Term	Topic content
Non-gonorrheal contagious diseases	A group of non-gonorrheal contagious diseases includes Genitourinary and extragenital lesions caused by Trichomonas, Chlamydia and some other pathogens.
Genitourinary Trichomonasis	*Genitourinary trichomonasis (trichomonasis)* is wide spread inflammatory disease of human genitourinary tract, which is, mostly, sexually transmitted and is caused by Trichomonas vaginalis. **Etiology.** The causative agent of genitourinary trichomoniasis is Trichomonas vaginalis *(Trichomonas vaginalis)* - a microorganism that has adapted through evolution to parasitism in human genitourinary system. *T. vaginalis* belongs to the genus of Trichomonas, which also includes intestinal *T hominis (intestinalis)* and oral cavity *T. tenax (elongate)* saprophytes. The colonization of genitourinary system with oral and intestinal species of Trichomonas does not lead to the development of pathological process. *T. vaginalis* is the only pathogenic species of Trichomonas for humans that does not cause disease in animals and cannot exist outside the human body. *T. vaginalis* is the simplest single-celled organism from flagellates *(Flagellata)* class, belonging to *Trichomanadidae* family of *Trichomonas* genus. There is only one form (or stage) of the development of vaginal trichomonas i.e. trophozoite. The causative agent, preferably, has oval pear-shaped nucleus. At the front end of the body of Trichomonas there are four free flagella extending from basal corpuscle. The fifth flagellum goes back to about one third of length of the body, thus forming an undulating membrane. There is an axostyle all along the cell. Dimensions of Trichomonas fluctuate significantly depending on the growth conditions and characteristics of the strain. Average sizes of *T vaginalis* are 10-30 Mm in length and 5 Mm in width. All the elements of Trichomonas vaginalis structure are detected by special staining methods, while in usual diagnostic tests specialists are guided by the size, shape and mobility of cells. Trichomonas is actively moving by means of free flagella and undulating membrane. Under certain conditions, they form pseudopodia, which ensure amoeboid movement. The main method of reproduction of Trichomonas is division of a cell into two daughters, but sometimes there is schizogony type of division into 8-24 cells. Multiple fission occurs more often under adverse conditions of existence, and Trichomonas do not form cysts, which explain their rapid death outside the human body. Today there are known three

	morphological forms of *T vaginalis* i.e. pear-shaped (flagellar), amoeboid and round (spherical). At the outbreak of chronic inflammation Trichomonas are often round-shaped resembling the nucleus of epithelial cells, making it difficult for the microscopic identification of pathogen. Trichomonas feed themselves endosmotically and by phagocytosis. The optimal pH for the existence of *T vaginalis* is 5.2-6.2. Trichomonas on artificial media develop at 36.5-37 ° C. *T vaginalis* culture, unlike other cultures, is not capable of hemolysis, plasma-coagulation, it cleaves glucose, maltose, starch well, but lactose - weak, it does not form a hydrogen sulfide and indole. Being out of human body, Trichomonas die quickly because of drying. Direct sunlight, a solution of carbolic acid (1 %) and chloramine B (1 %) are detrimental for *T vaginalis*. Trichomonas are resistant to low temperatures and can remain viable at -10 ° C up to 22-45 min. and for 95-115 minutes at 1 -4 ° C temperature. **Epidemiology.** Based on WHO data, more than 180 million cases of genitourinary trichomoniasis are registered each year all over the world. Trichomonas infection occurs through an infected person, most often it happens during sexual intercourse. So far, the epidemiology of genitourinary trichomoniasis has been studied badly, but the prevalence of infection is increased in older age groups, unlike gonorrhea and chlamydia, which are most common for those aged 19-29 years. In women with inflammatory diseases of pelvic organs, Trichomonas are identified in 5,6-20,6% of cases, in pregnant women - in 0,98-32%, while in patients with infertility - in 19.5% cases, in men with inflammatory diseases of genital area - in 0,2-8,5% cases. There is a connection between genitourinary trichomoniasis and premature birth and low birth weight of newborns. In recent decades, the incidence of genitourinary trichomoniasis has remained high. The infection occurs as a result of close household contacts. *T vaginalis* may also be the etiologic agent of pneumonia in neonates and such children may die if untreated with specific drugs. Perinatal trichomonas infection occurs in about 5% of infants born from infected mothers and *T vaginalis* may stay in the child's body from 3 to 9 months or more. **Classification.** Depending on the duration and intensity of organism's reaction to the presence of *T vaginalis* there are the following forms of

