

ELECTROCUTION DEATHS: MEDICO-LEGAL IMPLICATIONS AND PERSPECTIVES ON A STANDARDIZED PROTOCOL

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Abstract: Introduction. Electrocution deaths represent a significant medico-legal challenge due to the variability of lesions and the difficulty in identifying a single and constant thanatogenic mechanism. The present paper combines a review of the specialized literature with a series of selected cases from the archives of the Institute of Forensic Medicine (IML) Târgu Mureș.

Material and Methods. The study integrates data from international literature published between 1899 and 2023, correlated with a series of autopsy cases from the archive of the Institute of Forensic Medicine Târgu Mureș. Evaluated characteristics included cutaneous lesions (electrical marks, metallization), histopathological changes (epidermal and muscular aspects), biochemical markers (creatine phosphokinase, myoglobin, troponin), and physical parameters (voltage, current intensity, frequency).

Results. The analyzed cases revealed significant variability in lesions, ranging from the presence of pathognomonic electrical marks to their complete absence, depending on the specific circumstances of each incident. Distinctive histopathological changes (“nuclear palisades”, spiraled muscle fibers) as well as nonspecific alterations were also observed. Ventricular fibrillation remains the principal thanatogenic mechanism; however, other mechanisms of death are also documented.

Conclusions. Electrocution involves two main thanatogenic mechanisms: cardiac arrhythmias and respiratory tetanization. The medico-legal diagnosis of electrocution is based on the identification of characteristic electrical marks, suggestive histopathological changes, and relevant biochemical markers, supported by complementary investigations in atypical cases. We emphasize the necessity of developing a standardized medico-legal protocol that rigorously integrates all these components to ensure a more accurate and reproducible diagnosis.

Keywords: electrocution, electrical marks, thanatogenesis, histopathological diagnosis, ventricular fibrillation, tetanization of the thoracic muscles, asphyxiating petechiae, electromagnetic induction.

INTRODUCTION

Electrotrauma encompasses the spectrum of local and systemic injuries induced by the passage of electric current through the human body, with a potentially lethal outcome depending on various physical parameters. Based on the source of energy, electrotrauma can be classified as artificial (domestic

or industrial electrocution) or natural, resulting from atmospheric discharges (1-4).

Voltage, current intensity, frequency, and type of electric current, along with tissue resistance and the trajectory of the current through the body, all play essential roles in determining the severity of injuries. For instance, low-voltage alternating current, commonly encountered in domestic environments,

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poses significant danger due to its capacity to induce ventricular fibrillation (VF) even at voltages below 500 V (1). Furthermore, current pathways involving the thoracic region (superior or supero-inferior loops) significantly increase the risk of a fatal outcome due to the involvement of vital organs (1, 2). Modes of contact range from direct (unipolar, bipolar, multipolar) to indirect, such as contact with an electric arc, a conductive environment, or even with another electrocuted individual ("rescuer's electrocution") (1, 2). The majority of medico-legal electrocution cases are accidental, frequently associated with alcohol consumption and occurring in domestic or

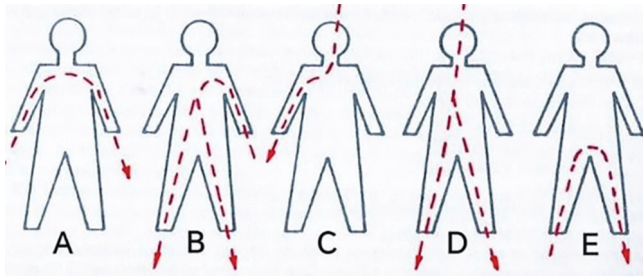


Figure 1. Main pathways of electric current propagation through the human body [1-3]. A and C: superior loop; B and D: supero-inferior loop; E: inferior loop.

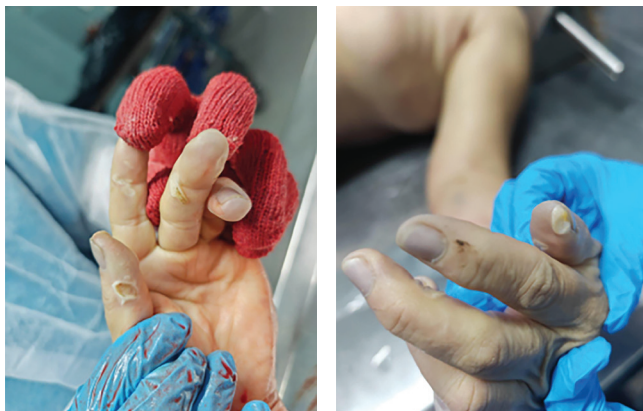


Figure 2. Electrical entry marks on the phalanges of the left hand fingers.



Figure 3. Absence of electrical exit marks.

occupational settings, with suicide by electrocution being far less common (1-4).

Although diagnosis traditionally relies on identifying characteristic lesions, these may be absent under certain conditions (e.g., moist skin, brief contact duration, large surface area exposure). Electric burns, skin metallization, and petechiae can provide important diagnostic clues, but are not universally present (1-4).

In this context, a detailed evaluation of clinical and morphopathological manifestations remains essential for establishing an accurate diagnosis. Electric burns typically occur at the points of current entry and exit, being characterized by coagulative necrosis, grayish-white or yellow coloration, induration, and crateriform appearance. These lesions often reflect the shape of the conductor and may include metallic deposits (metallization), depending on its composition. However, in certain situations - such as electrocution in a bathtub or brief contact - these pathognomonic lesions may be absent. Petechiae, indicative of acute vascular distress, can be observed on the skin, mucous membranes, or internal organs (1-4).

Systemic injuries may involve the heart (ventricular fibrillation, asystole, myocardial necrosis, epicardial hemorrhages), the central nervous system (hypoxic or anoxic encephalopathy, petechial hemorrhages in the brainstem, lesions of the basal nuclei, inhibition of cardio-respiratory centers), as well as the respiratory system, where pulmonary stasis, asphyxial syndrome, and subpleural and subepicardial petechiae may be present (1).

