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The Use of a Modern Algorithm for Prevention of Late Complications in Acute Poisoning with Acetic Acid

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ABSTRACT Acetic acid has a local cauterizing effect like coagulation necrosis and significant resorptive hemato-, nephro- and hepatotoxic effect due to hemolysis of erythrocytes, development of toxic coagulopathy, syndrome of disseminated intravascular coagulation. Developing severe hypoxia, microcirculation disorder, and impaired liver and kidney function significantly worsen proliferative processes in the area of chemical burn, leading to the development of such serious complications as late esophageal and gastric bleeding and cicatrical stenosis of the esophagus and stomach. Therefore, the prevention of these complications should include not only local treatment of the burn surface, but also complex therapy aimed at restoring the function of affected organs at the early hospital stage and rehabilitation stage. The article describes the case of practical application of an intensive care algorithm, which includes the use of microcirculation improvers, Cytoflavin — an antihypoxant on the basis of succinic acid, Actovegin — a stimulant of proliferation, prolonged use of glucocorticoids, active nutritional support with a protein-carbohydrate mixture, all initiated upon arrival.

Keywords: acetic acid, poisoning, hemolysis, chemical burn, therapy algorithm, antihypoxants, glucocorticoids, nutritional support

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During the clinical course of the disease, in severe acute poisoning with acetic acid, complications develop which are divided into early (1-2 days) and late ones (starting from the 3rd day). Early complications include mechanical asphyxia, early primary and secondary bleeding, acute renal and hepatic failure, acute reactive pancreatitis, and nephropathy with primary oliguria or anuria. Late complications are cicatricial deformities of the esophagus and stomach. The formation of the cicatrix of the esophagus and stomach occurs on average within day 30-90 after poisoning [1-3]. Also one of the frequent and terrible complications of acute poisoning with acetic acid of a severe degree is later esophageal-gastric bleeding, which develops on day 11–20 due to discharge of necrotic masses of the burn surface of the esophagus and stomach with its insufficient proliferation [1, 3-5].

Prevention of the development of late complications should be aimed at exclusion of all factors which reduce the activity of proliferative processes:

1. The management of tissue hypoxia on day 1–7 after poisoning. Among the drugs which relieve hypoxia, substrate antihypoxants based on succinic acid have become very popular [6, 7].

2. Since there is a dramatic increase in vascular permeability in acetic acid poisoning during hemolysis, endothelium protection is necessary.

3. Active enteral and parenteral nutrition in order to correct protein-energy deficiency [8].

4. The use of drugs stimulating proliferative processes [8].

5. The use of glucocorticosteroids to prevent fibrillogenesis. The use of hormones helps reduce tissue edema, relieves the inflammatory response, normalizes the neurohumoral processes of the stress associated with the onset of the post-burn period, has a positive effect on kidney function, metabolism and prevents the formation of rough scars [2, 7, 8].

6. After reducing the activity of the inflammatory process, it is necessary to use drugs improving the resorption of scar tissue and the prevention of coarse scars formation [1, 2, 4]. Considering the long period of development of proliferative processes, adequate provision of emergency care at all stages, i.e. at the prehospital and hospital stages, as well as during the rehabilitation period [4, 6].

To solve the above problems in the Scientific and Clinical Department of Toxicology at RSCEMC, we developed a complex of intensive care, including:

I. The hospital stage (day 1–28–30):

1. Introduction of drugs which improve microcirculation (derivatives of hydroxyethyl starch, Reosorbilact) within 3-5 days from the date of admission.

2. Introduction of the L-lysine Aescinate endothelioprotector up to 10.0 ml 2 times a day during the entire period of hemolysis.

3. High doses of glucocorticosteroids: 6–8 mg/kg of Prednisolone per day for 21–28 days.

4. Intravenous fluid drip of Cytoflavin up to 20.0 ml/day diluted in 5% glucose solution, Actovegin up to 10.0 ml 2 times a day for 15–20 days.

5. Nutritional support by infusion of amino acids (Infesol, Aminol, Gelofusine) every other day for 10–14 days. A balanced protein-carbohydrate mixture ("Atlant") with an energy value of 450 kcal/100 g at the rate of 1.5 g per 1 kg of body weight 3 times a day for 20–27 days is required.

