

High Tension Electric Current Injury and Silent Myocardial Infarction- A Case Report

Dr. R.N. Das MD, MRCP and Dr. Jaya Kumar MD

Department of Medicine, Manipal College of Medical Sciences, Pokhara, Nepal

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ABSTRACT: A 55-year-old male, non-diabetic, sustained severe electric current injury as evidenced by the grievous exit wound on the left dorsum of foot as well as entry wound in both palms. There was silent anterior wall myocardial infarction, discovered from incidental electrocardiograph.

KEY WORDS: Electric current injury, grievous exit wound, silent myocardial infarction

INTRODUCTION:

A variety of cardiac and non-cardiac abnormalities have been described following low voltage [<1000 volts] alternate current household [220-240 volts] electric shock¹. Among cardiac abnormalities, most lethal is sudden death owing to asystole or ventricular fibrillation. Electrocardiogram may show tachycardia, ST-segment changes, arrhythmias, rarely bundle branch block or complete heart block. Infrequently, acute infarction is also noted².

CASE REPORT:

A 45-year-old man sustained electric current injury on a rainy day, when by mistake the hand held iron rod fell on a live naked road-side high tension electric wire. He noticed loss of movement of left foot and pain in left hip but no angina. Local examination revealed 5cmX 3cm transversely placed linear white, swollen marks in both palms and a charred ulcer 4 cmX5 cm with raw necrotic base, black rolled out margin on left foot and toes [Fig1]. The systemic examination and vitals were normal. Biochemistry and hemogram were normal except

raised creatinine phosphokinase and creatinine phosphokinase myocardium-bound 4567 IU/ml and 14 IU/ml respectively. Electrocardiogram [Fig 2] showed ST elevation and T inversion in C1-C5. Echocardiogram showed left ventricular septal hypokinesia and anteroseptal infarction. He was observed and treated conservatively with aspirin, enoxaparin, β -blocker and alprazolam.



Fig 1: Entry and exit wound

Corresponding Author: Dr R N Das, Associate Professor, Department of Medicine, Manipal College of Medical Sciences, P.O. BOX: 341, Pokhara, Nepal. E-Mail: das_drdeepak2004@yahoo.com

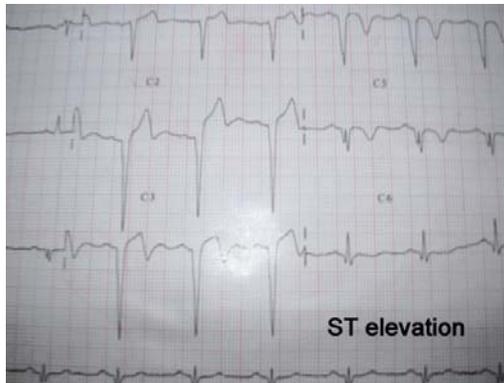


Fig 2: Anterior wall ST elevation and T inversion

DISCUSSION:

Electric current induced tissue damage depends upon amount of electric energy, duration of current, resistance offered by skin and the pathway of electric current³. Cardiac tissue is damaged directly by the passage of electric current and indirect damage is caused by altered serum potassium. Initially, there is hyperkalaemia owing to necrosed myocardium. In 2nd to 4th week, there is idiopathic hypokalaemic arrhythmia. Patients should therefore be closely observed for 4 weeks⁴.

The structural cardiac damage is localized necrosis and persists for weeks. It manifests as ST elevation and raised cardiac enzymes. The altered functions of myocardium due to changed cell membrane permeability by electric current persist for a short period. Victims of low voltage electric current frequently develop ventricular fibrillation which requires immediate defibrillation⁵.

The pathogenesis of electric current induced infarction is controversial. Besides damaging the coronary walls, it also has a direct thrombogenic effect. In the present case, this appears to be a possible mechanism in which myocardial lesions corresponded to acute occlusion of left anterior descending artery perhaps favored by presence of an atheromatous plaque. Here coronary arteriography would have been the most useful investigation.

Population studies suggest that many [20-60%] nonfatal infarctions, passed unrecognized by patients and are discovered only on routine electrocardiogram. Of these infarctions, half are truly silent as patients are unable to recall angina. The other half can recall angina when leading questions are posed⁶. Silent infarctions usually

occur in elderly, hypertensive and diabetics without any antecedent angina.

According to Cohen silent infarction patients are having *defective anginal warning system*⁷. Maseri has proposed that silent ischemia results from a combination of decreased sensitivity to painful stimuli and coronary microvasculature dysfunction⁸. There are many hypotheses to explain this type of silent or painless infarction e.g., abnormal autonomic neuropathy of diabetics, higher than normal pain threshold and lower reaction in elderly hypertensive, individual variation of higher pain threshold for different types of pain stimuli and variable levels of pain threshold-riser endogenous opioids and endomorphins.

CONCLUSION:

We infer that this type of silent infarctions occur rarely by electric current due to negligent handling of high tension live wires causing unexpected death. Secondly, local electrical department may be accountable for installing such road side electrical wires near residential places. Thirdly, attending doctors should observe such patients critically before instituting costly and unnecessary thrombolytics.

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