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Department of Infectious diseases

METHODOLOGICAL GUIDE

SALMONELLOSIS

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Salmonellosis is an intestinal zoonosis caused by numerous pathogens of the genus Salmonella, characterized in the manifest course of a clearly expressed intoxication and gastrointestinal symptoms, as well as the possibility of developing in some cases - a generalized form.

Historical information. Diseases that are epidemiologically and clinically similar to salmonellosis have been known to doctors for a long time. In 1885, D. salmon and T. Smith isolated B. suipestifer-the causative agent, as they believed, of swine fever. In 1888, A. Gertner discovered a microbe similar in properties to V. suipestifer in the organs of a deceased person and meat used for food, thus substantiating the bacterial etiology of salmonellosis in humans and animals.

In the future, reports began to appear about the isolation of a number of microorganisms similar in morphological and biochemical properties to the bacteria of Salmonella and Gertner. All of them were combined into a group of paratyphoid microbes and in 1934 received the name Salmonella.

Etiology. Pathogens of salmonellosis belong to the genus Salmonella, a family Of enterooacteriaceae intestinal bacteria Enterooacteriaceae. Morphologically, Salmonella are sticks with rounded ends and a size of 1-3-0, 2 - 0.6 microns. They, with few exceptions, mobile, have flagella all over the cell surface (peritricha). Spores and capsules do not form, gram-negative. They grow on normal growth media.

Salmonella are capable of producing exotoxins. Among them are enterotoxins (thermolabile and thermostable), which increase the secretion of fluid and salts into the lumen of the intestine, and cytotoxin, which disrupts protein-synthesis processes in the cells of the intestinal mucosa and affects cytomembranes.

When destroying bacteria, endotoxin is released, which is largely associated with the development of intoxication syndrome.

The antigenic structure of Salmonella is complex. They contain O - and N-antigens. O-antigen is associated with the somatic substance of the cell, is thermostable, H-antigen with the flagellated apparatus, is thermolabile. The antigenic structure is based on the International serological classification of Salmonella (Kaufman—white scheme). Differences in the structure of O-antigens allowed us to distinguish serological groups A, B, C, D, E, etc. Based on differences in the structure of H-antigens within each group, serological variants have been established. About 2000 serological variants of Salmonella have been described, including more than 700 in humans. The most common types of Salmonella are: S. typhimurium, S. heidelberg, S. enteritidis, S. anatum, S. derby, S. Ion-don, S. panama.

Salmonella is relatively resistant to various environmental factors, some of them do not die when frozen to 48-82°C and well tolerate drying. On various objects at room temperature, they persist for 45-90 days, in dry animal feces-up to 3-4 years. In water, especially at a low pH, Salmonella can survive for 40-60 days. In dairy and prepared meat products, Salmonella not only persists for up to 4 months, but also multiplies, without changing the organoleptic properties and appearance of the products. Salmonella is resistant to salting, Smoking and acids. The destruction of bacteria requires high-quality heat treatment. So, for complete inactivation of Salmonella, which are in a piece of meat weighing 400 g, it is necessary to cook it for at least 2.5 hours.

Epidemiology. The source of infection can be animals and humans, and the role of animals in epidemiology is the main one. Salmonellosis in animals is found in the forms clinically expressedgenego diseases and bacteria. Being outwardly healthy, they can secrete pathogens with urine, feces, milk, nasal mucus, saliva. The duration of bacterial transmission in animals can be different and is often calculated in months and years. The greatest epidemiological danger is infection of cattle, pigs, sheep, and horses. Salmonella-borne bacteria was also detected in dogs, cats, house rodents (mice and rats), and in many species of wild animals: foxes, beavers, wolves, Arctic foxes, bears, seals, monkeys, etc.

A significant place in the epidemiology of salmonellosis is occupied by birds, especially waterfowl, which serve as a powerful reservoir of the pathogen. Salmonella is found not only in meat and internal organs of birds, but also in eggs. Infected eggs do not differ in appearance, smell, or taste from normal eggs. In this regard, it is not recommended to eat raw eggs, especially duck and goose. Salmonella is also found in products made from raw eggs (egg powder). Salmonellosis disease and carrier pathogens are also noted among pigeons, sparrows, gulls and other bird species. There are data on the isolation of Salmonella from lizards, turtles, snakes, frogs, fish, crayfish and crabs.

Sources of salmonellosis can be people with salmonellosis or bacterial separators. The most important in this case are persons belonging to the category of "food processors".

The main route of infection transmission is food.

Salmonella transmission factors are food products. This includes meat from animals or birds. Infection of meat occurs endogenously (during the life of the animal during its illness), as well as exogenously, in the process of transportation, processing, storage. Food is often infected by improper cooking, cooking on contaminated tables, and using infected utensils.

Under certain conditions (close communication with a sick personor animals in case of non-compliance with basic sanitary and hygienic norms), a contact-household transmission path can be realized. This pathway is noted, for example, in nosocomial outbreaks of salmonellosis, usually caused by S. typhimurium. The disease is registered mainly in children under 1 year of age. Possible water and in rare cases the dust (if dust ingestion) routes of transmission of infection.

Children under 1 year of age and people with various types of immunodeficiency are most susceptible to salmonellosis. The latter explains the high incidence of salmonellosis in people with severe somatic pathology and serves as a prerequisite for outbreaks of the disease among hospitalized patients. In this case, salmonellosis can be considered as an "intra-hospital" infection.

Salmonellosis occurs throughout the year, but more often in the summer months, which can be explained by the deterioration of food storage conditions. There is both a sporadic and group incidence of this infection.

Pathogenesis and pathological anatomy. When ingested in the gastrointestinal tract, Salmonella overcomes the epithelial barrier of the small intestine and penetrates into the thickness of tissues, where they are captured by macrophages. Inside macro-

phages, microbes not only multiply, but also partially die with the release of endotoxin, which affects the neurovascular apparatus of the intestine and increases the permeability of cell membranes. This contributes to the further spread of Salmonella along the lymphatic pathways and their entry into the mesenteric lymph nodes.

