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Delayed Cardiac Complications in a Case of Electrical Burns

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Abstract

Electric shock is the set of pathophysiological events resulting from the action of an electric current on the human body. When the chest is situated along the path that connects the entrance and exit points, the heart is often affected. The two major cardiac complications of electrical shock are arrhythmias and myocardial tissue injuries. Here we discuss about a case of electric burns with the incident dating ten years back now having a non-healing scar over the lower back region of trunk. Patient had an unprecedented cardiac event while taken up for wound debridement under general anesthesia. Patient was managed accordingly and was stabilized afterwards.

Keywords: Electrical burns, cardiac complications

Introduction

3% to 5% of all admitted burned patients are injured from electrical contact. Electrical injury is unlike other burn injuries in that the visible areas of tissue necrosis represent only a small portion of the destroyed tissue. Electrical current enters a part of the body, such as the fingers or hand, and proceeds through tissues with the lowest resistance to current, generally the nerves, blood vessels, and muscles. The skin has a relatively high resistance to electrical current and is therefore mostly spared. The current then leaves the body at a "grounded" area, typically the foot. Heat generated by the transfer of electrical current and passage of the current itself then injures the tissues. During this exchange, the muscle is the major tissue through which the current flows, and thus it sustains the most damage. Blood vessels transmitting much of the electricity initially remain patent, but they may proceed to progressive thrombosis as the cells either die or repair themselves, thus resulting in further tissue loss from ischemia. Injuries are divided into high- and low-voltage injuries. Low voltage injury is similar to thermal burns without transmission to the deeper tissues; zones of injury from the surface extend into the tissue.[1] The syndrome of high-voltage injury consists of varying degrees of cutaneous burn at the entry and exit sites combined with hidden destruction of deep tissue. Initial evaluation consists of cardiopulmonary resuscitation if ventricular fibrillation is induced. The most common cause of death continues to be cardiac arrest after acute arrhythmias at the

scene of the incident secondary to either asystole or ventricular fibrillation. Late sequelae of cardiac complications are very rare in a case of electrical burns. Here we try to present case of electric burns who sustained the same many years back, now had a cardiac event in unusual circumstances

Materials and Methods

The study was carried out in a tertiary care hospital in South India after receiving approval from departmental ethical committee. Here we present a case of 31-year-old male who sustained an electrical injury in workplace suffered burn injuries to right hand, trunk, posture right and left thigh medial aspect. All the above wounds healed over a period of one year post burn except for a wound over the lower back. The lower back wounds healing was undulating over the past 8-9 years which history of episodes of serous discharges, fever and itching. Patient presented to plastic surgery OPD with above mentioned complaints and was admitted for further expert management. Patient has no other history pertaining to any systems that could be affected by electrical injury nor has any other comorbidities. Biopsy of the wide local excision specimen of the ulcer turned out to be squamous cell carcinoma positive. PET-CT was suggestive of a localized lesion with no systemic spread. After taking medical oncology and radiation oncology consultation patient was planned for debridement of the defect and flap cover or grafting of the defect under plastic surgery and later for adjuvant Radiotherapy.

After wide local excision patient was posted for debridement with or without skin graft or flap under general anesthesia. After inducing the patient with fentanyl, thiopentone and atracurium while proceeding with laryngoscopy, patient went into asystole. CPR and atropine injections were given as per ACLS guidelines. Adrenaline injections were also given. Patient was revived but he went in to ventricular tachycardia. Defibrillation was given with 200 joules of shock. Patient was again revived but to go into ventricular fibrillation. Defibrillation was given thrice with 200 joules followed by amiodarone injection. Patient was revived again, now into sinus rhythm with a HR-96/min and BP of 100/60 mm-hg on Noradrenaline support. From operation theatre patient was shifted to critical care ICU for further expert management.

Cardiology consultation was obtained on the same day but no significant changes were found in neither in ECG nor in echocardiogram. On post op day 1 patient was again reviewed by cardiology team. Repeat echocardiogram was showing mild global hypokinesia with an LVEF of 50%. On further detailed evaluation by the cardiology team patient was suspected have Paroxysmal AV-block (vagal induced prolonged asystole).