	trichomoniasis: 1) *recent trichomoniasis* (with disease duration of up to two months), which, in its turn, is divided into *acute, subacute and torpid (oligosymptomatic)',* 2) *chronic trichomoniasis,* which is characterized by torpid course and duration of the disease for more than two months; 3) *trihomonas-carriage,* when in the presence ofTrichomonas no any subjective and objective symptoms of the disease exist. **Clinical presentations.** The incubation period of genitourinary trichomonas infection is from 3 days to 3-4 weeks (usually 7-10 days). Colonization of genitourinary tract with *T vaginalis* leads to the development of mucosal, skin and affected organs sub-epithelial tissue inflamation. Morphological changes of epithelium are characterized by degeneration, desquamation, proliferation, metaplasia and formation of inflammatory infiltrate consisting of lymphoid elements, histiocytes and plasma cells with a mixture of leukocytes. Often genitourinary trichomoniasis runs subjectively, without any manifestations of symptoms. More than 30-40% of women and 60-70% of men with *T vaginalis* do not have any complaints. In pregnancy, postpartum or post-abortion period, with excessive sexual activity on the background of alcohol drinking and reduction of immunological reactivity, inflammatory process may manifest Asymptomatic genitourinary trichomoniasis is a serious danger to the sexual partners of patients who did not know about the disease, continue to lead normal life and, thus, become a source of further infection. Clinical manifestations of trichomonas infection depend on the location and severity of inflammatory process. *Clinical manifestations of trichomonas infection in women.* In women the inflammatory process may involve vulva, vagina, urethra, lacunar channels, cervix, uterus and its appendages, large vestibular gland, bladder and renal pelvis. In women, in 95-99% of cases, clinically genitourinary trichomoniasis manifests by affection of the lower region of genitourinary system, vaginitis, which is often combined with an infection of urethra, paraurethral ducts and large vestibular glands. Based on literature data, sometimes Trichomonas could be found in the cavity of the uterus, remote fallopian tubes and rectum. Trichomonas infection in women may be asymptomatic, but in a third of these patients clinical manifestations of genitourinary trichomoniasis develop within six months.

	The inflammation of large vestibular glands of trichomonas etiology is characterized by the appearance of painful formation in the lower third of the labia majora. Clinical manifestations ofTrichomonas vestibulitis are similar to those of gonococcal. In the course of colposcopical examination in a small number of patients with vaginitis (5.2%) there are single-point hemorrhages comparable with strawberries or wild strawberry - a symptom of "strawberry" ("wild strawberry") cervix uteri defined on the mucous membrane of the vagina and vaginal cervical with iodine-negative lesions when stained with 3% Lugol's solution. Clinical signs of the disease appear cyclically and are more pronounced before and after menstruation. *Clinical manifestations of trichomonas infection in men.* In men the inflammatory process involves urethra, prostate, seminal vesicles, bladder and renal pelvis. Thus, T. *vaginalis* spread throughout urethral mucosa, penetrating into its glands and lacunae. Trichomonas urethritis in men is often short-lived, transitory, apparently, due to unfavorable conditions for the existence of parasites in male urethra. In about 40% of case Trichomonas urethritis is complicated by prostatitis, which may run subjectively and without any symptoms for years. In a number of observations *T. vaginalis* causes inflammation of epididymis with tubular degeneration and infiltration of sub-epithelial and interstitial tissue, clinically, running like other non-specific epididymitis. Epididymitis is usually accompanied by trichomonas vesiculitis and/or cowperitis that occur with minimal clinical manifestations. The invasion of prostate with Trichomonas is usually asymptomatic; rarely do they register clinical inflamation in the form of catarrhal inflammation or parenchymal prostatitis. Dissemination of infection from primary lesion (urethra) is usually by transtubular way. As a rule, *T. vaginalis* colonize organs of genitourinary system, but they also can cause ulcers and erosions on the balanus skin in males and vulva mucous membrane in women, hence there is a need for differential diagnostics with other diseases with erosive and ulcerative elements that are characteristic of syphilis, herpes etc. *Clinical manifestations of trichomonas infection in children.* In children, before puberty, genitourinary trichomoniasis is rare. The classic manifestation of genitourinary trichomoniasis in girls is in the form of

