# Further Experience and Information in Management of High Power Electrical Burns, (A Study in a general hospital in Iran)

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***Summary***

***Aim:*** *Electrical burns are classified as high-voltage and low-voltage injuries. This retrospective study investigated high power electrical burn patients between 2003 and 2005 which showed a total number of 100.*

***Materials and Methods:*** *Compared to our last study between 2000 and 2001, the number of high power electrical burns has been dramatically increased. Due to high morbidity and mortality rates of high power electrical burns we paid our attention to study these cases and alleviate our treatment strategies. The study was Retrospective and SSPE software was used for statistical analysis.*

***Results :*** *In the vast majority of high voltage electrical burns to the limbs and chest, fasciotomy is indicated as the first emergency procedure. After the patient's general condition has stabilized, we perform early excision of nonviable tissues. Amputation may be performed as an emergency, or after demarcation lines become clear. The mean age of the patients was 44+-10 years. All of them were male and mostly worked for the ministry of power. High power burns are important due to multiple significant factors involving these victims. Damage to vital organs, fractures, head and neck traumas and undetectable damage to spinal cord, only contribute a small amount of many complications workings on high power burns.*

***Conclusion:*** *Our experience indicates that, in Iran, middle age men are at highest risk for electrical burns, mainly due to occupational hazards and lack of proper training. The encouraging finding in our study is the relatively low mortality rate in the high power electrical burn patients, which on the other hand, shows that our center is developing treatment strategies for these cases successfully.*

***Key words:*** *High Voltage injuries-Burns*

**Introduction**

The phenomenon of magical physical forces occurring between totally disconnected objects has been recognized for centuries, but it was Coulomb who first defined and explained the forces as "electrical". The understanding of electricity and magnetism that followed constituted one of the most significant advances of the 19th century. Inventors such as Edison and Westinghouse realized that electric energy could be generated in one location and used to perform work elsewhere.

As application of electric power increased, it became clear that electric forces can cause lethal tissue injury.

The first human fatality from an industrial accident apparently occurred in 1879 in Lyon, France.

Despite an increased awareness of the potential dangers, electrical injuries have increased with the widespread delivery of electricity and the application of electrically powered machinery.

Electrical burn injuries represent a special type of lesion in which disability is high and functional and aesthetic sequelae are significant. They occur less

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frequently than flame or liquid burns, and account for 2.7% of all admissions to burn centers.

In this issue we tried to collect a combination of scientific aspects of electrical burns from the literature and internet resources and also our experiences in Mehr General Hospital. In the end of issue, the statistics of our study on 100 high voltage electrical burns between 2003 and 2005 would be presented.

***Definitions and terms:***

Electrical injures can be categorized as high and low voltage injuries. The injuries result from direct effects of current, from conversion of electrical energy into thermal energy as the current passes through the body tissues, and from secondary effects. The severity of injury is determined by the voltage, current intensity, types of current, the current pathway, the duration of exposure, the resistance of the tissues, contact surface, the extent of multi system involvement, and the circumstances surrounding the incident. To fully understand such injuries, understanding certain basic electrical principles is necessary. Direct current (DC) flows in a constant direction. Batteries, for example, deliver DC. Alternating current (AC) changes direction.

A Volt is a unit of electromotive force or pressure that causes current to flow. An ampere is unit of electrical current. More precisely, it is the flow of a certain number of electrons per second. An ohm is a unit of electrical resistance. Conductance is defined as 1/ resistance. The resistance of a material to current flow depends on the physical and chemical properties of the material. The amount of current flow often determines the magnitude of injury. Because most voltages are known and skin resistance can be estimated, Ohm law, in which current is equal to voltage divided by resistance can be useful for calculating the current flow during electric shock. Most of our cases were injured by 20Kvolts current.

A watt is the unit of electrical power that is delivered when 1 ampere flows through 1 Ohm for 1 second. Energy is defined in terms of a watt-second. One watt- second is equal to 1 Joule. One watt of power delivered for 1 second produces 0.24 calories of heat.

