KAPITEL 11 / *CHAPTER 11* [11]
RESPIRATORY SYSTEM DAMAGE DUE TO ELECTRICAL INJURY

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Introduction.

Respiratory system damage due to electrical injury (EI) is relatively uncommon. The number of available foreign literature sources on this topic is significantly limited, and local reports mainly refer to pathological anatomy. 75% of cases of respiratory system damage due to ET occur at low voltage (< 1000 V), most often 220-240 V [1-3]. With a higher voltage difference and alternating current strength, respiratory arrest occurs more frequently [4]. The most dangerous and leading to respiratory arrest frequency of electric current is 200 Hz [5]. Apnoea occurs immediately after the injury [2].

Isolated lung damage due to EI has been reported relatively rarely [6]. The electrical resistance of lung tissue is relatively higher than in other chest structures, which may explain the lower incidence of direct lung damage, as more current passes through surrounding structures with lower resistance and therefore the lungs rarely suffer direct electrical injury [7-10].

Evidence collection. Literary sources were included in the study if they: 1) were published in Ukrainian, English or Spanish; 2) reported respiratory disorders and organic lung damage associated with electrical injury; 3) reported on the prevalence of respiratory disorders in electrical injury; 4) used observational designs (cohort or cross-sectional). A retrospective search for information was conducted using a spatial vector descriptive model, which was supplemented by a manual search for relevant articles. 27 literature sources were selected, all of which were published within the last 10 years.

Evidence synthesis. When the alternating current reaches 20 mA, tetanic contraction of the respiratory muscles occurs [3,7]. If the electric current passing through the body goes through the chest, there is a risk of paralysis of its muscles and cardiorespiratory arrest. [7-11].

[11]***Authors:*** *Kravets Olha V., Yekhalov Vasyl V., Khobotova Natalya V., Zinenko Mariia D.*
Author's sheets: *0,41*

The path of the current that causes sustained tetanic contractions of the respiratory muscles usually goes from hand to hand or from hand to foot and mostly does not involve the respiratory centre located in the medulla oblongata [7]. However, the vertical path of the current causes spasm of the vertebral arteries supplying the respiratory centre, primary electrically induced damage (inhibition or paralysis) of the respiratory centre of the brain, clonic contraction of the diaphragm, and the onset of 'electric shock' [2,3,7,12-15]. Spasm of the glottis (laryngospasm) with restrictive airways is the cause of 'electrical asphyxia' [3,5,7,12,16].

Electrical lung injuries can be classified as primary or secondary. Primary pulmonary gas exchange disorders are caused only during direct exposure to electric current and shortly after an injured person is rescued [15]. Respiratory acidosis with hypoventilation of the pulmonary areas occurs [17]. Primary parenchymal lung injuries directly caused by electric exposure are rare, as it was previously hypothesised that air is a poor conductor, making lung tissue less susceptible to damage. Chest pain, shortness of breath, and fever are commonly associated with electricity-induced lung injury (EILI). However, these symptoms may be masked by concomitant cardiovascular problems. Secondary apnoea occurs after asystole or ventricular fibrillation. Hypoxia and hypercapnia lead to secondary damage to the respiratory centre. Secondary pulmonary injuries are more common and include aspiration and nosocomial pneumonia (often due to loss of consciousness), pulmonary contusion (due to secondary trauma), inhalation burns (due to thermal trauma) and cardiogenic pulmonary oedema (caused by ventricular fibrillation or cardiac arrest) [2,5,6,11].

If strong electric current passes directly through the lungs, serious visceral injuries may occur. The respiratory system may be paralysed, heart rate may become rapid and irregular, or cardiac arrest may occur. Hypoxia is usually accompanied by asystole [12,18].

Bilateral changes in the upper areas may correspond to direct electrical damage to the lungs, as electrical marks are assumed to be present on the hands, forearms, and upper chest. Other factors, such as injuries caused by resuscitation measures, are considered less likely due to the exceptional upper/apical location of the affected areas.

On the other hand, consolidations in the right lower lobe are described, which may be associated with aspiration of gastric content [1,10]. However, mainly the lower parts of the lungs are affected [2,10,18].

There are two distinct patterns of electricity-induced lung injury (EILI): pneumomediastinum and parenchymal/interstitial injuries, ranging from non-cardiogenic oedema to consolidation [10].

Electric injuries can cause a number of physiological malfunctions. They can affect the endothelium, which in consequence increases the permeability of the pulmonary blood vessels. This can cause fluid to move from the blood capillaries into the interstitium of the lungs or even into the alveoli. The accumulation of fluid in the lung tissue can seriously affect respiratory function, causing a condition known as pulmonary oedema [3].