Along with the fact that endotoxin has a local effect, it contributes to the development of symptoms of General intoxication of the body. At this stage, the infectious process, acquiring a localized (gastrointestinal) form, can end.

However, even with localized forms of infection, the pathogen can enter the blood, but bacteremia in this case is short-lived.

With a deep violation of the barrier function of the intestinal lymphatic apparatus, the process of generalization occurs and a long-term bacteremia occurs, which clinically corresponds to the development of a generalized form of salmonellosis. As a result of bacteremia, Salmonella is introduced into various internal organs, causing dystrophic changes in them or the formationof secondary purulent foci (septicopyemic variant).

The mechanism of activation of adenyl cyclase (and guanyl cyclase) of enterocytes by salmonellosis enterotoxin with subsequent increase in the intracellular concentration of biologically active substances (camp, cGMP, etc.), which leads to the entry of a large amount of liquid, potassium, sodium and chlorides into the intestinal lumen, is the basis for increasing the secretion of fluid in the intestine. Patients experience vomiting and diarrhea. Symptoms of dehydration develop, and serum levels of sodium, chloride, and potassium decrease. Dehydration leads to tissue hypoxia with impaired cellular metabolism, which, in combination with electrolyte changes, contributes to the development of acidosis. In severe cases, oliguria and azotemia appear. These pathological phenomena are especially pronounced in the development of dehydration (more often), infectious-toxic and mixed shocks.

Pathomorphological changes in salmonellosis are diverse, depending on the form, severity and duration of the disease. The severity of pathoanatomic changes does not always correspond to the severity of the disease.

In *the gastrointestinal form* of the disease, catarrhal inflammation is morphologically predominant in all parts of the gastrointestinal tract. Macroscopically, the intestines show sharp fullness with hemorrhages of various sizes, edema of the mucous membrane, sometimes with superficial necrosis and a tender bran-like plaque. The lymphatic apparatus of the intestine may not be changed, and the spleen may not be enlarged. In all other organs, there is a sharp fullness and dystrophic changes. Microscopically, vascular changes with hemorrhages in the mucous membrane and submucosal layer are detected in the intestine. In the submucosal layer, there is a violation of microcirculation with a reactive leukocyte reaction and pronounced tissue edema.

In *the generalized form* of the disease with septic manifestations in the gastrointestinal tract, there is a slight fullness and small hemorrhages. There may be multiple metastatic ulcers in the internal organs. Diffuse and focal proliferation of reticuloendothelial cells is expressed. Salmonella is sown from pyemic abscesses, often in Association with other microbes (staphylococci, Proteus).

With *a typhoid-like course* of salmonellosis, the spleen and mesenteric lymph nodes are enlarged. In the intestine — swelling, fullness and hemorrhages in the mu-

cosa of the lower small intestine, especially in the group lymphatic follicles.

Clinical picture. The incubation period for salmonellosis is on average 12-24 hours. Sometimes it is shortened to 6 hours or extended to 2 days.

There are the following forms and variants of infection:

I. Gastrointestinal form: 1) gastritis; 2) gastroenteritis; 3) gastroenterocolitis.

P. Generalized form: 1) typhoid-like variant; 2) septicopyemic variant.

III. The bacterial excretion: 1) acute; 2) chronic; 3) transient.

The gastrointestinal form is most common. In this form, the disease can occur in the form of gastritis, gastroenteritis and gastroenterocolitis.

Gastritis (salmonellosis gastritis) develops rarely, is clinically accompanied by moderate symptoms of intoxication, pain in the epigastric region, nausea, repeated vomiting. Diarrhea in this variant of the course of the disease does not happen.

Gastroenteritis is the most common clinical variant of Salmonella infection. The onset of the disease is acute. Almost simultaneously, symptoms of intoxication and signs of damage to the gastrointestinal tract appear, which quickly, within a few hours, reach maximum development. Nausea and vomiting are noted in many patients. Vomiting is rarely single, often repeated, abundant, sometimes indomitable. Stool is liquid, plentiful, usually retains a fecal character, fetid, foamy, brown, dark green or yellow. Sometimes, the stool loses its fecal character and can resemble rice broth. The abdomen is usually moderately swollen, painful on palpation in the epigastrium, around the navel, in the ileocecal region (the so-called salmonellosis triangle), rumbling, "transfusion" in the area of loops of the small intestine can be detected.

Gastroenterocolitic variant of salmonellosis can begin as gastroenteritis, but then the symptom complex of colitis appears more and more clearly in the clinic. In this case, salmonellosis in its course resembles acute dysentery. The disease begins acutely, with a rise in body temperature and the appearance of other symptoms of intoxication. From the first days of the disease, the stool is frequent, liquid with an admixture of mucus and sometimes blood. There may be tenesmus and false urges. During rectoromanoscopy in such patients, inflammatory changes of various intensity are detected: catarrhal, catarrhal-hemorrhagic, catarrhal-erosive.

In the gastrointestinal form of salmonellosis, it is not possible to determine any characteristic type of temperature curve.

There is a constant, rarely remitting or intermittent type of fever. Sometimes the disease occurs at normal or subnormal temperatures. The pancreas is often involved in the pathological process in the gastrointestinal form of salmonellosis. Increases the activity of amylase in the blood and urine. Sometimes, there are clinical symptoms of pancreatitis. When salmonellosis occurs before liver damage, especially in the period of maximum taxinomie. In some patients, an increase in the liver is detected, sometimes subicteric sclera. The symptoms of lesion of the pancreas and liver are usually transient in nature.

The nervous system is often affected, which is caused by the action of endotoxin Salmonella, biologically active substances (such as histamine). Marked headache, dizziness, fainting. Damage to the autonomic nervous system is manifested by symptoms of hypermotor (spastic) dyskinesia of the stomach and intestines.

Disorders of cardiovascular activity develop in most patients: the Degree of its damage depends on the severity of General toxicosis. The frequency, filling and tension of the pulse change, and blood pressure decreases. In severe cases, collapse occurs, sometimes very quickly, in the first hours of the disease, even before the development of dehydration. As a result of intoxication and vascular insufficiency, dystrophic changes occur in the heart muscle. Heart tones are muffled or deaf, there is a systolic noise, arrhythmias may occur (most often extrasystole). Especially often these symptoms are expressed in the elderly, which is associated with a decrease in their adaptive ability of the cardiovascular system.