II. Rehabilitation (day 30–90):

1. Glucocorticosteroids: not less than 1 mg/kg of Prednisolone twice a day for 14 days, then 0.5 mg/kg for 10–14 days intramuscularly, then 10 mg of oral Prednisolone in the morning for 1 month.

2. Actovegin 5.0 ml/day intravenous for 1 month.

3. Lidase, 64 units 2 times a day intramuclurarly for 14 days, the cycle repeats in dynamics [9].

We present the description of a clinical case of treatment according to the developed algorythm of a female patient who entered our department.

The clinical case

A 22-year-old female patient S. entered the toxicological department of RSCEMC a day after taking acetic acid.

Complaints on admission of a sharp pain in the throat, along the esophagus, epigastria, shortness of breath at rest, nausea, vomiting, hypersalivation, hoarseness.

Anamnesis of the disease: the patient drank about 50 ml of 96% (glacial) acetic acid for suicidal purposes, 30 minutes before going to the regional hospital. The relatives took the patient to the RSCEMC sub-branch at the Yangiyul city hospital, where after gastric lavage with cold water through a probe she was hospitalized to the intensive care unit. At the same time, the patient showed signs of severe poisoning with acetic acid: burning unbearable pain in the region of the oropharynx, along the esophagus, in the epigastrium, vomiting of red blood and "coffee grounds", the appearance of red urine. There was shortness of breath at rest — the respiratory rate was 24–28 per minute, hemodynamic disturbance — BP 75/40 mm Hg, pulse — 118 beats per min of of low tension, central venous pressure — negative.

The content of free hemoglobin in the blood and urine was not determined when the patient was admitted to the hospital due to the absence of reagents in the sub-branch. Only the clinical blood test was performed (hemoglobin 134 g/l, erythrocytes 4.5 million/mm^3, leukocytes 11.2 thousand/mm^3, erythrocyte sedimentation rate (ESR) 18 mm/h, blood clotting time 3 min 10 s – 3 min 40 s) and urine, which showed the presence of gross hematuria with a large number of modified red blood cells.

A toxicologist from RSCEMC was invited for consultation, and recommended to initiate infusion therapy in the amount of 7,800 ml per day, including colloid (Reosorbilon, Volustim) and crystalloid preparations for blood alkalization (4% sodium bicarbonate), forced diuresis, intravenous drip of complex metabolic drug on the basis succinic acid Cytoflavin 20 ml 2 times a day on 5% glucose. Considering the signs of hemolysis, in order to improve microcirculation and prevent the development of early secondary bleeding, the patient received Heparin therapy 5,000 units IV daily [1], Prednisolone 360 mg/day IV, local therapy. After stabilization of the condition, it was recommended to transfer the patient to the specialized toxicology department of the RSCEMC. A day later, the patient was transferred to the Republican Scientific Center of Emergency Medical Care and hospitalized in the Toxicological Intensive Care Unit.

Upon admission the patient's general condition was severe, the skin was clean. She was breathing independently, respiratory rate was 24 beats/min. The pulse was 100 beats/min, rhythmic, intense, of satisfactory tension. BP 110/70 mm Hg.

From the time of admission laboratory and instrumental examination was carried out, which revealed:

1. Minor hemolysis: free hemoglobin in the blood 2.4 g/l, in the urine 5.7 g/l.

2. Moderate anemia and active inflammatory reaction: hemoglobin 106 g/l, erythrocytes 3.3 million/mm^3, leukocytes 16.5 x 10^9 /l, ESR 22 mm/h.

3. After managing the hemolysis there were signs of severe endotoxemia, toxic hepatitis, hemoglobinuria nephrosis: medium molecular peptides (MMPs) in blood 0.880 absorbance units, total protein 46 g/l, albumin 21.4 g/l, urea 12.6 mmol/l, creatinine 0.22 mmol/l (a norm of 0.06–0.11 SI units), aspartate aminotransferase (AST) activity 436.5 u/l and alanine aminotransferase (ALT) 380.2 u/l. The indicators of coagulogram showed signs of moderate hypercoagulation — the prothrombin index 94%, fibrinogen in the blood — 4.2 g/l, plasma recalcification time — 62 s. An ultrasound examination revealed diffuse induration of the liver, enlarged kidneys and thickened renal parenchyma.