A particular category is represented by natural electrotrauma, in which electromagnetically induced currents can lead to cardiac arrest without visible entry or exit lesions. Such cases present additional diagnostic challenges, requiring a high degree of medico-legal suspicion and careful integration of all available data (5-7).

Aim of the Study

Given the morphological variability of lesions and the potential absence of classical external signs, this study aims to analyze the key clinical and morphopathological aspects of electrocution by examining a series of six cases from the archives of the Institute of Legal Medicine (IML) in Târgu Mureș and through a review of the relevant literature, emphasizing the need for an integrated medico-legal approach. The central objective is to propose a standardized interpretative framework capable of guiding diagnosis in cases without visible electric marks, by correlating macroscopic and microscopic findings with the

circumstances and underlying pathophysiological mechanisms. The study seeks to optimize the accuracy of medico-legal expertise in inconclusive electrocution cases based on external examination, supported by the presentation of the six cases and a critical analysis of the literature.

MATERIAL AND METHODS

Domestic electrocution by unipolar contact - we present the case of a 47-year-old woman found in a flooded cellar (with water reaching 15-20 cm), near a hydrophore connected to an electric power source with damaged insulation. External examination revealed electrical entry marks on the phalanges of the left hand fingers. These were grayish-white to yellowish in color, with raised, indurated edges and a depressed, crateriform center (Fig. 2). No electrical exit marks were identified, likely due to the extensive contact surface, as the victim's feet were in direct contact with water in the flooded environment (Fig. 3). Internal examination revealed the presence of liquid blood in the explored cavities, massive pulmonary stasis and edema (Fig. 4), and bilateral asphyxial petechiae in the

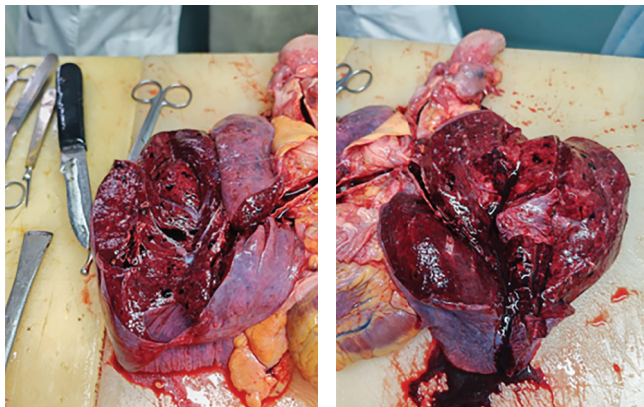


Figure 4. Pulmonary stasis and edema.

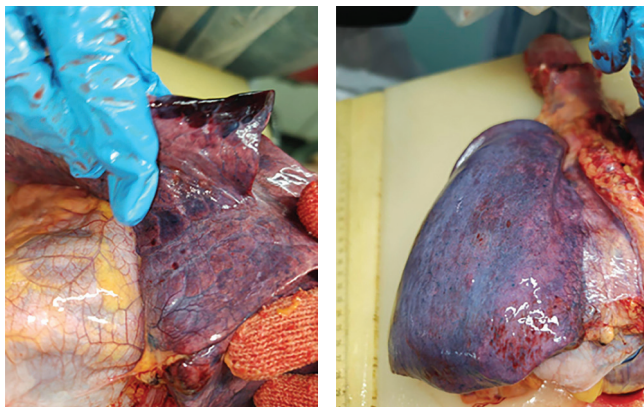


Figure 5. Subpleural asphyxial petechiae.

lungs, located subpleurally on the posterior surface and within the interlobar fissures (Fig. 5). Additionally, asphyxial petechiae were identified in the subepicardial region (Fig. 6). The death was violent, resulting from acute respiratory failure secondary to tetanization of the respiratory muscles in the context of accidental electrocution (with a supero-inferior trajectory: hand-to-foot, in the absence of a visible exit mark) due to direct unipolar contact with a low-voltage alternating current source.

Accidental electrocution by unipolar contact - A 39-year-old man was found deceased near a locality on the banks of the Târnava Mică River on May 26, 2020. Preliminary investigations revealed that the victim had been fishing and was using a carbon fiber fishing rod. The individual was located in close proximity to high-voltage power lines. External examination identified electrical entry marks on the right hand, palmar aspect, corresponding to the metacarpophalangeal joints of fingers II, III, and partially IV, including the proximal and middle phalanges of finger III. The lesions appeared blackish-reddish in color, consistent with burn injuries - dark brown to black, with indurated margins and perilesional skin metallization - measuring 6 × 2 cm (Fig. 7). Burn damage was also noted on the left sock, along with an electrical exit mark on the left plantar region, internal surface, posterior third. This lesion was indurated, with a blackened center and yellowish margins, round to oval in shape, and consistent with

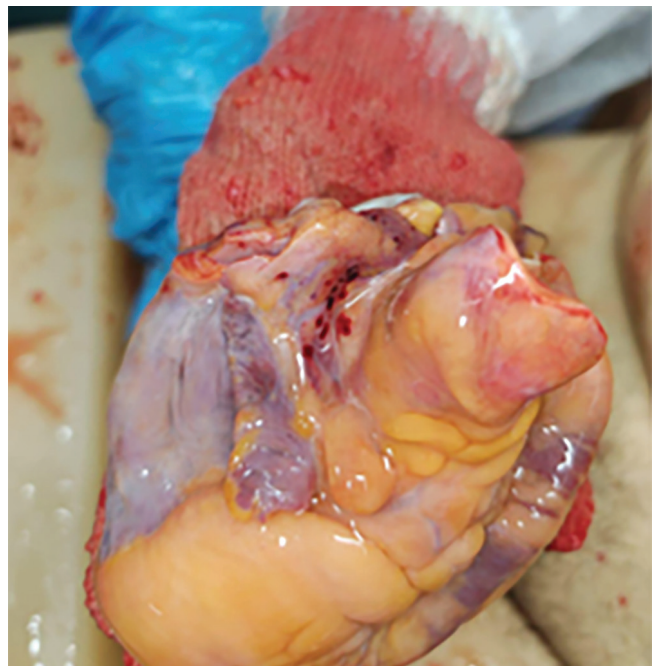


Figure 6. Subepicardial asphyxial petechiae.

a burn injury, measuring 2.5 cm in diameter (Fig. 8). Internal examination showed pulmonary stasis and massive acute pulmonary edema (Fig. 9). The death was violent, caused by cardio-respiratory arrest, most likely due to ventricular fibrillation (VF), resulting from electrocution through the creation of an electric arc between a high-voltage direct current source and the carbon fiber fishing rod (supero-inferior trajectory: hand-to-foot).