Toxic damage to the renal parenchyma is usually manifested by the syndrome of "infectious-toxic kidney": proteinuria, microhematuria, cylindruria. In very severe cases, in conditions of severe intoxication, a dropin cardiovascular activity, the development of collapse and significant electrolyte disorders, acute renal failure occurs.

The picture of peripheral blood in the gastrointestinal form of salmonellosis is different. With large fluid losses, blood thickening develops and erythrocytosis is possible. Occasionally, symptomatic thrombocytopenia develops. The number of white blood cells can be different — normal, reduced, but often increased, especially in severe salmonellosis. Leucocytosis is usually moderate, rarely exceeding 20-10/l. With great consistency, a shift of the leukocyte formula to the left is detected. ESR is normal or slightly increased. At the height of the disease, there may be violations of water-salt metabolism, leading to dehydration and demineralization of the body. Shifts in the acid-base state are detected, but they are detected only in the most severe cases.

Along the course of the gastrointestinal form of salmonellosis can be mild, moderate and severe. When *severe* intoxication is moderate, marked malaise, slight weakness, chilling. The temperature increases for a short time to supper strong numbers. Vomiting may not be present or it is a single event, abdominal pain is insignificant or absent, stool is mushy or liquid 1-3 times a day, quickly normalizes.

The medium-heavy course is accompanied by intoxication, the temperature rises to 39-40°C. Weakness, headache, dizziness, fainting, and cramps in the extremities are noted. Patients complain of abdominal pain, the localization of which depends on the degree of severity of gastritis, enteritis or colitis. Vomiting is excruciating, repeated, first with food eaten, then with bile or cloudy liquid. Stool up to 10 times a day, abundant, with a gastroenterocolitic variant — mucous. After 2-4 days, the patient's condition improves, abdominal pain disappears, the temperature and functions of the gastrointestinal tract normalize.

In *severe* cases, symptoms of intoxication reach maximum development in the first hours of the disease. The temperature quickly rises to 39-40°C, which is accompanied by chills. Fever is most often of a permanent nature with minor daily fluctuations; less often it takes a remitting character. In very severe cases, Hyper - or hypothermia develops, which is especially unfavorable in prognostic terms, since it indicates the occurrence of pronounced neurotoxicosis or acute vascular insufficiency. Simultaneously with the development of symptoms of intoxication, or somewhat later, there are strong cutting abdominal pain, excruciating nausea, then copious, repeated, sometimes indomitable vomiting. The chair 10-20 times a day, copious, watery,

foul-smelling, is sometimes resembles rice water. When involved in the colon process, the stool may be with mucus, rarely with blood. Symptoms of dehydration, demineralization and related acidosis develop. The patients look exhausted. The skin is pale, with a bluish tinge, dry, the face is drawn, the voice is weak, there are convulsions (from pulling pains in large muscles to total clonic ones), oliguria and anuria are possible. In this condition, reanimation detoxification therapy, rapid rehydration and remineralization are necessary.

Typhoid-like variant of salmonellosis usually begins with damage to the gastrointestinal tract, but can occur from the very beginning and without intestinal dysfunction. Clinically, it is very similar to typhoid fever and especially paratyphoid fever. The syndrome of intoxication is sharply expressed and accompanied by suppression of the Central nervous system functions. Patients complain of headache, sleep distortion (drowsiness during the day and insomnia at night), lethargy, sharp weakness. In severe cases, they become indifferent, adynamic, consciousness is clouded, delusions and hallucinatory syndrome are possible. Fever with a temperature reaching 39-40° C is often permanent. The duration of the febrile period ranges from 6-10 days to 3-4 weeks.

The skin of patients is usually pale, and a rash may appear. It is usually poorly visible and is represented by single small pale roseoles on the skin of the abdomen and torso. The pulse is often slowed down. Blood pressure is reduced. In some cases, there are phenomena from the upper respiratory tract, occasionally developing bronchitis and pneumonia.

By the end of the 1st week of the disease, there is an increase in the liver and spleen.

In the peripheral blood, leukopenia, aneosinophilia with a neutrophilic shift to the left is found, but there may also be moderate leukocytosis.

Septikopiemicheskoy variant of Salmonella from the beginning developed as Salmonella sepsis. But sometimes in a patient with a gastrointestinal form of salmonellosis, intestinal dysfunction stops, and intoxication increases. The disease loses its cyclical character, the temperature curve takes on an incorrect, remitting character, there are terrific chills and profuse sweat - salmonellosis takes a septic course. The clinical picture depends on the localization of metastatic purulent foci, which can occur in all organs. An increase in the liver and spleen is always detected. Typically a long-term severe course. Treatment presents significant difficulties, an adverse outcome is possible.

Bacterial release as a result of transferred salmonellosis can be acute or chronic. Acute bacterial discharge, in which the pathogen continues to be released for up to 3 months. After clinical recovery, it is much more common.

With *chronic* bacterial release, the pathogen is detected in the stool for more than 3 months. After a clinical recovery.

Transient bacterial release is diagnosed in cases where there is only one-two-fold Salmonella release with subsequent repeated negative results of bacteriological examination of feces and urine. In addition, the necessary diagnostic conditions are: the absence of any clinical manifestations of the disease at the time of examination and during the previous 3 months., as well as negative results of serological research per-

formed in dynamics.

Complications and outcomes of salmonellosis. Literature data and experience of clinical observations suggest that the widespread opinion about the successful course of the gastrointestinal form of salmonellosis is unfounded. Clinically severe generalized forms of the disease are usually accompanied by complications. A. F. Blugel et al. (1975) proposed a classification scheme, according to which the following groups of pathogenetic factors underlying the development of complications are distinguished:

1) excessive severity of the usual symptoms of the disease;
2) unusual secondary localization of infection foci;
3) superinfection, dysbacteriosis;
4) influence of salmonellosis on concomitant diseases.