In cases of electric injury, fibrillation and cardiac failure are associated with congestive pulmonary oedema. Congestion and oedema are the most common pulmonary signs of EILI, and the lungs are often hyperaemic and heavy. Rare cases of non-cardiogenic lung injury are described in the literature as an underdiagnosed clinical unit that occurs in any neurological or non-neurological event that can stimulate vasomotor centres. There are various theories explaining the cause of this rare phenomenon, but none of them has been reliably proven [19].

One mechanism of foam formation is destabilisation of the pulmonary vegetative nervous system. This may be the result of mechanical disruption or electrical failure of the vasomotor centres of the central nervous system, ultimately leading to pulmonary complications. Taking these factors into account is a vital part of the diagnostic process, as it provides opportunities to narrow down the range of potential causes and guide the medical team towards a more accurate understanding of the condition. These changes are accompanied by constriction of the pulmonary vessels, increasing hydrostatic pressure in the pulmonary capillaries, damage to the alveolar membrane, fluid leakage into the intra-alveolar and interstitial spaces, bleeding, and damage to the alveolar membrane [3]. In contrast, haemoptysis (except for the appearance of pink frothy sputum, which indicates pulmonary oedema) is a distinctive feature of

neurogenic pulmonary infarction [2,3]. Haemoptysis was present in 25% of cases of EILI, sometimes at a mild and self-limiting stage [2].

Lung contusion is sometimes the result of secondary trauma due to an explosion or fall. However, pneumothorax is a very rare complication of EI [2,9,11,20].

Respiratory tract injury can also occur as a result of inhaling smoke or toxic gases from flames, which are formed indirectly due to the action of electric current. The presence of inhalation injuries in burn trauma correlates with high morbidity and mortality rates. Inhalation injury is assessed by fibreoptic bronchoscopy; a direct correlation has been established between the severity of inhalation injury observed during bronchoscopy and patient mortality. Almost a third of ICU patients with electrical injuries had inhalation injuries of varying severity, which led to a worsening prognosis [21].

Acute pulmonary complications are sometimes isolated to pleural involvement, leading to exudative pleuritis and lobular pneumonitis between the entry and exit points of the current, which usually manifest at the end of the first week. Complaints of shortness of breath in patients are more often due to uraemia and the presence of pleural effusion, the formation of which is influenced by an imbalance between fluid production, its absorption and several protective forces, such as plasma osmolality, hydrostatic/venous pressure and capillary wall perfusion. Pleural effusion in this case may be caused by increased capillary wall perfusion due to electroporation and rhabdomyolysis, which causes acute tubular nephrosis [22].

Another potentially late indicator is pulmonary embolism due to post-traumatic deep vein thrombosis [11].

Electrical lung injury can resemble conditions such as drowning, poisoning, and neurological disorders, which are often characterised by the presence of foam in the lungs [3].

However, EILI generally has a favourable prognosis, often resolving spontaneously within 3 to 10 days in the absence of severe multi-organ damage [2].

Macroscopic and microscopic changes in the lungs in EILI manifest themselves in ruptures of lung tissue, the presence of necrotic foci, and the integrity of blood

vessels walls destruction and bronchi destruction on the background of significant haemorrhages. Bronchospasm with epithelial oedema and swelling of the interstitial lung tissue are observed, and isolated eosinophilic infiltrates are seen [5,23]. Histological findings are characterised by diffuse and watery pulmonary oedema due to changes in hydrostatic pressure between the vascular and interstitial spaces. Therefore, attention should be paid to the type and extent of pulmonary oedema. The distinction between functional and protein oedema, which occurs in diffuse alveolar damage, and the presence of intra-alveolar haemorrhages are fundamental and useful for differential diagnosis. The presence of hyaline membranes, inflammatory elements and/or pigment inclusions against a background of oedema are defined as pathological aspects that may be useful in assessing the likelihood of other causes of death [19].

In cases of high-voltage electrical injuries, even if there is no specific visible damage to the lungs or airways, one of the main causes of sudden death is respiratory arrest [3,14]. With EI, a pause in breathing lasting up to 20 seconds is usually not fatal, apnoea lasting 20 to 30 seconds most often leads to death, and more than 30 seconds is always fatal [12]. Pallor of the skin is determined in 'white asphyxia', which occurs as a result of primary cardiac arrest or simultaneous circulation arrest and respiratory arrest; cyanosis of the skin is observed in 'blue asphyxia', which occurs in primary respiratory arrest [24]. Due to the small number of cases, the pathogenesis of EILI has not been fully studied [18].

Given the life-threatening nature of the condition, timely diagnosis is essential, as massive haemoptysis can cause airway obstruction, leading to hypoxia and death [1].

