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QUINCKE'S EDEMA. UNUSUAL COURSE OF THE DISEASE

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Annotation: The article describes the clinical observation of an atypical course of angioedema caused by a delayed and prolonged course of the pathological process with fulminant end, unclear underlying cause of the allergic reaction, ineffective pathogenetic treatment and unfavorable outcome. The delay in hospitalization, as well as the shortcomings of the intensive care, the overload of the infused fluid, in particular, were the factors that predetermined the fatal development of the pathological process.

Key words: allergy, angioedema, diagnosis, infusion therapy.

Angioedema (AO) or Quincke's edema was described by the German physician Heinrich Quincke in 1882 [1-3]. There are two types of this syndrome - allergic and pseudoallergic. They differ in the mechanism of development and the causes that cause a dangerous condition. Allergic edema occurs in half of patients with urticaria and is a manifestation of an immediate allergic reaction. Suddenly, limited or diffuse edema of subcutaneous fat and mucous membranes develops. Most often, this is the result of taking medications such as beta-lactam antibiotics, cephalosporins, aspirin and other nonsteroidal anti-inflammatory drugs, sulfonamides, B vitamins, ACE inhibitors, muscle relaxants, X-ray herbal preparations, dextran, thiazide diuretics, streptokinase, as well as the use of a number of foods - chicken eggs, whole milk, fish, alcoholic beverages. Quincke's edema is exposed-wives are adults and children, but mostly the disease occurs at a young age. Allergic edema is based on an allergic antigen-antibody reaction. Biologically active mediator substances released in a pre-sensitized organism (histamine, kinins, prostaglandins, cytokines) cause local expansion of capillaries and veins, an increase in microvascular permeability occurs and tissue edema develops. Non-allergic or pseudoallergic Quincke edema is less common with a frequency of 1:10-15 thousand in the general population. Its development is related to heredity. With Quincke's edema of pseudoallergic genesis, the main changes are reduced to a genetically determined violation of the activation of the complement system, while genetically determined AO (autosomal dominant) is distinguished. In this case, there is a deficiency of the C-inhibitor (type I), functional insufficiency of the C1 inhibitor (autosomal dominant inheritance is characteristic of type II), linked to X-chromosome and observed only in women (type III). The cause of pseudoallergic Quincke's edema may be minor physical, thermal or chemical effects. The most threatening complication of the syndrome is laryngeal edema with increasing symptoms of acute respiratory failure. Symptoms of laryngeal edema are hoarseness of voice, barking cough, progressive difficulty breathing up to asphyxia. The complex of urgent measures in these cases, in addition to pathogenetic therapy, requires readiness for resuscitation, which can be optimally carried out in the conditions of the intensive care unit, where the patient should be hospitalized . Deviation from the existing standard of emergency care for angioedema is fraught with the development of undesirable, even catastrophic consequences. An example of this is the following description of a clinical case. The SMP team

arrived on call to patient K., born in 1983, on 10.08. at 19:05. The patient complained of increasing pain of a pressing nature "all over his head", general weakness, malaise. I got sick yesterday. This condition is associated with alcohol consumption. I turned to a narcologist for medical help, who prescribed treatment. Objectively: adequate, space- and time-oriented. The skin is physiological, clean, dry. Blood pressure is 120/80 mmHg. The pulse is rhythmic - 71 beats / min, satisfactory filling and tension. The heart tones are clear, rhythmic. Nasal breathing, free, vesicular, no wheezing. SaO2 - 98%. There are no focal neurological symptoms. Muscle strength is preserved, d=s. The belly is soft, painless. Stool and diuresis without features. No visible signs of injury or damage were found.