4. Bacterial culture from the wound showed the presence of moderate growth of Staphylococcus aureus.

5. According to chest X-ray, bilateral focal pneumonia was diagnosed.

6. ECG: significant dystrophic changes in the myocardium.

7. Esophagogastroduodenoscopy: the mucous membrane of the esophagus, beginning at the mouth, is covered with dirty fibrin, which is easily removed when in contact with the device and diffusely bleeding. Erosive and ulcerative lesions with a purple-bluish tinge and areas of necrotic changes on the mucous membranes are noted throughout the esophageal mucosa. Peristalsis of the esophagus is not visible. The gastric mucosa is edematous, along with the entire mucous membrane erosive and ulcerative lesions with a purplish-bluish tinge and areas of necrotic changes on the mucosa are also revealed.

Conclusion: a chemical burn of the oropharynx, esophagus and stomach of the III degree (according to the classification by V.I. Volotskov, 1988). Laryngoscopy: significant hyperemia and edema of the laryngeal mucosa. The tip of the epiglottis is covered with fibrin. The vocal cords are movable, not fully closed due to edema of the mucous membrane.

According to the claimed algorithm we performed:

1. Intravenous drip administration of Cytoflavin antihypoxant in a dose of 20.0 ml, diluted in 400 ml of 10% glucose solution 2 times a day, starting 1 hour after administration, for 5 days.

2. Introduction of L-lysine Aescinate endothelioprotector intravenously diluted in 200.0 ml of a physiological solution, 10.0 ml 2 times a day for the entire period of hemolysis, 3 days.

3. Intravenous administration of Actovegin proliferation stimulator 10.0 ml 2 times a day, starting from the 2nd day, for 5 days.

4. Introduction of Prednisolone according to the scheme: 120 mg 2 times a day for 3 days, then 60 mg 2 times a day for 10 days, then 30 mg 2 times a day for 10 days, then 30 mg per day (in the morning) for 14 days, then 2 tablets (10 mg) in the morning for 14 days.

5. Introduction of Lidase, 64 units intramuscularly 2 times a day, starting from the 28th day for 14 days. Also, the patient underwent a set of measures in the intensive care unit, corresponding to this pathology, which included infusion therapy with blood alkalization (4% sodium bicarbonate solution), H_2-blockers, given the low level of total protein and albumin — albumin and plasma transfusion, antispasmodics, local therapy (Sea Buckthorn Oil, Almagel), nebulizer therapy (Hydrocortisone, Euphyllin, Dimedrol), antibiotics according to sensitivity (Ceftriaxone and Levofloxacin), and Heparin 5,000 U/day for 3 days in order to improve microcirculation and prevent secondary bleeding [1, 3].

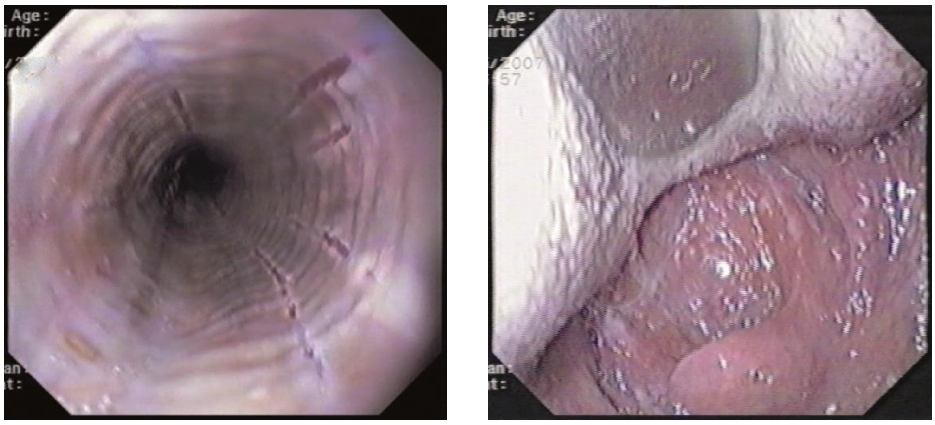

Fig. 1. The endoscopic view of the third-degree pharyngeal and esophageal burn of a patient S., day 2 from the moment of poisoning

As a result of intensive therapy, the patient's condition stabilized, there were positive changes: elimination of dyspnea, recovery of swallowing, appetite on day 5-6, relief of hemolysis just a day after hospitalization, normalization of clinical and biochemical analyzes by day 9-10 (hemoglobin 112 g/l, erythrocytes 3.4 million/mm^3, leukocytes 7.1 x 10^9 /l, ESR 9 mm/h, MMPs 0.230 absorbance units, urea 4.9 mmol/l, creatinine 0.07 mmol/l, AST 76.4 u/l and ALT 52.5 u/l). The patient was discharged on day 25 after poisoning in a satisfactory condition.

