FOOD POISONING

Foodborne diseases - acute, short-term diseases caused by opportunistic bacteria capable of producing exotoxins outside the human body (in food), and occurring with symptoms of upper gastrointestinal tract (gastritis, gastroenteritis) and impaired water salt metabolism.

Clinically, these diseases are characterized by a sudden onset, a combination of intoxication syndromes,
gastroenteritis and frequent development of dehydration.
The diagnosis of FP is collective and combines a number of
etiologically different, but pathogenetically and clinically similar diseases.

Historical information

Even in ancient times it was known that the use of food can cause diseases accompanied by vomiting and diarrhea. It was assumed that in this state based on the body are incompatible food, natural pathogenic properties or the content of toxic substances. In the last century it was noticed that some "food poisoning" associated with the consumption of meat of sick animals. Later it was found that these diseases can cause opportunistic infectious bacteria and their toxins, food animal and non-animal origin. To date, there is a large amount of information about the role of conditionally pathogenic microflora and exotoxins produced by it in the development of the FP. These data suggest that, unlike other infectious diseases for the emergence FP prerequisite is not only the presence of microorganisms in food products, but, more importantly, build them a sufficient dose exotoxin produced by bacteria.

Bacterial food poisoning divided into poisoning and toxicosis (intoxication). The latter include diseases caused by Cl. botulinum and enterotoxigenic strains St. aureus. Because of the pronounced differences in the mechanism of action of the toxin (neyroplegii effect) allocated Cl. botulinum, and the identity of the clinical picture of botulism is described separately. Staphylococcal same intoxication, similar in the clinical picture of foodborne disease are presented in this section.

Etiology

For foodborne pathogens include many kinds of opportunistic bacteria capable of producing exotoxins during their livelihoods outside the human body in a variety of foods.

Among exotoxins - enterotoxins (heat-labile and heat-stable), enhances the secretion of fluids and salts in the lumen of the stomach and intestines, and cytotoxin damaging membranes of epithelial cells and disrupts their protein synthesis process.

Etiology

EXOTOXIN

C.Perfringens
Proteus vulgaris
P.Mirabilis
Bacilus cereus

ENDOXIN

Klebisella
Enterobacter
Citrobacter
Serratia
Pseudomonas
Aeromonas
Edwardsiella
Vibrio

CYTOTOXIN

Klebisella pneumoniae
Enterobactercloacae
Aeromonas hydrophila
C. Perffringens type C
C. Difficile
Vibrio parahaemolyticus
St. aureus

The most common pathogens FP capable of producing enterotoxins are Clostridium perfringens, Proteus vulgaris, Proteus mirabilis, Bacillus cerreus.

Enterotoxins are also formed FP pathogens belonging to the genus Klebsiella, Enterobacter, Citrobacter, Serratia, Pseudomonas, Aeromonas, Edwardsiella, Vibrio.

In the majority of pathogens FP enterotoxins are thermolabile.

Thermostability properties different enterotoxin St. aureus. It is not inactivated by boiling up to 30 minutes (according to some estimates, up to 2 hours), and retains the ability in the absence of the bacteria itself cause a clinical picture of the disease.

Among the pathogens FP ability to produce cytotoxin have Clebsiella pneumoniae, Enterobacter cloacae, Aeromonas hidrophilia, Clostridium perfringens type G and Clostridium difficile, Vibrio parahaemolyticus, St. aureus and some other microorganisms.

It should be noted that not each of the above bacteria strain capable of forming exotoxins. Therefore, the use of food containing a large number of bacteria, not lead by itself to the development of FP. The disease occurs only when infected with toxin-eating strains of bacteria.

Pathogens are resistant to the action of physical and chemical environmental factors;

capable of reproduction both in the conditions of a living organism and outside it, for example, in food products (in a wide temperature range).

Epidemiology

Sources of pathogens can be people and animals (patients, carriers), as well as environmental objects (soil, water).

