Hepato-porto-biliary changes following a high energy electrical shock

Osama Alnuaimi¹, Mihai Lazăr^{2,3,*}, Cătălin Apostolescu^{2,3}, Cristian Scheau¹, Daniela Adriana Ion^{2,3}

Abstract

Present day increasing dependence on electricity, in both home and work environments, can lead, if the necessary precautions are not met, to a wide type of injuries, from cutaneous burns to important visceral lesions. We report the case of a 44 year-old male with cutaneous and severe visceral lesions, paresis and paresthesia of the right upper limb induced by a high energy electrical shock. No psychiatric manifestations were observed. This case followed an unusual course due to the particular evolution of the hepatic lesions, which included necrosis, abscesses, thrombosis and angiocholitis.

Keywords Electric shock lesions, portal thrombosis, liver abscess, electric necrosis.

Introduction

The first reported case of a fatality due to electricity was approximately 120 years ago and today the number of electrical injuries is increasing,¹ but the exact number of individuals who suffer 'mild shocks' and do not seek medical care is actually unknown. Electrical injuries are most common at work (repairing power lines, operating tall machinery near power lines) but can also occur around the home (installing electrical devices, dropping electrical appliances).

The type and the severity of the lesions induced by the electrical shock depend on many factors - type of the electric current (EC): alternating current (AC) or direct current (DC), duration of exposure, site of entry and exit, body mass, surface area that has been crossed by the

energy and tissue resistance, voltage and amperage.

Electrocution with AC is more dangerous than with DC - the patient may present tetany since muscular contractions range between 50-120 per second, and the victim tends to grasp the current source harder, increasing the exposure time and aggravating the injuries, while high-voltage DC can induce a single muscular contracture, followed by the throwing of the victim (increasing the risk for a traumatic lesion). The electrocution with DC also has a higher risk of associating with disturbances in the heart's electric activity.

The adult human skin has a resistance of approximately 5000 ohms. This value can increase or decrease depending on a number of factors (e.g. dry skin resistance could reach 100.000 ohms due to the outer layer of cells in the *stratum corneum*, while immersion in water greatly reduces its resistance).² When an electric current passes through a circuit, heat is generated depending of structure resistance - higher resistance means more heat generated and greater damage inflicted to the adjacent structures. Impedance represents a broader term (used for AC), extending the concept of resistance (used for DC), characterizing beside the magnitude also the phase of the EC. Primary produced lesions are necrotic organ lesions due to the direct effect of electric current, while secondary lesions are related with cardio-vascular, hepatic, respiratory decompensations and/or with associated infections. The location of lesions is related to the path of the electrical current through the

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¹Clinical Institute Fundeni, Bucharest, Romania,

²National Institute for Infectious Diseases 'Prof. Dr. Matei Balș', Romania,

³Carol Davila University of Medicine and Pharmacy, Bucharest, Romania

*Corresponding author: Mihai Lazăr, MD, National Institute for Infectious Diseases 'Prof. Dr. Matei Balș', 1 Dr. Calistrat Grozovici street, Bucharest, Romania, University of Medicine and Pharmacy 'Carol Davila', 37 Dionisie Lupu street, Bucharest, 020022, Romania, mihai.i.lazar@gmail.com

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body – the path of least resistance as the current searches for a grounding source. The tissues in the body have different resistance levels (in decreasing order: bone, fat, tendon, skin, muscle, blood, nerves),³ and in consequence EC travels mostly along nerves and blood vessels; however if the electricity is high enough, it will travel through whatever is in its path on the route to the exit point, regardless of the resistance of the tissues, causing extensive lesions also in muscle, bones and fat.

Electrical injuries can be divided into high or low voltage using 1000 V as dividing mark, with no significant clinical impact (deaths have been recorded from contact with voltage of 25 V⁴ and survivors have been reported after contact with voltage of 34.000 V⁵ – in our case the voltage was 12.000 V). The higher the voltage is, the more intense the thermodynamic effect is (the same amount of electric particles carries more energy in high voltage EC than in low voltage EC), however, the most important characteristic which conditions the destructive effect is represented by the intensity.⁶

Because the difference in potential in electrocuted patients is mostly constant, the amount of tissue damage is proportional with the square of the intensity of the EC, explaining why an increase in the amperage determines more extensive lesions than an increase in the resistance or in the voltage ($Q=I^2 \cdot R \cdot t$ - the heat (Q) generated is equal to the square of the intensity (I) multiplied by resistance (R) and time (t) – Joule's law).

The amperage reflects the number of electrons passing in one second through the circuit; it is proportional with the voltage and inversely proportional with the resistance. If the voltage of the EC is usually known and constant, the resistance/impedance of the EC varies, depending on the tissue; we must also consider that as the tissue becomes necrotic, the resistance (R) of the tissue will change ($R=\rho \cdot l/s$, where ρ is the resistivity of the material, l is the length of the resistive material and s is the cross-sectional area of the specimen; a structural change of the tissue determines a change in the resistivity of the tissue) and as a result the amperage of the EC will

also change, according to Ohm's law ($I=V/R$, where I is the current, V is the potential difference and R is the resistance), making it difficult to establish the exact intensity of the electric current.