Domestic electrocution by multipolar contact - A 76-year-old man was found deceased on August 19, 2024, at approximately 7:30 p.m., in the yard of his vacation home located in Mureș County. Preliminary findings indicated that the decedent had been performing household tasks at the time, using a metallic fruit grinder that was connected to an electrical power source (Fig. 10). External examination revealed multiple electrical marks, characterized by areas of epidermolysis alternating with zones of cutaneous necrosis, displaying a blackish-gray-yellowish coloration, dry and firm texture, depressed compared

to the skin surface. Some lesions reproduced the shape of the electrical conductor. The marks were located in the following regions: left zygomatic-temporal area, right hemithorax, right forearm (antebrachial), right cubital region, left deltoid, left brachial region, and left forearm (entry marks) (Figs. 11 and 12). On the left patellar region, a round to oval electrical exit mark was observed, crateriform in aspect, with gray-yellow coloration, whitish, indurated, slightly raised margins. Surrounding the lesion was a small pale area bordered by a reddish rim (Fig. 13).

Internal examination showed massive acute pulmonary stasis and edema (Fig. 14).

The death was violent, caused by acute cardio-respiratory failure of central origin due to inhibition of the brainstem neural centers, resulting from electrocution (with a supero-inferior trajectory: head → lower limb), through direct multipolar contact with a low-voltage alternating current source, in the context of a domestic accident.



Figure 7. Electrical entry marks (right hand).

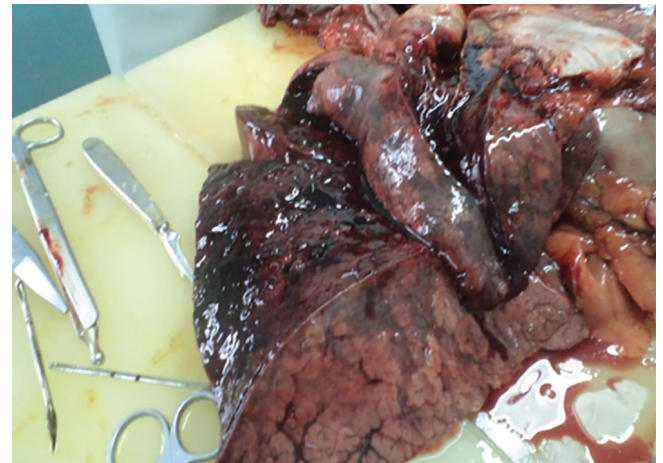


Figure 9. Massive acute pulmonary stasis and edema.



Figure 8. Burn injury (sock); electrical exit mark.



Figure 10. Metallic fruit grinder.



Figure 11. Electrical entry marks: left zygomatic area, right hemithorax, left deltoid, left forearm.



Figure 12. Electrical entry marks: right cubital and right forearm regions.



Figure 13. Electrical exit mark on the left patellar region.

Cases of suicide by electrocution

First case - A 55-year-old male was found deceased, having been electrocuted by the contact wire located above the railway on Railway Line 300. Investigative authorities at the scene reported that the victim had established multipolar contact (hand, neck) with improperly insulated electric wires connected to a high-voltage power source.

Further information from investigators revealed that the individual was a professional electrician and had expressed suicidal intentions to a woman from whom he had purchased half a liter of strong alcohol (pălincă) approximately three hours before the body was discovered. External examination revealed burned clothing in the abdominal region, left arm, right thigh, with melted socks and slippers (Fig. 15). Around the left radiocarpal joint, a piece of wire with melted ends was wrapped, and in the left palm, another piece of wire was found, with one end melted and the other cleanly cut (Fig. 16).

Signs of real death included purplish livor mortis, dorsally distributed and in the diffusion stage,

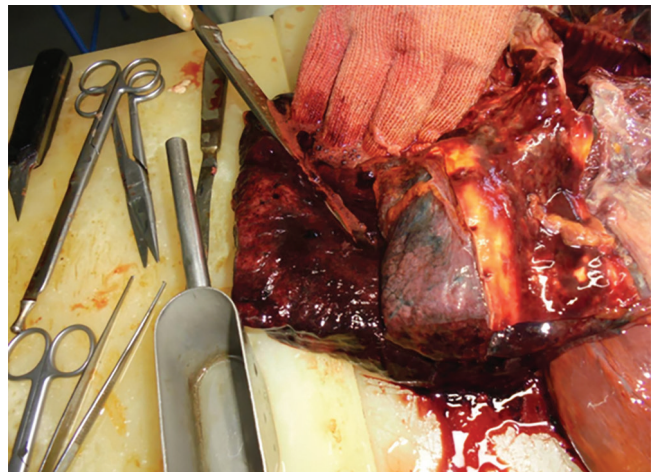


Figure 14. Massive acute pulmonary stasis and edema.



Figure 15. Burned clothing, melted socks and slippers.

along with generalized rigor mortis, firm and difficult to overcome. Additionally, third-degree contact and mixed electrical burns were observed externally, with the skin and subcutaneous tissues carbonized, blackened, and necrotic in the cervical region (Fig. 17),



Figure 16. Left radiocarpal joint.



Figure 17. Cervical region with third-degree contact and mixed electrical burns, carbonized skin and subcutaneous tissue (entry mark).



