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Ischemic Stroke Induced by an Electric Shock in a Patient with Ischemic Cardiomyopathy: An Unusual Association of Events

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Abstract **Case Report**

Electrocution is a well-known accident that can lead to a highly variable clinical presentation, including cardiac and neurological complications. Some studies have shown that electric shock contributes to arterial ischemia; either through vasospasm or by inducing thrombotic reactions. Here, we present a 66-year-old male patient with unrecognized cardiac disease who developed brachio-facial paresis related to ischemic stroke immediately after being electrocuted with domestic electricity.

Keywords: Ischemic stroke; ischemic cardiomyopathy, electric shock, echocardiography, electrocution.

INTRODUCTION

The severity of electrocution injuries depends on the strength, type of circuit of the current and duration of contact. Electrocution is a well-known accident that can result in a highly variable clinical presentation, ranging from a simple benign accident to a life-threatening cardio-neurological condition [1].

The primary cause of death in this context of electric shock is cardiopulmonary arrest. Electric shock (ES) can cause damage to organs such as the heart, kidneys, skin, muscles, blood vessels and nervous system. Electrical currents in the home are of low voltage. Concerning the high voltage currents often cause serious complications with a high mortality rate [2].

Myocardial damage and ischemic stroke are rarely reported after being exposed to an ES. It may be either isolated cardiac damage, isolated cerebral damage, or both at the same time [3].

In this observation, we report the case of an ischemic stroke that occurred immediately in a male patient who had experienced an ES at home and in whom investigations had shown the evidence of previous cardiac disease.

CASE REPORT

66-year-old male patient with a history of chronic smoking. Two months ago, he had a sudden onset of right brachio-facial weakness, which resolved completely after 1 week. The patient did not consult a physician during this neurological episode. He was recently admitted to the emergency department of our hospital because of sudden onset of left brachio-facial weakness following an ES accident at home (220 volts) which occurred 3 days ago. There was no evidence of seizure, muscle or chest pain, altered consciousness or headache. It is worth highlighting that the patient developed this recent muscular weakness immediately after the electrocution.

Clinical examination revealed a well-conscious patient who had a supple neck with good spatial and temporal orientation. Neurological examination revealed left brachio-facial paresis with dysarthria. On cardiological assessment, the patient was found to be hemodynamically stable with a blood pressure of 125/80 mmHg, a heart rate of 70 bpm and an oxygen saturation of 95% on room air. Cardiac auscultation was normal. There was no evidence of heart failure. Peripheral pulses are easily palpable without murmur on auscultation along the major vascular axes. Dermatological examination revealed superficial erythematous skin lesion on the back of the right hand related to the site of electrocution. The remaining of somatic examination was normal.

In view of this neurological presentation, a brain MRI angiogram was performed (Figure1), showing a bilateral superficial sylvian ischemic stroke; new on the right with a hemorrhagic component and old on the other side.

