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Morphological Changes in the Small Intestine Wall in Experimental 3-Month-Old White Breed Rats with Burns of the Digestive Tract Caused by Acetic Acid

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Abstract In case of chemical burns, gastrointestinal and respiratory burns can be caused by accidental or suicidal use of the substance. The circumstances of the accident and the chemical nature of the substance determine the degree of damage and the toxicological risk. The early period after a chemical burn is characterized by swelling of the larynx, perforation of the esophagus, stomach and intestines, gastrointestinal bleeding and the possibility of pancreatitis.

Keywords Chemical burn, Gastrointestinal tract, Resorption, Moxibustion

1. Introduction

Active ongoing research in the study of chemical burns of the gastrointestinal tract confirms the complexity of this problem and its relevance. According to the American Association of Poison Control Centers, more than 1.6 million child poisoning occurred in 2008 alone, with alkaline esophageal burns recorded in 18-46% of cases after consuming various household chemicals.

In chemical burns of the digestive tube with acetic acid, accidental or suicide of the substance can cause gastrointestinal and respiratory burns. The circumstances of the accident and the chemical nature of the substance determine the degree of damage and toxicological risk. The initial period after chemical burns is associated with the possibility of laryngeal edema, perforation of the esophagus, stomach and intestines, gastrointestinal bleeding, and pancreatitis.

Currently, chemical burns of various degrees of digestion are a very urgent medical, social and economic problem. Severe burns of the upper gastrointestinal tract affect 10-33% of adult patients, the mortality rate is observed up to 10%.

Two age groups are at the greatest risk: children aged 2-6 years, who involuntarily consume household cleaning products and burn up to 80% of ingestion, but usually with mild injuries; and adults aged 30-40 years who use strong corrosive substances with the intention of suicide and who resort to serious, life-threatening injuries. Suicidal attempts predominate in women as reasons for taking burning fluids. Most often they are observed in young women and are seasonal (spring and autumn). In some patients, knowing the possible death and the nature of the fluid causes fear.

Patients who decide to commit serious suicide take a large amount, more than 50-100 ml of burning fluid in one sip, and they experience deep burns not only of the oral cavity, throat and esophagus, but also of the stomach, duodenum and hungry intestine. Such patients die of severe general intoxication and extensive tissue necrosis within the first two to three days.

Severe burns can not only cause extensive damage to the skin and subcutaneous soft tissues, but also cause damage to many organs due to ischemia, hypoxia and inflammation, and the intestine is one of the most vulnerable organs to burn damage. Burn injury significantly reduces blood perfusion in the intestine, as a result of which the structure and function of the intestinal mucosa is disrupted. The intestinal mucosa is the main place responsible for the digestion and absorption of food, and its barrier function is also the main element responsible for maintaining the ecological stability of the body.

Damage to the intestinal mucosa barrier after a burn injury can lead to the migration of bacteria and toxins, and this translocation can lead to intestinal infections and upper intestinal metabolism, which leads to a poor prognosis. Studies have shown that loss of the mucous barrier can exacerbate intestinal damage caused by various pathological factors. Failure of mucous secretion under various pathological conditions and damage to the intestinal mucosa as a result of changes in mucous composition are some of the starting factors leading to intestinal damage.

The late consequences of burns include retrograde changes in the oral cavity, esophagus, stomach or respiratory system. In addition, this can lead to the formation of strictures in the organs. The formation of stricture can have serious systemic consequences for the patient, including poor overall condition,

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significant weight loss, malnutrition-related diseases, repeated aspiration leading to respiratory infections, and potential respiratory failure. To assess the severity of the injury in patients with acetic acid poisoning, it is recommended to determine the presence and level of free hemoglobin in the blood and urine. [1]

2. The Purpose of the Study

White breed rats digest what to use the biological method of correction using black sedana oil in order to detect trophological changes in the wall of the small intestine in burns caused by various degrees of oxalic acid, to reduce the side effects of the chemical substance.

3. Research Materials and Methods

For experimental purposes, 120 white non-breeding bats of both sexes were used, which were kept in normal vivarium conditions of 3 months. All experimental rats were kept in vivarium in plastic cages, at room temperature, in accordance with the standards for keeping laboratory animals. At the beginning of the experiment, all rats were quarantined for a week, and after the elimination of somatic or infectious diseases, they were transferred to a normal vivarium regime. Based on the model developed in the study on the chemical burn of white-breed rats digested nai No. 2269349, a61k 35/00, a61n 5/067, a61r 1/00, publ. 10.02.2006 held. In the experiment, the animals of the experimental group were knocked unconscious under general narcosis using a general ether, a solution of acetic acid (70% of the total dose) was sent through an 8 cm probe with a diameter of 1.5 ml. Animals were divided into 3 groups (n=110): Group I-3 months of Control (n=25); Group II rats with a 3 – month (total dose of 70% li) dose of acetic acid (n=40); Group III - 3 months (total dose 70%,) after receiving a dose of acetic acid, rats were ingested through a black sedana oil probe for 30 days (n=45); [2].

