, or even simple waiting for spontaneous resolution.

Conclusion

We report a rare case of an electrical injury-induced AF, which was converted to sinus rhythm by applying drug therapy. Although most cases of electrical injury-induced AF represent self-limited benign conditions, cardiac monitoring as a routine measure should be considered.

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