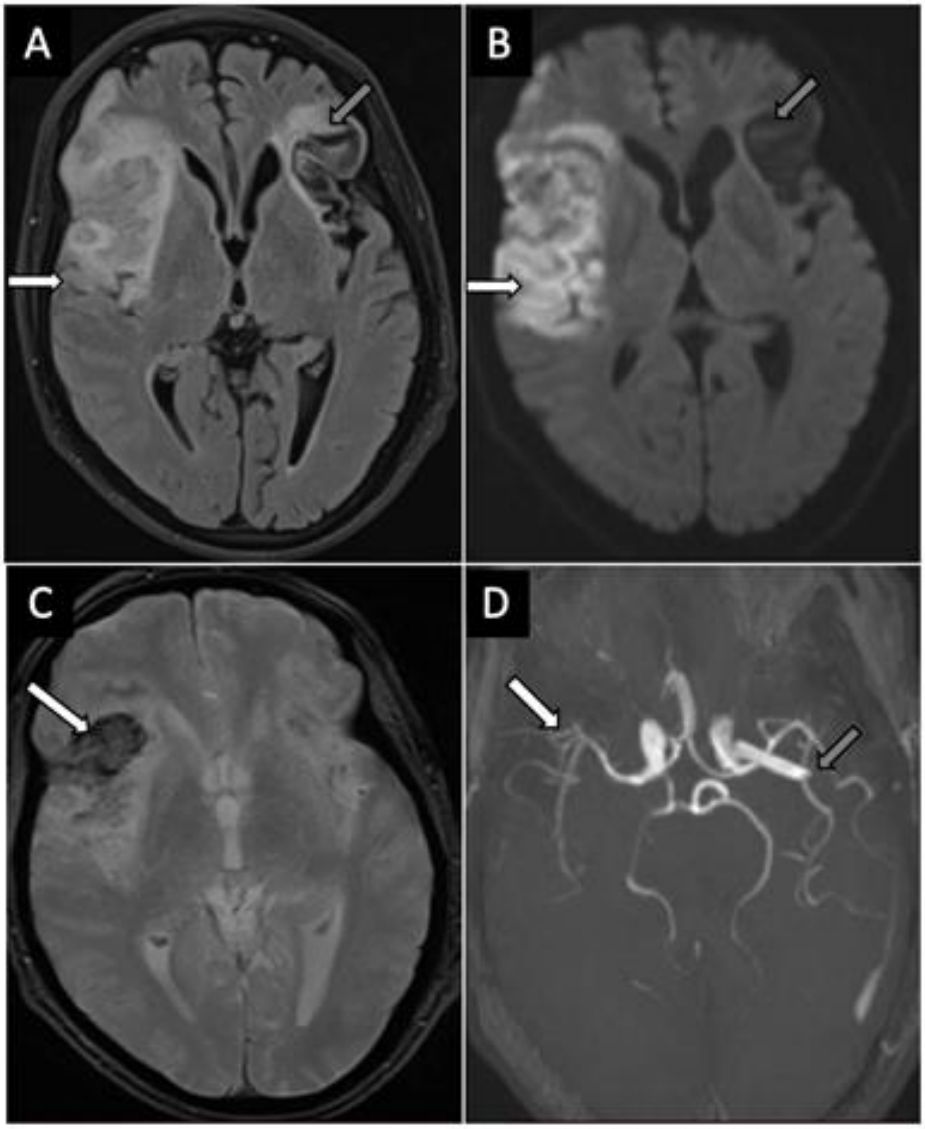

Figure 1: Axial brain MRI (A: FLAIR sequence, B: diffusion sequence, C: echo-gradient sequence) and TOF image: showing a new sylvian ischemic stroke on the right side with hemorrhagic infarction (white arrows) and an old sylvian stroke on the left side (grey arrows)

Electrocardiogram (Figure 2) showed regular sinus rhythm with a heart rate of 70 cycles per minute. In addition, we noted sequela of anteroseptal necrosis without conduction disturbance. Transthoracic echocardiography showed a slightly dilated left ventricle and segmental impairment of myocardial contractility in the left ventricle with akinesia of the anteroseptal wall and lateral hypokinesia (Figure 3; A, B, C). Left ventricular ejection fraction is severely impaired, estimated at 30% on Simpson's biplane. Global strain was estimated to be 5%. The right ventricle is of normal size with preserved systolic function. There is no pulmonary hypertension or intracavitary thrombus. The valve structures are thin and flexible. Left and right atria of normal size. The rest of the examination was unremarkable.

Arterial Doppler of the supra-aortic trunk showing slight atheromatous plaques, which is not significant throughout the arterial network (Figure 3 D).