In our case, the patient suffered a high energy electrocution (12.000 V) with alternate current, following a right shoulder-right thigh path, with no involvement of vital organs, explaining the good survival chance of the patient; however the complications after the accident might have been responsible for additional risks if not properly diagnosed and treated.

Case report

A 44 year-old male who suffered in 30.10.2010 an electrical shock accident, presenting second and third degree skin burns on 24% of the body surface was referred to our hospital 2 months after the accident with multiple hepatic abscesses.

Before referral to our hospital, the patient was surgically treated – 3 skin autografting procedures and 1 compartment release intervention; during this period, *Pseudomonas aeruginosa* was isolated from the right elbow wound and treated according to the antibiogram.

The abdominal CT scan performed on 22.12.2010 showed portal vein thrombosis, biliary duct enlargements and multiple diffuse hypodense areas in both hepatic lobes, suggesting necrotic lesions. The patient started antibiotic and antipyretic therapy, without changes in the clinical status and was transferred to our institute.

During the clinical examination, the patient was conscious, cooperative and he presented healing skin burns (friable skin, pink to violet, with varying amounts of granulation tissue), covering 90% of the upper right limb, the right side of the face and neck, right ear and right hemithorax (ribs I to VIII), internal and anterior segment of the right thigh, white deposits on the tongue and non-palpable superficial lymph nodes (in the context of multiple hepatic abscesses and right elbow wound infection). The patient displayed cardiovascular balance, presenting

decreased alveolar murmur in pulmonary bases without abnormal breath sounds, a slightly increased liver (4 cm below the rib cage) and abdominal discomfort on palpation in the right hypochondrium. The neurological exam described paresis of the right upper limb; no other relevant signs or symptoms were present.

Biologically: moderate inflammatory syndrome (ESR 100 mm/h, fibrinogen 693 mg/dl, leukocyte count 12100), moderate hepatocytolytic syndrome (AST 69, ALT 62, GGT 2205, FAL 737), thrombocytosis (631000/cmm), hypochromic normocytic anemia (Hb 9.4 g/dl), indirect bilirubin 0.3 mg/dl, direct bilirubin 0.5 mg/dl.

The abdominal ultrasound examination showed:

- Inhomogeneous hepatic structure, predominant in the right lobe, with presence of hypoechoic ill-defined areas alternating with normal areas, suggestive for necrotic lesions/possible abscesses;
- Enlarged biliary ducts with thickened walls in the left hepatic lobe, and hyperechoic biliary ducts with posterior reverberation in the right lobe – suggestive for cholangitis and pneumobilia (figure 1);
- Thrombosis in the right portal vein and the right intrahepatic branches (figure 1).

Figure 1. Abdominal ultrasound examination revealing thrombosis of the right portal vein and enlarged, hyper-echoic right biliary duct.

The CT scan confirmed the presence of hepatic abscesses (figure 2) and biliary ducts enlargement, with a slowly favorable evolution – progressive decrease in size and number upon successive examinations.

Figure 2. CT scan showing hepatic abscesses in the right hepatic lobe.

Due to a persistently high level of FAL and GGT, an MRI scan was also performed in order to further assess the biliary ducts. T2-weighted sequences showed an irregular biliary tree in the right hepatic lobe, with multiple intraductal signal-void areas, increased signal in the adjacent liver parenchyma (compatible with coagulation lesions) and contrast enhancement of periportal tissues and biliary-wall on T1-weighted images. Slight enlargement of biliary ducts in the left lobe, portal vein thrombosis and necrotic areas were also confirmed.

The etiology of the hepatic abscesses could not be established and therefore a broad spectrum therapy (effective on Gram-negative bacterial strains, *Staphylococcus aureus* and fungal infection) was initiated; during the hospitalization period, the patient also developed ventilation-associated pneumonia with *Acinetobacter baumannii*, which was successfully treated. Medication included: meropenem (3 g/day – 37 days), linezolid (1200 mg/day – 43 days), colistin (9 MU/day – 14 days), tigecycline (15 days), posaconazole (400 mg/day – 23 days) and fluconazole (19 days), associated with gastric protectors (ranitidine 300 mg/day – 19 days), anticoagulant therapy: enoxaparin (40 mg/day – 5 days) followed by acenocoumarol (2 mg/day – 24 days), and symptomatic treatment (phenobarbital 100 mg once daily at bedtime – 37 days, paracetamol 500 mg/day – 9 days, lidocaine spray for the right elbow area – 2 puffs/day). Under this treatment, the patient showed slowly favorable evolution, with gradual diminution of the inflammatory syndrome (ESR decreased from 100 mm/h to 30 mm/h,

fibrinogen from 693 mg/dl to 405 mg/dl and the leukocyte count decreased from 12100 to 7800), regression of the hepatocytolytic syndrome (AST decrease from 69 to 45, ALT from 62 to 51, GGT from 2205 to 464, FAL from 737 to 281), with normalization of platelet count (from 631000/cmm to 322000/cmm). The symptoms registered during admission in our hospital were also ameliorated – no abdominal discomfort described upon palpation and skin burns, skin grafts with normal aspect (healing of the right elbow wound) and improvement in moving the right arm.

