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Department of Infectious diseases

METHODOLOGICAL GUIDE

FOOD TOXICINFECTION

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Food toxicoinfections (PTI) are acute, self - limiting diseases caused by opportunistic bacteria that can produce exotoxins outside the human body-in food products and occur with symptoms of the upper gastrointestinal tract (gastritis, gastroenteritis) and disorders of water and salt metabolism.

Microbial food poisoning is divided into toxicoinfections and toxicoses (intoxication). The latter include diseases caused by CL.lbotulinum and enterotoxigenic strains of St. aureus. Due to the pronounced
difference in the mechanism of action of the toxin (neuroplegic effect) released by CL.botulinum, and the originality of the clinical picture, it is described separately.

Etiology. Pathogens of food toxicoinfections are various conditionally pathogenic bacteria (UPB), individual strains of which are able to produce exotoxins outside the human body - on food products. Among the exotoxins formed by UPB are enterotoxins (thermolabile and thermostable), which increase the secretion of fluid and salts into the lumen of the stomach and intestines, and cytotoxin, which damages the membranes of epithelial cells and disrupts protein-synthesis processes in them.

The most common pathogens that can produce enterotoxins are Clostridium perfringens, Proteus yulgaris, Proteus mirabilis, and Bacillus cereus. Enterotoxins are also formed by pathogens belonging to the genera: Klebsiella, Enterobacter, Citrobacter, Serratia, Pseudomonas, Aeromonas, Edwardsiella, Vibrio. Most of the enterotoxins are heat-labile pathogens.

St. aureus enterotoxin has pronounced thermostable properties. It is not inactivated when boiled for up to 30 minutes. And it retains the ability to cause a clinical picture of the disease in the absence of microbes themselves.

Klebsiella pneumoniae, Enterobacter cloacae, Aeromonas hydrophila, Clostridium perfringens type C and Clostridium difficile, Vibrio parahaemolyticus, St. aureus and a number of other microbes have the ability to produce cytotoxin.

Epidemiology. Sources of food toxicoinfections can be people working in the food industry and suffering from various pustular skin infections, angina, upper respiratory tract diseases, pneumonia, etc.

Among the zoonotic sources of PTI can be ball mastitis animals-cows, goats, etc.

However, the wide spread of UPB in the external environment often allows us to identify the source of the disease.

The path of distribution of PTI is alimentary. Among the transmission factors are solid (sausages, jellies, eggs, canned meat, fish, etc.) and liquid (soup, milk, juices, compotes, jelly, kvass, lemonade, beer, cocktails, etc.) food products that are a breeding ground for bacteria. Staphylococcal intoxication is more often associated with eating infected confectionery creams, meat, fish and vegetable dishes, and dairy products. Proteus and clostridia reproduce well on protein products (meat, fish, including canned ones, sausages, milk). BAC.cereus is very unpretentious, it multiplies rapidly in various food products: vegetable salads and soups, puddings, meat and fish dishes.

Susceptibility to this group of diseases is high. It is not uncommon for 90 - 100% of people who consume an infected product to become ill. Characteristic of PTI is not only a group, but also an explosive (explosive) nature of the incidence, in which in a short time (in a few hours) all participants of the outbreak fall ill.

The incidence of PTI is registered throughout the year, but more often - in the warmer months.

Pathogenesis. Penetration into the stomach along with food not only of the UPB itself, but also a large number of exotoxins formed by them, causes the development of the shortest incubation period in infectious pathology.

Clinical and pathogenetic features of PTI largely depend on the type and dose of exotoxins, as well as other toxic substances of microbial origin that contaminate the food product.

Enterotoxins (thermolabile and thermostable), binding to epithelial cells of the stomach and intestines, affect the enzymatic systems of epithelial cells, without causing morphological changes in these organs. Among the enzymes activated by enterotoxins are adenylate cyclase and guanylate cyclase, which increase the formation of biologically active substances in the cells of the mucous membrane - camp and cahmp. Under the influence of toxins increases the rate of formation of prostaglandins, his-

tamine, intestinal hormones, etc. All this leads to an increase in the secretion of fluid and salts into the lumen of the stomach and intestines and the development of vomiting and diarrhea.

Cytotoxin damages the membranes of epithelial cells and disrupts protein-synthesis processes in them. This can increase the permeability of the intestinal wall to various types of substances of microbial origin, and in some cases, the microbes themselves. All this leads to the development of intoxication, impaired microcirculation and local inflammatory changes in the intestinal mucosa.

Thus, the clinical manifestations of PTI caused by pathogens capable of producing mainly only enterotoxins are less severe, the diseases in most cases occur without hyperthermia and any significant inflammatory changes in the gastric and intestinal mucosa. The same cases when there is an accumulation of both enterotoxins and cytotoxin in food products are incomparably more severe, with short - term, but high hyperthermia, inflammatory changes in the gastrointestinal mucosa.

The short-term nature of the course of PTI is associated with a short stay of pathogens in the human body, but the Action of toxins that bind to epithelial cells of the stomach and intestines stops after desquamation of these cells. Unbound toxin molecules are inactivated by proteases.

Only under certain conditions, when previous diseases have violated the systems of antibacterial protection of the small intestine, pathogens can stay in the intestine for a longer time. In some cases, as is the case, for example, in patients with malnutrition, after gastrectomy, with blind loop syndrome, colonization of the small intestine Cl. perfingens type C leads to severe necrotic enteritis.

The pathoanatomic picture of PTI has been little studied. In rare cases of death, there is edema, hyperemia of the stomach and small intestine mucosa, sometimes desquamation of the epithelium. In other organs, dystrophic changes of various degrees of severity are detected, developing as a result of intoxication and violations of hemodynamics.

Clinic. The duration of the incubation period in most cases is 2-6 hours (from 30 min. to 24 h.).

The onset of the disease is acute. Most often, the PTI will make their debut with the appearance of nausea and vomiting. A little later, there is diarrhea of the small-bowel type. Stool is liquid, watery from 1 to 15 times a day, and does not contain pathological blood and mucus impurities.

In some patients, PTI occurs without the development of diarrheal syndrome. In other cases, there may be no vomiting.

Depending on the pathogenetic features of the disease caused by the predominance of enterotoxins or cytotoxin (and bacterial lipopolysaccharides) in a contaminated food product, 2 types of PTI can be distinguished.

In the first case, vomiting and diarrhea are not accompanied by any strong pain syndrome, hyperthermia and inflammatory processes in the gastrointestinal tract. In the second case, there are cramping pains in the EPI-and mesogastrium, a short-term, within a few hours, increase in body temperature and inflammatory changes in the mucous membrane of the stomach and small intestine. 12-24 hours after the onset of the disease, pain symptoms and temperature reactions usually disappear.