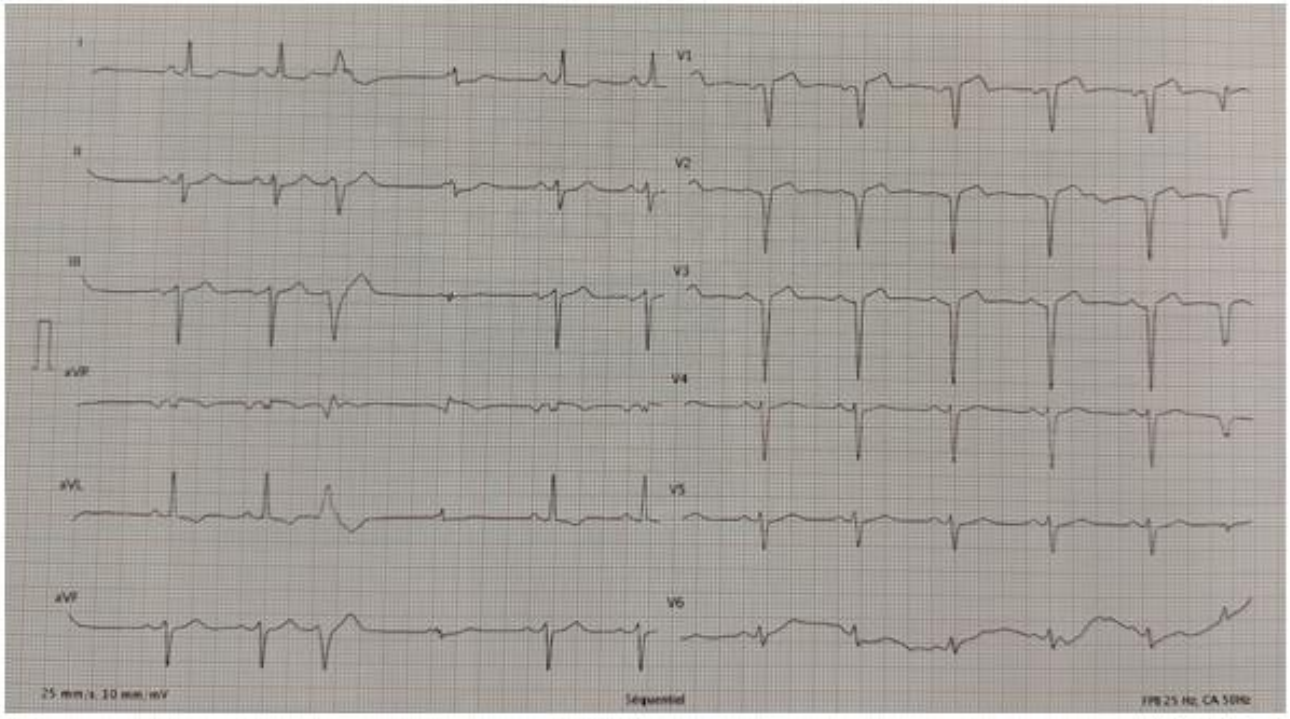

Figure 2: Electrocardiogram showing signs of old myocardial ischemia with normal PR interval. There is ventricular extrasystole with left anterior hemi-block

