Electrical Burns

6

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6.1 Introduction

Electrical burns are rare but can be particularly severe or injuring and sometimes fatal. They represent approximately 5 % of burns [1]. In addition, it is estimated that 4,000 people every year undergo an electrocution in France.

This type of burn affects mainly *two categories of patients:*

- The young child exploring his environment
- The adult in his workplace

They are *two types:*

- Damage by direct contact with the electric current. The lesions spreading from an entry point to an exit point of the current (our focus of interest in this chapter).
- Injury by electric arcs in accidents at very high voltage. That is mainly thermal burns but at a very high temperature (>2,000 °C).

They can be divided into *two groups:*

- Low voltage injuries (<1,000 V) occurring mainly at home
- High voltage injuries (>1,000 V) occurring more often in the workplace

They mainly concern *two locations:*

- The upper limb
- The face

Mechanisms of tissular injury appear to be of *three different types:*

- The Joule effect: generating heat depending on tissue resistance – "$J = R\ I^2\ T$". The amount of the heat intensity generated (J depends indeed on voltage U because $U = RI$. T is the duration

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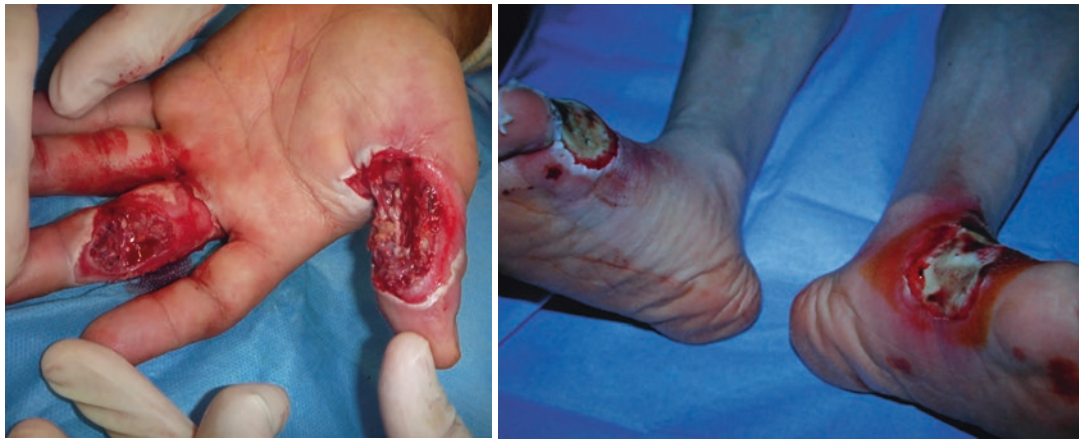

Fig. 6.1 Example of multiple points of entry and exits in the same patient

in seconds of the contact, R is the resistance in ohms, and I is the intensity in amperes).

- The higher the resistance, the greater the heat generated will be and the more serious the injuries are, but less current will travel through.
  Actually two parameters influence tissue resistance:
  - Its category (with decreasing resistance): Bone > fat > skin > muscle > mucosa > vessel > nerve
  - Its diameter: the smaller the diameter (wrist, elbow and ankle), the higher the resistance, and thus the damage related to the Joule effect is significant [2].
- Cell membrane destruction by electric shock (electroporation) [3] increasing tissue damage and promoting the release of myoglobin.
- Massive depolarisation, which will result in the damage of muscle, cardiac and nervous cells. The "shock" causes phenomena of tetanisation, which increase the contact time of the victim with the electric current source (cable grasps, feeling of being "stuck" to the source). Furthermore, tetanisation allows the joint's jump of current by hyperflexion of the joints [4].

6.2 Tissue Injury

1. *Entry and exit skin points*
   These points are most often located at the extremities. The entry point is centred by a sore indicating carbonisation. This area is surrounded by a burn of decreasing depth ("cockade aspect"). On the way to the exit point, an area of deep burn should be suspected, following theoretically the path of sensory and motor nerves (the superficial veins also).