According to A. F. Bluger et al. (1975), the most frequent complication in terms of excessive severity of the usual symptoms of salmonellosis is collapse, which appears in 1, -6% of cases. More often, the collapse develops in the first day of the disease at the height of clinical manifestations. The important role of endotoxinemia in the Genesis of collapse is evidenced by cases of rapid development of collapse in the first hours of the disease, even before dehydration.

When the collapse occurs, there is weakness, lethargy and adynamia, pallor or cyanosis of the skin, cold extremities, sweating. There is a short-term loss of consciousness, a decrease in blood pressure, a sharp tachycardia, a small, frequent pulse (120-160 beats per minute).

We do not share the opinion of A. F. Bluger et al. (1975) about the rarity of toxic-infected shock in salmonellosis. It should be emphasized that the very concept of toxico-infectious shock in the clinic of infectious diseases is not yet fully established. The main reason for the development of shock in infectious diseases, according to I. V. Davydovsky (1953), is not the damaging effect of microbes or toxins themselves, but a peculiar response of the body to them. According to the definition of R. p. Chetkareva, V. S. Enaleeva and P. S. Gurevich (1983), toxic-infectious shock should be understood as an extreme state of the body that occurs as a result of the action of toxic substances of pathogens, pathogenic immune complexes (sometimes other factors) on the organs and tissues of the body, accompanied by an acute violation of metabolism in them. A prerequisite for the development of shock is the presence of a large number of pathogens and their toxins, mass penetration of antigens into the blood. In its pathophysiological basis, shock is an inadequate perfusion of tissues and organs with oxygenated blood, resulting from bacteremia and toxinemia (Spink W., 1977).

It is generally accepted that the pathophysiology of toxic-infectious shock includes:

1) peripheral vascular collapse, leading to severe local lesions and culminating in particularly severe cases of systemic circulatory decompensation;
2) the development of disseminated intravascular coagulation with symptoms

of hemorrhagic diathesis;

3) inhibition of the immune response, up to its decompensation;

4) in many cases - immune inflammation (Chetkareva R. P. et al., 1983).

To these factors, according to the authors, we should add various secondary processes that are a consequence of the above-mentioned phenomena. These include, in particular, hyperstimulation of the pituitary-adrenal system with its subsequent decompensation.

According to Ferraris et al. (1974) and Klessler et al. (1973) (cited In: Lytkin M. I. et al., 1980), the development of endotoxin (toxic-infectious) shock causes the release of a large number of biologically active substances from the lymphoid cells, in particular prostaglandins.

The pathogenesis of toxic-infectious shock in salmonellosis is insufficiently studied. It is possible that the mechanisms of its development are the same in various infectious diseases, which is reflected in stereotypical hemodynamic disorders, the development of disseminated intravascular coagulation syndrome, disorders of the acid-base state, etc. Clinical observations on the development of toxicoinfectious shock patients with gastrointestinal form of salmonellosis confirm this assumption.

Along with severe hemodynamic disorders, the clinical picture was dominated in some cases by acute renal failure, in others by edema of the brain and lungs, and in others by hemorrhagic syndrome.

Clinical signs of brain edema are quite difficult to detect against the background of a severe course of salmonellosis. According To L. Bakay and D. Li (1969), acute brain edema is characterized by intense headache, impaired consciousness, repeated vomiting, motor restlessness, convulsions, disorders of the cardiovascular system, and increased intracranial pressure. It should be borne in mind that the violation of consciousness can be of various degrees - from a slight dazing to a deep coma. Disorientation and psychomotor agitation, tonic and clonic convulsions are characteristic. The pupils are initially narrow, then dilated, anisocoria is noted. Tendon reflexes are elevated, meningeal syndrome is clearly expressed, sometimes there are pyramid signs. Movement of the eyeballs is erratic. Gradually, the reflexes fade, mydriasis appears, signs indicating the insertion of the medulla oblongata into the large occipital foramen. Cardiovascular disorders are characterized by an initial increase and then a sharp drop in blood pressure, as well as bradycardia. Te's breathing **is** sharp and deep, sometimes Chane - Stokes type. In a significant number of cases of brain edema, hyperthermia is expressed, which makes the patient's condition heavier and contributes to the suppression of vital functions (Brodov L. E., 1970). Hyperthermia is the first phase of neurotoxicosis, followed by a hypoxic encephalopathy phase, followed by a terminal phase. At the same time, there is a pronounced metabolic acidosis, respiratory alkalosis, hypocapnia, hypoxia of the brain and myocardium (Bulychev V. V. et al., 1975; Blair E. Et al., 1969).

Pulmonary edema in severe salmonellosis is usually diagnosed postmortem, with a pathoanatomic autopsy. According to J. Riordan and G. Walters (1968), M. S. Covern (1972), in toxic-infectious shock, pulmonary edema may develop. It should be borne in mind that with salmonellosis, which ends in death, pulmonary edema does not always develop. Pulmonary edema occurs not only with rehydration therapy,

but also in cases when the fluid was not injected intravenously or was introduced in a small volume. So, J. Riordan and G. Walters reported 7 cases of bacterial shock caused by an infection unrelated to respiratory pathology and complicated by pulmonary edema. In no case were the authors able to explain its development by hyperhydration. H. S. Nugmanova (1971) noted the polyethologicity and polypatogenicity of pulmonary edema. O. G. Solomatina (1973) emphasized the importance of violations of water - electrolyte metabolism, acid-base state and increased excitability of the sympathetic Department of the autonomic nervous system for the development of pulmonary edema. The effect of all these factors is combined, but the significance of each of them is not the same in individual cases. Violations aerogematiceski barrier and concerning histophysiologic features have important pathogenetic significance. According to V. K. Kulagin (1975), the development of pulmonary edema in toxic-infectious shock is associated with the action of ultra-extreme factors and a clear lack of mechanisms of compensation and adaptation, which determine the regulation of homeostasis. The decisive factors in the development of pulmonary edema include multiple thrombosis in the pulmonary capillaries, damage to the vascular endothelium due to hemodynamic disorders, hypoxia, acidosis (D. Alexandrov And Wishnatzki-V. Aleksandrov, 1975). J. Riordan and G. Walters (1968) noted the role of direct exposure to gram-negative bacteria and their toxins responsible for the development of increasing vasoconstriction and disseminated intravascular coagulation. V. McGovern (1972) emphasized the role of focal necrosis in the lungs associated with pulmonary edema. E ^w of cases, the development of pulmonary edema in toxicoinfective shock was associated with the appearance of brain edema (Bunin K. V. et al., 1974). The role of hypoxia in the development of pulmonary edema was indicated by G. Moss and S. Staunton (1972), and on the role of neuro-reflex influences - G. Kessler et al. (1968).