Objectively, patients have pale skin, sometimes cyanosis of their extremities, cold. The tongue is covered with a white-gray coating. The abdomen on palpation is soft, painful in the epigastrium, less often around the navel. Naturally, we suffer from a cardiovascular system: bradycardia is determined (with hyperthermia-tachycardia), blood pressure is reduced, systolic noise is heard at the top of the heart, the heart tones are deaf. In some cases, fainting spells and short-term collaptoid States develop. With repeated vomiting and profuse diarrhea, symptoms of dehydration, demineralization and acidosis may appear. Possible cramps in the muscles of the extremities, a decrease in diuresis, a decrease in turgor of the skin, etc.with timely and adequate therapy, these phenomena are quickly stopped. The liver and spleen are not enlarged. In the hemogram, leukocytosis, neutrophilosis, a moderate increase in ESR.

The duration of the disease in most cases is 1-3 days.

Complications of PTI include dehydration shock and acute heart failure associated with impaired electrolyte (hypokalemia) metabolism. Other complications, including septic ones, are rare and are largely associated with the patient's premorbid condition.

Although the manifestations of the disease do not depend much on the type of pathogen, in some cases, you can find some etiologically determined originality of the clinical picture of the disease.

Thus, the range of clinical manifestations of PTI caused by Cl. perfringens is quite wide. Along with easily occurring diseases, in which the clinic is dominated by symptoms of gastritis or gastroenteritis, there are also severe forms of disease, accompanied by the development of necrotic enteritis and anaerobic sepsis.

In PTI caused by Proteus, the stool has a pungent fetid smell. In some patients, there is a short-term decrease in visual acuity.

Staphylococcal intoxication often occurs without diarrhea. The clinical picture is dominated by gastritis symptoms in the form of repeated vomiting, cramping pains in the epigastric region. There are signs of vascular dystonia. The body temperature in most patients is normal or subfebrile.

The forecast is usually favorable. Fatalities are rare and are caused by complications such as dehydration shock, acute heart failure, necrotic enteritis, and anaerobic sepsis.

Diagnostics. Clinical and epidemiological data are of the greatest importance in the diagnosis of PTI. Among them: 1) acute onset and dominance in the clinical picture of symptoms of gastritis or gastroenteritis; 2) absence of hyperthermia or its short-term character; 3) short incubation period and short duration of the disease itself; 4) group character of morbidity and its connection with the use of the same food; 5) explosive (explosive) the nature of the disease. In laboratory diagnostics, a bacteriological method is of great importance, including the study of the toxogenic properties of isolated pathogens. The material for the study is vomit, gastric lavage, stool of the patient, the remains of uneaten food, etc. In food toxicoinfection, the isolation of a particular microbe from a patient does not yet allow it to be considered a pathogen of the disease. It is necessary to prove its identity with the strains that were isolated from simultaneously ill patients, as well as with those obtained from the contaminated product.

General characteristics of the group of conditionally pathogenic bacteria, their typification. A certain role in the occurrence of food diseases of people can play some bacteria, combined as opportunistic pathogens. They include groups of Escherichia coli and Proteus, which are more often the culprits of food diseases. These bacteria are quite widespread in the external environment, found or constantly live in the intestines of animals and humans. Like the bacteria of the genus Salmonella, morphologically they are sticks with rounded ends or oval shape, 1-4 μm long and 0.5-0.6 μm wide. With the exception of some, are motile, Gram-stain negative, spores and capsules do not form, aerobic, grow well on ordinary nutrient media.

The name "Escherichia coli" is collective, since it includes a large number of varieties that differ from each other in cultural, biochemical, serological and pathogenic properties. According to Minkiewicz, this group includes the subgroups B. coficitrovorum, aerogenes and paracoli. The name "Escherichia" this group received in honor of the German scientist Escherichia, who was among the first in 1885 to isolate Escherichia coli. Bacteria of the Escherichia coli group have a complex antigen structure. Unlike Salmonella, they have not two, but three different antigens: O (somatic), H (flagellated) and K (capsular). Among all this group of bacteria there are pathogenic serotypes, conditionally pathogenic and even useful for humans. Useful for human Escherichia coli groves is reduced to their participation in the synthesis of vitamins complex B and K, as well as in the antagonistic effect on anthrax and dysentery sticks, staphylococci, etc. Serological typing of Escherichia coli by O-antigen allows distinguishing pathogenic strains from non-pathogenic ones.

Biochemically, Escherichia coli are very active. They all break down lactose, mannitol, maltose, dextrose, galactose, and xylose; dilute gelatin, reduce nitrates to nitrites, and the vast majority forms indole, but they do not decompose Inositol or form hydrogen sulfide. For isolation of E. coli from various objects and differentiation of their subgroups in laboratory conditions, elective media of Endo, Levin, Heifetz, V. M. Kartashova, Simons, Clark, "nitrin-6", for determination of colitis, Kessler medium, etc. are widely used.

Bacteria of the Proteus group also have a different antigenic structure, which Kaufman and perch put on the basis of serological typing and diagnosis. On the basis of a number of cultural and biochemical signs, such Proteus species as Proteus vulgaris, Pr. Mirablis, Pr. Morganii, Pr. RettgeriM, etc. are described.

The most permanent feature for all types of Proteus is the ability to decompose urea. All oppor-

tunistic bacteria are relatively resistant. They are resistant to high concentrations of table salt and to drying on various environmental objects, do not die in sub-zero temperatures, are viable in raw well and tap water, etc.These bacteria quickly die at a temperature of 68 C and above.

Pathogenicity. To date, about one hundred pathogenic serotypes of Escherichia coli have been systematized, causing diseases in humans, animals, including birds;

From the representatives of the Escherichia coli group, the most pathogenic subgroup is considered to be A. Aerogenes (I. S. Zagaevsky). These bacteria often cause colibacteriosis in calves and children, severe mastitis in cows, and acute inflammation of the lungs and genitourinary tract in humans and animals. In addition to the disease, some types of E. coli bacteria cause spoilage of milk and dairy products. Bacteria of the Proteus group in animals sometimes cause severe gastritis and gastroenteritis. They can also aggravate the underlying disease (in humans – wound) infection, in young animals-paratyphoid), causing a secundary infection.