Figure 18. Electrical entry marks with tissue metallization on both hands and right forearm.

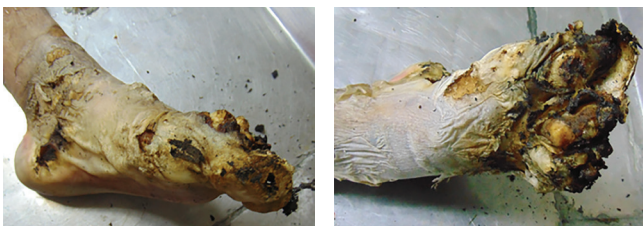


Figure 19. Electrical exit marks on both soles.

right forearm, and both hands (Fig. 18) (entry marks). On the left lateral cervical region, an electrical burn affected the internal jugular vein, which presented a 0.2/0.3 cm discontinuity with hemorrhagic infiltration of the margins.

In the right thigh, electrothermal burn lesions covered approximately 80-90% of the anterior surface of the segment, and on the anterior abdominal wall, approximately 45% of the surface was affected.

On the soles of both feet, carbonization, osteonecrosis, and calcination of the distal phalanges were observed (exit marks) (Fig. 19), along with electrothermal burns caused by ignition of clothing. Internal examination revealed cerebral hyperemia and edema, and massive pulmonary stasis and edema. Toxicological analysis indicated a blood alcohol concentration of 1.92‰ and urinary alcohol concentration of 2.57‰.

The death was violent, resulting from acute cardio-respiratory failure due to inhibition of the brainstem neural centers, following direct multipolar contact with a high-voltage direct current source. Second case - A 68-year-old male was found deceased at his residence on January 18, 2023, by a neighbor. On-scene investigation determined that the victim showed visible traumatic lesions on both wrists, having established multipolar contact with improperly insulated electrical wires connected to a low-voltage alternating current source. The body was in a state of decomposition, with multiple spots and lesions characteristic of putrefaction noted on the body surface. External examination identified a complete circular electrical entry mark at the left radiocarpal level, consistent with an electrical burn, showing



Figure 20. Electrical entry mark on the left radiocarpal region.



Figure 21. Electrical entry mark on the right radiocarpal region.

surrounding tissue metallization (blackish-green, dry, cardboard-like in consistency), maximum width of 3 cm on the anterior side, with irregular depth, and dorsal exposure of underlying bone tissue (Fig. 20). A superficial circular entry mark was observed at the right radiocarpal joint, dark brown-black in appearance, with shiny greenish electrical metallization, measuring 0.3 cm in width (Fig. 21). An irregular electrical exit mark was found in the right preauricular region extending to the cheek, with metallized surrounding tissue (blackish,

brittle, cardboard-like), measuring 7×4 cm (Fig. 22).

Internal examination revealed pulmonary stasis and edema.

The death was violent, resulting from acute cardio-respiratory failure of central origin due to brainstem neural center inhibition following electrocution (infero-superior path: hand → head) through direct multipolar contact with a low-voltage alternating current source.

Atypical case of electrocution without visible external traces - The practical applicability of theoretical notions can be supported by several cases, including an older case from the archive of the Târgu Mureș Institute of Legal Medicine (2002), involving a 5-year-old child who was found deceased next to a metal bar he had been leaning on. In close proximity, there was an electric corn grinder. According to the findings at the scene, the machine was operating and connected to a power source via a poorly insulated cable. It was determined that a puddle of water had formed on the entire ground surface—resulting from recent rainfall—where both the child and the device were located, and the child was barefoot. During the external examination, only a parchment-like excoriation measuring 2 ×



Figure 22. Electrical exit mark in the right preauricular region.