The clinic of pulmonary edema is described in numerous works. However, in patients with salmonellosis at the height of the disease, it is quite difficult to diagnose pulmonary edema due to the bright severity of the underlying disease, complicated by shock, and the paucity of clinical manifestations of pulmonary edema. Auscultative changes in the lungs were detected quite late, the appearance of typical pink sputum was the exception rather than the rule (Alexandrov D., Vyshnacka-Alexandrov V., 1975).

With a severe course of salmonellosis, complicated by shock, acute renal failure can be observed. Shock causes a violation of systemic and regional, including renal, hemodynamics, characterized by a prolonged spasm of the arterioles and arteriovenous shunting. Already in the first 2-3 hours, anoxia negatively affects the function of the kidneys. With a longer anoxia, necrobiotic changes in the renal tubular apparatus occur. According to O. S. Bukhantseva (1976), at the height of the disease, oliguria was detected in 77.8% of patients, a decrease in glomerular filtration rate - in 68.4%, an increase in tubular reabsorption of water - in 87.7%, an increase in azotemia - in 57.4 %. V. N. Nikiforov et al. (1973) radiosotope renography revealed abnormalities of the absorption and excretory functions of the kidneys due to intoxication. A relationship was found between the amount of renal plasma flow and glomerular filtration, on the one hand, and the severe course of salmonellosis - on the other. T. I.

Dmitrovskaya (1977) observed the development of uremia in severe salmonellosis. In patients who died 7 to 8 hours after the onset of the disease, the section revealed parenchymal dystrophy of the kidneys, fullness of the stroma and glomerular capillaries with focal hemorrhages. In cases of fatal outcomes occurring 24 hours after the onset of the disease, necrobiotic changes in the proximal tubules were observed. The development of acute renal failure is facilitated by dehydration, hyponatraemia, hypokalemia, and hypochloremia that often occur during severe salmonellosis. Thus, kidney damage is caused by a severe course of salmonellosis, complicated by shock.

The analysis of the medical history of 32 patients hospitalized for salmonellosis was carried out. All observations were complicated by the development of toxic-infectious shock, and in 30 cases there was a fatal outcome.

The diagnosis of salmonellosis was diagnosed during life in all patients and confirmed in 30 cases by pathoanatomic autopsy. Edema of the brain during life was established in 12 patients, and in pathoanatomic research - in all 30 deceased, pulmonary edema-in 3 of 12 patients, respectively. Oliguria was observed in all 32 patients.

Among the 32 patients, there were 23 men and 9 women. On the 1st day of the disease, 30 patients were hospitalized, on the 2-3-th day-9, 3 people were hospitalized after 3 days. Upon admission to the hospital, 28 patients were in a serious condition, 4 were in moderate condition.Normal body temperature was in 10 patients, 37-39 C - in 14, over 39 C - in 8 patients. All patients had chills, repeated vomiting, frequent stools, and half of the patients were the color of "meat slops", sometimes with streaks of mucus. Abdominal pain occurred in 25 patients, cyanosis-in 14, acrocyanosis-in 18. Low blood pressure was diagnosed in 26 patients, tachycardia in 20, bradycardia - 9. Dyspnea was observed in 11 patients, breathing such as Cheyne - Stokes -4, cough with difficult expectoration - in 2, crackles in the lungs - 3. 26 patients had progressive increase of headache. In 21 patients, despite rehydration therapy, vomiting did not stop. Disorders of consciousness in varying degrees, from mild dazing to deep coma, were found in 26 patients. Visual hallucinations occurred in 6 patients, auditory-in 3. Hyperthermia developed in 6 patients, tonic or clonic seizures-in 13. Motor restlessness was observed in 6 patients, anisocoria - in 3, mydriasis-in 3, miosis-in 2, smoothness of nasolabial folds - in 1, reduction of tendon reflexes-in 1, pathological reflexes - in 2, severe meningeal syndrome - in 3 people. A progressive drop in blood pressure, preceded by a rise that lasted several hours, was observed in 18 patients. Electrocardiograms recorded in 14 patients revealed diffuse dystrophic changes in the myocardium. Oliguria took place in 16 patients, anuria - in 16 patients.

The development of edema of the brain and lungs in salmonellosis was not associated with the amount of injected fluid, nor with the speed of its administration, nor with the qualitative composition of infusion solutions.

Intravenous administration of solutions was carried out at a rate of 20 to 100 drops per 1 min. Ten patients received up to 20 mg/kg of fluid per day, 14 patients - 20 - 50 ml/kg, 4 patients - 50 - 80 ml/kg and only 2 patients-90 ml / kg. The total number of solutions ranged from 500 to 10,400 ml. Two patients did not receive infusion solutions, but they also developed brain edema and pulmonary edema. It should be noted that brain edema and pulmonary edema developed against the background of both massive dehydration therapy (diuretics, glucocorticosteroids, plasma substi-

tutes, oxygen) and targeted correction of water-electrolyte metabolism and acid-base state.

A pathoanatomic study revealed brain edema in 30 cases, and in 7 cases brain edema was complicated by the insertion of the tonsils of the cerebellum into the large occipital opening.

