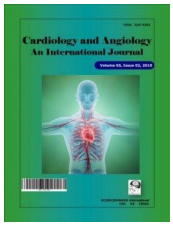

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Death Due to Low Voltage Electric Shock Induced Myocarditis

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Authors' contributions

This work was carried out in collaboration between all authors. Author RK wrote the first draft of the manuscript. Authors RK and RSP managed the literature searches. Authors RSP and DH revised the manuscript. All authors read and approved the final manuscript.

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Case Report

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ABSTRACT

Low voltage electric shock resulting in myocarditis induced delayed death is a rarity and has not been reported so far, to the best of our knowledge. The definitive diagnosis is autopsy based as it has variable clinical presentations. We report such a case where in the histopathologic findings of myocarditis came as a surprise during microscopic evaluation of the autopsy sections in a case with an apparently normal heart on gross examination. The present case mandates a careful microscopic examination of autopsy sections in cases of electrocution.

Keywords: Myocarditis; electrical burn; heart; autopsy.

1. INTRODUCTION

Heart is characteristically vulnerable to electrical injury because of the electromechanical nature of its work dynamics. Direct necrosis of the myocardium, cardiac dysrhythmia, asystole and ventricular fibrillation are the cardiac complications of electrical injury of serious nature [1,2]. Sinus tachycardia, supraventricular tachycardia, bradycardia, sick sinus syndrome, atrial fibrillation and nonspecific ST-T wave changes; conduction defects like heart block,

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prolongation of the QT interval and bundle branch block are all the aftermaths of electrocution.

Dilated cardiomyopathy, pericardial effusion, acute myocardial infarction, cardiac rupture, and left ventricular failure due to cardiac fibrosis after electric injury, have been reported [1]. Delayed death due to myocarditis, as a consequence of low-voltage electricity shock, is a rare event. We report such a case.

2. CASE REPORT

A 22-year-old unmarried male hailing from North India was brought to the hospital in unconscious state after an alleged history of incurring an electric shock due to short circuit in a factory machine. He was a worker in a food processing factory. Cardiopulmonary resuscitation was not given to the patient after the accidental electric shock. He was put on mechanical ventilation in the intensive care unit. The electrocardiogram (ECG) revealed sinus tachycardia with non specific ST, T wave changes and occasional premature ventricular contractions. After a week, the patient was tracheostomised and mechanical ventilation was continued for 3 more days after which he was put on spontaneous ventilation with oxygen supplementation through tracheostomy T-piece. During this period the sensorium of the patient remained altered. He could not be revived and died a month after the electric shock, due to heart failure. History elicited from the relatives failed to reveal any flu like symptoms and the deceased was apparently healthy with no history of any medication. There was no history of alcoholism/drug abuse.

On autopsy, there were no effusions. Uncut heart, portions of; lungs, liver, spleen, kidneys and brain were subjected to histopathology. The heart measured 10 x 8.5 x 4.0 cm and weighed 220 gm. All the chambers were grossly normal. The circumferences of the aortic, pulmonary, mitral and tricuspid valve were within normal limits. There were no pale or necrotic areas in the myocardium to suggest myocardial infarction. The coronaries were traced as far as possible and were found patent. On microscopy, there were multiple foci of necrosis of the cardiac myocytes with lymphomononuclear cell infiltrate in the myocardium (Fig. 1) Sections from rest of the heart showed no significant pathological change.

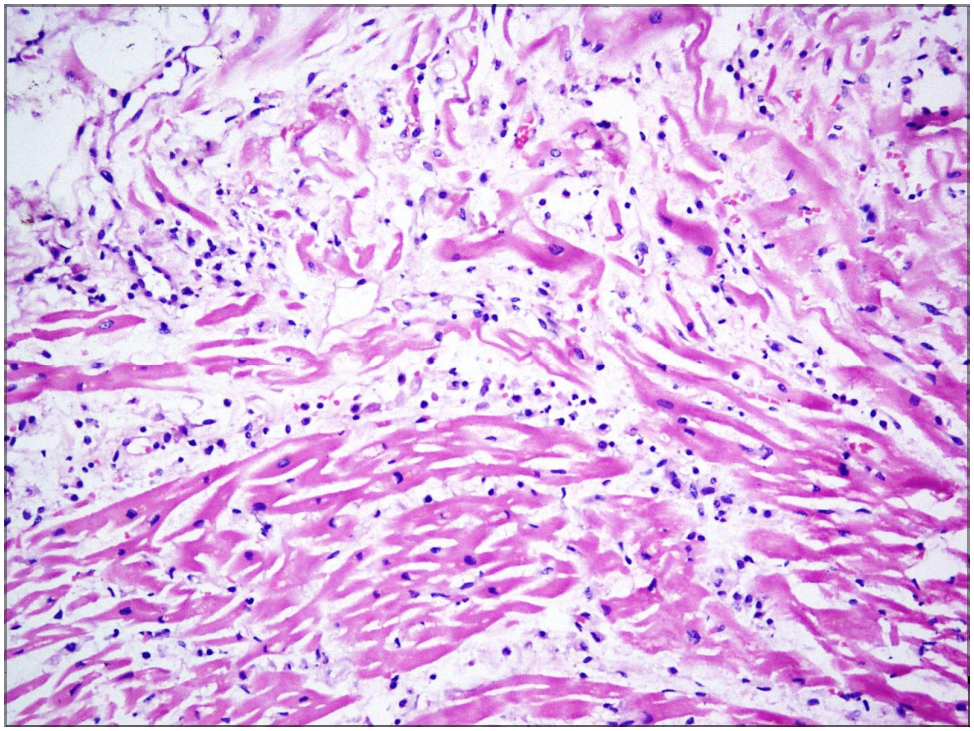

Fig. 1. Histopathologic section showing lymphomononuclear cell infiltrate in the myocardium along with patchy myocytolysis (H&E, x20)