Allergological history is calm. At 21:20 on 10.08. the patient independently applied to the emergency department of the CRH with complaints of difficulty breathing from 20:00. The condition is satisfactory. Blood pressure - 120/80 mmHg. Pulse - 80 beats/ min. BH - 18 per minute. Breathing is free. The mucous membrane of the pharynx is slightly swollen, without hyperemia. D-Z: Laryngeal stenosis. 60 mg of prednisone I / V and 2 ml of suprastin were administered. A record has been made of the patient's refusal to be hospitalized, there is no signature of the patient in the refusal. Accompanied by his father, he was allowed to go home. At 0:15 on 11.08. the patient independently applied to the emergency department of the CRH with complaints of difficulty breathing. Deterioration from 23:30, blood pressure -120/90 mmHg, pulse - 86 beats /min, BH - up to 20 per minute. Breathing is free. Local status "The mucous membrane of the pharynx is somewhat swollen. The swelling is mild, without hyperemia. The airway is narrowed to 2.5 cm." An infusion of phys. a solution of 400.0 ml + 8 mg dexamethasone, diphenhydramine and lasix IV were introduced. The time of observation and the results of treatment are not specified. The patient was released home, hospitalization was not offered. Then on 11.08., at 04:46, the NSR was called. Complaints about a feeling of lack of air at rest, inability to swallow independently, general weakness, malaise. I got sick at about 21:00 on 10.08. I went to the CRH 2 times, after being examined by the doctor on duty, treatment was prescribed, after assistance there was improvement. Nausea, vomiting is not noted. This condition is unrelated to anything. Before the arrival of the SMP, he did not take any medications. Since 04:00, a sharp deterioration has been noted, the SMP brigade has been called, delivered to the CRH. Objectively: adequate, space- and time-oriented. The skin is clean, dry, and there is no rash. Blood pressure is 140-120 / 80 mmHg. Pulse is rhythmic 78 beats / min, satisfactory filling and tension. SaO2 - 98%. The heart tones are clear, rhythmic. Nasal breathing, free, vesicular, no wheezing. BH - 22 per minute. Lymph nodes are not palpable. There is bright hyperemia in the pharynx, the mucous membrane of the pharynx is edematous, the tonsils are enlarged, edematous, there are no plaque. Mixed shortness of breath. There are no focal neurological symptoms. The belly is soft, painless. Stool and diuresis without features. No visible signs of injury or damage were found. The diagnosis of the ambulance crew: An allergic reaction according to the type of Quincke's edema of unclear etiology. Therapeutic measures: 04:50. A peripheral catheter was inserted into the right ulnar vein, 04:52 intravenously injected: prednisone - 90 mg + phys. solution -10.0 ml; 04:56 - phys. solution - 200.0 ml, prednisone - 60 mg. At 5: 00, he was hospitalized in the GBUZ PK CRH. From the record of the doctor of the emergency department: "The general condition is closer to satisfactory. The voice is practically unchanged - slight muffling. Blood pressure -120 /90 mmHg. pulse - 84 beats/min. In the lungs, respiration is vesicular, harsh in the interscapular areas, wheezing and shortness of breath were not detected. The soft tissues of the neck are slightly swollen, the larynx is not changed, the mucous membrane of the pharynx is swollen. The swelling is mild. The swelling is mild. The airway clearance is 1.5 cm". Diagnosis: allergic reaction according to the type of Quincke's edema. He was hospitalized in a day hospital bed. An infusion of 16 mg dexamethasone + 1000.0 phys. solution. At 5:50 a.m., an I-graph of the chest organs was performed. I went up to the 3rd floor from the office on my own. He lost consciousness at 6:15 a.m. and was diagnosed with clinical death. Intensive care measures have been initiated: NMS, ventilator with an Ambu bag, 1.0 mg of adrenaline + 20.0 ml of phys. solution, an intensive care specialist has been called. At 6:30 - failures- the first attempt at tracheal intubation. From the description of the resuscitator, "Pronounced swelling of the larynx, vocal folds. The entrance to the esophagus is not visualized." NMS was continued, ventilation with an Ambu bag, 1.0 mg of adrenaline + 20.0 ml of phys. was injected. Finally, a tracheostomy was performed at 6:33 a.m. Cannula No. 8 with a cuff is inserted. Breathing is performed on both sides. CPR was continued at 6:40 a.m., adrenaline was administered 3 times. Pulmonary-cardiac resuscitation for 30 minutes without effect. Death was pronounced. The final clinical

diagnosis is Quincke's edema.

Forensic medical diagnosis: Basic. An allergic reaction to an unknown substance. Laryngeal edema: the entrance to the larynx and esophagus is narrowed to a point, sharp edema of the epiglottis, laryngeal walls and vocal cords, edema of the subepithelial layers of the larynx, plasmorrhagia and lymphostasis at the level of the submucosal layer. Edema of the lungs and brain. The accompanying one. Fatty hepatosis. Cardiomyopathy.