During the morphological study of kidneys (30 deceased), uneven blood filling of the renal tissue was noted. Cortical ischemia was observed in 24 deceased patients, and fullness - in 5. Fullness of the cerebral Substance and at the border of the cerebral and cortical layers took place in 30 cases. In 6 deceased patients, there was an edema of interstitial tissue with a sharp compression of the lumen of the tubules. These hemodynamic disorders were considered as a reflection of General hemodynamic shifts in shock. The epithelium of convoluted tubules in both the proximal and distal parts was in a state of granular dystrophy (30 cases). Dystrophic changes in the epithelium of straight tubules were observed only in one deceased person. In 2 cases there was rupture of the basal membrane of the seminiferous tubules. Necrobiotic changes of the proximal convoluted tubules was noted in 8 cases, distal - from 5 people, direct channel - 1. Regeneration of tubular epithelium was observed in only 2 deaths. Hyaline cylinders were found in the lumen of the tubules in 7 cases. Dystrophic and necrobiotic changes in the convoluted tubules were regarded as manifestations of kidney damage caused by circulatory-ischemic and nephrotoxic effects.

Symptoms of edema of the brain and lungs constantly prevailed in the clinical picture of shock over the manifestations of acute renal failure.

Prognosis for gastrointestinal form and typhoid-like

Septikopiemicheskoy variant of salmonellosis from the beginning developed as Salmonella sepsis. But sometimes in a patient with a gastrointestinal form of salmonellosis, intestinal dysfunction stops, and intoxication increases. The disease loses its cyclical character, the temperature curve takesan unfavorable, remitting character, there are terrific chills and profuse sweat — salmonellosis takes a septic course. The clinical picture depends on the localization of metastatic purulent foci, which can occur in all organs. An increase in the liver and spleen is always detected. Typically a long-term severe course. Treatment presents significant difficulties, an adverse outcome is possible.

Bacterial release as a result of transferred salmonellosis can be acute or chronic. *Acute* bacterial discharge, in which the pathogen continues to be released up to 3 months after clinical recovery, is much more common.

With *chronic* bacterial release, the pathogenis found in the stool for more than 3 months after clinical recovery.

Transient bacterial release is diagnosed in cases where there is only one-two-fold Salmonella release with subsequent repeated negative results of bacteriological examination of feces and urine. In addition, the necessary diagnostic conditions are: the absence of any clinical manifestations of the disease at the time of examination and during the previous 3 months, as well as negative results of serological research performed in dynamics.

Complications of salmonellosis are numerous and varied. In the gastrointestinal form of the disease, the development of vascular collapse, hypovolemic shock, acute

heart and kidney failure is possible. Salmonellosis patients are prone to septic complications: purulent arthritis, osteomyelitis, endocarditis, abscess of the brain, spleen, liver and kidneys,meningitis, peritonitis, and appendicitis. In addition, there may be pneumonia, ascending urinary tract infection (cystitis, pyelitis), infectious and toxic shock. In all clinical forms of the disease, the development of relapses is possible.

The prognosis for gastrointestinal form and typhoid-like variant of salmonellosis is favorable, especially in cases of early diagnosis and proper treatment. The prognosis for septicemia is always serious. The mortality rate is 0.2—0.3 percent.

Diagnostics. Diagnosis of salmonellosis is carried out on the basis of epidemiological, clinical and laboratory data. Laboratory examination of patients is an important link in the diagnosis, especially if we take into account the polymorphism of clinical manifestations. Apply bacteriological and serological methods of research. Bacteriological examination is performed on vomit, gastric lavage, stool, duodenal contents, blood, and urine. Material from the patient should be taken as early as possible and before the start of treatment.

In serological studies (7-8-th day of the disease), the reaction of agglutination (RA) and indirect hemagglutination (RIGA) is used.

RA is considered positive with a serum dilution of at least 1: 200. Especially important diagnostic value is the increase in the titer of antibodies in the dynamics of the disease. Rnga is more sensitive and gives positive results on the 5th day of the disease. For the diagnostic titer, a serum dilution of 1:200 is taken.

For group diseases with salmonellosis, rapid diagnostic methods are used: MFA, rnga with antibody diagnosticums, etc.

Differential diagnosis of salmonellosis depends on the clinical form of the disease. Most often, the gastrointestinal form has to be differentiated from other acute intestinal infections — dysentery, food toxicoinfections, escherichiosis, cholera. It is often necessary to differentiate this form from acute surgical diseases — acute appendicitis, pancreatitis, cholecystitis, mesenteric vascular thrombosis and acute gynecological pathology — ectopic pregnancy and adnexitis, from therapeutic pathology - from myocardial infarction, exacerbation of chronic gastritis, enterocolitis, peptic ulcer disease. There are also difficulties in the differential diagnosis of the gastrointestinal form of salmonellosis and poisoning with inorganic poisons, pesticides, fungi, and some plants.

The generalized form of salmonellosis should be differentiated from other bacteriemic infections, sepsis of various nature, pneumonia, malaria, acute pyelonephritis, tuberculosis, and lymphogranulomatosis.

Treatment. The complexity of pathogenetic mechanisms in salmonellosis, the variety of clinical forms of the disease dictate the need for an individual approach to treatment.

Currently, there are no sufficiently effective chemotherapeutic drugs (including antibiotics) for the treatment of the gastrointestinal form of Salmonella infection. In this form of the disease, the main methods of pathogenetic therapy are: The main directions of pathogenetic therapy of salmonellosis are: 1) detoxification; 2) normalization of water and electrolyte metabolism; 3) fighting hypoxemia, metabolic acidosis; 4) maintaining the physiological level of hemodynamics, as well as the functions of

the cardiovascular system and kidneys.

All patients with a gastrointestinal form of salmonellosis in the first hours of the disease are shown gastric lavage.

Patients with a mild course of the disease do not need a wide range of therapeutic measures. It should be limited to prescribing them a diet (#4) and a plentiful drink.

For oral rehydration, glucose-electrolyte solutions can be used (for example, "Oral": sodium chloride 3.5 g, potassium chloride 1.5 g, sodium bicarbonate 2.5 g, glucose 20 g per 1 liter of drinking water). They are given to drink in small portions in an amount corresponding to the loss of fluid.