For a long time, it was believed that these opportunistic bacteria do not cause food diseases in humans. This statement was justified by the fact that Escherichia coli4 constantly lives in the gastrointestinal tract of humans, and" B. Proteus vulgaris in 6-8% of cases is found in the intestines of healthy people. Based on numerous studies and observations in recent decades, the epidemiological role of opportunistic bacteria, especially Escherichia coli and Proteus, in the occurrence of food toxicoinfections in humans has been fully proven: It is also proved that not all strains of Escherichia coli are capable of causing foodborne illness in humans, and toxicoinfection is caused only by those that have acquired and have a known degree of pathogenicity. One of the conditions for the occurrence of toxicoinfections of this etiology is the massive contamination of food products with these bacteria.

The incubation period for toxicoinfections of colibacterioid etiology in humans is from 8 hours to one day. Clinically manifested by cramping pains in the abdomen, Nausea and liquid multiple stool. The body temperature is usually normal and rarely rises to 38-39 C, recovery occurs in 1-3 days. Food toxicoinfections caused by Proteus Bacillus usually develop in 8-20 hours after eating. The disease can have a violent beginning, accompanied by cutting pains in the intestines, nausea, vomiting, diarrhea. The disease lasts 2-3, sometimes 5 days. In severe cases, there is cyanosis, convulsions, weakening of heart activity, leading to a fatal outcome (mortality up to 1.5 - 1.6 %). The process of occurrence and development of the disease is similar to that of food salmonellosis, since an indispensable condition is also the ingestion of live bacteria into the human body with food.

Food toxicoinfections caused by Cl. Perfringens.

Morphologically, the microbe is a short, spore-forming, gram-positive rod, it is an anaerobe. There are six types of Cl. Perfringens, designated by initial letters of the Latin alphabet. Some representatives of these types can be pathogenic. Types B, C, D,E are pathogens of enterotoxemia in various animals, and type C is also a pathogen of necrotic enteritis in humans.

Of the slaughtered animals, enterotoxemia is more common in sheep.

Of course, the meat of animals forcibly killed with enterotoxemia is a danger of occurrence among " people. However, it is established that the most common food diseases in humans are caused by type A. they are Characterized by diarrhea and abdominal pain, sometimes nausea, and only a few victims-vomiting and fever. The incubation period ranges from 5-6 hours to one day. The disease usually lasts about a day. Mass outbreaks with pronounced symptoms of toxicosis are more often observed in children and weakened elderly persons.

A prerequisite for the occurrence of toxicoinfection is the accumulation of a large number of live bacteria in the food product. Criteria for the sanitary assessment of products contaminated with Cl. Perfringens, however, remain insufficiently developed. In some meat products (pasteurized canned food), the presence of Cl. Perfringens is not allowed. It is recommended to consider food products subject to long-term storage as benign, if in 1 g of the product the vegetative cells of the microorganism contain up to 10,000, and spore cells-up to 1,000. In General, the recommendations come down to the fact that When establishing CL contamination. Perfringens of meat and meat products the latter must be cooked. When controlling food products, it should be borne in mind that the contamination of milk with these microorganisms often causes damage to cheeses.

Food toxicoinfections caused by B. Cereus.

This microbe is a fairly large, up to 3 - 5 μm in length, gram-positive rod belonging to the group of aerobic or facultatively anaerobic spore bacteria. It grows well on conventional, nutrient media, quickly forming oval spores located terminally. According to H-antigen, there are more than 20 serological variants of this microorganism. It is widely distributed in the surrounding environment (soil, air, water), is found on the skin of animals, the Surface of equipment for the production of food products, as well as in meat, offal, meat semi-finished products during their storage, plant food products, etc. In food products, it usually penetrates exogenously, without changing their organoleptic parameters.

Vegetative forms of the microbe die when exposed to temperatures of about 70 C, at 4 - 6 C do not reproduce, and at subzero temperatures for a long time remain viable (at minus 20 C to 4 months.). Spores of B. cereus are quite stable. They can withstand pasteurization modes, and at 105 — 125 C show viability to 101 - 13 min. They are preserved for a long time at low plus and minus temperatures.

Food toxicoinfections, the cause of which is B. Cereus, occur when eating food of animal, vegetable, and mixed origin. The incubation period of the disease is short - from 3 to 4 hours to a day. The disease in humans is manifested by gastroenteritis (colic-like abdominal pain, nausea, diarrhea) at normal or slightly elevated body temperature. Less often, the disease is accompanied by a sharp headache, vomiting, convulsions and even loss of consciousness (in children and the elderly). The duration of toxicoinfection up to 3-6 days and fatal outcome is extremely rare.

Toxicoinfections caused by poorly studied microorganisms.

Recently, reports of food toxicoinfections caused by poorly studied pathogens belonging to the genera Yersinia, Campylobacter, Pseudomonas, Citrobacter , etc. have appeared in the literature. These bacteria are gram-positive, in the absolute majority mobile, aerobic. They live in the intestines of animals and humans, from where they enter the environment, including food. Their resistance does not differ from the resistance of conditionally pathogenic gram - negative bacteria (E. Coli, Proteus), as well as vegetative forms of bacilli (B. Cereus).

Food toxicoinfections caused by these microorganisms occur after consuming meat, milk, meat and dairy products, fish, etc. They have an acute course lasting up to 3-5 days. Clinically manifested by fever, headache, weakness, nausea, vomiting, diarrhea. In most cases, the disease ends in recovery after 5-6 days.

The diagnosis of food toxicoinfection is of a clinical and epidemiological nature. The leading and constant component is the clinical picture of the disease.

The clinical picture of food toxicoinfection consists of a different combination of symptoms of acute gastritis, enteritis and colitis, signs of General intoxication characteristic of an acute infectious disease and developing dehydration.

Food toxicoinfections have a cyclical course: a short incubation period (from 1-6 hours to 2-3 days), an acute period of the disease (1-5 days) and a period of reconvalescence.

They differ in the degree of severity of the condition to light, medium, severe and severe. The severity criteria of the patient's condition are the severity of intoxication, the degree of dehydration, the amount of blood pressure, pulse rate, stool frequency, changes in peripheral blood with leukocytosis and a rod-shaped shift of the leukocyte formula to the left.

In food toxicoinfections, the stomach and intestines are involved in the pathological process. The degree of damage to different parts of the gastrointestinal tract is not the same, which causes the predominance of symptoms of enteritis, colitis, enterocolitis in the clinical picture, and in some cases the dominance of the phenomena of acute gastritis.