According to the ecological and epidemiological classification, FP caused by opportunistic microflora are classified as anthroponoses (staphylococcosis, enterococcosis) and sapronosis - water (aeromonosis, plesiomonosis, parahemolytic and albinolytic infections, edwardsiellosis) and soil (cereus infection, clostridium, pseudomonosis, klebsiellosis). , proteosis, morganellosis, enterobacteriosis, erviniosis, hafnia and providence infections).

However, in some cases, when the sources are people working in the food industry, and suffering from various pustular skin diseases (pyoderma, felon, sores, etc..), or angina, rhinopharyngitis, laringotraheobronhitami, pneumonia, their identification is not only necessary, but also possible. Among the zoonotic FP sources can be identified mastitis diseased animals - cows, goats, sheep, etc.

The pathogen transmission mechanism is fecal-oral; transmission route - foodborne.
Transmission factors are varied.
Usually, the disease occurs after eating food contaminated with microorganisms brought in by dirty hands during the cooking process; undisinfected water; finished products (in case of violation of the rules for storage and sale in conditions conducive to the reproduction of pathogens and the accumulation of their toxins).
***Proteus and clostridia* are capable of active reproduction in protein products (jelly, jellied dishes), *B. cereus* - in vegetable soups, meat and fish products. In milk, mashed potatoes, cutlets, enterococci rapidly accumulate.**

Halophilic and parahemolytic vibrios **that survive in marine sediment infect many marine fish and molluscs.**

Staphylococcus **gets into confectionery, dairy products, meat, vegetable and fish dishes from people with pyoderma, tonsillitis, chronic tonsillitis, respiratory diseases, periodontal disease and working in catering establishments. Zoonotic source of staphylococcus - animals with mastitis.**

Foods are to bacteria medium.

Staphylococcal intoxication	→	is most often associated with the consumption of infected milk and dairy products, confectionery creams, meat, fish and vegetable dishes.
Proteus and Clostridium	→	breed well in protein foods (meat, fish, including canned, sausage, milk).
B. cerreus	→	very unpretentious, multiplies rapidly in a variety of foods: vegetable salads and soups, puddings, meat and fish dishes.

Epidemiology

Practice has shown that, despite the diverse etiology of FP, the food factor is crucial in maintaining a high level of morbidity.

FP are "dirty food" diseases.

- **Outbreaks of FP have a group, explosive nature, when most people (up to 90-100%) who have consumed an infected product fall ill within a short time. Frequent family outbreaks, group diseases of passengers of ships, tourists, members of children's and adult organized groups.**
- **Diseases are most often recorded in the warm season.**
- **The natural susceptibility of people is high. More susceptible are newborns, patients after surgical interventions, long-term antibiotics, patients suffering from impaired gastric secretion.**

❑ **Immunity after past illnesses is short-term and species-specific.**

Pathogenesis

With food toxic infections (and intoxications) to the moment food enters the stomach in it, except bacteria, contains a significant amount exotoxin.
This causes the development of the shortest incubation period in infectious pathology.
From the moment of exposure to toxins on the mucosa the lining of the stomach before the development of clinical symptoms
in some cases, no more than 30 minutes pass (usually 2-6 hours).

Pathogenesis and clinical picture FP largely depend on the type and dose of exotoxin A, as well as other toxic bacterial substances contained in the food product.

PATHOGENESIS

For the occurrence of the disease is important:

- **infectious dose - not less than 10^5-10^6 microbs in 1 g of substrate;**
- **virulence and toxigenicity of strains of microorganisms**
- **a sufficient period of time for the accumulation of the pathogen and its toxins in the food product.**

Of primary importance is intoxication with bacterial exo- and endotoxins of pathogens contained in the product.

Enterotoxins (thermolabile and thermostable)

contacting epithelial cells of the stomach and intestine

influence on enzymatic systems of epithelial cells without causing morphological changes of the bodies.

Among the activated enzymes enterotoxins - guaniltsiklaza adenylcyclase and enhancing the formation of the mucosal cells of biologically active substances - cAMP and cGMP.

Under the influence of toxins , also increases the rate of formation

of prostaglandins

histamine

other gastrointestinal hormones.