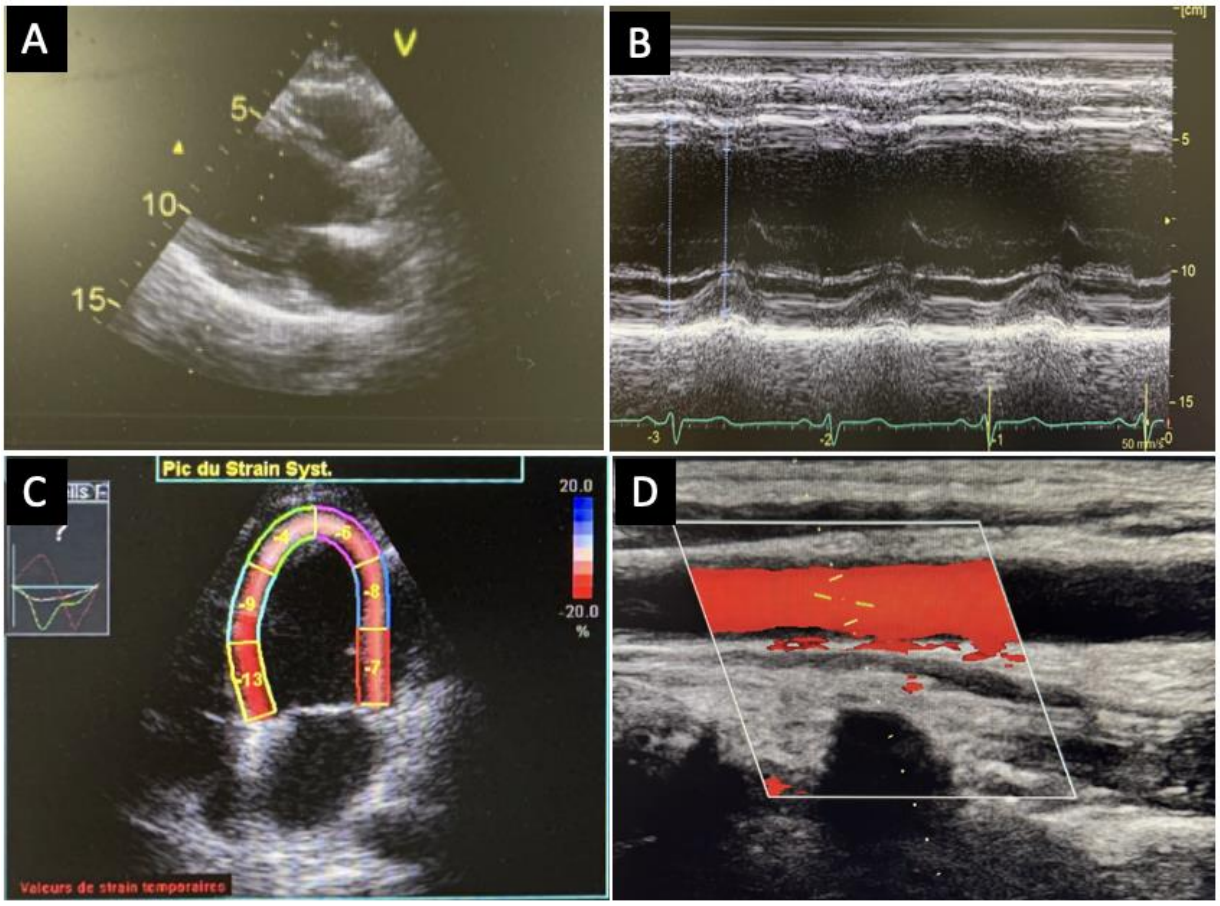

Figure 3: A, B, C images: transthoracic echocardiography images demonstrating a dilated left ventricle with dysfunction. D: Arterial echodoppler of the left carotid artery showing slight atheromatous deposits

Creatine kinase, troponin with extensive biological tests were normal (complete blood count, prothrombotic tests, hepatic and renal tests, hemoglobin electrophoresis, glycemia, autoantibody tests, syphilis serology, hepatitis and HIV tests).

The patient did not show any electrical changes in the ECG monitoring, and no rhythm or conduction abnormalities were observed. Biologically, troponin and CPK levels were normal in follow-up tests.

After stabilization, coronary angiography was performed and showed intermediate lesions in the right coronary artery and in the middle interventricular artery which required medical treatment. The rest of the coronary network was normal.

The patient was treated with aspirin (150 mg/day), rosuvastatin (10 mg/day) and physical therapy. The course on day 10 was characterised by complete recovery of the motor deficit.

DISCUSSION

The nervous system, blood vessels and mucous membranes are more susceptible to being injured because they offer less resistance to the flow of electricity [2]. Neurological complications due to electrocution are multiple and may include peripheral and central nervous system. Acute ischemic stroke due to ES are uncommon and rarely reported as case reports and small series. Most commonly, they are reported in young men who have suffered a low-voltage shock [2].

Cardiovascular complications of ES occur in approximately one-third of cases and may include acute arterial hypertension, pericarditis, heart failure, myocardial ischemia and necrosis, conduction disorders, arrhythmias and cardiac arrest [4, 5].

Generally, ischemic lesions and myocardial necrosis in the setting of ES involve non-systematized territories that do not belong to a coronary territory. This finding was not seen in our patient because he who had anteroseptal and lateral involvement of the anterior interventricular and circumflex artery territories on echocardiography [6].

Some studies have shown that ES contributes to arterial ischemia, either through vasospasm or by inducing thrombotic reactions 15, 16. In a study conducted by Hunt and colleagues about vascular lesions after electrocution; a vasospasm was demonstrated on angiograms of 8 cases among 12 patients [3].

Based on the clinical, electrical, biological and echocardiographic data in our case; it is most likely that our patient had an unrecognized chronic coronary syndrome which was discovered after the electrocution. Concerning the ischemic stroke; the most likely cause was the electrocution due to the immediate stroke after ES. In addition, this brain complication was likely precipitated by the cardiac disease.

All reported cases of ischemic stroke after ES were treated according to standard guidelines for the treatment of acute stroke [2].

Most patients had a favorable outcome with clinical improvement, which is consistent with our clinical case [2].

CONCLUSION

The manifestations of electrocution are diverse and can sometimes be fatal. Our case is interesting because, on the one hand, it shows that electric shock can cause an ischemic stroke and, on the other hand, it shows how to make a good clinical and paraclinical approach to maintain or exclude the involvement of ES in the myocardial lesions. Our patient's myocardial infarction was old and unrecognized until he developed an ischemic stroke after electrocution.

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