Food toxicoinfection is characterized by an acute onset, most often by the type of acute gastroenteritis or gastritis. The leading symptoms are: weakness, nausea, runny stools, abdominal pain, chills, and elevated body temperature. The disease usually begins with abdominal pain. Most often, they are localized in the epigastrium, somewhat less often - in the amniotic region or have a spilled character. In a significant number of patients, abdominal pain is cramping, less often it is permanent. Nausea is observed in 85-90% of cases, it often precedes the onset of vomiting. Vomiting is observed in 75-80% of patients. At first, the vomiting is plentiful, then more sparse mucosa. Often the vomit is colored green due to an admixture of bile. Vomiting can last up to two days, in some cases it is Long and painful. After vomiting, sometimes simultaneously, in 95-96% of cases, liquid stool appears. Usually it is of a wa-

tery nature, less often it has the appearance of a liquid porridge. The stool may be copious or sparse, sometimes the color of "bog ooze" or containing flakes of mucus. Significantly less often, an impurity of blood is detected, which indicates the involvement of the colon in the pathological process. Tenesmus is not observed in patients. The frequency of defecation acts, to a certain extent, reflects the severity of the disease and the severity of intoxication. With a mild course of the disease, the number of acts of defecation does not exceed 5-10 times a day, with medium - severe reaches 10-20 or more times. In a significant number of patients, bloating and rumbling are detected, the tongue is densely overlaid, and with significant fluid loss it is dry. Chills and an increase in body temperature are observed in 60-70 % of patients. Fever is often subfebrile, but there may be a high temperature reaching 38-40 C. The duration of fever is determined in 2-5 days. In severe cases, hypothermia or hyperthermia may occur.

Intoxication syndrome in patients, in addition to fever, is manifested by General weakness, headache, dizziness, muscle and joint pain, shortness of breath, cramps in the muscles of the extremities. The latter can also be a consequence of dehydration. Most often, cramps of the calf muscles are noted. Dehydration in most patients is characterized by thirst, dryness of the mucous membranes, in severe cases - hoarseness of the voice. Dehydration of II - III degree, marked pallor and dryness of skin, decreased skin turgor, sharpen facial features, sunken eyes, acrocyanosis and cyanosis, tachycardia and arterial hypotension, decreased urine output. Distinguishes four degrees of dehydration (according to V. I. Pokrovsky): I degree – fluid loss within 3 % of body weight; II degree-4' - 6 %; III degree-7-9 %; IY degree - 10 % or more of body weight.

It is advisable to distinguish between light, medium-heavy and severe intoxication. Food toxicoinfections are characterized by an increase in leucocytosis in peripheral blood and a rod-shaped shift of the deucocyte formula to the left. Due to the severity of intoxication syndrome in the blood serum of patients, an increase in citric acid and the value of average molecular metabolites is noted. In venous blood, hypokalemia, hypocatremia, an increase in the Na index is determined, in some cases - hypocapnia.

In food toxicoinfections, hypovolemia due to fluid loss is noted, and on the other hand, blood redistribution due to toxic paresis of the capillary network. In severe food toxicoinfections, dehydration is caused by the loss of extracellular and intracellular fluid, and in moderate cases, the deficit of extracellular space is combined with the transition of water into cells. Dehydration is of an isotonic nature, and is accompanied by hemoconcentration and an increase in blood viscosity, a shortage of electrolytes, metabolic acidosis in the capillary blood, and deformation of red blood cells. Dehydration is associated with a decrease in heart size, shock index, circulating plasma volume, right ventricular overload, pulmonary hypertension, bypass surgery in the large and small circulatory circles, significant microcirculation disorders, thrombohemorrhagic syndrome, hypoxia, and respiratory insufficiency. And although in food toxicoinfections, violations of water-electrolyte metabolism do not determine the entire complex of clinical and pathophysiological changes, however, in many cases they become dominant and become crucial for the outcome of the disease.

In patients with food toxicoinfections, changes in the cardiovascular system are often detected, which are manifested by a decrease in the sonority of heart tones, tachycardia (less often - bradycardia), arterial hypotension. On the ECG, diffuse changes in the myocardium of a dystrophic nature are observed, due to intoxication, water-electrolyte and circulatory disorders, hypoxia. These changes are characterized by a decrease in the t-wave and depression of the t-segment. These changes may persist in speech for 6 to 10 days, and then disappear. Impaired blood supply to the kidneys and changes in the water-electrolyte balance lead to acute renal failure, which is mainly prerenal and characterized by the occurrence of oliguria or anuria, azotemia. At the same time, nausea, vomiting, anorexia, weakness, headache are observed. Venous blood tests reveal increasing decompensated metabolic acidosis.

In patients, along with the symptoms of acute gastroenteritis, symptoms of colitis can be noted. In these cases, the disease is relatively more severe: abdominal pain becomes pronounced, cramping and localized in the lower abdomen or in the left iliac region. Stool becomes more frequent, but its volume may decrease, and the impurity of mucus with streaks of blood increases. Palpation determines the soreness of the sigmoid and other parts of the colon.

Approximately 4-5 % of cases in the clinical picture of the disease can be observed only symptoms of acute gastritis. These ним include loss of appetite, nausea, repeated copious vomiting first with food, and then with mucus and bile, pain on palpation in the epigastric region, an increase in body temperature, rapid pulse, and a decrease in blood pressure. The duration of the acute period with timely treatment is up to three days.

In the elderly and senile age, the clinical picture of the disease is characterized by a longer duration than in young patients: nausea and vomiting, a tendency to arterial hypotension.

Atherosclerosis, hypertension and coronary heart disease increase the possibility of developing a hypertensive crisis, acute or transient cerebral circulatory disorders, acute coronary insufficiency, etc.

In patients with chronic alcoholism, food toxicoinfections contribute to the development of severe variants of abstinence syndrome and metal-alcohol psychoses.

Lethality in food toxicoinfections ranges from 0.04 to 0.6 % and largely depends on the organization and level of qualified emergency care, the use of modern methods and means of therapy, timely correction of the resulting violations. In the vast majority of cases, the cause of fatal outcomes is complications from the cardiovascular system: myocardial infarctions and acute coronary insufficiency (23.5%), mesenteric vascular thrombosis (23.5%), pulmonary vascular thrombosis (2.6%), acute cerebral circulatory disorders (7.8%), aortic ruptures (4.3 %). The second place in the frequency of fatal outcomes (15.6 %) is occupied by pneumonias combined with food toxicoinfection (mixed infection), or developing in patients after suffering from pulmonary edema with infectious and toxic shock, which complicated food toxicoinfection. The third place among the causes of fatalities in food toxicoinfections is occupied by shock, which in some cases is hypovolemic, in others-infectious and toxic. When shock occurs, acute hemodynamic and microcirculatory disorders, metabolic shifts. There are General (or systemic) and regional disorders of microcirculation (brain, kidneys, and lungs). Their causes are toxic effects of microbes and their toxins, vasomotor disorders and the resulting increased capillary permeability, violations of the acid-base state and water-electrolyte metabolism.