This leads

to increased secretion of

salts and liquidsin the lumen of

the stomach

and

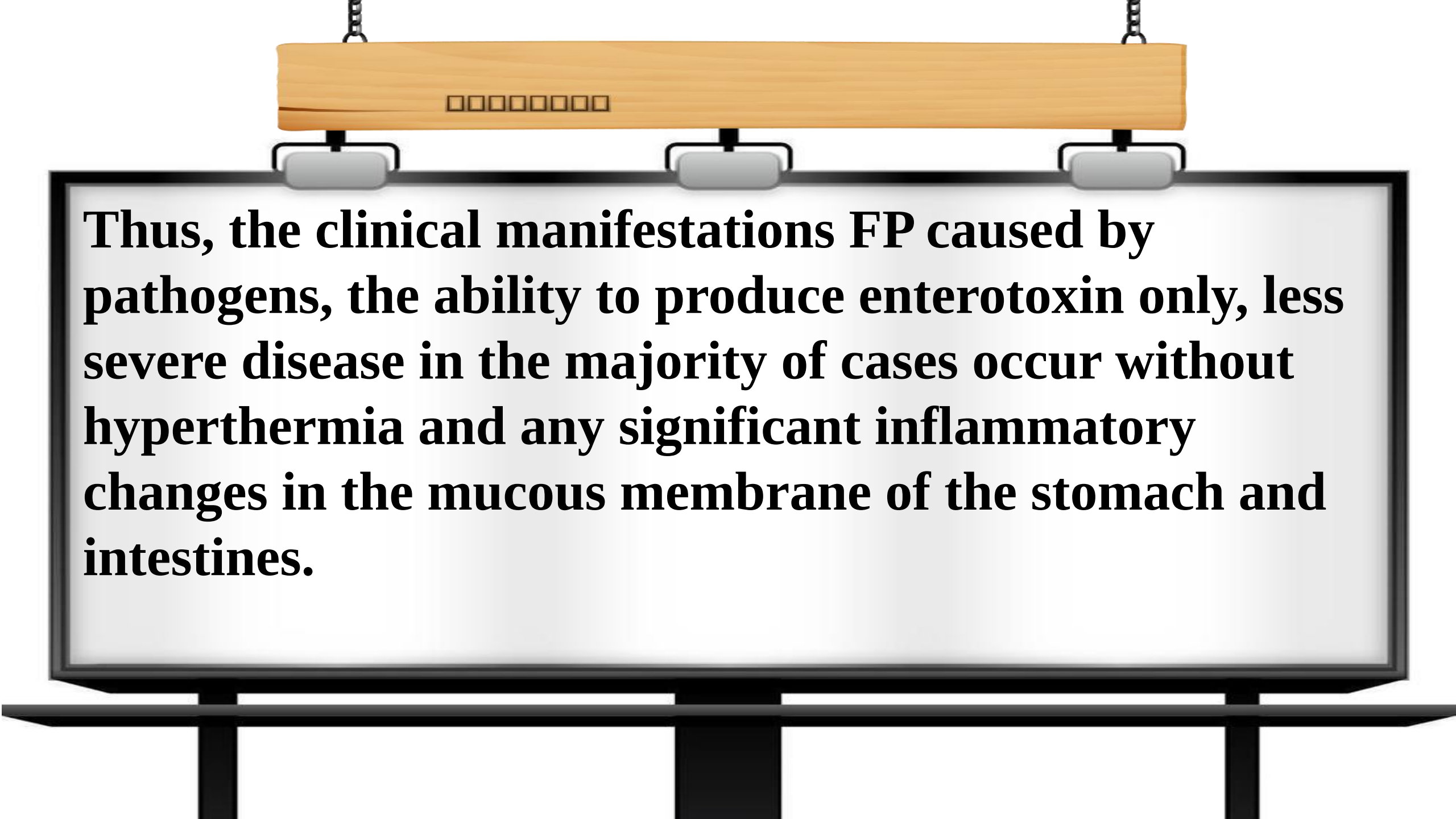
intestines

and the development of

diarrhea

and

vomiting


Thus, the clinical manifestations FP caused by pathogens, the ability to produce enterotoxin only, less severe disease in the majority of cases occur without hyperthermia and any significant inflammatory changes in the mucous membrane of the stomach and intestines.

