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Wound Congress & Clinical Dermatology Congress 2018: Inhalation burn, crush-syndrome and rhabdomyolysis syndrom - Dalamagka Maria - UK University Medical Centre Schleswig-Holstein

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Inhalation burn is responsible for 50% of the mortality associated with thermal burn. Inhalation burns are usually observed in exposure to smoke, heat, toxic gases, and combustion components. Inhalation burn causes damage to airway epithelium, mucosal edema, and reduces surface activity. These conditions are clinically manifested by airway obstruction, bronchospasm and atelectasis. The area above the tongue is particularly vulnerable to thermal damage. Often the burn develops swelling and obstruction of the upper airways, which may not be immediately apparent. The larynx can be affected not only by thermal burn but also by the direct toxic action of irritant gases, showing early tibial swelling and laryngospasm.

Unlike the upper airway lesions, lesions of the tracheobronchial tree are almost never caused by heat. Heat burn of the lower airways is only observed in exceptional cases of fire in a saturated water vapor environment. The lesions are usually of a chemical nature of irritating gases and soot. The diagnosis is usually based on clinical behaviors such as: facial burns, soot deposition in the rat, pharynx, epiglottis and tongue, voice gurgle, laryngospasm image and bronchospasm. Smoke exfoliation is also a positive diagnostic point.

The burning of natural material (wood, wool, silk) and certain synthetic substances such as nylon and polyurethane causes the release of cyanide with the most important hydrogen cyanide. Their poisonous action is due to the binding of cytochrome oxidase and inhibition of oxygen uptake at the cellular level. Cyanide poisoning should be suspected of any victim of fire in an enclosed space where unexplained metabolic (lactic) acidosis occurs. Concentration of lactate> 10 mmol / L indicates a high probability of cyanide poisoning. Blood cyanide concentration greater than 40 mmol / L is considered to be toxic, with a concentration of 100 mmol / L being fatal. The clinical picture includes tachypnea, tachycardia, confusion, convulsions, metabolic acidosis, and at higher concentrations of respiratory depression and circulatory insufficiency.

The specific treatment of cyanide intoxication involves the administration of 25% sodium thiosulphate at a dose of 50 ml at an infusion rate of 2.5 ml / min, which converts cyanide into thiocyanates that are less toxic and are eliminated by the kidneys. Also reported is the administration of hydroxybutylamine (Vit B12) which binds cyanide to the formation of hydroxycyanocobalamin. The dose used is 5 g at slow intravenous infusion. Burn injury is characterized by the development of burnout. Burning shock is due to a combination of hypovolemia and local and systemic secretion of a large number of mediators of inflammation. The most popular equation for calculating liquids is the Parkland equation: R / L 4ml × body weight (kg) ×% EU; 50% of the volume of fluid is given in the first 8 hours, the rest - 16 hours; If the EU assessment is not feasible, it is recommended to administer 20 ml / kg of body weight; crystalline solutions during the first hour of injury the first 24 term is recommended to use only crystallized solutions. Factors which increase the needs of the liquids delivered: Inhalation burns; Delay in fluid delivery; Electric burn; Extensive extent of burn surface; Concurrent injuries; In recent years, the so-called "fluid creep" phenomenon has been described Crush-syndrome and rhabdomyolysis syndrome: It was first described after a London bombing during the Second World War.

The syndrome occurs during natural disasters, wars, explosions, industrial accidents. Compression of muscle mass leads to tissue ischemia, an increase in tissue pressure, which exceeds the capillary filtration pressure. After lifting the external pressure, the muscle tissue is reperfused. The mechanism of ischemia-reperfusion injury is the major pathophysiology-logical mechanism of this syndrome. Often, pressure damage is associated with vascular damage, traumatic vascular rupture, thrombosis and stroke. Occlusion syndrome is clinically manifested by hypoemia sequelae due to high accumulation of fluids in damaged tissues and by seo resulting from the release of large amounts of toxic substances from the injured tissues. The first manifestation of the syndrome is usually hypoaemic shock, which is the most common cause of death during the first 4 days after the injury. Hyperkalemia with its effect on heart function is the second cause of premature death. Myoglobin is a protein that accounts for 1-3% of the dry weight of the muscle tissue. Normally, myoglobin binds to actoglobin and α-2 globin and is eliminated through the duodenal endothelial system.

The saturation of aptoglobin (when a large amount is produced of myoglobin) leads to an increase in free myoglobin in plasma that is excreted by the kidneys. In the renal tubules, myoglobin forms casts, especially in acidic environment. Urine alkalinisation reduces the generation of these complexes. Myoglobin causes immediate action in the renal tubules, helping to increase free radicals of oxygen and promoting fat peroxidation. This action of myoglobin is also limited to alkaline environment. Patients usually experience: extensive soft tissue injury with ischemia-reperfusion effect, edematous edges, compartment syndrome, dark-colored urine, positive test for hemoglobin, absence of red blood cells in the urine, elevation of CPK levels in the blood.

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