Electrical current less then 600 volts is considered low- tension and above 600 volts, high- tension current. High tension wires usually carry alternating current of higher voltage and amperage. Extensive Burns after high- tension accident are partly caused by arcing of the current, which consists of ionized air particles, and much of the electrical energy is then consumed resulting in considerable voltage drop.

The resistance of the body tissues to the flow of a current, in order of decreasing magnitude is: bone, fat, tendon, skin, muscle, blood, and nerve. The skin resistance, being the most important, varies within wide limits depending upon its thickness, moisture and cleanliness. If the cutaneous resistance is low at the site of current entrance, systemic damage may be greater; conversely if the skin resistance remains high, local damage is more severe and systemic injury is minimized. After injury skin is charred and its resistance drops to nearly zero, diminishing any protective effects.

The most severe local lesions, which also has a higher incidence of complications, are found in the pathway of greatest resistance, i.e./ from hand to hand, or form hand to foot, while current flows through the lowest resistance pathway- in and out or the same extremity causing less serious damage. If the heart of brain stem has been traversed, respiratory paralysis is more frequent in injuries due to high voltage alternating currents.

The typical types of tissue destruction resulting from electrical injury are: Entry and exit wounds (contact and grounding points) Arc Burns (or "markings") and thermal burns. Distinction between entrance and exit wound is not always obvious.

Team working is the term that we frequently use in our lectures to mention that working on burn patient needs a close cooperation between medical professionals to reduce complications of frequent mandatory anesthesia.

***Pathology and Pathophysiology;***

One third of all electrical traumas and the vast majority of high-voltage injuries are job related. All of our cases were workers of utility companies.

The resulting transformation of electrical energy into heat is maximal at the contact site, thus affecting the skin and the adjacent tissues. As the tissue resistance of the trunk is lower and its cross- sectional area much greater, the amount of heat released is considerably less, and thermal damage to internal organs is therefore rare.

The principal pathologic change caused by electricity is coagulation necrosis. In contrast to thermal burn, electrical injury is classically deep and accompanied by muscle damage, vascular lesions dominate the clinical picture in many electrical injuries. Arterial changes in the vicinity of electrical burns are due to the heat rather than the specific action of the electrical current. Thrombi are apt to form within the damaged vessels and ischemic necrosis is therefore common in high- tension injuries.

The 3 major mechanisms of electricity- induced injury are (1) electrical energy causing direct tissue damage, altering cell membrane rest potential, and electro conformational alteration of membrane proteins. (2) The conversion of electrical energy into thermal energy causing massive tissue destruction and coagulative necrosis, which is called joules heating; and (3) mechanical injury with direct trauma resulting from falls or Violent muscle contraction. When contact is through an electric are, thermoacoustic blast force can significantly add to the injury.

In the low-tension group the victim often gets locked to the contact and occurs most frequently when the current went in and out of the same extremity. In the high- tension accidents, locking to the contact is not common. The infrequency of locking with higher voltage is probably explained by the fact that the circuit is completed by arcing before the person touches the contact. Meanwhile in voltage more then 500 volts, if the person touches the contact especially in alternative currents, sever tetany makes the victim unable to disconnect from the source, this phenomenon is celled in terms "let go voltage".

Disruption of cell membrane occurs at temperatures greater than 420C. At slightly higher temperatures (> 450C) disruption of intramolecular bonds in proteins, with loss of conformation (denaturation) takes place. At temperatures above 650C, DNA denatures.

Heat damage is also related to duration of exposure. In most cases of high- voltage electrical shock, heat damage occurs instantaneously at skin contact points and current passage for 1-3 seconds is enough to thermally burn tissues between contact points. Electroporation is the process of structural breakdown of membranes by strong electric fields which was first identified in the mid-1980. The normal transmembrane potential for mammalian cell is less than 0.1 volts.