Those cases where food is accumulated and enterotoxin and cytotoxin, occur much harder to short-term, but high fever, inflammatory changes in the mucous membrane of the gastrointestinal tract.

The short-term nature of the course of FP is associated with short stay of their pathogens in human body.

The action of toxins that bind to the epithelial cells of the stomach and intestines stops after the desquamation of these cells.

Unbound toxin molecules are inactivated by proteases.

CLINICAL FEATURES

Incubation period - 2 hours
(with FP of staphylococcal etiology – 30 minutes) to 1 day.
The acute period of the disease is from 12 hours to 5 days, after which a period of convalescence begins.
In the clinical picture come to the fore:

- ❑ **general intoxication,**
- ❑ **dehydration and**
- ❑ **gastrointestinal syndrome**

Classification FP:

- **According to the prevalence of the lesion:**
  - ✓ **gastritis variant;**
  - ✓ **gastroenteric variant;**
  - ✓ **gastroenterocolitic variant.**
- **According to the severity of the course of the disease:**
  - ➢ **mild;**
  - ➢ **moderate;**
  - ➢ **severe.**
- **For complications:**
  - ▪ **uncomplicated;**
  - ▪ **complicated.**

The clinical features of FP caused by various pathogens has much in common and is represented by similar symptoms.

- ✓ **The onset of the disease is acute.**
- ✓ **There is nausea, which is accompanied by vomiting.**
- ✓ **Vomiting is rarely single, more often it is repeated, sometimes indomitable, painful, debilitating, diarrhea begins almost simultaneously with vomiting.**

- ✓ **The stool is liquid, watery, from 1 to 10-15 times a day, usually has an enteric character and does not contain mucus and blood.**
- ✓ **In a significant proportion of patients, the disease is not accompanied by any severe pain in the abdomen and fever.**

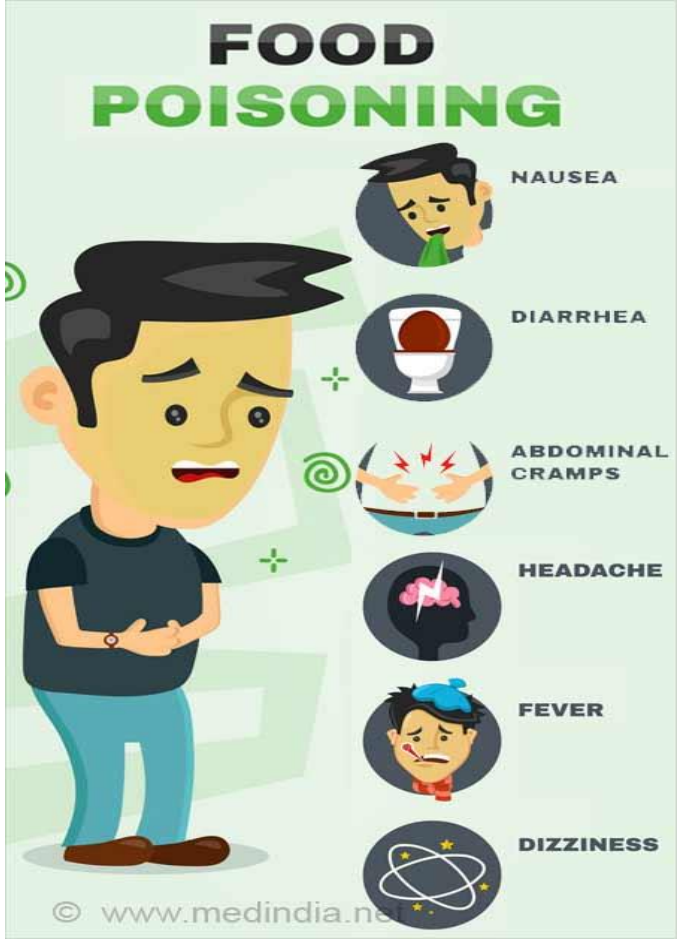
Clinical signs of intoxication -

- **pallor of the skin,**
- **dyspnea,**
- **muscle weakness,**
- **chills,**
- **headache,**
- **joint and bone pain,**
- **tachycardia,**
- **arterial hypotension.**