	vulvovaginitis; rarer occur urethritis and cervicitis. Clinical picture is characterized by profuse vaginal discharge, often of frothy nature, accompanied by intense itching and formation of erosions, not only on the mucosa of anogenital region, but also on the inner thighs. Often the course of trichomonas infection is latent, thus the frequency of the child's examination and the use of effective methods of laboratory identification of *T vaginalis* are of great importance. Complications of genitourinary trichomoniasis: *In women* salpingitis, salpingo-oophoritis, pyosalpinx, endometritis in various combinations; *In men* prostatitis, orchiepididymitis. **Diagnostics of Trichomoniasis.** The diagnosis of trichomoniasis is set by means of identification of causative agent in biological materials. Today there is microscopic, culture, immunological and molecular and biological methods for the identification of *T vaginalis*. *Microscopic method involves* the study of native preparation and smears stained with methylene blue and by Gram's (to simultaneously identify *N gonorrhoeae)* or Romanowsky-Giemsa method. When examining men, they prepare a smear of discharge from urethra or the sediment of centrifuged urine, while with women – they take discharge from posterior vaginal fornix. In chronic inflammatory process the agent can be localized in prostate gland, which requires microscopic examination of its secret. Negative results of microscopic examination should not be regarded as final; the survey should be repeated in 2-3 weeks. On the other hand, epithelial cells, leukocytes and macrophages present in the smears are often perceived as a modified form of *T vaginalis,* which leads to over-diagnosis of trichomoniasis. *Immunological methods* combine the methods of direct and indirect immunofluorescence (DIF and IIF). *Molecular and biological techniques* involve polymerase and ligase chain reaction (PCR and LCR). They have a high specificity and sensitivity, simple and easy to use and allow detecting in one sample such bacteria as *T vaginalis, Candida spp., Gardnerella vaginalis,* which is essential in case of co-infection of genitourinary tract. **Treatment.** The treatment shall be applied to persons who were found positive for *T. vaginal is* regardless of clinical manifestations of the disease and patients who were negative for this type of bacteria, but their sexual partners were positive. The only group of drugs

	that are effective against *T vaginalis* is derivatives of 5-nitroimidazoles, the antiprotozoal activity of which had been proved yet in 1956. It was in 1960 when Metronidazolum was first applied to treat trichomonas infection. In the following decades there were synthesized analogues of metronidazole and a series of new drugs in this group with high activity against protozoa and anaerobic bacteria has been developed. These drugs include Ornidazole, Nimorazolum, Secnidazole. Based on modern international guidelines on selection of single-dose and course antiprotozoal drugs to treat patients with trichomoniasis, the preference is given to single oral administration of Metronidazolum or Tinidazolum at a dose of 2.0 g *Alternative techniques* involve prescription of Metronidazolum 500 mg orally two times a day for 7 days or Tinidazolum 500 mg orally two times a day for 5 days; Methods of treatment of patients with genitourinary trichomoniasis proposed in Ukraine are as follows: Metronidazolum (Trichopol etc.) 250 mg 2 times a day for 10 days or 250 mg three times a day for the first 4 days and 250 mg 2 times a day for the next 4 days: or 500 mg 2 times a day - in the first day, 250 mg 3 times a day on the second day and 250 mg 2 times a day - on the fourth and fifth days; or 500 mg 4 times a day for 5-7 days (recommended for chronic or complicated trichomoniasis): or 750 mg 4 times a day in the first day, 500 mg 4 times a day - on the second day (recommended for recent trichomoniasis with a small disease duration); or Metronidazolum 500 mg in 100 ml of intravenous solution (by drop infusion) 3 times a day for 5-7 days (recommended for chronic continuous course, frequent relapses, complications of trichomoniasis); 2.0 g single dose of Tinidazolum; Ornidazole 500 mg 2 times a day for 5 days. After inflammation relief you should conduct local treatment that must be justified by uretroscopic survey. Male urethra is washed with solutions of potassium permanganate (1 1000-1:6 000) Rivanolum (1-1 000), Furacilin (1:5 000) Gibitanum (1:5000). Women patients with genitourinary trichomoniasis are prescribed local antitrichomonad drugs in the form of vaginal suppositories or vaginal tablets (Trichopol, Tergynan). Tergynan preparation (vaginal tablets) includes: ternidazol. neomycin sulfate, nystatin, prednisolone. Respective components of Tergynan preparation determine its pharmacological

