



Morphological Changes in the Colon in Burns With Acetic Acid

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Abstract. Actively ongoing research into chemical burns of the digestive tract confirms the complexity of this problem and its relevance. When taking acetic acid, a burn disease of chemical etiology develops due to the local destructive effect on tissue and its resorptive effect as a hemolytic poison. The article presents an analysis of literature data on morphological changes in the colon during burns of the gastrointestinal tract with acetic acid under experimental conditions.

Keywords: morphology, large intestine, acetic acid, gastrointestinal tract.

Relevance of the problem: Currently, chemical burns of the digestive tract of varying degrees of severity represent a very pressing medical, social and economic problem. Severe burns of the upper gastrointestinal tract affect 10-33% of adult patients, mortality is up to 10% [Rollin M. et al. 2015].

Sharp hyperemia of the burned mucous membrane of the stomach and intestines contributes to the rapid penetration of acetic acid into the bloodstream. The absorption of acetic acid causes severe disturbances in the acid-base balance, such as sub- or decompensated metabolic acidosis, which are caused by the participation of exogenous factors - anions of acid residues and endogenous factors - under-oxidized metabolic products formed during a chemical burn of the digestive tract and its various complications. The consequence of resorption is hemolysis of red blood cells. The undissociated acetic acid molecule is the main hemolytic agent. In the presence of acetic acid, hemoglobin is broken down into globin and heme, and the latter is oxidized to hemin. Hemoglobin, iron ions, especially heme compounds, accelerate the decomposition of hydroperoxides with the formation of free radicals capable of activating new oxidation chains. Hemoglobin in this regard is 100 times more active than iron ions. Hemoglobin catalysis is enzymeless and cannot be inhibited [Luzhnikov E.A., Kostomarova L.G., 2016].

A common chemical burn of the digestive tract in 37% of cases is accompanied by the development of exotoxic shock. Psychomotor agitation, followed by confusion and complete indifference to the environment, pallor and hypothermia of the skin are noted. Blood pressure increases due to systolic pressure to 150–160 mm Hg. Art., then drops sharply, central venous pressure decreases, tachycardia, shortness of breath, cyanosis increase, and diuresis decreases. Exotoxic shock has a pronounced hypovolemic character, and is also accompanied by profound disturbances in the acid-base state with the development of decompensated metabolic acidosis. Mortality with the development of shock reaches 64.5% [Ostapenko Yu.N., 2014].

The experiment showed that already one day after intragastric administration of acetic acid, signs of edema are observed in the lungs with thickening of the walls of the alveoli, a decrease in the volume fraction of the alveoli, an increase in exudate, and the appearance of red blood cells in the lumen of the alveoli. Local atelectasis occurs. Destructive processes intensify in the first 7 days. The volume fraction of the alveoli during this period can decrease to 55%, which negatively affects the respiratory function of the organ. From 7 to 14 days there is a slow recovery process. Late effects of burns include retrograde changes in the oral cavity, esophagus, stomach or respiratory system. In addition, it can lead to the formation of strictures in organs. The formation of strictures can have



serious systemic consequences for the patient, including poor general condition, significant weight loss, malnutrition, recurrent aspiration leading to respiratory infections, and potential respiratory failure [Cheng H.T. et al. 2018].

During a burn disease with severe acetic acid poisoning, the following stages are distinguished: I – stage of exotoxic shock and initial manifestations of intoxication (1-1.5 days); II – stage of endotoxemia (2-3 days), which is characterized by hyperthermia and often observed acute intoxication psychoses; III – stage of infectious complications (from 4 days to 2 weeks). During this period, clinical signs of esophagitis, gastritis, tracheobronchitis, pneumonia, reactive pancreatitis, reactive peritonitis appear; IV – stage of stenosis and burn asthenia (from the end of the 3rd week), accompanied by a violation of protein and electrolyte balance, a decrease in body weight in severe poisoning up to 15-20%; V – stage of recovery [A.A. Stopnitsky, R.N. Akalaev, 2017]. A study of the literature data showed that there is practically no study of the morphological parameters of the large intestine during burns with acetic acid and this requires further study and scientific research in this area.

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