In the differential diagnosis of food toxicoinfections, it is necessary to remember that a number of acute intestinal infections have similar clinical symptoms, including the gastrointestinal form of salmonellosis, gastroenterocolitic form of dysentery, the gastrointestinal form of iersiniosis, rotavirus gastroenteritis, botulism, cholera, NAG infection, campylobacteriosis. Known food poisoning caused by arsenic and heavy metal salts, fluorine compounds, tetraethyl lead, phosphorous and organochlorine compounds. There are food poisoning associated with eating poisonous or conditionally edible mushrooms.

The second component of the diagnosis is information of an epidemiological nature. As a rule, clinicians do not have sufficiently complete epidemiological data, especially in cases of sporadic diseases. For example: the identification of the product that was the factor of infection, as well as the isolation of the pathogen from this product, which, however, is not an obstacle to establishing a diagnosis.

In an epidemiological sense, the most important task remains to identify the conditions that contribute to outbreaks and sporadic diseases. These materials are important for organizing targeted preventive work.

When analyzing laboratory data, it should be taken into account that:

I. Two-time isolation of a monoculture of conditionally pathogenic bacteria in the first three days of the disease from the patient's stool is possible on average in 50 %, and a single isolation-in 30 % of cases;

II. The increase in antibody titer in the blood depends not only on the type of pathogen, but also on the state of reactivity of the patient's body. It is necessary to take into account the presence of concomitant chronic diseases of the digestive system in the patient and the use of antibiotics in the period preceding the present disease.

Patients with food toxicoinfections are hospitalized in infectious hospitals.

In some cases of a mild course of the disease, the doctor of the infectious diseases office (or a district therapist) can leave the patient for treatment at home after a thorough examination, provided that active daily medical supervision is carried out.

At the same time, it is necessary to keep in mind that at first a mild course of the disease can pro-

gress. The volume of medical care for food toxicoinfections is directly dependent on the form and severity of the disease.

Therapeutic measures aimed at detoxification, combating dehydration and restoring hemodynamic disorders are carried out. At the same time, background and concomitant diseases should be treated. All patients with food toxicoinfections should receive a sparing diet. In the first day, table No. 4 is usually assigned, and as diarrhea decreases, table No. 13 is assigned. The diet excludes products that have an irritating mechanical and chemical effect on the stomach and intestines: milk, canned food, smoked foods, spicy and spicy dishes, raw vegetables and fruits. It should be remembered that errors in the diet can lead to an exacerbation of functional disorders of the gastrointestinal tract.

All treatment measures for food toxicoinfections should be carried out in the shortest possible time, and in severe cases of the disease - immediately upon admission of patients to the hospital. Treatment of patients begins with gastric lavage. It is performed regardless of the time that has elapsed since the onset of the disease, and is carried out with a solution of sodium bicarbonate or a 0.1% solution of potassium permanganate. The volume of solutions used for washing is determined, on average, by 3 liters at a temperature of 18-20 C. The most effective t gastric lavage is performed with the help of a gastric probe. Usually, gastric lavage is performed before the outflow of washing waters. Contraindications to gastric lavage are: ischemic heart disease with angina pectoris, hypertension with high blood pressure figures, atherosclerosis with a predominant lesion of the brain vessels, peptic ulcer of the stomach and duodenum.

Scheme of treatment of patients with PTI

STRATEGY	TACTICS
I. Elimination of microbial toxins from the gastrointestinal tract.	1. Gastric lavage (2-4 % sodium bicarbonate solution or 0.1 % potassium permanganate solution). 2. The purpose of adsorbents. - Preparations of cellulose (polyphepan, lignosorb, activated carbon, vaulen, etc. on 20g. 3 times a day). - Attapulgitis preparations: neo intestopan, reaban; - cation-Binding preparations (enterocate M: 20-30g., then 10 g. 3 times). - Derivatives of polyvinylpyrrolidone (use adsorbents 5 g/100 ml 3 times per day).
II. Relief of diarrheal syndrome	Preparations of CA++: gluconate, glycerophosphate, lactate-5g. or calcium carbonate-30-50g.
III. Rehydration and remineralization.	1. Glucosinolate oral rehydration solutions (rehydron, tsitraglyukosolan, etc.) 2.Nasogastric rehydration option. 3. Parenteral rehydration (solutions "Kvartasol", "Acesol", "Lactasol", "Trisol", "Methusol" (for intravenous infusion in patients with dehydration III-IV art.)
IY. Restoration of the gastrointestinal mucosal barrier.	Cytoprotectors (smekta, POLYSORB MP).

In mild cases of food toxicoinfections without signs of dehydration, gastric lavage exhausts the entire volume of medical care. The fight against dehydration in the case of moderate diseases is performed with rehydration solutions that are administered orally. For the majority of patients, rehydration therapy should be performed by oral route and only 5-15 % of patients with food toxicoinfections need intravenous infusions. The volume of injected fluid should be determined by the amount of losses dur-

ing vomiting and diarrhea. Practically, it is determined by the degree of dehydration and body weight of patients.

When conducting rehydration therapy, there are two stages:

I. recovery of pre-treatment losses;

P. correction of continuing losses.

In severe cases of the disease and in some cases of moderate food toxicoinfections rehydration therapy is performed intravenously with polyionic crystalloid solutions: "Kvartasol", "Chlosol", "Trisol".

The most effective solution is considered to be "Kvartasol", which contains in 1 liter of pyrogen-free distilled water: sodium chloride-4.75 g., potassium chloride-1.5 K., sodium acetate-2.6 g., sodium bicarbonate-1.0 g.

Trisol solution, despite the fact that it is used in various regions of the world, has a number of disadvantages, and first of all, incomplete compliance of the salt composition of the solution with the structure of electrolyte losses in patients with acute intestinal infections. It contains an excessive amount of Na+ ion, a clearly insufficient amount of K+ ion and an excessive amount of sodium bicarbonate, which contributes to the development of alkalosis. In addition, bicarbonate is unstable in aqueous solutions, it can not be sterilized by boiling, and after production it can be stored for no more than 12 hours. The solution "Kvartasol" is devoid of these disadvantages, the duration of its storage when manufactured in pharmacy conditions - 1 month, in industrial production-2 years.

Before intravenous infusion, the solutions are suspected to reach a temperature of 38 C and try to maintain the specified temperature throughout the entire transfusion period.