	properties. Ternidazol has trichomonicidal, also it is active against anaerobic bacteria, including gardnerellas. Neomycin sulfate is a broad-spectrum antibiotic of aminoglycoside group. Nystatin is an antifungal antibiotic from polyenes group and it is active against *Candida* fungi. Prednisolone is a glucocorticoid with anti-inflammatory action. The indications for use of Tergynan drug (vaginal tablets), except for trichomoniasis, include bacterial vaginitis that is caused by simple pyogenic organisms; vaginitis caused by *Candida* fungi; vaginitis caused by mixed infection (Trichomonas, anaerobic infection, yeast-like fungi). Pregnant women patients with genitourinary trichomoniasis should be prescribed antiprotozoal systemic therapy not earlier than the end of the II - III trimester. In case of diagnosing genitourinary trichomoniasis in women in early pregnancy, you should prescribe specific (antiprotozoal) local treatment by applying vaginal suppositories or vaginal tablets (Trichopol, Tergynan), 1 per day for 8-10 days. **Follow-up examination.** Follow-up examination shall be carried out 7-10 days after the end of treatment; further examination is carried out twice at an interval of one month. Sexual partners should be treated. Patients should be advised to avoid sexual contact until the end of the treatment and control of laboratory tests. **Prevention.** Preventive measures for trichomoniasis are similar to measures for gonorrhea. These include the earliest possible detection and treatment of trichomoniasis, identifying and bringing to examination and treatment of persons who are the source of infection, as well as preventive health screening and health communication among people.
Genitourinary chlamydia infection	*Genitourinary chlamydia infection* is now the most common among the diseases that are transmitted mainly via sexual contact. The world is witnessing a constant increase in the incidence of chlamydia, particularly among young people who had just entered the period of sexual activity. The prevalence varies widely among different age groups in different regions of the world, but everywhere the disease is much more common than gonorrhea. Slow development of symptoms and often a complete lack of them results in late request of patients for specialized medical care. Medical and social importance of chlamydia infection is conditioned by high incidence of morbidity and complications that significantly affect the demographics, because Chlamydia infection is the most common cause of male and female infertility.

	Etiology. Genitourinary chlamydia infection is caused by *Chlamydia trachomatis*, a representative of *Chlamydiales* order, which includes 4 families, 6 genera and 13 species. Modem classification of microorganisms involves the use of strict criteria of genosystematics to describe various levels of taxonomic groups. Based on new classification, *C trachomatis* is an obligate human parasite responsible for a wide range of diseases, i.e.. trachoma, genitourinary infections, some forms of arthritis, conjunctivitis and pneumonia of newborns. The structure of chlamydiae cell wall is similar to the structure of gram-negative microorganisms. All chlamydiae are similar in morphology, they share genus-specific antigen presented by lipopolysaccharide (LPS) of the outer membrane of cell wall and a variety of species-, subspecies-and type-specific antigens that are protein in nature and characterized by thermolability. Chlamydia elementary bodies are surrounded by tight rigid cell wall, which is separated from plasma membrane by electronically non-transparent periplasmatic space. The cell wall of chlamydiae has typical of gram-negative bacteria two-layer structure: it is composed of proteins, phospholipids and lipopolysaccharides. Unlike other prokaryote, chlamydiae cell wall doesn't contain peptidoglycan that is necessary to maintain its rigidity. Approximately 60% of the total weight of membrane protein makes Omp 1 the major outer membrane protein (or MOMP *Major Outer Membrane Protein)*. Molecular weight of MOMP varies depending on the serotype from 38 to 42 kDa. MOMP protein is a dominant antigen of *C trachomatis*, which determines its serotype. At the current stage, as an alternative for determination of chlamydiae, it is proposed to use the analysis of polymorphism of DNA restriction fragment length. This method was called genotyping and serotypes were called genotypes. This analysis allows detecting new subtypes of known serotypes. Serotype F is often associated with damage to the upper genital tract and pronounced clinical symptoms. E serotype is associated with asymptomatic infection or mild clinical signs of infection. Identification of genotypes of more virulent and capable of causing serious diseases of the upper genital tract, may serve a prognostic indicator of complications. The life cycle of chlamydiae has been studied well enough. It provides for the change of two forms of pathogens metabolically inactive extracellular elementary corpuscles (ECs) and metabolically active non-infectious reticular corpuscles (RC). ECs are adapted to extracellular survival. They are the carriers of specific signs of chlamydiae and at the same time represent highly infectious form of the parasite.