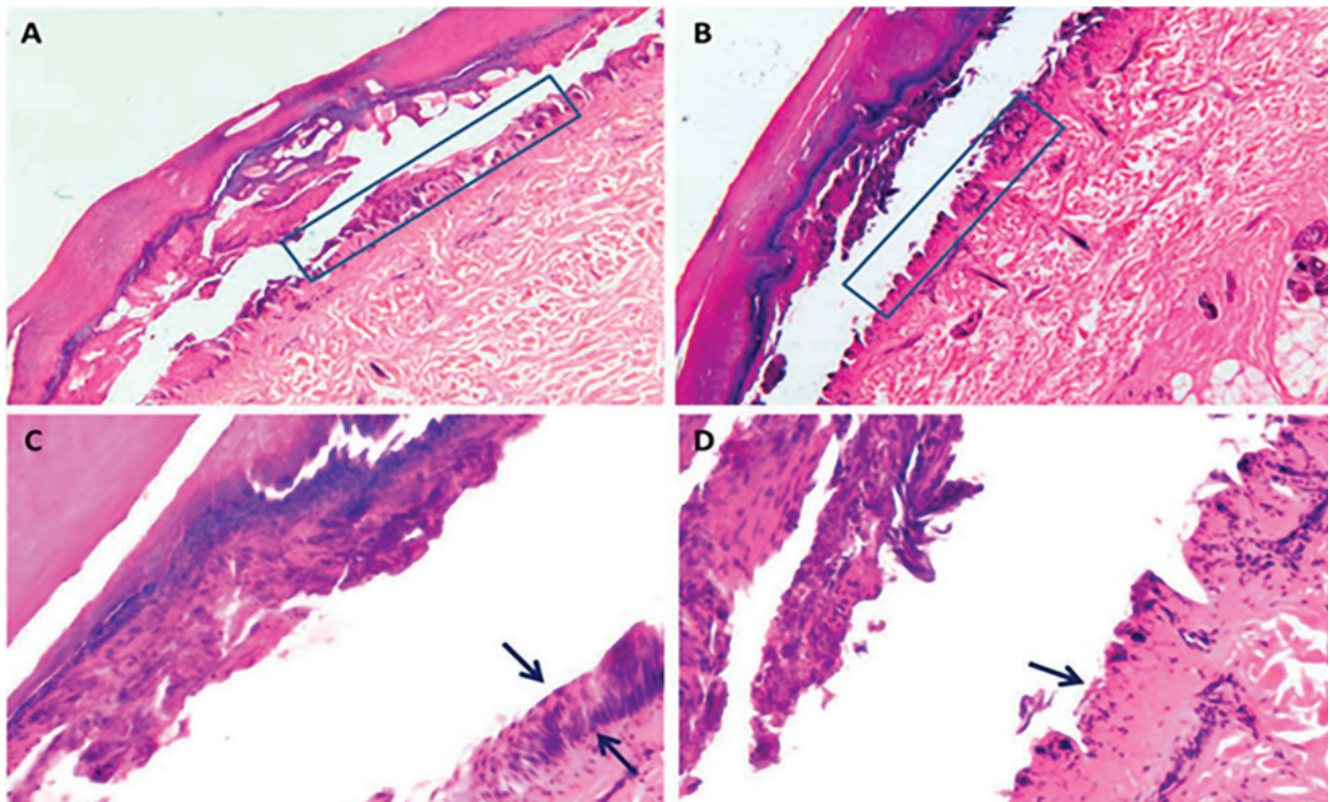


Figure 23. Skin histology in low- and high-voltage electrocution. Low-voltage electrocution (A–C) and high-voltage electrocution (B–D). The preserved basal epithelial layer is observed in (A) (blue frame), and the detachment of the dermo-epidermal layer in (B) (blue frame). In (C), the black arrow indicates the presence of the basal epidermal layer with palisading nuclei, whereas in (D), the loss of the basal epidermal layer is observed (8).

0.5 cm was observed on the anterior surface of the right knee, with no other visible external traumatic lesions. Internal examination revealed acute visceral congestion, asphyxial petechiae in the subconjunctival, subpleural, and subepicardial regions, and fluid blood. The death was violent, resulting from acute respiratory

failure due to tetanization of the respiratory muscles, as a consequence of a domestic electrocution accident (creating an infero-inferior current pathway: foot → foot) through direct contact with a low-voltage alternating current source in wet ground conditions, without visible electrical marks.

DISCUSSION

According to the literature, multiple scientific pieces of evidence and studies, ranging from 1899 (in Europe and the United States) to the present day, support ventricular fibrillation (VF) as the primary mechanism of death and reject asphyxia. These studies have also demonstrated the theoretical possibility of death due to respiratory arrest; however, they assert that accidental human electrocutions are predominantly caused by the induction of VF, as the duration of the electric shock is typically too short to produce death by asphyxiation. A current of sufficient magnitude, following a path that involves the cardiac trajectory, can

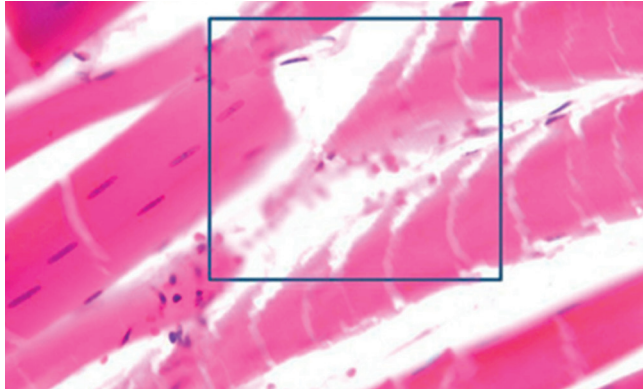


Figure 24. Rhabdomyolysis due to tetanization. Fragmented striated muscle fibers with necrosis and erythrocytes (the blue box highlights the details) (8).