   However, the current path remains unpredictable. Meanwhile, the exit point more often represents a whitish area. During the impact, it links the body to the ground or other external elements connected to it (Fig. 6.1).
2. *Muscle injury*
   It is always more severe than suggested by skin lesions and is due to the action of depolarisation and Joule effect. It represents the most important vital and functional prognosis factor in this type of burn. Muscles submitted to high voltage will undergo a very significant oedema, which can lead quickly to a compartment syndrome (>30 mmHg). This syndrome, if not managed by a fasciotomy, will significantly increase muscle, nerve and vascular damage, by direct compression, thrombosis [5] and necrosis leading to local acidosis. This vicious cycle is to be broken as soon as possible (Fig. 6.2).
3. *Myocardial damage*
   Except the acute cardiac fibrillation, approximately 10 % of patients admitted for electrical burn present electrocardiographic abnormality. This is most often represented by bundle branch block, supraventricular tachycardia or

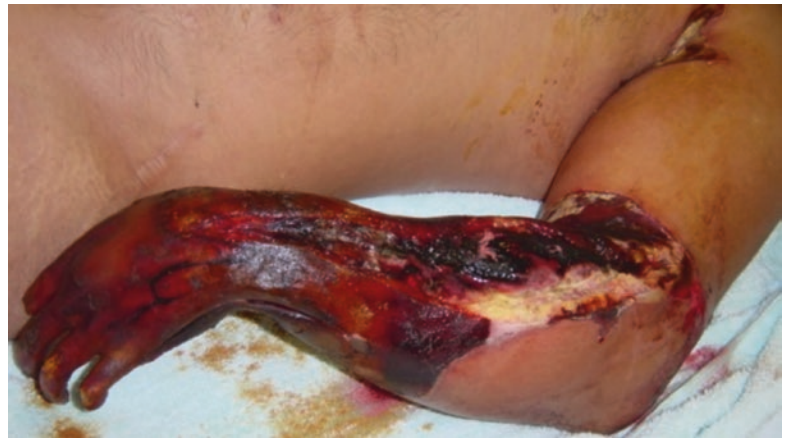

Fig. 6.2 Carbonisation of upper limb responsible for major and composite tissue lesions

nonspecific repolarisation disorder. To these mechanisms is added necrosis by coronary thrombosis according to the same mechanisms mentioned above.

4. *Buccal mucosa damage*
   It is typical of young children biting electric cables. The lesions are most often at the commissures, gums and tongue. Full necrosis occurs most often before the end of the second week. Spontaneous wound healing is often adequate but sometimes secondary interventions are required [6]. Their objective is in fact to reconstruct the anatomical subunits. The establishment of a shaper must be compulsory if there is a risk of microstomia.
5. *Nerve damage*
   It is most often a direct injury of axons by the current, causing paralysis or sensory disturbances more or less permanent. Indirect injury, often persistent, is caused by thrombosis or compression.
6. *Deep damage (except viscera)*
   They are the consequences of the Joule effect. With the bone and fascia being poor conductors, the heat effect is very significant, causing periosteal bone necrosis. In addition to that, fractures and serious sprains (typical posterior glenohumeral dislocation) are not uncommon due to tonic muscles tetanisation.
7. *Other damages*
   - Renal: damage by renal parenchymal necrosis, thrombosis or DIC (*disseminated intravascular coagulation*) and acute tubular necrosis by accumulation of myoglobin.
   - Visceral damage represented by gastrointestinal perforation, paralytic ileus, hepatorenal, liver injury or acute pancreatitis. Liver enzymes as well as amylase/lipase are to be obtained.

6.3 Medical Management

1. Monitoring
   The intensive care management (cardiovascular monitoring, rehydration, coagulation, CPK, K^+, etc.) must be rigorous and precautionary [7]. Compartment syndrome is to be ruled out (increased pressure of the compartments, hypoaesthesia, impaired distal perfusion, etc.).
2. Assessment of the lesions
   If entry and exit skin points are usually obvious, the path and the internal damages are sometimes more difficult to assess. Scintigraphy (99mTc; 133X) and MRI can provide important information on the condition about the deep integuments [8].