During electrical shock, electrical current flowing around the cell causes supraphysiologic transmembrane potential to be established across the membranes of very large cells. Skeletal muscle and nerve cells are much larger than other cells, experience this effect more severely. When the transmembrane potential exceeds 8-10 times normal, the membrane swells with water until it ruptures. This process is called electroporation and it can happen quickly, in less than 1 millisecond. Tissue field- strengths in the 100 volt/Cm range can electroporate skeletal muscle and peripheral nerve cells, whereas fields of 1000 times larger are required to electroporate fibroblasts and endothelial or other smaller cells.

During electrical shock, fields in the tissue can be expected to range between 1 to 1000 volts/Cm.
When a victim comes into contact with a high power source the epidermis is usually destroyed by heat within milliseconds. Large currents can then pass and produce tissue damage, particularly to skeletal muscle and nerve.

Recently, another type of injury mechanism has been proposed; electroconformational denaturation of membrane proteins, particularly ion channels.

When the transmembrane potential exceeds 0.6 to 0.8 volts, the potassium channel is damaged, and this leads to cell dysfunction. Other channels have higher thresholds. Disruption of membrane integrity by heat, electroporation, or electroconformational protein damage leads to osmotic swelling of tissues. Alternately, this can precipitate, as it commonly does, muscle compartment syndromes. When muscle compartment pressure exceeds 30-35cm of water, the vascular circulation is compromised, and this can cause ischemic necrosis.

Typically, it is at least 1 to 3 days before the true extent of damage can be recognized. Furthermore, healthy skin and fat often conceal injured muscles, nerves and bone. Thus, it is very difficult to accurately diagnose and localize tissue damage scattered throughout the current path before irreversible cellular degeneration has occurred.

***Initial evaluation and diagnostic methods:***

Electrical injuries occur from direct contact or as an indirect contact with a power source. Indirect contacts can be separated into arc, flash, thermal and blunt trauma injuries. The typical entry wound is charred and depressed and blisters may be produced by vaporization of water within the tissues.

Circumscribed zone of tissue coagulation necrosis is seen at the exit (grounding) point.

Electrical injury produced by current coursing external to the body from the contact point to ground is called Arc Burn, which is the most destructive of all of the indirect mechanisms. The heat generated by the arcing burns is often in the range of 3000 to 200000C and the depth of these burns is dependent upon the proximity of the current to the skin. This injury occurs when the victim becomes part of the arc itself. Electrical arcs can ignite Clothing and cause thermal burns. Arcing wounds occur when current takes a direct path, often between joints in close apposition to one another at the time of injury. Burns of the volar aspect of wrist, the antecubital fossa when the elbow is flexed at the time of injury, and the axilla are most common. The immediate evaluation of the patient with a major electrical burn is similar to that of any severely injured patient and includes evaluation of airway, breathing and circulation.

The high voltage direct current electrocution tends to cause a single muscle contraction, throwing its victim from the source. These patients tend to have blunter trauma. Direct current electrocution can also cause cardiac cycle affected. Alternating current (AC) electrocution is three times more dangerous than DC current at the same voltage.

High voltages shocks may cause blunt trauma, usually when the electrical energy causes the victim's muscles to contract violently. This intense and diffuse muscle contraction can cause the victim to fall or fracture bones, especially in AC electrocution. Central nervous system injury is common with electrocutions, as is blunt head trauma, which results from falls or from being thrown from a high voltage contact.

Change in mental status is also common. Corneal burns, intraocular hemorrhage or thrombosis, uveitis, retinal detachment, and orbital fractures may also result.

Cardiac arrest, either from asystole or ventricular fibrillation, is common in electrocution. Vascular damage is greatest in the medial layer of the vessel. The damage to the small arterioles, along with marginal muscle viability as a direct result of the electrical energy, can lead to progressive tissue necrosis. Coagulation necrosis, intravascular hemolysis, and delayed vessel rupture may also occur.

Acute renal failure usually results from rhabdomyolysis or hypovolemia. Hepatic and pancreatic necrosis, paralytic ileu's, bowel perforation, esophageal hemorrhage Curling's ulcers and gastrointestinal bleeding have been reported.