Pieces from both lungs together measured 12 x 8.0 x 2.5 cm and were subcrepitant. On microscopy there was pulmonary oedema. The inter-alveolar septa were widened and showed congested blood vessels. The alveoli had eosinophilic proteinaceous edema fluid in their lumina. Sections from the liver showed maintained hepatic lobular architecture. The central veins and sinusoids were dilated and congested. There was necrosis of the centrilobular hepatocytes. The other viscera did not show any abnormality both on gross and microscopy. The final histopathologic impression was myocarditis (lymphocytic), with pulmonary edema and chronic venous congestion of liver.

3. DISCUSSION

Electricity can be either of high voltage (≥1000 V) or low-voltage (≤350 V) [3]. Life threatening cardiac dysrhythmias with loss of consciousness and ventricular asystole are likely to occur in persons experiencing high-tension electrical injury. Dilated cardiomyopathy has been described as sequelae of both low and high voltage electric current. The gravity of involvement of the heart is dependent on a plethora of factors which include voltage, magnitude, current frequency, duration and location of the entrance and exit wounds.

A large study spanning over a long period of time concluded that patients in good health without co-morbidities, having normal ECG findings and no loss of consciousness resulting from low voltage electricity, are unlikely to develop serious cardiac dysrhythmias and hence require no observation in the medical emergency [4]. Our case was without any co-morbidity but the ECG revealed sinus tachycardia with non specific ST, T wave changes and occasional premature ventricular contractions. The patient died a month after the low voltage electric shock, due to myocarditis.

Fatal myocarditis has generally uncharacterized epidemiological profile in general population owing to the variability in clinical presentations and demonstrable findings and the definitive diagnosis being available largely on autopsy [5]. The reported frequency of myocarditis in large autopsy studies ranges from 0.11-0.55% in the general population [6]. The clinical presentations of myocarditis range from being asymptomatic to mild “flu-like” symptoms to life threatening events like cardiogenic shock [6,7]. Abnormalities that may be observed on ECG are ST segment and T wave deviations. About one third of patients experience chest pain. Clinical manifestations akin to acute myocardial infarction may be seen on occasion. Less than half of the patients have left ventricular dysfunction. The initial presentation may be syncope with A-V block or ventricular arrhythmias and sudden cardiac death from complete heart block or ventricular tachycardia can also occur in some. Approximately 10% of recent-onset cardiomyopathy in adults, in the absence of structural heart diseases, is attributable to myocarditis [7]. A meta-analysis on patients with myocarditis reported spontaneous improvement in about half of the patients, a downhill course in 15 percent and subsequent mortality in up to 30 percent cases [8,9].

Cardiac involvement in electrocution is unpredictable and may affect the myocardium or the conduction system of the heart per se, causing sudden cardiac death or delayed mortality. Life-threatening ventricular arrhythmias develop due to unstable myocardial substrates; inflammatory infiltrate, interstitial edema, necrosis of cardiomyocytes and fibrosis [10]. The gross appearance of the heart is not characteristic in myocarditis and it can be normal. The coronary arteries are usually patent. The heart was grossly normal with patent coronaries, in our case also. It was only during the microscopic evaluation that the features of myocarditis were demonstrable.

There was a lack of standardized histological criteria for the autopsy diagnosis of myocarditis till the ‘Dallas’ and ‘Marburg WHO’ criteria were proposed [11]. Myocarditis, by definition, is a process characterized by inflammatory infiltrate in the myocardium in association with myocyte necrosis and/or degeneration which is not typical of ischemic damage. Multiple foci of necrosis of cardiac myocytes and lymphomononuclear cell infiltrate in the myocardium were seen in the current case. The wide etiologic spectrum of myocarditis comprises of infectious and non-infectious causes (drugs, toxins, or autoimmune disorders) [7]. History elicited from the relatives in our case failed to reveal any flu like symptoms and the deceased was apparently healthy with no history of any medication/ alcohol/drug abuse. Histopathology remains the gold standard for a confirmatory diagnosis of myocarditis either on endomyocardial biopsy or autopsy specimens [6].

4. CONCLUSION

The histopathologic autopsy diagnosis of myocarditis came as a surprise in the index case, emphasizing the importance of careful microscopic examination in cases with electrocution and an apparently normal heart on gross evaluation. Furthermore, myocarditis causing mortality subsequent to low voltage electric shock, although uncommon, needs to be considered in all cases of delayed death due to electricity.

CONSENT

All authors declare that written informed consent was obtained from the next of kin for publication of this case report and accompanying images.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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