6.4 Surgical Management

1. First surgery
   It must be determined by the existence of a compartment syndrome, which must be

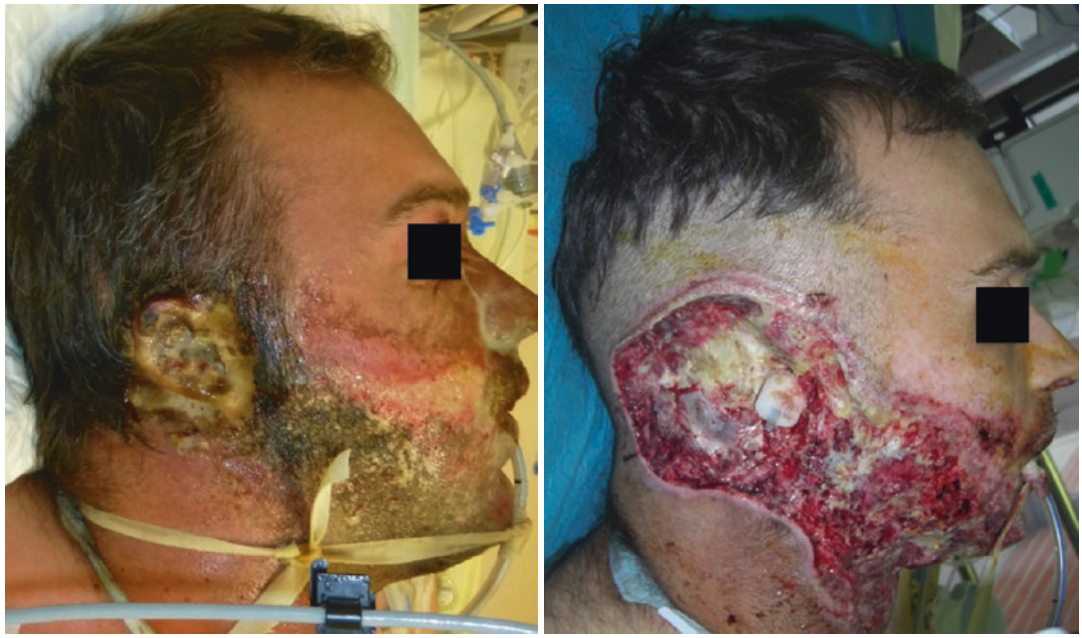

Fig. 6.3 Deep burn of the lateral side of the face due to a very high voltage electric arc

managed within 6 h of the injury [9]. Deep exploration is to be done while carrying out escharotomies and fasciotomies. Necrotic tissue should be removed; the damaged muscles and nerves have to be preserved if we consider a possible recovery especially after fasciotomy (Fig. 6.3).

Immediate flap coverage is recommended by many authors to limit devascularisation, but in emergency cases we think that it must be reserved for vital organs coverage [10]. Besides these situations, we believe that we must avoid to perform locoregional or free flaps before 3 weeks to allow time for oedema to decrease and promote drainage of all local toxins (free radicals, lactate) leached after the trauma. Immediate amputation is limited to extreme cases with anuria or shock; it will aim to keep a length always compatible with future equipment.

2. Second look
   It is carried out 2–3 days later. We have to spare the maximum of tissue (tendon, nerve, etc.) even if they fall in a grey zone. Skin coverage remains our priority; the damaged nerves will be repaired in a second time. Even if not widely practised, these interventions bring some interest as they will allow being less aggressive in the first surgery and opening a window for a new debridement of secondary necrotic tissue after the removal of the ischemia-reperfusion syndrome (when fasciotomy is performed). Ultimately, a third or a fourth revision is sometimes necessary to achieve complete debridement of large areas (Fig. 6.4).

   Furthermore, a polymicrobial infection of necrotic tissue can occur with plurimicrobiens processes often including anaerobes or *Pseudomonas aeruginosa*. Bacteriological samples are systematically taken and antibiotics are given as needed.