Long bone fractures, vertebral compression fractures, and scapular fractures may result from electrical shock, either from violent and intense muscle contraction or from secondary blunt trauma. Shoulder dislocations may result from tetanic spasm of rotator cuff muscles. Periosteal burns, bony matrix destruction, aseptic necrosis, and osteomyelitis have been reported as delayed complications. Severe muscle necrosis may require fasciotomy. A low threshold for fasciotomy is indicated because an early fasciotomy may prevent limb ischemia and prevent or limit the extent of amputation.

Altered mental status is usually transient unless associated with significant head injury. The victim may have confusion, agitation, emotional lability, amnesia (with loss of both short-term and long-term memory), cognitive impairment, aphasia, headache, seizures, and prolonged coma. Cerebral edema, hypoxic encephalopathy and peripheral neuropathy have been reported. Central and peripheral nervous systems symptoms may improve, but long-term disability is common.

In high voltage electrocution, the spinal cord may be injured as a result of fractures or ligamentous injury to the spinal column caused by muscle contraction or blunt trauma.

In addition, the electrical current may cause direct injury to the spinal cord. This spinal cord may cause weakness and paresthesia within hours of the initial injury. Neurologic deficits are more common in lower extremities than in upper extremities. These patients have a good prognosis for partial or complete recovery. On the other hand, delayed injury may be manifested days to years after electrocution.

Delayed injuries include ascending paralysis, amyotrophic lateral sclerosis, or transverse myeltitis. Motor deficits are common in delayed injury, and deficits are typically patchy and do not correlate well with the level of motor deficit. Although recovery has been reported, the prognosis is usually poor.

The cutaneous system is typically involved in electrocution. Importantly, the initial size of the electrical burn is not an accurate reflection of the amount of tissue actually involved because the subcutaneous tissue is commonly involved. Therefore, for diagnosis and treatment, the Rule of nines should not be used for calculating fluid resuscitation. Instead, adequate tissue perfusion, vital signs, and urine output should guide resuscitation.

Indicated lab studies in first step include urinalysis with urine for myoglobin and serum myoglobin level, if urine is positive for myoglobin, complete blood cell count (CBC), serum electrolyte levels (sodium, potassium, chloride, carbon dioxide, urea, glucose), creatinine level, liver function tests, creatine phosphokinase (CPK) level, total and fractionated, if elevated, determination of creatine Kinase- MB (CK-MB), and arterial blood gas determinations. More severely injured patients who require surgery may need blood typing or cross matching, prothrombin time, and activated partial thromboplastin time studies (APTT).

The need for imaging studies is dictated by other elements of the history or by patient complaints. Violent tetanic contractions may lead to focal areas of injury or injuries resulting from fall. Cervical spine, chest, and pelvis radiography should be performed on any victim who was previously unconscious. Appropriate extremity films in victims with obvious extremity injuries should also be obtained.

Electrocardiogram readings in all patients are recorded.

The development of increased myofascial compartment pressures should cause great concern. If this is suspected, each compartment must be measured. If signs and symptoms of compartment syndrome exits, decompression is necessary. The hall mark of compartment syndrome is pain with passive motion in the compartment containing the muscle groups responsible for that motion. Characteristically, the pain is unrelenting and may appear out of proportion to the visible injury. Patients may experience paresthesia, hypoesthesia, or decreased motor function. Loss of pulses is a late sign of compartment syndrome. Advanced diagnostic methods may help to diagnose occult damage to muscle, nerve and bone- which may later be aggressively treated surgically. New strategies come in magnetic resonance (MR) imaging to locate and quantify tissue injury and monitor response to therapy. In some burn centers a very low field Instrumentarium MEGA-4(0.1 Tesla) unit has been installed to permit rapid study before a patient is transported to the intensive care unit. The 0.1 T unit permits higher sensitivity to tissue protein- water interactions, and it allows simultaneous use or resuscitation equipment during imaging. In the laboratory, more advanced strategies are being developed:

MR angiography can both detect damage to vasculature and monitor tissue perfusion, and MR sodium imaging, by distinguishing changes in extra-cellular and intra-cellar ion concentrations, can locate and quantify cell membrane damage in tissues.