The development of dehydration is evidenced

- **thirst,**
- **dryness of the skin and mucous membranes,**
- **decrease in skin turgor,**
- **sharpness of facial features,**
- **retraction of the eyeballs,**
- **pallor,**
- **cyanosis (acrocyanosis),**
- **decreased diuresis,**
- **muscles cramps in the limbs.**

From the cardiovascular system are noted -
deafness of heart tones, tachycardia
(less often - bradycardia), arterial hypotension, diffuse dystrophic changes on the ECG (decrease in the T wave and depression of the ST segment).
Changes in the kidneys are due to both toxic damage and hypovolemia.
In severe cases, the development of acute renal failure with oligoanuria, azotemia, hyperkalemia and metabolic acidosis is possible.
Changes in hematocrit and specific gravity of plasma make it possible to assess the degree of dehydration.



Bacterial food poisoning has features depending on the type of pathogen.

Staphylococcal food poisoning is caused by enterotoxigenic strains of pathogenic staphylococci. They are resistant to environmental factors, tolerate high concentrations of salt and sugar. Staphylococcus enterotoxins withstand heating up to 100 ° C for 1-2 hours. In appearance, taste and smell, products contaminated with staphylococcus are indistinguishable from benign ones.

Enterotoxin is resistant to the action of digestive enzymes,
which makes it possible to absorb it in the stomach.
It affects the parasympathetic nervous system,
contributes to a significant decrease in blood pressure,
activates the motility of the stomach and intestines.

The onset of the disease is acute, stormy.
The incubation period is from 30 minutes to 4-6 hours. Intoxication is pronounced, body temperature is usually elevated to 38-39 ° C, but can be normal or low.
Characterized by intense pain in the abdomen, localized in the epigastric region.
Weakness, dizziness, nausea are also noted. In 50% of patients, repeated vomiting (within 1-2 days), diarrhea (within 1-3 days) is observed.
In severe cases, acute gastroenteritis (acute gastroenterocolitis) occurs.
characteristic tachycardia,
deafness of heart sounds,
arterial hypotension,
oliguria.
A short-term loss of consciousness is possible.
The duration of the disease is from several hours to 2-3 days.

Clostridial toxin food poisoning occurs after eating foods contaminated with Clostridium and containing their toxins. Clostridium is found in soil, human and animal feces. Poisoning is caused by the use of contaminated home-cooked meat products, canned meat and fish. Canned food contaminated with Clostridium
(*Cl. Perfringens*), there is an accumulation of gas - "bombing", they acquire the smell and taste of rancid oil.
The disease is characterized by a severe course, high mortality.
Toxins damage the intestinal mucosa,
interfere with absorption. When released into the blood, toxins bind to the mitochondria of the cells of the liver, kidneys, spleen, lungs, the vascular wall is damaged and hemorrhages develop.

Clostridiosis occurs in the form of acute gastroenterocolitis with signs of intoxication and dehydration.

The incubation period is 2-24 hours. The disease begins with intense, stabbing pains in the abdomen.

In mild and moderate cases, an increase in body temperature, repeated vomiting, loose stools (up to 10-15 times) with an admixture of mucus and blood, abdominal pain on palpation are noted.

The duration of the disease is 2-5 days.

Cereosis in most patients is mild. The clinical picture is dominated by symptoms of gastroenteritis. A severe course is possible in the elderly and in immunodeficiency states. Isolated cases of infectious-toxic shock (ITS) with a fatal outcome are known.

Klebsiellosis is characterized by an acute onset with an increase in body temperature (within 3 days) and signs intoxication. The clinical picture is dominated by acute gastroenterocolitis, less often by colitis. The duration of diarrhea is up to 3 days. The moderate course of the disease prevails. It is most severe in people with concomitant diseases (sepsis, meningitis, pneumonia, pyelonephritis).