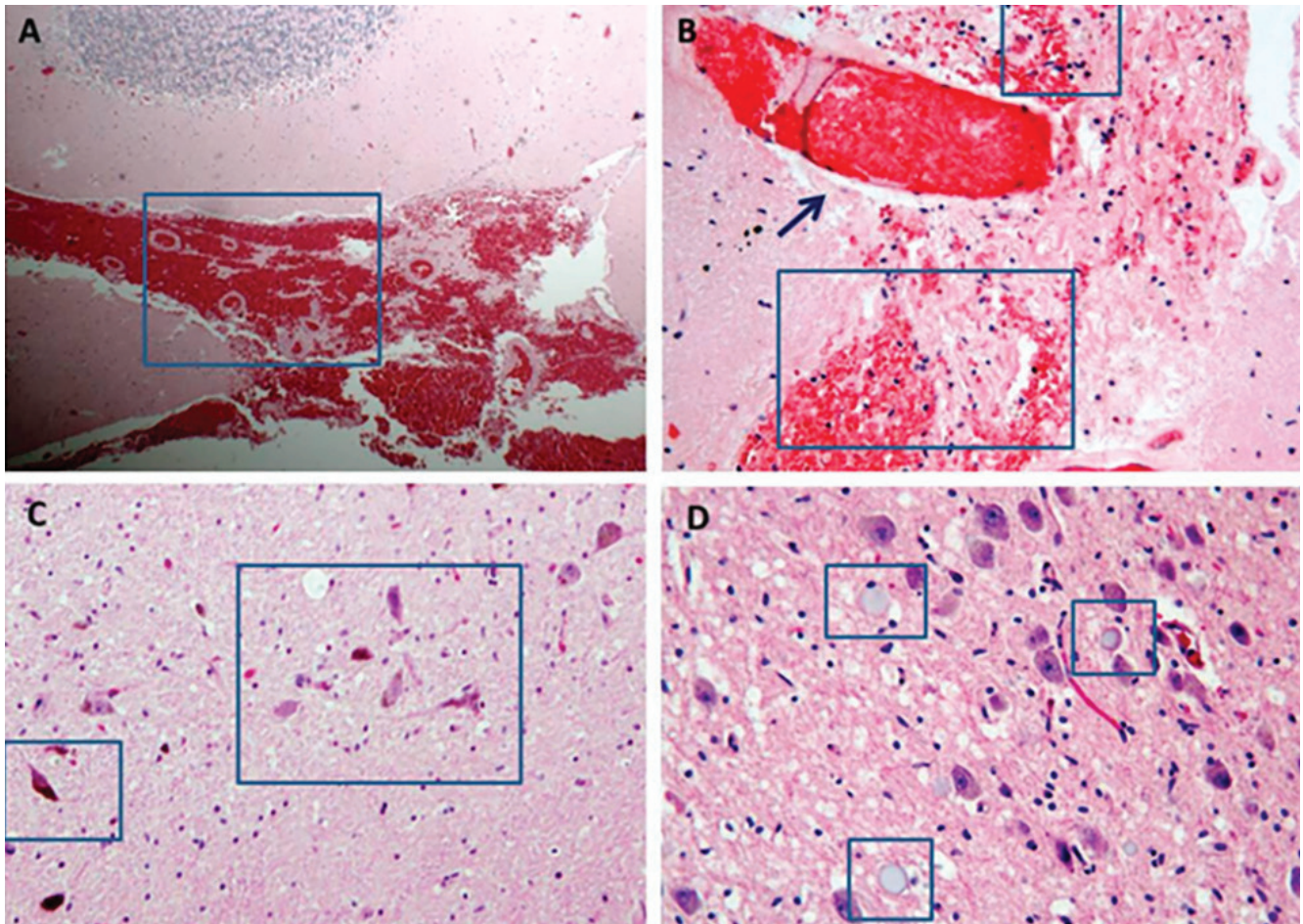


Figure 25. Brain histology in death by high-voltage electrocution. (A–B): Massive cerebellar hemorrhage and cerebral hemorrhage, respectively (blue frame), with intravascular coagulopathy in (B) (black arrow). (C): Dark neurons as a marker of ischemic neuronal injury due to vasoconstriction. (D): Axonal ballooning degeneration (blue frame) within a background of necrosis and white matter degeneration (8).

induce fibrillation and result in death within seconds. Georges Weiss investigated the issue of asphyxia versus VF induction and concluded that electrocution results from VF induction, since lethal respiratory arrest required an exposure duration of at least 10 minutes using medium-strength currents in dogs (9). In low-voltage electrocution victims, typical electric signs such as burns or other characteristic electrical lesions may not always be present. In such cases, the absence of specific external findings can hinder the identification of the cause of death. Electrocution produces a wide array of paraclinical and histopathological alterations, the accurate interpretation of which is essential for establishing causality in complex forensic cases. The integration of biochemical markers with macro- and microscopic findings enables the formulation of a solid differential diagnosis, especially when external lesions are absent or atypical. From a biochemical standpoint, electrocution is accompanied by a significant increase in markers indicative of skeletal muscle and myocardial injury: myoglobin, total creatine kinase (up to 5,000-6,000 IU/L), CK-MB (≤ 35 IU/L), and troponin I (1-4). These changes, also observed in therapeutic electroshock (defibrillation), correlate with early subepicardial hemorrhages and interstitial necrosis, detectable within the first 72 hours (1-4). Histopathologically, cutaneous lesions provide valuable diagnostic clues.

At the entry point, flattening of the stratum corneum and homogenization of the epidermis are typically observed, while at the exit point, vacuolization within the mucosal layer is evident. Additional features include the so-called “electrical channel,” dermal hyalinization, vascular thromboses, and follicular sheath undulation (1-4). At the ultrastructural level, the Somogyi phenomenon highlights electron-microscopic changes such as obliteration of membrane pores, mitochondrial damage, and detachment of the basement membrane, along with loss of desmosomal junctions (1-4).

Musculo-tendinous alterations resulting from tetanization include hyaline bands, corkscrew-shaped myofibrils, loss of striations, and coagulative necrosis (1-4). Cardiovascular damage is complex and encompasses cardiomyocyte fragmentation, pericapillary hemorrhages, interstitial edema, and focal necrosis (1-4). The specialized literature additionally describes cardiomyolysis, separation of myofibrils, disintegration of myocardial fibers, and alternating hypercontracted myocytes (8-10). However, to date, no strictly specific histopathological pattern of electrocution has been identified. Consequently, interpretation of these changes must consider known electrical parameters and the thanatogenic circumstances (10). Severe rhabdomyolysis, secondary to intense muscular tetanization, may lead to acute renal injury. This