With an average severe course of the gastrointestinal form of salmonellosis, but without pronounced hemodynamic disorders and rare vomiting, oral rehydration is also performed. However, with increasing dehydration, pronounced violations of hemodynamics, frequent (indomitable) vomiting, polyionic solutions are administered intravenously. After recovering the initial fluid loss and absence of vomiting, oral rehydration can be continued.

In severe cases of the disease, treatment is carried out in the mode of intensive therapy and resuscitation. For the implementation of the above principles of pathogenetic therapy, intravenous administration of polyionic solutions is mandatory. Their volume depends on the amount of liquid lost with feces, vomit and urine, as well as on the degree of intoxication, amounting to a day from 4 to 8 liters. In infusion therapy solutions "Trisol", "Acesol", "Latosol", "Kvartasol", "Chlosol".

In the development of dehydration shock, reanimationtherapy is carried out, approaching that which is carried out in severe forms of cholera.

With the development of infectious-toxic shock, in addition to multi-injected colloidal solutions (gemodez, reopoligljukin) and corticosteroids.

In the complex of pathogenetic measures, especially with a prolonged course of the disease, stimulating therapy is of great importance. Multivitamins, non-steroidal anabolics (methyluracil, potassium orotate) increase the body's resistance to infection, promote tissue regeneration, and stimulate the production of immunity.

Antibacterial therapy, including antibiotics, sulfonamides and other chemotherapy drugs, is ineffective..

One of the main reasons for this is mainly the intracellular location of microbes, characteristic of the gastrointestinal form of salmonellosis.

In the generalized form, along with pathogenetic therapy, etiotropic treatment, including antibiotics, is indicated. The course of treatment is prescribed individually, depending on the form and severity of the disease. Levomycetin, ampicillin, kanamycin, gentamycin, and ceporin are used.

An unsolved problem is the treatment of patients with prolonged Salmonella discharge. As a rule, the strains of the pathogen that caused the bacterial carrier are resistant to many antibiotics. In some cases, it is possible to get an effect in the treatment of patients with ampicillin or negram, especially in combination with injections of prodigiosan or other bacterial lipopolysaccharide (3-5 injections per course).

In the complex treatment of patients with salmonellosis, polyvalent salmonellosis bacteriophage is also used.

Special attention in the treatment of patients with salmonellosis should be paid to

concomitant pathology, as well as the rehabilitation of chronic foci of infection.

Prevention. Prevention includes veterinary-sanitary, sanitary-hygienic and anti-epidemic measures. Veterinary and sanitary measures are aimed at preventing the spread of Salmonella among domestic mammals and birds, as well as at organizing a sanitary regime in meat processing plants and dairy enterprises. The purpose of sanitary and hygienic measures is to prevent Salmonella contamination of food products during their processing, transportation and sale. Proper cooking and sufficient heat treatment of food products is of great importance in the fight against Salmonella. Anti-epidemic measures are aimed at preventing the spread of the disease in the collective. In the event of sporadic diseases and epidemic outbreaks, it is necessary to identify ways of transmission of infection and submit to bacteriological research questionable food products, vomit, washing water, blood and feces of patients in the foci of the disease, the current and final disinfection is carried out. Patients are hospitalized for clinical and epidemiological indications. Patients are discharged after clinical recovery and negative results of control bacteriological studies of bowel movements.

An important role in the prevention of Salmonella disease is played by the industrialization of the food industry, the mechanization and automation of basic technological processes, and the improvement of the food trading system.

LITERATURE

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TASKS

1. Sick T., 39 years old, riverman, kapian-mentor. He became acutely ill 6 hours aftereating a jelly that had been stored for more than 5 days. I felt a pronounced weakness in the entire body, chills, cutting pains in the subglotalny area, nausea, the temperature increased-first 37.8°, and then 38.5°. Repeated vomiting began, followed by diarrhea. Vomitingbegan with the food she had eaten, and then bile, painful with sharp vertigo. Stool every hour, liquid, abundant. Delivered by ambulance 10 hours after the onset of the disease. Withstate of moderate severity. Vomiting and diarrhea continue. Complains of spilled abdominalpain, headaches, dizziness, and General weakness. The face is hyperemic, cyanosis of the lips, fingers; temperature 39.5°; pulse 120 V min., weak filling, rhythmic; blood PRESSURE-90/80 mm Hg., art.; dyspnea-28 per minute. In the lungs, catarrhal changes are not noted. The tongue is thickly coated with a white coating, dry. The abdomen is somewhat retracted, and on palpation there is spilled soreness, distincturination. The sigmoid colon is not spasmodically altered. Faeces are liquid, plentiful, fetid with "greens", there are no lumps of mucus and streaks of blood. There were no clinically pronounced signsof dehydration (the skin turgor was preserved, the voice was not changed). However, a bloodtest revealed a thickening (4.9 million red blood cells, HB - 98 units). Leukocytosis (9600) with neutrophilosis (75%) and a pronounced left shift (p - 37%, y-7%), ESR - 15 mm/h. Koprivshtitsa - a singlestar leukocytes. A diagnosis of food toxicoinfection was made. S are selected from the stool S.typhimurium.

The final diagnosis is acute salmonellosis gastroenteritis. After treatment, a critical drop in temperature and rapid recovery.

2. Sick R., 63 years old, watchman. Delivered together with his wife, children and grandchildren. They got sick thenext morning after eating hot smoked mackerel. Incubation is about 12hours. General condition of admission of moderate severity. Complains of abdominal pain, fever, chills, cramps in the calf muscles, diarrhea. There was no nausea or vomiting, t-38.5° (at home 38.0°). Pulse-88 V min., satisfactory filling, respiration-16 V min., blood PRESSURE-110/70 mm Hg. St. heart Tones are muted. The tongue is moist, slightly overlaid. The stomach is retracted. If Palacio - rumbling, diffuse pain along the intestines. The sigmoid colon is not spasmodically altered. Stool is frequent, liquid, abundant, fetid with greens, lumps of mucus and streaks of blood are not found.From fish sediments and from feces, S are distinguished.newport 1:640. The diagnosis is acute salmonellosis enteritis. After receiving assistance, a rapid recovery occurred.