Positron emission tomography (PET) and single photoelectron emission computed tomography (SPECT) are also being employed to map and monitor tissue injury. Some scientists have implemented protocols for measuring tissue perfusion, muscle membrane integrity, and glucose metabolism. Soon to be routinely performed at the bedside, these techniques employ radiotracers to reflect physiological conditions including cellular metabolism, blood flow, and cell membrane integrity.

Nerve injuries are particularly unavoidable following electrical shock because of the complex composition of nerve tissue, the pattern of injury that often results from electrical shock is more complex than commonly used neurological tests allow detecting.

To more accurately describe the electrical nerve injury more sophisticated spectral analysis techniques are adapted. These studies- if available- are very helpful in making diagnosis and planning therapy.

***Management:***

High voltage electrocution injuries are very costly, not only for the victims and their families but for the employers and the society in general. In a study in California, electrical injuries cost to employers has been approximated as 15.75 million dollars per case in direct and indirect costs. Major high-Voltage electrical trauma can produce one of the most devastating of physical injuries. Repeated debridements, amputation, and extensive rehabilitation are common. It is not unusual for a surgeon to debride viable tissue and then return 48 hours later to find most of the forearm muscles necrotic and an above-elbow amputation necessary. Additional debridements may be repeated over several days. The condition of devitalized muscle can be further complicated by acute infection. In general, most victims who survive high-voltage electrical shock are left permanently disabled.

Initial management of all patients with electrical injury includes evaluation of airway, breathing, and circulation. These patients should be assessed as trauma patients. Supplemental Oxygen, cardiac monitor, pulse oximetry, and two large bore intravenous catheters are essential. Applying intravenous routs is difficult in most of high-voltage electrical victims due to involvement of extremities, and so we usually perform a central venous rout such as subclavian or sapheno-femoral catheter, which makes IV fluid therapy, CVP assessments and serial blood sampling examinations more practical.

Nasogastric decompression of the gastrointestinal tract should be considered. H2-blockers or Sucralfate may be beneficial.

Injured or fractured limbs should be splinted in a functional position. Tetanus prophylaxis should be administered.

Fluid resuscitation should begin as soon as possible to maintain a urinary output of 0.5 to 1.0 ml/kg/hr in the absence of pigmented hemoglobin in the urine or 1.0 to 1.5 ml/kg/hr in its presence. A urine output of 1.0 to 1.5 ml/kg/hr should be maintained until the urine is clear of myoglobin. Additionally, the blood PH should be maintained at 7.45 or higher with sodium bicarbonate to prevent the precipitation of myoglobin into the renal tubules. The extent or volume of tissue damage involved with an electrical injury is difficult to assess. The unpredictable nature of electrical injuries makes estimating fluid deficits much more difficult. Many professionals increase fluid replacement after an electrical injury. Based on parkland formula, fluid replacement should be increased by 2-3 times, depending on the total surface area potentially involved. For example it should be increased by 3 if the surface area is 20% and by 2 (or less) according to an increased percentage of burned skin. These formulas estimate necessary initial resuscitation volume over the first 24 hours (started at the time of the burn).

We use isotonic balanced saline solution (e.g., Ringer lactate) for fluid resuscitation. As mentioned before urinary output should be followed closely as an indicator of hemodynamic status and kidney function. Installing an indwelling urinary catheter is mandatory. Bicarbonate should be administered at 1-2 mEg/kg. Furosemide or mannitol may be used to maintain urine output. The initial recommended dose of mannitol is 25 g, followed by 50to 200 g/day.

Cutaneous burns should be treated with silver sulfadiazine or mafenide (sulfamylon). Silver sulfadiazine is preferred in extensive cutaneous burns. Sulfamylon is preferred in full-thickness burns because of its superior penetration of the Escher. Sulfamylon may cause electrolyte abnormality in burns involving more than 15% to 20% of body surface.