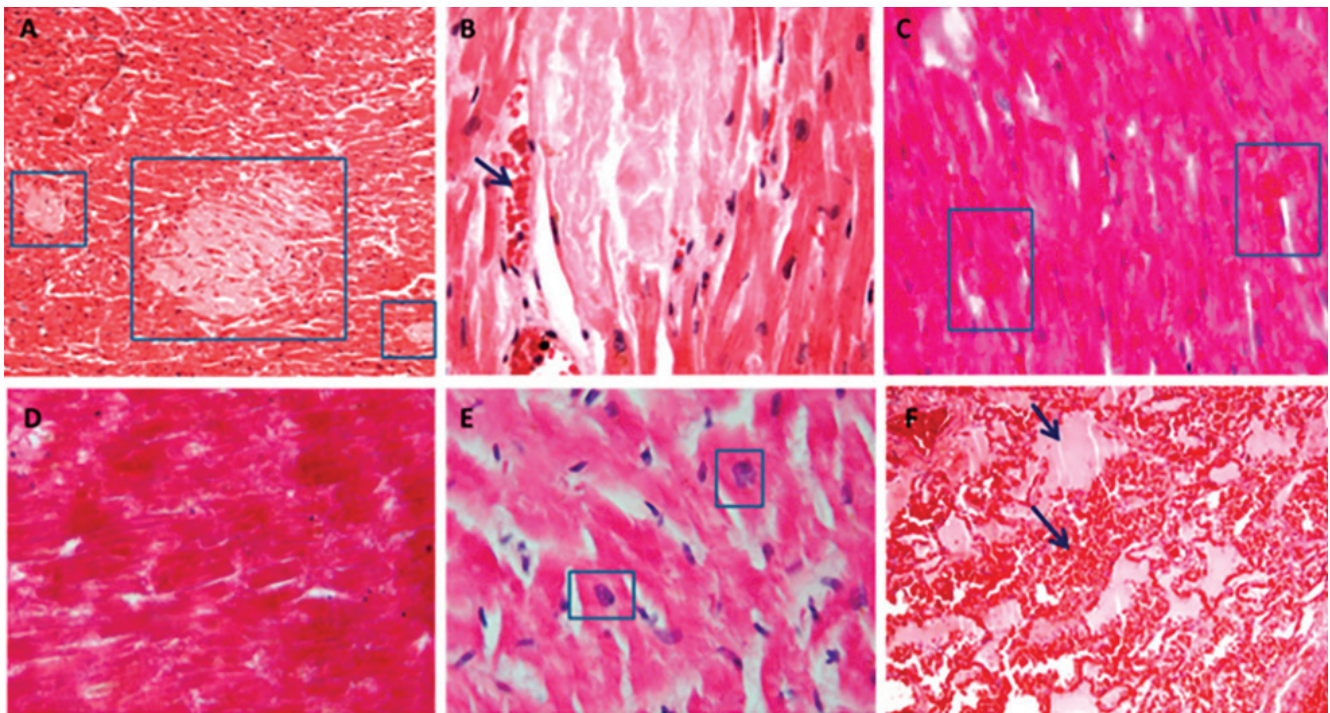


Figure 26. Heart and lung histology in high-voltage electrocution. Different locations of necrosis. (A–B): Multiple and diffuse foci with absence of inflammatory infiltrates (blue frame) and erythrocyte extravasation (black arrow). (C–D): Large areas of coagulative necrosis with nuclear loss and erythrocyte extravasation in (C) (blue frame). (E): Myofiber fragmentation with enlarged and distorted nuclei (blue frame). (F): Intra-alveolar pulmonary edema and hemorrhage (black arrow) (8).

condition is characterized by muscle fragmentation, necrosis, and erythrocyte extravasation, followed by acute tubular necrosis induced by myoglobin, especially in post-electrocution survivors (1-4). It is imperative to differentiate these lesions from other causes of rhabdomyolysis, such as cocaine use, extreme physical exertion, or seizures (8). Cerebral and systemic alterations further complete the morphological picture. Uneven hemorrhages, predominantly located in the basal ganglia and brainstem, along with perivascular necroses, vacuolar neuronal degeneration, and reactive gliosis, constitute classical signs of electrical aggression to the central nervous system (1-4). At the pulmonary level, interstitial edema and eosinophilic infiltration are observed, while the kidneys exhibit acute tubular necrosis (1-4).

Therefore, the correlation of complex biochemical and histopathological data with the physical parameters of the electric current remains essential for the assessment of thanatogenesis in electrocution. Their integration into a standardized interpretative framework will reduce diagnostic ambiguities and enhance the accuracy of forensic evaluations in cases lacking external electrical marks.

Alternative Thanatogenic Mechanisms in Electrocution: Between Ventricular Fibrillation and Respiratory Failure

Despite the fact that a significant number of studies strongly support ventricular fibrillation (VF) as the predominant thanatogenic mechanism in electrocution, the literature also acknowledges the development of asphyxial petechiae (which may be observed during autopsy at the subpleural and subepicardial levels) as indicative of death resulting from respiratory causes. These findings suggest their appearance as part of the macroscopic asphyxial syndrome occurring during the agonal period. The macroscopic asphyxial syndrome—mainly characterized by the presence of asphyxial petechiae—occurs very frequently in deaths of respiratory origin (94%) and relatively often in cardiac deaths (75%) (11). Supporting these observations, electric current can directly stimulate the motor nerves of the respiratory musculature, causing involuntary and prolonged muscle contractions. These contractions may lead to asphyxia due to the inability to perform normal respiratory movements. Lee WR *et al.*, in an experimental study on rabbits, demonstrated that electric shocks can induce tetanic contractions of the respiratory muscles, leading to respiratory arrest. The authors noted that, at currents

up to approximately 200 mA, respiratory arrest occurred exclusively due to tetanic muscle contractions (12). Bradford A *et al.* investigated the effects of low-voltage electric shocks on respiration in anesthetized cats. They observed that the application of electric currents (15-110 V AC, 8-45 mA, 50 Hz) caused respiratory arrest due to tetanic contractions of the respiratory muscles during shock administration. Following cessation of the shock, significant increases in ventilation, oxygen consumption, and carbon dioxide production were recorded, with some of these parameters remaining elevated even 60 minutes post-shock. These findings indicate that low-voltage electric shocks can induce marked respiratory, circulatory, and metabolic responses (13). Guimarães F *et al.* report the case of a 31-year-old man who suffered a low-voltage electrocution due to a water heater malfunction while turning on a shower tap. He experienced cardiac arrest but was successfully resuscitated. Subsequent investigations revealed direct pulmonary injury, manifested as bilateral ground-glass opacities, attributed to the passage of electric current through the thorax and possible tetanic contraction of the respiratory muscles (14). From a histological standpoint, current- and heat-related changes have been demonstrated, such as hypercontraction bands in the intercostal muscles and coagulative changes in peripheral nerves (within the nerve fascicle sheath) (15). These observations highlight the importance of a systematic evaluation of electrocution cases that considers not only ventricular fibrillation mechanisms but also the potential effects of asphyxia.

Forensic interpretation protocol for electrocution cases

1. Purpose of the Protocol: This protocol aims to standardize forensic evaluations in cases of electrocution, particularly in the absence of classic electrical marks. Through an integrated approach that considers macroscopic, histopathological, and biochemical data, the protocol will enable more accurate identification of thanatogenic mechanisms and contribute to improved documentation of such cases.

2. Evaluation components: The protocol will include the following components:

Macroscopic examination:

Identification of cutaneous and mucosal lesions: visible external injuries (entrance and exit electrical marks) or other associated trauma, including their appearance and location.