Discussion

Considering the path of the electric current: right shoulder – right thigh, without involvement of the heart and brain, without additional tests, we can assume that:

1. The liver should be the first inner organ to be evaluated.
2. The probability for the patient to develop cardiac or psychiatric symptoms is remote.
3. 24% coverage of surface skin burns (second and third degree) in the right arm, flank and right thigh are usually associated with a good survival rate.

Besides the typical lesions in an electrocuted patient, a particularity of this case is represented by the wide polymorphism of hepatic lesions – biliary tree lesions, portal thrombosis, necrotic lesions and abscesses. Necrotic liver areas can be induced directly by the EC conducted mostly through the portal or biliary branches, following a linear lesional pattern, in the proximity of the portal/biliary branches; however the lesional pattern in this case was also pseudo-nodular, leading to another diagnostic possibility – associated vascular lesions. The occurrence of thrombosis can be explained either by an excessive activation of the coagulation cascade on the external pathway, secondary to an increased tissue thromboplastin level or by the direct effect of the electric current on the vascular branches with extensive endothelial lesions. Usually the necrotic lesions regress until they are replaced by fibrotic scars or cysts; in this case the necrotic areas were transformed into abscesses, in the

context of ineffective local defense mechanisms and deteriorated anatomic barriers.

The presence of air and inflammatory changes in the biliary tree suggested ascension of intestinal bacteria in the intrahepatic biliary tree with possible proximity contamination of the necrotic areas, via biliary ducts. Presence of air in the biliary ducts can also be found after ERCP (endoscopic retrograde cholangiopancreatography) interventions or in biliodigestive anastomosis – in these cases, when the patient is examined (through either ultrasound, CT or MRI) the air is present mostly in the left hepatic lobe due to the anatomic disposition of the left biliary ducts (antero-posterior/vertical) in comparison with the right biliary ducts (latero-medial/horizontal); therefore when air is detected in the right biliary tree, angiocholitis must be strongly considered even without a thickened duct wall.

The extensive involvement of both the portal and the biliary tree can be explained by the fact that the electric current is preferentially conducted through liquid mediums (biliary fluid, blood), the cells and connective tissue having a higher resistance to the electric current propagation.

The arguments for the infection of the necrotic lesions have been provided by the radio-imaging investigations (contrast enhancement of the biliary-wall on MRI, hepatic lesions with nodular pattern, peripheral enhancement and median density of 21 HU on CT evaluation). Invasive diagnostic and therapeutic approaches for the hepatic lesions (puncture and drainage) were not possible, the patient being under anticoagulant therapy; however the patient had a positive response under antibiotic treatment, and the lesions regressed, providing another argument for an infectious etiology.

The ultrasound examination of the biliary ducts revealed not only the enlargement but also the adjacent inflammatory changes, with similar sensitivity for the intrahepatic ducts to the MRI scan, while on the CT scan only the increased dimension could be evaluated. For the extrahepatic bile ducts, the MRI characterization was more exact, providing more details.

The most accurate data regarding portal thrombosis were provided by the CT scan and the ultrasound examination (we used color and power Doppler). The MRI scan could provide good quality images only for the main branches of the portal vein; the third and fourth degree branches could not be well evaluated due to the impossibility of the patient to hold his breath long enough during T1 sequences.

The necrotic areas were detected and described by all three imaging methods that we used, however the aspect suggesting infection of the necrotic lesions was detected on CT after contrast administration.

The patient presented paresis of the right upper limb; because the nerves have low electric resistance, a spinal cord injury was also considered, but the unilaterality of symptoms and the good recovery after compartment decompression suggested peripheral neuropathy.

It is important to administer antibiotic treatment not only to prevent the infection of the skin burns but also to prevent a potential infection of the necrotic areas located in the inner structures (the liver in our case).

The dynamic radio-imaging evaluation of the patient, correlated with the clinical and biological frame, is mandatory due to the complexity of the lesions. Ultrasound examination is an appropriate method for

evaluating the evolution of the lesions, however for a proper therapeutic management of the patient, a CT or MRI scan must also be performed.

The risk in case of electrical shock depends on the type of current – AC or DC – the voltage and amperage, the circuit of current through the body, the person's overall health and the promptitude of treatment.

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