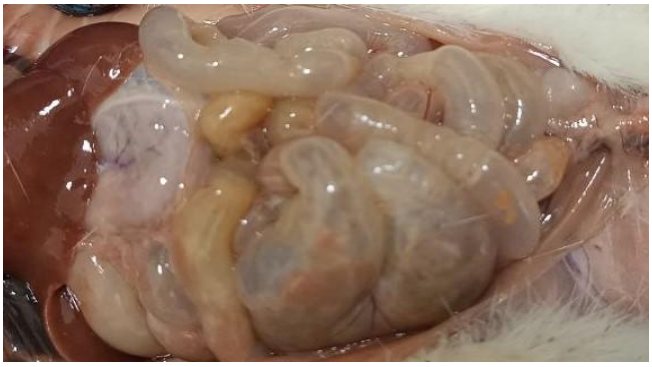

Figure 1. 70% acetic acid of the small intestine of a 3-way white-breed rat macroscopic appearance after exposure

A total of 110 rats were used in the experiments, of which 35 died during the experiments. Rats in all groups on the 1st, 7th and 30th day, animals were slaughtered in the morning, on an empty stomach, at appropriate times under general narcosis. After opening the abdominal cavity, a small intestine handle was taken to conduct a Morphological Study, and then its resection was cut with a microcracked. [3] After that, the length, width of the small intestine was measured using a millimeter gauge in the initial, middle and final parts Group I control group (n=30) for morphological and morphometric studies, 3-month-old rats (male and female) and 3-month-old (male and female) rats with an average weight of 211.6±8.05 G were selected. A morphological and morphometric examination of 12-toed and hungry intestinal tissue obtained after autopsy of 3-month-old white non-breeding bats in the control group found that it had a normal 4-layer structure. In 3-month-old white non-breeding rats, differences in the structure and age of the 12-finger and hungry intestinal floors were found:

Figure 2. a 3-month-old white broodless rat is a microscopic view of the duodenum, the norm. The paint was painted vandoon. AB 4x20 ok Intestinal vortices (1). Intestinal crypts (2). Inner circular floor sparse fibrous adder (3). mucous membrane (4)

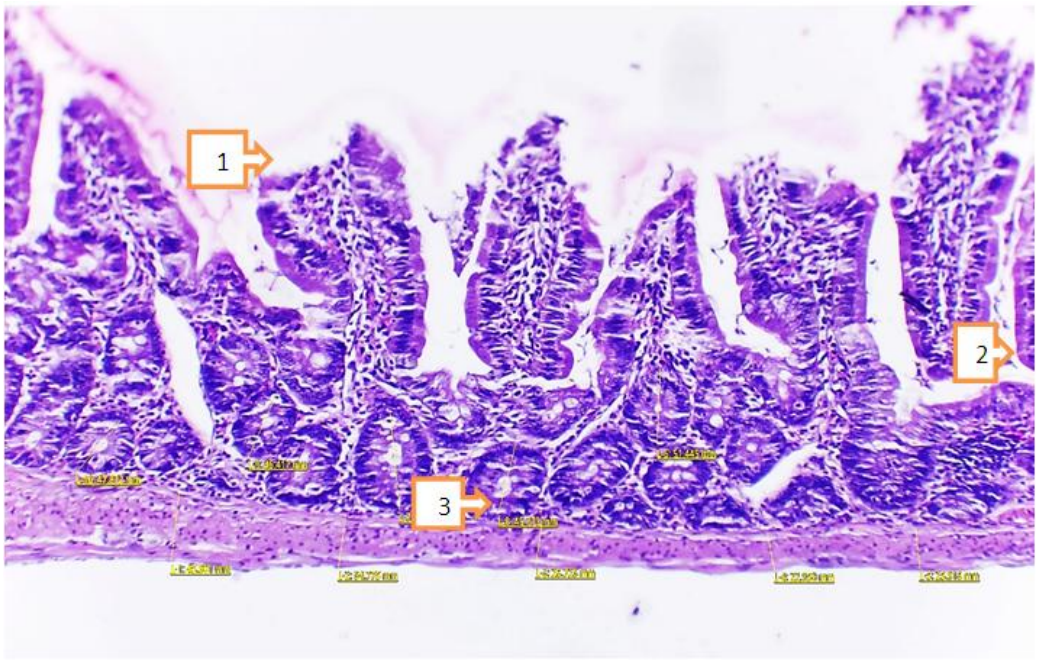

Figure 3. a microscopic view of a 1-month-old white zotless rat dead two-toed intestine, after a 6% burn with acetic acid. The dye is dyed in hemotoxilin-eosin. AB 4x20 ok. Deep-hearth atrophy of the intestinal vortices (1). The appearance of signs of inflammation in the intestinal crypts (2). On the inner circular floor, signs of inflammation and edema are detected (3)