Proteosis in most cases proceeds easily. The incubation period is from 3 hours to 2 days. The main symptoms are weakness, intense, unbearable pain in the abdomen, sharp pain and loud rumbling, fetid stools. Cholera-like and shigellosis-like variants of the course of the disease are possible, leading to the development of infectious-toxic shock .

Streptococcal FP is characterized by a mild course. The main symptoms are diarrhea and abdominal pain.

Complications.

- • Dehydration shock, infectious-toxic shock (ITS).

- • Regional circulatory disorders: coronary (myocardial infarction); mesenteric (thrombosis of mesenteric vessels); cerebral (acute and transient disorders of cerebral circulation), mesenteric thrombosis.
- • Pneumonia.
- • Acute renal failure
- Lethality is low. The main causes of deaths are myocardial infarction and acute coronary insufficiency, mesenteric artery thrombosis, ischemic cerebral infarction, pneumonia, dehydration shock, ITS.

DIAGNOSTICS

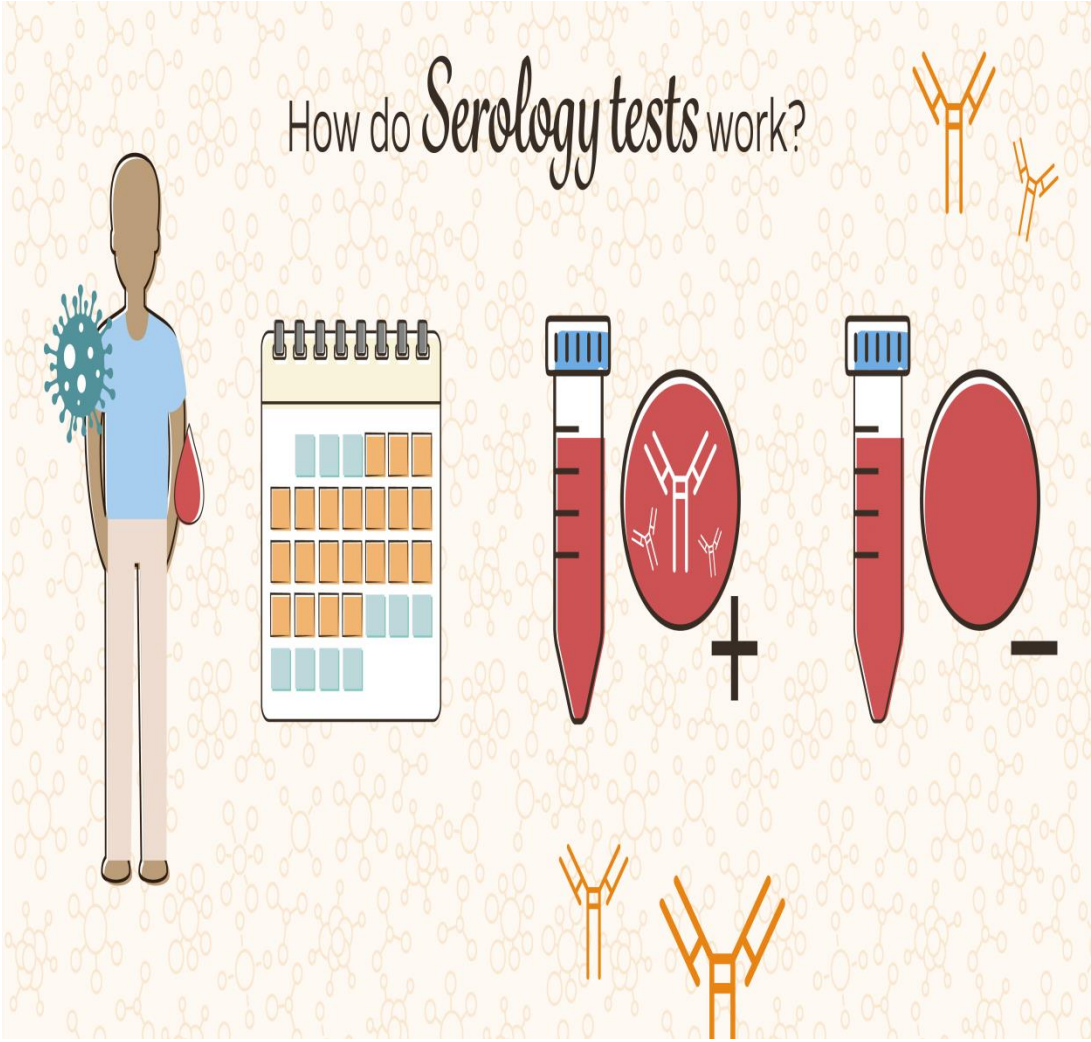
It is based on the clinical picture of the disease, the group nature of the disease, the connection with the use of a certain product in violation of the rules for its preparation, storage or sale.