The diagnosis can be confirmed by the presence of fresh haemoptysis and the results of bronchoscopy, chest X-ray and ultrasound examination [1,3]. When performing fibrobronchoscopy, blood is observed in the bronchial branches, Gram-stained bronchial secretion samples show the presence of erythrocytes. These data are consistent with pulmonary alveolar haemorrhage due to electrical injury. Fibrobronchoscopy has certain advantages in the diagnosis of EILI, as it is useful in ruling out aspiration pneumonia in patients with a history of loss of consciousness or falls, ruling out pulmonary infections, and confirming the diagnosis of EILI. However,

the procedure should be carefully considered in cases where there is a risk of progressive respiratory failure or when the patient's condition does not allow bronchoscopy to be performed [2].

In terms of imaging characteristics, EILI shows the following features: 91% of cases have bilateral distribution, with 64% showing predominant consolidation in the lower lobes. Various patterns have been reported, ranging from alveolar to interstitial damage, including consolidation, nodules, thickening of the interlobular septa, pleural effusion, cavitation, and 'ground glass opacity' (GGO). These data are coherent with radiological signs of diffuse alveolar haemorrhage (predominantly with damage to the lower regions) or acute respiratory distress syndrome (ARDS) with damage to the gravity-dependent or posterior basal regions. Diffuse alveolar haemorrhage is the predominant pathological feature in cases of EI. This condition results from widespread damage to high-flow pulmonary and bronchial vessels, leading to haemorrhages into the alveolar spaces. GGO is observed around bronchovascular fascicles, spreading from the pulmonary hilum to peripheral areas, diffusely affecting both lungs, with consolidation predominantly determined in the basal areas, where the blood supply is more abundant. In some cases, blood was found in the alveoli without signs of other inflammatory exudates. EILI without damage to the chest wall can be explained by two mechanisms. The first is that electric current affects tissue through the thermal energy generated by tissue resistance. This mechanism leads to protein denaturation and coagulation necrosis. The second, electric current causes tissue damage at the cellular level by altering the resting membrane potential, leading to electroporation, damage to channel proteins, and disruption of the cell membrane. This causes massive tissue destruction, necrotic and conformational changes in EILI, characterised by damage to the alveoli, interstitium, parenchyma and pulmonary vessels. Haemorrhagic areas and erythrocytes are found in the alveoli, indicating diffuse alveolar haemorrhage. There are also signs of vascular congestion and interstitial fibrosis. Pulmonary infarction can be caused by embolism or thrombosis due to vascular damage, arterial spasm, or impaired blood flow in the capillary bed. Coagulation necrosis, typical ARDS pattern, or fat embolism have also been reported. Haemoptysis may be the result of vascular

damage caused by electrical injury, leading to rupture and subsequent pulmonary haemorrhage [1-3,6,10].

Computed tomography of the chest shows patchy infiltration and focal consolidation with pulmonary infiltrates in both lungs, opacity behind the GGO and thickening of the interlobar septa distributed throughout both lungs, with consolidations predominantly located in the dependent areas of the lungs after exposure to low-voltage electric current [1,2,6,10,25].

When the heart rhythm restarts spontaneously after cardiac arrest, breathing may not occur due to paralysis of the respiratory centre. This may be caused by damage to the brain stem respiratory centre due to the hyperthermic effect of electric current [26,27]. Normal breathing is restored after the power is turned off, provided that the duration of its action is less than 4 minutes. If prolonged contractions last longer than this time interval, death from suffocation occurs. This consequence can be avoided by starting artificial lung ventilation (ALV) [7]. Up to one-third of patients admitted to the ICU with electrical burns required mechanical ventilation. The minimum duration of mechanical ventilation was 7 hours, the maximum was 7,200 hours, and the average was 427 hours. Long-term mechanical ventilation determines a poor prognosis for the patient, causing numerous complications. Patients with extensive burns who require prolonged mechanical ventilation and multiple surgical procedures are candidates for tracheostomy [21].

EILI can be potentially fatal if vital treatment has not been administered [18], which includes oxygen therapy, artificial ventilation, lobectomy, antifibrinolytic agents (for haemoptysis) and bronchodilators (for bronchospasm). In addition, treatment of other organs and systems affected by electricity is crucial, including resuscitation measures, infusion therapy, urine decontamination, anticoagulant therapy, and antibiotics. Closed thoracostomy may be used to remove pleural effusion, which largely prevents its re-accumulation [2].

Pulmonary infection can develop as a result of systemic infection or inhalation injury. Some authors recommend early lobectomy or removal of necrotic tissue to prevent nosocomial infections, as delaying surgery can lead to multiple organ failure.

However, there are currently no effective clinical guidelines for the treatment and prevention of EILI. Nevertheless, the majority of patients recover without lung resection, indicating that symptomatic treatment of EILI may be considered at the onset of the pathological process [2].

Conclusions.

1. Electrically induced lung injury is a rare and therefore insufficiently studied complication of electrical injury.

2. Respiratory disorders can be primary or secondary.

3. Early diagnosis and intensive treatment of electrically induced lung injury can prevent death from electrical injury in most cases.