The time of surgical debridements is individualized according to the extensiveness of tissue destruction, the type of injury.

There are two reasons for early operation in a patient with a high-voltage electrical burn. If the burn is making the patient sick (e.g., myoglobin will not clear or acidosis will not resolve), the wound should be explored and all grossly necrotic tissue removed. In general, an escharotomy is indicated for a circumferential deep burn when there is evidence of impaired distal perfusion.

Fasciotomy is indicated for concomitant electrical injury to underlying muscle when there is evidence of increased compartment pressure, myoglobinuria, tense muscle compartments, or nerve or vessel comportment pressure, is mandatory, and escharotomy and fasciotomy should be performed at the slightest suggestion of progression. Routine compartment pressure measurements may be helpful. But any of signs of impending compartment syndrome mandate prompt compartment release in the operating theater. One unique injury that must be considered in patients with electrical injuries to the hand is a carpal tunnel injury.

With the associated swelling, relatively small electrical injuries can lead to swelling in the carpal tunnel that manifests as increasing paresthesia and numbness in the distribution of the median nerve. If the injury is sufficiently devastating to create a mummified hand, carpal tunnel release probably has no role in the treatment plan. In severe cases, early amputation remains the only safe choice.

In general, necessary amputations can most safely be accomplished on the second or third day post-injury after patient is fully stabilized and is a better candidate for general anesthesia. Indications for amputation are, massive circumferential devitalized muscle with gross evidence of gangrene, carbonization and for surgical revision of auto amputation by electric current. The initial use of fasciotomy often defines the extent of debridement or level of amputation, which will ensure the least number of complications and the best chance for survival. The most common problem with early amputation is the exposure of large areas which are of questionable viability.

Entry and exit wounds are treated initially with topical chemotherapy. Once the resuscitation is completed and the patient is stable, debridement of the nonviable tissue should be done within 48 hours. For covering the open wounds we usually use antimicrobial dressings but sometimes we consider the use of biological dressings.

A second look procedure under general anesthesia in the operating room may be indicated in extensive deep injury 48 hours later. Definitive wound closure should be attempted after completed debridement of all nonviable tissue has been achieved and no further progression of necrosis is observed. In the majority of cases, auto grafts and secondary closure of amputation stumps may be sufficient. In cases, where vital structures are exposed, flap coverage may be necessary. Septic complications most often arise from hidden extensive deep tissue necrosis which was over looked in the initial evaluation.

Arc burns vary in extent and depth. The deep second or third degree wounds involving extensive skin surfaces are managed as ordinary thermal burns. These wounds are treated like the entry and exit wounds.

Due to extensive protein catabolism in major high-voltage burns, the patient fails to gain or maintain body mass that can affect all aspects of management including wound healing and reproduction of blood cells. We use anabolic steroids (Nandrolone Decanoate- one injection every 3-5 days) to improve protein anabolism.

In areas that debridement has been completed and there is no more need to topical ointments, we use wet dressings (piperacillin soluted in normal saline) to maintain a humid antimicrobial environment helping a healthy granulation tissue formation.

A variety of wound coverings are also available. In high-voltage electrical burns due to different healing stages even in one extremity, use of these dressings is limited, but in our experience collagen sheets and lipido-colloid dressings are more applicable. Collagen gives strength and structure to the tissues, support to the hard tissues like bone, form and integrity to soft tissues, provides stiff layer to vessel wall, thus preventing its bursting, and degrades in the biological environment. It has low antigenicity, supports cellular growth. It has homeostatic properties.

***Sequelae:***

It is very important that families and Co-workers of injured persons understand that even with no visible burn, survivors may be faced with long-term muscular pain and discomfort, fatigue, problems with peripheral nerve conduction and sensation, inadequate balance and coordination and other symptoms.