6.5 Global Management

This type of patients requires hospitalisation in specialised burn unit with experienced teams. Supervision by physiotherapists to limit retractions is necessary, but also the psychological side should not be neglected.

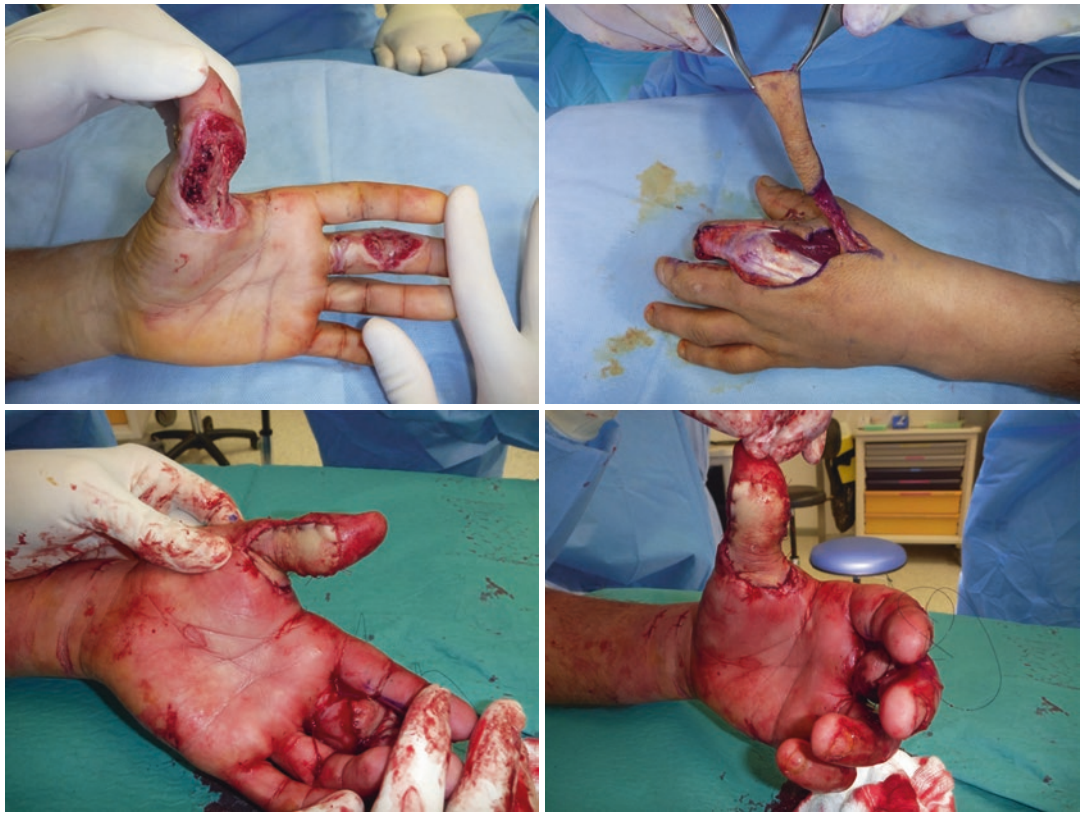

Fig. 6.4 Realisation of an island flap for the reconstruction of the proximal defect. Skin graft was used for the middle finger

It is indeed known that electrocution can have a psychological impact and even cause psychiatric diseases.

The final healing is often long and delayed. Thus, 3–4 weeks may be required to obtain granulation tissue after debridement and 2–3 months to hope for healing of the entry and exit skin points.

Conclusion

The electrical burns are rare but often severe. The initial management is dominated by the detection of deep lesions and the prevention of organ failure. Management of muscle injury is important for vital and functional outcomes; however, it remains very difficult to assess in the early days. Surgery is often delayed and should usually aim, after a second look, to restore the original anatomy and function. Cosmetic and functional sequelae will be supported later, usually at 18–24 months.

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