3-month-old white-breed rats in the control group Complex relief as in the 12-finger and hungry intestinal mucosa has not yet been formed, the thickness of the 12-finger and hungry intestinal mucosa: in 3-month-old white-breed rats along the entire 12-finger intestine from 655.17 to 688.79 µm, on average from 677.97±17.82 µm, and in 650.03±54.34 µm. 3-month-old white broodless rats were found to contain 12-finger and light intestinal mucosa: epithelial structures, a private plate, and a muscle plate. By epithelial structure, of course, suckers (vorsinka) and crypts on the mucous membrane of the 12-finger and hungry intestine are lowered. 3-month-old white broodless rats in the control Guru were found to differ between suckers and vorsinks in the 12-finger and hungry intestinal mucosa. Suckers located in the 12th-toed intestine were found to be lower and wider in height than the ratio, and in 3-month-old white non-breeding bats, suckers were found to increase in height from 345.79 to 352.92 µm, to 348.37 ±3.58 µm. Their shape was found in 3-month-old rats to be mostly leafy, covered with a single-layer cylindrical epithelium and containing a small amount of goblet cells. The suckers located in the hungry intestine are longer in height, and in 3-month-old white non-breeding bats, the suckers have been found to range in height from 325.58 to 375.84 µm, to 348.8±24.12 µm. It has also been found in two young guruhis that the shape is mainly finger-shaped, narrower than that of the 12-finger intestine and differs from the 12-finger intestine by the large number of goblet cells. Group II 70% acetic acid sent 3-month-old white brood rats In the results from white non-breeding rats, in which chemical burns were called and not biocorrected under experimental conditions, it became known that Group II was found to have undergone morphological changes in the small intestine in all laboratory animals sent with 70% acetic acid (100.0%, n=30) (Figure 3).

In 3-month-old rats, the thickness of the 12-finger intestinal mucosa averaged 400.8±9.3 µm, while in the hungry intestine this indicator was 350.4±7.8 µm. But in 3-month-old rats, necrosis reached mainly to the muscle plate, although in the muscle and serous floors, strong collagenization and scarring processes were noted. Stationary tissue changes were formed in areas with Necrosis. Crypts: crypts were also observed to have nearly died as a result of the action of 70% acetic cislata, and epitheliocytes had been denucleated, and significant changes were also observed in the depths of crypts. In 3-month-old rats, this indicator was 150.7±9.1 µm in the 12-finger intestine and 130.8±8.3 µm in the hungry intestine. Private plate: due to Necrotic processes, the thickness of the private plate has also seriously decreased. In 3-month-old rats, however, 93.5±4.7 µm was found in the 12-finger intestine and 90.3±4.2 µm in the hungry intestine. Muscle plate: in 3-month-old rats, this indicator was 170.2±2.1 µm in the 12-finger intestine and 140.9±2.4 µm in the hungry intestine (slightly increased due to inflammation and swelling). [4]

Group III-3-month-old white zotless rats that were correted with sedana oil after administration of 70% acetic acid-in experimental conditions, chemical burns were called and results from biocorrected White zotless rats revealed that Group III was correted with sedana oil for thirty days after administration of 70% acetic acid-in all laboratory animals (100.0%, n=30) found to have undergone morphological changes in the small intestine. In 3-month-old rats, suckers in the 12-toed intestine were 135.7±9.4 µm in height and 123.6±8.1 µm in the hungry intestine. The crypt depth was 147.4±9.6 µm in the 12-finger intestine and 130.7±8.3 µm in

the hungry intestine. Although the recovery process was in a slow phase, there were significant signs of renewal. Private plate: In 3-month-old rats, the thickness of the private plate in the 12-finger intestine was 113.5±8.7 µm, in the hungry intestine-97.3±7.5 µm. Necrotic changes were limited to the muscular floor, and the serous floor retained swelling and inflammation. Muscle plate: In 3-month-old rats, muscle plate thickness was 167.6±6.2 µm in the 12-finger intestine and 155.4±5.7 µm in the hungry intestine. The muscle floor is in the recovery phase, and the serous floor retains swelling and inflammation.

4. Conclusions

Conclusion the following morphological changes in the small intestine in chemical burns of the experimental gastrointestinal tract were found, including furnace atrophy of the intestinal vortices, dystrophic and necrobiotic changes of the crypts and versine, reduced musculature, the appearance of signs of inflammation in the intestinal crypts, signs of inflammation in the inner circular floor as well as swelling, signs of infiltration Their detection indicates dystrophic changes in the small intestine, an inflammatory process.

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