Conclusion: The death of Mr. K. occurred as a result of an allergic reaction to an unknown substance, accompanied by severe swelling of the larynx, namely the epiglottis, laryngeal walls and vocal cords, with acute respiratory failure, as evidenced by the pathohistomorphological signs of rapid death found during the examination of the corpse. Comment. Indeed, the death of patient K. occurred from asphyxia as a result of an allergic reaction corresponding to Quincke's edema or angio-neurotic edema. It is also impossible to exclude that this could be a pseudoallergic variant of Quincke's edema. The conditions of the medical institution excluded the possibility of differential diagnosis. But in any case, we can talk about the atypical course of the syndrome, which is due to the delayed and prolonged course of the pathological process with the development of a lightning-fast ending, an unclear root cause of edema, the lack of effect from the pathogenetic treatment and an unfavorable outcome, since the prognosis for Quincke's edema, according to the literature, is more often favorable provided timely and adequate emergency care. Swelling of the larynx without proper help is usually fatal. Treatment of patients with Quincke's edema in the acute period should be comprehensive, it is aimed at eliminating an allergic reaction, reducing edema, reducing the sensitivity of the body to histamine. Urgent treatment requires laryngeal edema, in which it is necessary to immediately subcutaneously inject 0.1% adrenaline solution at an age-appropriate dose (0.3-0.5-0.8 ml); intravenously or intramuscularly, one of the antihistamines (diprazine, diphedrol, suprastin, etc.), hydrocortisone hemisuccinate (75-125 mg) or prednisone hexuccinate (30-60 mg). A patient with laryngeal edema needs urgent hospitalization in the intensive care unit or intensive care unit. The rate of development of respiratory failure can be catastrophically fast, and the degree of severity of the narrowing of the entrance to the trachea is so strong that even intubation with direct laryngoscopy is difficult or even impossible. In these cases, life-saving manipulation (on-site surgery) is tracheostomy (conicotomy). Most likely, these are medications that were administered by the SMP team as prescribed by a narcologist. They included injections of vitamins B1, B6, thiosulfate Na, etc. - drugs, potential allergens. Thus, the instructions for the use of vitamin B1 indicate that sometimes injections of B1 cause sweating and tachycardia, they can also contribute to an allergic reaction. The symptoms of allergy to B1 are not specific. Some people have hives and rashes, while others have more severe symptoms up to Quincke's edema. In addition, vitamin B1 is pharmacologically poorly compatible with vitamin B6, which can also contribute to the development of anaphylaxis. So, most likely, angioedema in patient K. it was the result of the administration of drugs, the side effect of which may be allergic reactions. It is quite difficult to predict the likelihood of such complications, especially with a calm allergic history. The team serving the call correctly diagnosed the complication. The peripheral vein was catheterized for IV infusion, prednisone was administered 2 times with phys. with a solution, and the patient was taken to a medical institution. If breathing was impaired, adrenaline and antihistamines could be used. But in this case it is not essential, because the patient was taken to the hospital in a compensated condition.

In the hospital, there was an underestimation of the severity of the condition in relation to the prediction of possible complications. In all cases of clinical manifestations of Quincke's edema, emergency hospitalization of the patient to the intensive care unit or intensive care unit is indicated. In this episode, the patient with breathing difficulties sought medical help 3 times and was admitted to the therapeutic department only on the 3rd time. Intensive monitoring of the patient was not provided, which led to a catastrophic development of events and the unavailability of staff to provide urgent and effective care. Special attention should be paid to the fact that within a relatively short time (from 19 h to 5:45) 2500 ml of liquid was injected: 2000 ml - phys. solution and 500 ml of 5% glucose, with the last portion of 1000 ml of phys. The solution was administered between 5:00-5:45 and, moreover, it was insufficiently justified, since hemodynamic parameters were stable and decongestant therapy was shown to the patient. It can be assumed with a high degree of probability that the administration of such an amount of fluid for a short time against the background of local edema, such as Quincke's edema, could contribute to the catastrophic progression of laryngeal edema. In the hospital, only dexamethasone is prescribed as a decongestant, which is not a first-

line drug. Adrenaline, antihistamines, diuretics, and oxygen therapy were shown. With the development of clinical death, time was lost. Ventilation with an Ambu bag with such a stenosis turned out to be ineffective, and intubation was impossible. The tracheostomy was applied 18 minutes after the start of intensive care, i.e. late. By this time, the cerebral cortex had naturally died and resuscitation measures, which were also not carried out in full (there was no monitoring, defibrillation was not performed) turned out to be ineffective. Thus, untimely and non-core hospitalization, lack of intensive care-research, incomplete and sometimes inadequate volume of therapeutic measures, and unavailability to carry out resuscitation aids have become defects, which contributed to the unfavorable outcome. Analyzing this clinical situation in terms of intensive care and bearing in mind the interpretation of the fatal role of infusion of phys. of the solution, especially the last portion, it can be concluded that in this case the main postulate of medicine "do no harm" was violated, which we often declare, but far from always extrapolate to our activities. The role of infusion therapy in the treatment of critical patients cannot be overestimated, but at the same time it should be remembered that this is a potentially dangerous treatment method associated with the possibility of severe complications, requiring the use of modern technologies, adequate monitoring, a differentiated approach and a clear understanding of the goals and objectives of this type of therapy.

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