MYOCARDIAL INJURY IN ELECTRICAL BURNS

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Introduction

Electricity has been a boon to mankind. The technological advancements and increasing use of electricity have however resulted in many accidental injuries. Electrical injuries comprise 2-8% of all acute burn unit admissions and 15% of deaths [1]. In United States approximately 1000 individuals die each year from electricity related injuries [2]. Majority of those affected are young males between 20-34 years of age. One third of the patients manifest a cardiologic component and many of them die as a result of it [3]. The extent of the problem in India is not known. We describe a case of high voltage electrical injury and discuss the problems encountered in its management.

Case Report

25 year old male construction worker sustained accidental electrical shock while working on a construction site, from an overhead high tension wire (33000V AC). The electric arcing set his clothes on fire and he fell down unconscious on the ground five feet below. Patient was thereafter rescued by bystanders and brought to the hospital within 30 minutes. At the time of admission he was conscious, confused, uncooperative and violent. He was unable to recall the events leading to the injury. He denied having chest pain or dyspnoea. Examination and resuscitation was possible only after sedating and restraining him. The pulse was 104/minute and blood pressure 120/90 mm of Hg. There was no entry or exit wound. The face (including eyebrows), neck, thorax, upper abdomen and thighs were burnt. These comprised 50% body surface area. Burns were full thickness in lower limbs. He also had a lacerated wound (1 cm x 0.25 cm) on the right frontal region. Examination of the abdomen, cardiovascular and respiratory system was normal. There was no congestive heart failure, focal neurological deficit or features of smoke inhalation. A large bore intravenous line was established and an indwelling urinary catheter was placed. The urine was reddish brown in colour (Fig.1) suggesting myoglobinuria. The cutaneous burn was debrided and antibiotic cream was applied locally. Tetanus prophylaxis was updated. Patient received 14 litres of Ringer Lactate on the first day and 10 litres on second day. The investigation results were as follows : Hb 18gm/dl; TLC 8200/cmm; Urine haemoglobin +, albumin +++, 2-4 pus cell; Total protein 9 gm/dl; Blood urea 28 mg/dl; Serum creatinine 0.8 mg/dl; Sodium 129 mEq/L; Potassium

5.9 mEq/L; Chest radiograph NAD. The Electrocardiogram (ECG) (Fig-2) showed pathological 'Q' waves and inverted 'T' waves in inferior leads. The serum transaminases were normal. Urine output was initially maintained at 75 ml/h, however it gradually diminished over 24-h, despite adding a diuretic. Patient became febrile on the second day and Inj Cefotaxime 1 gm 6 hourly was exhibited empirically pending culture results. Upper gastrointestinal haemorrhage and hyperkalemia (S. Potassium 6.5 mEq/L) further complicated the course of illness. A glucose insulin drip was added. Periodic evaluation did not reveal evidence of compartment syndrome. The condition of patient progressively worsened and he died 36 hours after admission due to acute renal failure and septicemia.

Fig. 1 : Sample of Burgundy coloured urine suggestive of myoglobinuria

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Fig. 2 : Electrocardiogram shows pathological 'Q' waves and 'T' wave inversion (arrowhead) in II, III and AVF.

Discussion

Of the 33 cases of burns admitted and treated at this hospital since 1993, only 2 (6%) cases had electrical burns. Although electrical burns constitute a mere 3% of all burns [4,5], there are significant differences between them and conventional thermal burns. Cardiac arrest, acute renal failure, amputation rate and delayed onset organ dysfunction occur in greater frequency in former. Electrical burn is classified as high voltage (>1000V) or low voltage (<1000V). Factors that determine the extent and severity of injury include the type of current, voltage, amperage, resistance offered by the body, pathway of current and duration of contact. At low voltages, alternating current is three times more dangerous than direct current. It has a propensity to produce ventricular fibrillation, central respiratory arrest and asphyxia due to tetanic contraction of respiratory muscles. Majority of high voltage injuries are sustained by electrical and construction workers. The injuries result from conversion of electrical energy to heat. High voltage current produces burns by electrical arcing on external surface or by power dissipation inside tissues. Electrical arcing can ignite the clothing, as had occurred in the present case, leading to severe thermal burns in a dazed or unconscious patient. Soft tissues with low resistance are more liable to damage compared to skin and the true extent of injury may not correlate with the extent of cutaneous burns.

There are conflicting reports about the prevalence of cardiac injury in electrical burns. Studies based on creatine phosphokinase (CK) and ECG indicate myocardial involvement in 36% of cases. It is unclear whether the elevated CK-MB and ECG changes actually represent evidence of myocardial injury, since technetium pyrophosphate scans in the patients are

normal. Mc Bride et al [5] studied 36 cases of high voltage electrical injury to determine the incidence and possible sources of elevated CK-MB. They found that although more than 50% of patients had elevated CK-MB, only 2 sustained myocardial infarction based on history, ECG and clinical course. The elevated CK-MB was attributed to associated skeletal muscle injury. Our patient did not have history of premature IHD, chest pain or features of heart failure, and the ECG showed features of inferior wall 'Q' wave infarction. Inferior wall involvement and absence of precordial pain, have been reported [3,7]. Congestive cardiac failure is uncommon following electrical injury. Myocardial necrosis may be focal or diffuse [6]. Non-ischaemic mechanisms of myocardial injury have been postulated, since occlusive thrombi of coronary arteries have not been demonstrated. Thrombolysis is therefore not indicated. Elevation of total CK and CK-MB are not diagnostic and technetium 99m pyrophosphate scan is needed to confirm the injury. We could not undertake both these tests, as they were not available locally. Other cardiac manifestations of electrical burn include immediate cardiac arrest, pseudo infarction, myocardial ischemia without necrosis, dysrhythmia, conduction abnormalities, acute hypertension and nonspecific ECG abnormalities. Cardiac arrest needs aggressive resuscitation since the chances of revival are high even after prolonged cessation of vital functions. All patients with pre-hospital arrest, loss of consciousness and abnormal ECG need admission and cardiac monitoring for at least 48 hours.

Fluid replacement is essential in initial management to combat hypovolemia from the rapid loss of fluid into the damaged tissues. There is a risk of underestimating the fluid requirement due to little cutaneous burn coupled with extensive inapparent deep tissue injury. Regulation of rate of administration of fluid is best determined by urinary output rather than by routine burn formulas. The presence of myoglobin in urine requires administration of diuretic and alkali to prevent precipitation of pigment in the kidney. Urine output should be maintained at a rate of 75-100ml/h.

Debridement of dead and devitalized tissue is essential to reduce the risk of infection and post-injury hyperkalemia. Extent of devitalised tissue can be determined by arteriography, which will show pruning of nutrient vessels in injured muscle. Other methods available include xenon-133 washout, 99m-technetium pyrophosphate scan and resistivity to 60-Hz current. It is important to pack open amputation or exploration wounds and re-examine them 24-48 hours later for necrotic tissue. Multiple debridement in separate set-

tings are necessary including revision of stumps. Other injuries like vertebral compression fractures, delayed onset neurological deficit and cataracts can occur. These should be looked for by repeated physical examination. The overall mortality rate has been reported to be between 3 and 14%. This case has been reported to illustrate the problems faced and complications encountered in the management. In conclusion, despite their low incidence electrical burns continue to be a challenge, requiring expertise in intensive care medicine and wound management.

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EME Talk

RMO : What is the problem?

Veh Mech : Sahib pait me bahut dard hai, khana bhee hazam nahi ho rha hai.

RMO : Any other problem?

Veh Mehh : Yes sir, exhaust bhi bahut chalta hai.

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