Assessment of other findings, such as: asphyxial

petechiae, acute pulmonary edema, congested organs, and the presence or absence of fluid blood.

Histopathological examination:

Analysis of affected tissues: recommended sampling includes muscle (intercostal, diaphragm), heart, brain, and lungs.

Detection of specific changes such as muscle fiber fragmentation, myocardial necrosis, and cerebral hemorrhages.

Histopathological assessment of pulmonary and renal lesions.

Biochemical evaluation:

Measurement of serum levels of myoglobin, CPK (including MB fraction), and troponin I, as indicators of muscular and cardiac injury (nonspecific in electrocution), with a rigorous differential diagnosis. Analysis of other relevant biochemical markers that may support the differential diagnosis.

3. Interpretation of Results

Identification of the Thanatogenic Mechanism:

Determining the cause of death by correlating macroscopic findings, histopathological data, biochemical results, and investigative information.

Differentiation between death due to ventricular fibrillation and that caused by asphyxia, considering the presence or absence of electrical marks and/or other signs such as asphyxial petechiae and fluid blood.

Distinguishing between death caused by inhibition of brainstem neural centers and other causes, based on the intensity of the electric current, the entry point, and the presence or absence of other relevant macroscopic signs.

Additional Considerations: Correlation with the medical history, accident circumstances, including alcohol or drug consumption that may influence the results.

Integration of forensic medical data with information obtained from criminal investigations.

4. Innovation brought by this Protocol

This integrated approach has not been systematically explored in the national literature, thus offering a valuable framework for forensic specialists. By standardizing evaluations and integrating various types of data, this proposal may lead to a deeper understanding of electrocution-related mechanisms of death and to improved differential diagnosis in complex cases.

In conclusion, the current specialized literature uniformly supports the notion that ventricular fibrillation (VF) represents the most plausible cause

of death in the presented context, due to its well-understood pathophysiological mechanism and the availability of modern diagnostic methods. This makes VF more amenable to being demonstrated with greater precision compared to other potential thanatogenic mechanisms.

Although some experimental studies provide evidence for the mechanism by which tetanization of the thoracic musculature, induced by electrocution, may lead to asphyxia and death, it is important to note that recent literature lacks a significant number of such documented cases. The distinction between these thanatogenic mechanisms in electrocution-related deaths remains a matter of scientific debate in the absence of advanced electrophysiological and histopathological studies that could establish definitive criteria for their differentiation.

While certain paraclinical markers (e.g., myoglobin, CPK) may be significantly altered in electrocution, these are nonspecific, highlighting the need for increased attention to differential diagnosis.

Moreover, investigative findings may provide crucial clues in determining the cause of death—especially in the absence of specific findings such as cutaneous and mucosal lesions (burns/electrical marks), the latter being pathognomonic for electrocution. The presentation of this medico-legal interpretation protocol, based on an integrated evaluation, aims to emphasize the need for increased attention in investigating and documenting the thanatogenic mechanisms of electric current. It further underscores the relevance of a systematic approach in contemporary forensic practice, with the potential to enhance the quality of assessments and conclusions in electrocution cases.

Conflict of interest

The authors declare that they have no conflict of interest.

References

1. Bulgaru, Ilieșcu, Schițco, Curcă, Enache (coord.) (2022). *Tratat de traumatologie medico-legală*. Vol. II. Leziuni fizice, agenți chimici, agenți biologici. ași: Timpul. ISBN: 978-973-612-972-8.
2. Beliş V. (red.) (1995). *Tratat de medicină legală*. Vol. II. București: Regia Autonomă „Monitorul Oficial”. ISBN: 973-39-0262-4 / 973-39-0272-1.
3. Valentin I. (2009). *Medicina legală*. București: Editura C.H. Beck. ISBN 978-973-115-479-4.
4. Saukko P, Knight B. *Knight's Forensic Pathology*. 3rd ed. London: Hodder Arnold, part of Hachette Livre UK; 2004. ISBN: 978-0-340-76044-4.

5. Hecser L. *Medicina Legală*. Vol. II: Teorie, legislație, practică. Târgu Mureș: University Press; 2006. ISBN: 973-86119-0-3.
6. Cherington M. Lightning Injuries in Sports. *Sports Med*. 2001; 31(4): 301-308.
7. Wetli CV. Keraunopathology: An Analysis of 45 Fatalities. *Am J Forensic Med Pathol*. 1996; 17(2): 89-98.
8. Mansueto G, Di Napoli M, Mascolo P, Carfora A, Zangani P, Pietra BD, Campobasso CP. Electrocution stigmas in organ damage: The pathological marks. *Diagnostics*. 2021; 11(4): 682.
9. Kroll MW, Luceri RM, Efimov IR, Calkins H. The electrophysiology of electrocution. *Heart Rhythm O2*. 2023; 4(7): 457-462.
10. Favia M, Mele F, Introna F, De Donno A. Morphological cardiac changes in electrocution deaths: A literature review. *Med Sci Law*. 2021 Jan;61(1_suppl):130-135.
11. Tăbircă DD. Asphyxia syndrome in underlying versus direct causes of death. The Publishing House of the Romanian Academy. „Sf. Apostol Andrei” Emergency Hospital, Department of Legal Medicine, Constanța, Romania. 2019.
12. Lee WR, Zoledziowski S. Effects of electric shock on respiration in the rabbit. *Br J Ind Med*. 1964;21(3):220-226.
13. Bradford A, O'Regan RG. The effects of low-voltage electric shock on respiration in the anaesthetized cat. *Q J Exp Physiol*. 1985;70(1):115-127.
14. Guimarães F, Camões J, Mesquita A, Gomes E, Araujo R. A Case Report: Low Voltage Electric Injuries Culminating in Cardiac Arrest and Direct Lung Injury. *Cureus*. 2020;12(10):e11261.
15. Anders S, Schulz F, Tsokos M. Intramuscular haemorrhages of vital origin in a case of suicide by electrocution. *Rechtsmedizin*. 2000;10:105-109.