Along with the salt solutions for food poisoning to detoxification used synthetic colloidal solutions gemodez, reopoligljukin, polyglukin.

Hemodesis has the ability to form a complex with toxins, increases renal blood flow and eliminates red blood cell stasis in the capillaries. Rheopolyglucin reduces the aggregation of shaped blood elements, promotes the movement of fluid from tissues to the bloodstream. When it is administered, the viscosity of blood decreases, blood flow in small capillaries is restored and the detoxification effect is carried out. Polyglucin retains fluid in the bloodstream for a long time and has a hemodynamic effect. All colloidal solutions can be administered only in the absence of dehydration in patients. When introducing: colloidal solutions into the vein, fluid moves from the interstitial space to the vascular bed. This increases blood pressure and creates an idea of the resulting clinical effect. However, the condition of water-salt metabolism in patients worsens.

It is most appropriate to use hemodesis to combat intoxication, and rheopolyglucin - to restore microcirculation disorders. The disadvantage of colloidal solutions should also be considered an increase in metabolic acidosis that occurs after treatment.

Intravenous therapy with polyionic crystalloid solutions is carried out at the 1st stage of treatment for severe food) toxicoinfections in the volume of 60-120 ml per kg. Body mass index with a volume rate of 70-90 ml/min, with a moderate form of the disease in the volume of 55 - 75 ml per kg of body weight with a volume rate of 60 - 80 ml/min.

When dehydration of the III-IY degree, treatment begins with intravenous jet administration of polyionic crystalloid solutions with a volume rate of 80-120 ml/min. When introducing solutions in the volume of more than 90 ml/kg in elderly people and those suffering from cardiovascular diseases, there is a need for dynamic monitoring of the state of hemodynamics (measurement of blood pressure, ECG). The total amount of fluid required for infusions is determined based on the degree of dehydration of the patient and body weight. Treatment should be preceded, if possible, by a study of the acid-base state(CBS), hematocrit index and concentration of electrolytes in blood plasma and in red blood cells. After the end of stage 1 of treatment, repeated laboratory tests are necessary. If necessary, the second stage of treatment is performed, using the same polyonic solutions, taking into account the measurement of continuing fluid loss with stool, vomit and urine for a certain period of time (for large losses - every 2 hours, for small - for 4-6 hours). The rate of administration of liquid in the second period of treatment may vary depending on the amount of continuing losses and is 40-60 ml/min. The balance of losses and the amount of fluid administered every two hours are recorded in the medical history.

Intravenous fluid administration is stopped after the disappearance of vomiting, stabilization of hemodynamics and restoration of excretory function of the kidneys. An important criterion for stopping intravenous transfusion is the predominance of the amount of urine released over the amount of stool in the last 4 hours. The total volume of intravenous administration in severe and prolonged cases of the disease can reach 10-20 liters of solution.

Colloidal liquid (gemodez, reopoligljukin, polyglukin) are administered intravenously at a speed of 40 - 60 drops per minute. The volume of administered hemodesis is determined in 400 ml (no more than 800 ml) per day, rheopolyglucin in 400 - 800 ml, polyglucin in 500 -1000 ml. In some cases, the use of crystalloid solutions, providing the elimination of hemoconcentration and hemodicamic disorders, does not lead to the correction of metabolic acidosis. In these cases, after completion of water - electrolyte therapy with polyonic solutions, it is necessary to resort to correction of metabolic acidosis with a 4% solution of sodium bicarbonate.

The amount of solution is calculated using the formula:

$$\text{The amount of 4\% growth. NSOZ, ml} = \frac{\text{body weight in kg x for base deficiency}}{3}$$

To combat metabolic acidosis, trisamine can be used instead of a 4% sodium bicarbonate solution. Trisamine (TNAM) is a buffer substance that reduces the concentration of hydrogen ions when administered intravenously, increases the main blood reserve and eliminates acidosis. At the same time, the CO2 Content in the blood does not increase. The drug penetrates well through cell membranes into cells and produces a diuretic effect.

The amount of necessary trisamine solution administered intravenously is determined by the formula:

K=B x E, where

K - the number of milliliters of 3.66% trisamine solution;

B - defecit of bases in mEq/l (BE, according to Astrup); \

E - patient's body weight in kg.

Metabolic acidosis, observed in patients with food toxicoinfections, can be caused not only by these diseases, but also by the exacerbation of background diseases (diabetes mellitus, chronic renal failure), about which some patients may not know. When correcting acidosis, it should be borne in mind that colloidal solutions not only do not eliminate metabolic acidosis, but also increases it.

The method of oral rehydration is applicable in patients with mild and moderate course of food toxicoinfection, which occurs with dehydration of the I - II degree and in some patients with the III degree. Oral route of liquid administration is more physiological than intravenous, safe and widely used in conditions of a shortage of sterile solutions.

Currently, the oral rehydration solution ORS, recommended by who, "Oralit", and containing in 1 liter of boiled water: sodium chloride-3.5 g, sodium bicarbonate-2.5 g, potassium chloride-1.5 g, glucose-20g.

It is advisable to recommend another oral rehydration solution ORS ("Regidron"), containing sodium hydrocitrate and successfully used in the clinic of the Central research Institute of epidemiology of the Ministry of health of the Russian Federation. In 1 l. solution of ORS with sodium hydrocitrate contains sodium hydrochloride - 3.5 g, potassium chloride - 2.5 g, sodium hydrocitrate - 4.5 glucose - 17.0 g.

The volume of administered oral solutions should be determined by the amount of fluid loss during vomiting and diarrhea and is practically determined by the severity of intoxication, degree of dehydration and body weight of the patient. The volume rate of administration of the solution through the mouth is 1-1. 5 l / hour and depends on the absorption capacity of the mucosa (up to 2 - 3 l/hour). Oral rehydration therapy is performed in two stages - stage 1-primary rehydration to eliminate signs of dehydration and IIstage II - maintenance therapy aimed at stopping ongoing losses. In some cases, especially with persistent vomiting, there is a need for the introduction of a solution; ORS through a nasogastric probe. Regulation of the speed of the solution - this is carried out with the help of a Mora clamp, the temperature of the administered solutions is 40 C, which contributes to better absorption in the intestine. Difficulties in implementing oral rehydration occur with decompiled dehydration and signs of

infusion-toxic shock; prolonged olinoauria, indomitable vomiting, glucose absorption disorders in individual patients; diabetes mellitus. It is also possible to assume the failure of oral rehydration therapy in cases when the loss of fluid in patients with acute diarrhea exceeds the volume of the ORS solution administered over a period of time.