3. The patient is 62 years old, a locksmith. Delivered in a serious condition with a diagnosis of food toxicoinfection6 hours after the onset of the disease. He was taken ill at dawn, and the previous evening he had been ona call. In the future, the hospital began to receive other people who were there. The condition is serious. Profuse diarrhea. The chair is literally continuous, without any attempts. Abundant, liquid, watery, odorless. Repeated vomiting is also plentiful. The temperature was

36.3°, at home it was 37.6°.Pulse 96 V min., weak filling, blood PRESSURE-70/40 mm Hg. The tones of the heart are deaf. Shortness of breath 0-20 breaths per minute. Spilled cyanosis. Clear signs of dehydration: turgor of the skin is lowered, the voice is hoarse, difficult to distinguish, convulsive contractions of the calf and other muscle groups. Myseparation has stopped. The tongue is covered with a white coating, dry. The stomach is drawn in, soft, a spilled rumble. Specific gravity of plasma-1030, protein content-8.8 g%. Uncompensatedmetabolic acidosis (pH-7.29; pCO2-38 mm Hg; BB-48 m-EQ/l, base deficiency VE-8.8 m-EQ/l). Leucocytosis (9700), erythrocytosis (5.2 million), delayed ESR (2mm / h). A significant increase in blood viscosity (with a minimum speed gradient of 79.3, with a maximum of 26.9). S are selected from the stool S.heidelberg. An epidemiological investigation found that the source ofcontamination was poor-quality meat that had been stored for more than 5 days. The diagnosis is acute salmonellosis gastroenteritis, severe course, with the development of hypovolemic shock. The patient was treated in the intensive care unit, after which a rapid convalescence occurred.

4. Ill N., 31, programmer. It was received in connection with a family outbreak of salmonellosis, which occurred as a result of eating jelly, from the remains of which S were subsequently isolated S.mission. I got sick first, 4 hours after eating. The beginning of the disease is stormy. There was weakness, dizziness, headache, abdominal pain, nausea, vomiting, diarrhea. A little later, I felt a fever, chills, increased t - 38°. The condition is very serious upon admission. The patientis agitated, tossing about in bed. Her face was pale despite her high fever, and there was a cold sweat on her brow. Pulse is frequent, filiform, not counted, blood PRESSURE 70/50 mm Hg. art., later ceasedto be determined. The heart tones are muted. Shortness of breath 28 in min., spilled cyanosis. Convulsive contractions of the calf muscles. The turgor of the skin is preserved, the voice is resonant. The abdomen is soft, with palpation rumbling and spilled soreness along the course of the intestine. UD. weight of plasma-1025, protein content-6.3 g%. Compensated metabolic acidosis. Blood viscosity at a fixed rate gradient: minimum-60.2 SP, maximum-13.4 SP. Stool is liquid, fetid, green, without lumps, mucus and blood (s are isolated from the feces S.mission). The urination stopped. He was diagnosedwith acute salmonellosis gastroenteritis of severe course with the development of infectious and toxic shock of the P-sh stage. The patient is placed in the intensive care unit. Immediately carried out gastric lavage with a soda solution to clean flushing waters. Intravenous hemodesis infusion (400ml) was started, followed by Trisol solution (500ml) and 5% glucose (500ml). 250 mg of hydrocortisone was added to the glucose-saline solution. At the same time, 125 mg of the drug was administered *intravenously.* Assigned to repeated injections of sulfokamfokain and ephedrine. After 2 hours from thestart of intensive care, there was a noticeable improvement. The patient's face turned pink,-and the cold sweat on her forehead disappeared. The pulse filling increased noticeably, it could already be counted (120-136 in min.). blood Pressure increased-90/60 mm Hg. cyanosis Significantly decreased. The patient became calmer,the cramps in the calf muscles stopped, and there was no more vomiting. Indicators of acid-base balance have normalized, t-39°. The next day, urination was restored.Further flow is favorable, on the 3rd day the temperature normalized. At the same time, diarrhea

stopped.

5. Patient P., 64 years old, watchman. Connects the disease with an egg eaten the day before. At onetime, my wife fell ill. Fell ill at dawn (9 hours after eating). Vomiting and diarrhea appeared. Stool is very frequent, abundant, liquid, without attempts. Vomiting was threefold, very weak, t-37.8°C.Delivered 6 hours after the onset of the disease. The condition is serious. Consciousness is preserved. The voice is hoarse and hard to distinguish. Spilled cyanosis. Extremities are cold, t-35,6°. Shortness of breath or out of breath. in min. Painful cramps in the calf muscles. Pulse is frequent, weak filling, not counted. AD cannot be measured. The tones of the heart are deaf. The urination stopped. The profuse diarrhoea. The allocation of copious, liquid, whitish, with cereals (like rice-water), and odorless. Repeated copious vomiting. Obvious signs of dehydration. Turgor of the skin is sharply reduced. The tongue is dry, covered. His features sharpened. The abdomen is retracted, and on palpation there is a spilled rumbling sound. UD. weight of plasma is increased-1030, hyperproteinemia (8.6 g%). Compensated metabolic acidosis. Blood viscosity at a fixed speed gradient: minimum-73.8 SP, maximum-24.4 SP. The diagnosis was made of a severe form of food toxicoinfection with the development of hypovolemic shock IIIof stage III (Later s were isolated from the feces S.heidelberg). The patient washospitalized in the intensive care unit. Copious gastric lavage was performed and at the same time jet intravenous administration of heated Acesol saline solution was started. Within 2 hours, the patient was injected with 7 liters of solution (the initial weight of the patient is 70 kg). The condition has improved,the cyanosis has decreased, the pulse has become fuller-116 b min, blood PRESSURE 110/60 mm Hg, t-36.4°. Vomiting has stopped, the specific weight of plasma is 1025. Further infusion of the solution was carried out intravenously,according to continuing diarrhea. Urination was restored after 36 hours,and diarrhea stopped on the 4th day of the disease. By this time, the indicators of acid-base balance were normalized. Reconvalescence is smooth. He was discharged after 3 weeks in a satisfactory condition.