The control examination after discharge was performed 1 month after poisoning. According to the clinical and laboratory examination, there were no pathological processes. There were no complaints, no dysphagia, food passed the esophagus freely, including solid pieces.

Esophagogastroduodenoscopy: the esophagus was freely passable, the mucous membrane of the esophagus and stomach was completely covered with areas of granulation tissue, isolated erosions on the mucous membrane of the esophagus of the stomach, no edema, in the lower third of the esophagus there were small areas of fibrin plaque, there were no signs of the Schatzki's ring constriction.

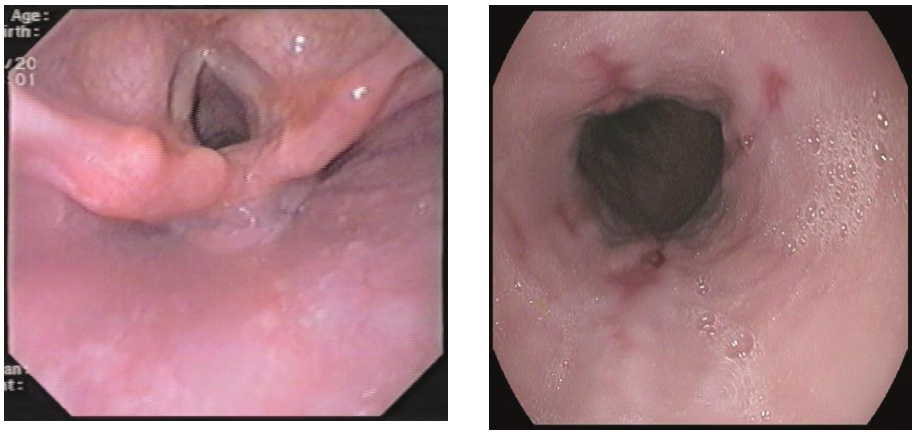

Fig. 2. The endoscopic view of the pharyngeal and esophageal burn of a patient M., day 34 from the moment of poisoning

Control examination after discharge, 3 months after poisoning: no complaints, no dysphagia, food passed freely, including solid pieces.

Esophagogastroduodenoscopy: freely passable esophagus, pale pink mucous membrane of the esophagus and stomach, no fibrin plaque and no signs of the Schatzki's ring constriction..

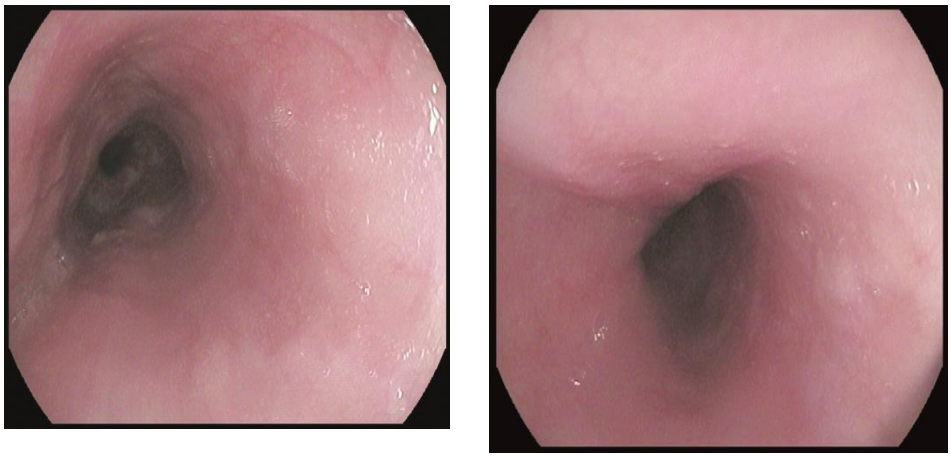

Fig. 3. The endoscopic view of the esophageal burn of a patient M., day 92 from the moment of poisoning

CONCLUSION

The introduction of an improved algorithm for staged emergency care in the complex treatment of severe acute poisoning with acetic acid is an effective measure for the prevention of late complications such as esophageal-gastric bleeding, and cicatricial deformity of the esophagus and stomach in this category of patients.

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