In the vast majority of cases, oral rehydration therapy has a convincing therapeutic effect. In the treatment of food toxicoinfections in conditions of developing arterial hypotension, there is no indication for the appointment of glucocorticosteroids and analeptics (cordiamine, sulfokakfokain). Past adrenomimeticalkie substances (norepinephrine, mezaton) is contraindicated, because their introduction is exacerbated by the peripheral vasoconstruction. If the patient did not receive cardiac glycosides (strophanthin, corglycone, digoxin) for the treatment of background diseases before the food toxicoinfection, then their use is not indicated. After the acute symptoms subside it is advisable to appoint enzyme preparations (panzinorm, festal, deferment etc.) and antioxidants (tocopherol acetate, unital).

Treatment of complications.

SHOCK Treatment of hypovolemic shock is carried out by jet injection of polyonic crystalloid solutions ("Kvartasol", "Khlosol", "Trisol") with a volume rate of 100-120 ml/min. Most often, transfusion is performed through one , and sometimes simultaneously, through two peripheral veins. If it is impossible to perform transfusion to peripheral veins, subclavian vein catheterization is performed. The volume of infusion depends on the degree of dehydration and body weight of the patient and is determined taking into account arterial and venous pressure, respiratory rate and pulse, hematocrit and acid - base status. Usually after 30 - 60 minutes of treatment, cyanosis and shortness of breath disappear, the pulse rate is restored and blood pressure normalizes, nausea and vomiting stop. Stopping diarrhea occurs later. Hypovolemic shock, as a rule, can be eliminated with the help of infusion therapy and the use of any other drugs is not necessary.

In case of infectious-toxic shock, treatment should begin with transfusion therapy performed with polyionic crystalloid solutions. Main goals of treatment:

1) filling the volume of liquid in order to eliminate hypovolemia;
2) the fight against intoxication;
3) restoration of disturbed hemostasis parameters.

Replacement of the fluid volume should be performed under the control of determining the Central venous pressure (CVP). The increase in CVP to 12 - 15 cm water column indicates the emergence of a threat of overload of the heart. The transfusion rate is then slowed or stopped. With a CVD of more than 15 cm.water. St. transfusion therapy is contraindicated and in these cases, the use of sympathomimetics. Simultaneously with infusion therapy with crystalloid solutions, oxygen therapy is performed by insuflating oxygen in a volume of 4 l/min through a probe inserted into the nose. Further administration of oxygen is performed under the control of the level of blood gases. At the same time, correction of metabolic acidosis is performed using a 4% solution of sodium bicarbonate or 0.3 m solution of trisamine. These measures belong to the 1st therapeutic stage of treatment of infectious toxic shock. In the absence of an effect or incomplete effect of treatment, resort to the second therapeutic stage of treatment. Prescribe sympathomimetics (dopamine). Selective action on various parts of the organ's circulatory system makes dopamine the drug of choice. The dosage of the drug is controlled by the level of blood pressure and heart rate. Dopamine is administered intravenously by drip: 25 or 200 mg. The drug is diluted, respectively, in 125 or 400 ml of 5% glucose solution. The content of dopamine is 200 and 500 micrograms, respectively. The initial rate of administration is 1-5 micrograms/kg/min. or 2-11 drops of 0.05 % solution. If necessary, the speed of dopamine administration is increased to 10-25 mcg/kg/min. The action of the drug comes quickly and ends in 5-10 minutes after the end of the infusion.

An infusion of a dopamine solution is made continuously for a time from several hours to several days. The daily dose of dopamine is up to 400 mg per patient with a body weight of 70 kg. When using dopamine, constant monitoring of the level of hemodynamics (including pulse rate, blood pressure and electrocardiographic monitoring) is necessary. The treatment of such patients should be carried out in the Department of resuscitation and intensive therapy. It should be borne in mind that when using high doses of dopamine, myocardial ischemia, tachycardia and renal vasoconstriction can occur.

In the presence of high CVD figures, the appointment of digoxin (0.025 % - 1 ml. intravenously in a drip), celanide (0.02 % - 1 ml. intravenously in a drip) is indicated. For the treatment of infectious - toxic shock, intravenous administration of 5 glucocorticosteroids in large doses - 15-30 mg/K of prednisolone is indicated. Sometimes combine the appointment of prednisolone with dexazone and hydrocortisones. Steroids will reduce the overall peripheral resistance and have a favorable protective effect on the cell structure, preventing the violation of its function.

Given that disseminated intravascular coagulation (DIC - syndrome) occurs with ITS, we should expect a positive effect from the appointment of heparin, to prevent serious coagulopathic disorders and irreversible disorders of kidney and liver function. Its dose depends on blood clotting indicators and usually makes 20-30 thousand rubles. ED/day. The most effective is its intravenous drip introduction at the rate of 8-10 thousand rubles. U/50 ml of transfusion solutions.

It is advisable to use proteolysis inhibitors: kontrikala and gordoks. Single doses of kontrikala 10-20 thousand UNITS., gordoks-100-500 thousand UNITS. Drugs are administered intravenously drip.

With persistent oliguria, but only after eliminating hypovolemia and normalizing blood pressure, it is advisable to prescribe diuretics. The most effective are lasix in large doses (250 mg - 1 kg) and mannitol. Therapeutic single dose of mannitol 1-1. 5 g/kg, daily dose-no more than 140-180 g.

After the elimination of hypovolemia, appropriate combined use of crystalloid ("Kvartasol", "Trisol", "Chlosol») and colloid (gemodez, reopoligljukin, polyglukin) solutions. Especially effective is rheopolyglucin, which promotes the restoration of microcirculation.

With progressive shock, in some cases, there is a need for artificial ventilation, which is able to prevent alveolar collapse and prevent the development of pulmonary edema. The consumption of oxygen and carbon dioxide production decreases, which is a favorable factor. The prognosis for ITS, which complicates food toxicoinfections, is always very serious.;

Acute renal failure. Most often, it is the result of a late start or not rational treatment. Its occurrence is facilitated by a long state of hypovolemia and arterial hypotension, the use of Pressor amines (norepinephrine, mezaton). Especially dangerous is the Use of these drugs in conditions of dehydration. Elimination of hypovolemia, rapid recovery of diuresis, correction of the acid - base state and water - electrolyte metabolism are an indispensable condition for a successful fight against acute kidney injury.