The following clinical and epidemiological data are of the greatest importance in the diagnosis of FP:

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) **a short incubation period and the short duration of the disease itself;**
4) **the group nature of the incidence and its relationship with the use of the same food product;**
5) **explosive (explosive) nature of the incidence.**

In the laboratory diagnosis of FP is of great importance
has a bacteriological method, including
study of toxigenic properties of isolated pathogens.
The material for the study is vomit, gastric lavage, feces of the patient, the remains of uneaten food, etc.
With PTI, the isolation of a particular microorganism from a patient does not yet allow us to consider it as the causative agent of the disease.
It is necessary to prove its identity with the strains that were isolated from simultaneously sick, as well as those derived from a contaminated product.

The serological method in the diagnosis of FP has no independent significance, since only an increase in the titer of antibodies to the autostrain of the isolated microorganism is conclusive.



Differential Diagnosis

FP should be differentiated from

- **acute intestinal infections and exacerbations of chronic diseases of the gastrointestinal tract,**
- **chemical poisoning,**
- **poisoning with poisons, mushrooms,**
- **surgical diseases of the abdominal organs,**
- **gynecological pathology,**
- **myocardial infarction,**
- **CNS diseases.**

TREATMENT

Patients with severe and moderate course, socially unsettled persons with FP of any severity are shown hospitalization in an infectious diseases hospital.
A sparing diet is recommended with the exclusion from the diet of milk, canned foods, smoked meats, spicy and spicy dishes, raw vegetables and fruits.

Pathogenetic therapy depends on the degree of dehydration and body weight of the patient, is carried out in two stages:
I - elimination of dehydration;
II - correction of continuing losses.

Treatment of food poisoning

In establishing the clinical and epidemiological diagnosis of FP should:

- **Carry out a thorough and repeated gastric lavage until the clean wash water. Washing is carried out 2-4% sodium hydrogen carbonate or 0.1% potassium permanganate.**
- **In severe diarrhea designate activated carbon or other adsorbents (Polyphepanum, calcium carbonate).**
- **In the absence of the stool makes a high enema siphon type.**
- **Accelerate the relief of diarrhea calcium supplements (gluconate, lactate, glycerophosphate) - 5 g per reception.**

Treatment of food poisoning

Further treatment is carried out according to the degree of dehydration of the patient.

- Dehydration I-II degree (body weight loss of up to 3-6%) and the absence of uncontrollable vomiting carried oral rehydration glucose electrolyte solution.
- In severe cases, the dehydration stage III-IV (body weight loss of more than 6%) showed intravenous polyionic solutions "Kvartasol", "Acesol", "Laktasol", "Trisol" and others.

Administration of antibiotics, sulfonamides and other chemotherapeutic agents in patients with uncomplicated PTI inappropriate.

During the disease and during convalescence important dieto- and vitamin therapy.

Prevention

The key to a successful fight against FP is widely conduct public events: the creation of modern, mechanized and automated food processing enterprises, the development and introduction of new methods of preservation and storage of perishable products.

Of great importance are the increasing demand for quality food, efficient and universally existing sanitary service in the food industry, trade and catering.

The most important preventive measure when staphylococcal intoxication is a suspension from work of persons with pustular skin diseases, angina, and others., Come into contact with food (food business employees, cafeterias, grocery stores). Great responsibility lies with the Veterinary Services, responsible for overseeing the health of dairy cattle.

FOOD POISONING PREVENTIONS