Electrical injury often leads to problems with neurocognitive function affecting speed of mental processing, attention, concentration, and memory. In addition, as any traumatic experience in a life-threatening situation, electrical injury may result in post traumatic disorders, which can be as life-changing as a major physical deformity.

The incidence of premature cataract development may be as high as 5% to 10% following electrical injury. Surprisingly, they occur equally in patients who do and do not have obvious contact points on the face or head. The latent period may range from weeks to years, with the average being 6 months. Therefore, complete ophthalmologic examination for cataract at the time of hospitalization and, subsequently, with any subjective decrease in visual acuity is warranted.

Other common sequelae are observed in the gastrointestinal tract (cholelithiasis), and bone sequestra.

The electrical injury has more than one victim. Co-workers and family members are often affected as well. Successful therapy has multiple components including successful re-integration in to the work place.

***Materials and Method:***

A total of 100 high- Voltage electrical burn patients were admitted to Mehr General Hospital between 2003 and 2005. This retrospective study investigated these patients according to burn size, methods of treatment, complications, and treatment results. Data were collected from patient records and were analyzed using the software program SPSS for windows.

***Results:***

The mean age of the 100 high- voltage electrical burn patients was 39 years. All the patients were male and worked for the utility companies. The mean period of hospital stay was 24 days and all of them were results of Job related accidents.

Presence of underlying diseases was 4% for cardiovascular, 4% for respiratory and 22% for endocrine diseases.

The burn sites were right upper extremity in 74%, left upper extremity in 71%, Right lower extremity in 29%, left lower extremity in 22%, trunk in 55%, face in 71%, perinea in 4% of cases. The mean total body surface area (TBSA) burned in the 100 patients overall was 28%.

16% of patients experienced loss of consciousness after electrocution.

The accompanying damage in the cases were ophthalmic injury in 57%, Respiratory injury in 20%, vital organs injury in 3%, Muscular burn or damage in 42%, damage to tendons in 35%, bone damage in 13%, cartilage damage in 32%, Vascular damage in 35% and damage to peripheral nerves in 29%.

Management complications rated as 3% for gastrointestinal problems, 3% for pulmonary emboli, 13% for respiratory infections, 5% for ARDS and 6% for pulmonary edema.

In the lab studies myoglobinuria was present in 45% and significant rise in CPK in 58% of cases, all of which had experienced direct contact.

The average surgical intervention was 6.65 times for each case that was extensive in 61%, limited in 39% and surgical intervention was done for accompanying traumas in 3% of cases.

Amputation performed in 29 cases and was 6% for Right hand, 22% Left hand, right foot 9%, left foot 3% and 51% of cases needed skin grafts. In 9% of cases some sort of skin substitutions were used.

Blood was infused to 61% of cases.

Central intravenous routs were maintained in 39% of patients and no infection was reported from that origin.
In 23% of cases fasciotomy was performed.

Systemic infection in 10% of cases and local infection in 29% of cases were noted. In 7% patients infections of ear cartilage was noted.

7% of case had complications during hospitalization period and 89% of cases needed out patient care and surgery in future. Psychiatric problems involved 32% of cases.

Accompanying trauma occurred in 7% of cases. Maximum hospital stay period was 12 weeks and was seen in 16% of severe cases.

Other analyzed information in this study included Foley catheter insertion in 55%, Nasogastric tube insertion in 6% and 100% of grafts was successfully maintained.

***Discussion:***

The results indicated that in Iran, middle aged men are at highest risk for high voltage electrical burns, mainly due to occupational hazards and lack of proper training and safety facilities. The study also revealed that complications and morbidity in high-voltage electrocutions are significantly higher in comparison with any other burn groups.

So there is a serious need for improved preventative measures on industrial sites in particular and prompt treatment of high-voltage cases is absolutely vital.

Better training of technicians and workers on these sites would also help reduce this type of injury.

The encouraging finding was the rate of mortality which was 4% compared to 11% in our previous study between 2000 and 2001.

This shows that our center is using successful treatment strategies for these cases.

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