Patients with acute renal failure need to perform forced diuresis. After the introduction of 1500 - 2000 ml of polyonic crystalloid solution, an osmodiuretic is introduced - a 15 % solution of mannitol in the amount of 1-1.5 g per 1 kg. patient's body weight. The daily dose of the drug should not exceed 100 g. The use of urea in acute renal failure is contraindicated. It should be borne in mind that the osmodiuretic contributes to an increase in the volume of circulating blood. Saluretics reduce the BCC, so the use of lasix requires caution. Doses of lasix are 2-10 mg/kg, administered intravenously by jet. If the desired effect is not present, then the introduction is not repeated. Intravenous administration of 10-20 ml of 2,4% euphyllin is advisable in order to enhance renal blood flow. It is recommended to introduce dopamine using low concentrations in the blood, achieved at doses of 150-200 mcg/min. Higher concentrations of domamine (300-400 mcg/min) are contraindicated. With increased blood pressure figures in patients with acute renal failure, it is advisable to use ganglioblockers (pentamine, hexonium). In some cases, the effect of papaverine is observed. Can you recommend a gastric lavage through catheters introduced through the nose. Thanks to this, it is possible to reduce vomiting, remove excess toxins from the stomach. It is advisable to wash the intestines in the form of siphon enemas at the rate of 5-10 liters of solution per wash. In cases where the above measures do not give a clinical effect, resort to extra-corporeal hemodialysis using the device "artificial kidney". Indications for transfer of patients to ekstrakorporalny memodules are:

I)progressive deterioration of patients ' condition, continued vomiting and persistent anuria, despite treatment;

II) hyperazotemia, hypercreatiminemia, high rates of azotemia increase (with an increase in the level of urea by more than 8.3 mmol/l per day);

III) hyperkalemia.

In the treatment of ARF it is necessary to carefully measure and record in history the number of

introduced fluid, the magnitude of diuresis, to systematically monitor the frequency of heart rate, arterial and Central pressure, determine the amount of hematocrit and acid – base status, the content of electrolytes in plasma and in erythrocytes, concentration of urea and creatine.

Features of treatment of food toxicoinfections in patients with hypertension, coronary heart disease and chronic alcoholism. Patients with food toxicoinfection with severe dehydration or intoxication, suffering from hypertension and coronary heart disease, crystalloid solutions should be administered in full, based on the degree of dehydration and body weight. All patients with a history of hypertension, who have high blood pressure, headaches and dizziness when admitted to the hospital, at the same time with infusion therapy, intravenous or intramuscular administration of antihypertensive drugs (clofelin, Dibazol) is indicated. Patients whose admission to hospital was noted a distinct anginal syndrome: no signs of myocardial infarction, concurrently with infusion therapy is the purpose of antianginal drugs (nitrosorbid, sustak, nitrong, sustonit). Colloidal solutions (polyglucin, rheopolyglucin, hemodesis) are contraindicated in patients of these groups, especially in the presence of high blood pressure.

In food toxicoinfections occurring in patients with chronic alcoholism and who develop withdrawal syndrome; or metal alcohol psychosis, treatment is carried out by intravenous drip introduction of polyonic solutions.

The volume of fluid administered to the patient is determined by the degree of dehydration and the patient's body weight. Control of diuresis is an indispensable condition for the effectiveness and safety of treatment. Rehydration therapy in this group of patients should be Preceded by stopping the Central excitement by using tranquilizers (seduxen, phenozepam) and nootropic drugs (sodium oxybutyrate).

Prevention. On a national scale-the creation of modern mechanized enterprises of the food industry, the development and introduction into practice of new methods Of processing and storing products.

At food enterprises-careful sanitary control over the production, storage, transportation and sale of food products. Preventing persons With signs of infectious diseases, pustular lesions of the skin and mucous membranes from working.

There is also a need for sanitary and veterinary control at dairy farms and other livestock enterprises.

TASKS

1. Patient D., 50 years old, a nurse of the therapeutic Department became "ill" - dizzy and aching head, weakened, nausea appeared, then vomiting and repeated liquid watery stool. It turned out that in the morning she had had Breakfast with sausage sandwiches, which had been lying outside the window for 3 days. In the Department, the patient was given a gastric lavage, after which she felt better. However, the liquid stool continued, and there were short-term cramps of the calf muscles. After 2 hours, the temperature is 38.5°C, the state of moderate severity, the patient is pale, she has cyanosis of the lips, the pulse is 102 beats per minute. Min., low filling, blood PRESSURE 90/60 mm Hg. The tongue is covered with a white coating, dry, thirsty. The abdomen is soft, slightly painful in the epigastrium and in the navel area, increased rumbling. There are no symptoms of irritation of the peritoneum. Since the morning, the patient did not urinate, the beating on the lumbar region is not painful. There are no meningeal symptoms. The stool was plentiful, green, and the muscle spasms became more frequent and prolonged.

1) Your preliminary diagnosis? 2) Give an assessment of the severity of the disease?

2. An ambulance was called to the patient V., 55 years old. He became acutely ill at 5 o'clock in the morning, when there was frequent vomiting, liquid, abundant stool more than 20 times, cramp-like abdominal pain, weakness, chills, a temperature of 39°C. By the time the doctor arrived, the patient had developed cramps in the calf muscles and was losing consciousness. Stool on examination is liquid, fetid.

The patient's condition is severe, acrocyanosis, turgor of the skin is reduced. In the lungs vesicular breathing, respiratory rate - 28 per min. Heart tones are dull, arrhythmic. Pulse of weak filling, frequent. The AD LIMIT is not detected. The tongue is covered with a white coating, dry. The abdomen is soft, swollen, rumbles on palpation in all departments, there are no signs of irritation of the peritoneum. The liver protrudes from under the edge of the costal arch at cm. The spleen is not palpable. Pasternatsky's symptom is negative on both sides. He didn't urinate. There are no meningeal signs. After starting intensive rehydration therapy in the ambulance, the patient regained consciousness. He was taken to an infectious disease hospital, wherehe continued treatment and performed laboratory tests: pH 7.37; MS 29 mm Hg; SB 16.5 mmol/l; BE-8 mmol/l; hematocrit 57%; plasma potassium 3.2 mmol/l; plasma sodium 115 mmol/l.

1) Make a clinical diagnosis. 2) Suggest a treatment plan for the patient (his weight is 80 kg).

THE ANSWERS

1.1) Diagnosis of " Food toxicoinfection»

2) Severe course of the disease.

2. 1) Diagnosis "Food toxicoinfection, severe course. Dehydration of the III-IV degree»

2) Intravenous rehydration - a solution of quartasol 8 liters (10% loss of body weight at a weight of 80 kg). The liquid is introduced in 2 stages: 1-rehydration 1-1. 5 hours (120 ml/min). 2-correction of continuing intravenous losses by infusion.

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