Since the patient would require further surgeries, Temporary cardiac pacing with Jugular TPI inserted into right ventricle was done. Further procedures with TPI insitu, patient did not have any cardiac events intra-op.

Discussion

When the chest is situated along the path that connects the entrance and exit points, the heart is often affected because the electric current actually follows vascular axes, which present the lowest resistance (along with nerves). Horizontal (hand to hand) as well as vertical current passages (hand to foot or head to foot) can thus lead to cardiac injury. The two major cardiac complications of electrical shock are arrhythmias and myocardial tissue injuries. Thereafter, if the initial electrocardiogram findings are abnormal or there is a history of cardiac arrest associated with the injury, continued cardiac monitoring is necessary along with pharmacologic treatment for any dysrhythmias. The most serious derangements occur in the first 24 hours after injury. If patients with electrical injuries have no cardiac dysrhythmias on initial electrocardiogram or recent history of cardiac arrest, no further monitoring is necessary. The mechanism behind electrically induced cardiac arrhythmias is not entirely understood but biopsies reported arrhythmogenic foci in patchy myocardial fibrosis with increased numbers of Na^+ and K^+ pumps, potentially associated with transient and localized changes in sodium and potassium transport as well as concentrations with resultant changes in membrane potential.[2] As a consequence, arrhythmias can be triggered by these areas of repolarization heterogeneity, with possible abnormal enhanced automaticity or after depolarization and triggered activity several hours after the injury. Furthermore, if necrotic areas/scars persist, this substrate can favor the occurrence of re-entry arrhythmias remotely. Conduction disturbances, like sinus bradycardia or standstill, bundle branch blocks or various degrees of atrioventricular blocks can occur following electrical shocks. Indeed, electrical injuries seem to have a predilection for the sinoatrial and atrioventricular nodes. The reason for this vulnerability remains unclear. It has been hypothesized that the sinoatrial and atrioventricular node ion channels are the easiest to disrupt and

that ischemia and infarction in the right coronary artery distribution (running closest to the chest surface and supplying both nodes) make the nodes more vulnerable to electrical current. Interestingly, conduction pathway injuries secondary to an electric shock underlie the development of ablative techniques since the description of the first fulguration, in 1979, where an external shock had accidentally induced a total atrioventricular block due to the propagation of the electric current to an endocavitary electrode positioned on the His bundle. Other cardiac manifestations reported include hemorrhagic pericarditis, transient arterial hypertension and transient autonomic dysfunction. Electricity has been documented to cause myocardial necrosis, infraction, dysrhythmia, and contractile dysfunction, all of which may be delayed as well as persistent. Dilative cardiomyopathy (DCM) is an uncommon cardiac complication of an electric shock.[3] Only three cases of DCM caused by electrical injury have been reported in the international literature. The electric shock could cause myocardial necrosis, myocardial infraction, arrhythmia, conduction disturbances as well as contractile dysfunction. DCM appearing after an electrical injury could be a result of direct injury to the myocardium (contraction band necrosis) or a result of host's inappropriate response to injury leading to a cytokine-induced myocardial dysfunction.

So here in this case of a young male with no other comorbidities or previous cardiac events it is very unlikely for him to have gone into asystole. But since he had a history of electrical burns in the past, there could have been pathological changes in the conducting system of the heart or in the myocardium which was now precipitated when the patient was induced for general anesthesia. Hence there could be a possibility that electrical burns which affects the cardio vascular system can have late sequelae of complications. The exact cause cannot be pinpointed. It could be due to changes in the conducting system, myocardium or thrombosis of blood vessels of the heart.

Conclusion

In a nutshell we have tried to present a case of rare complication of electrical burns affecting the cardiovascular system. We should also take into consideration that electricity not only has acute effects on the heart but also significant changes in the normal anatomy and physiology of the heart which may come into the picture later in life of the patient.

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