Epidemiology. The source of infection with genitourinary chlamydia is a person with an acute or chronic form of the disease. The main routes of transmission are sexual, contact-household. Given the common ways of transmission of sexually transmitted infections, chlamydia often occur in association with other organisms, such as gonorrhea, trichomonas, mycoplasma, ureaplasma etc.

Pathogenesis. Chlamydiae are profoundly responsive of columnar epithelium that covers mucous membrane of urethra, cervix canal, rectum, conjunctiva and the nasopharynx. The ability to infection is attributed only to EC chlamydiae. The experiments on cultivation of chlamydiae in cell culture have defined that susceptible to infection only those cells whose membrane due to the action of certain factors has lost mechanisms preventing adhesion and intrusion of EC. ECs adhesion on cell membrane and their inside penetration are the first step in the interaction between cells and chlamydiae. By penetrating into the cell, chlamydiae inhibit fusion of lysosomes with phagocytic vacuole. EC penetrate into the cell by pinocytosis, being protected from destruction by phagosome membrane. A few ECS can be present in the cell at the same time, i.e. some groups of chlamydia microcolonies can happen to be in the cytoplasm of cells. Being in the cytoplasmic vacuoles (endosome), ECs consistently across the stage of TC (transitional corpuscles) are transformed in the RCs, which, in their turn, are subject to binary fission. At the end of fission period RCs are subject to reverse transformation into ECs. Newly formed ECs go out the cell, destroying it and infecting new cells.

The study of immune responses to this infection showed that a lot of complications are combined with severe destructions of immune regulation. Immune response in chlamydia infection is diverse and is characterized by production of secretory Ig of A, M, G classes, inflammatory mediators (cytokines), such as IFN, IL-1, IL-4, IL-6, tumor necrosis factor etc. The type of disease course depends on human immunity, the massiveness of infection, pathogenicity and virulence of the pathogen, and many other reasons. Complications of genitourinary chlamydia infections are often combined with severe disorders of immune regulation, in particular, with a reduction in the concentration of T-lymphocytes, T-helper cells, lowered IFN-status of the patient.

Clinical picture. There are the following peculiarities in the course of chlamydia infection in men, women and children.

Clinical picture of genitourinary chlamydia infection in men. In vast majority of cases, chlamydia infection in men is oligosymptomatic. The most common form of the disease in men is urethritis. Based on clinical classification similar to gonorrhea classification there are three forms of urethritis: *recent urethritis* (with disease duration of up to two months), which may occur *acutely, subacutely and torpidly, chronic urethritis* (with disease duration of more than two

	months, or with unknown duration of illness), runs torpidly, with exacerbations by type of acute or subacute urethritis; *latent chlamydia urethra infection* (singled out by some authors). Recent and chronic urethritis are divided into *anterior,* when columnar epithelium cell. Romanovwsky-Giemsa inflammatory process is limited to the anterior segment of urethra, and *total,* when inflammatory process is spread proximal to the external urethral sphincter. *Acute urethra inflammation* is rare, and the patients are concerned with serous or seropurulent urethral discharge, painful and frequent urination. Even without treatment within a few days or weeks acute urethritis symptoms subside, and the inflammation becomes subacute or torpid. In *recent torpid chlamydia urethritis* inflammation, in most cases, is limited to the defeat of the anterior segment of urethra. In practice, the more common are subacute or torpid courses of urethritis, when patients do not complain at all and chlamydiae detection occurs by accident, or complain of a little itchy in the urethra and meager discharge. When examining, you may notice slight swelling and redness of urethral lips. During exacerbation *of chronic chlamydia urethritis* patients' complaints and clinical picture are consistent with recent acute and subacute urethritis, and lesions totally cover the anterior and posterior segments of urethra. Exacerbation occurs after consumption of alcohol, spicy food, sex, exposure to cold or other factors that reduce protective properties of the microorganism. Ureteroscopy in chronic chlamydia urethritis reveals mucosal changes that are consistent with the picture of soft, transitional or solid infiltrate. In *latent chlamydia infection* objective and subjective symptoms are absent; the diagnosis is set on the basis of detection of chlamydia in urethra scrapings. There is possible transformation of latent infection into clinically apparent disease, the cause of which may be associated with concomitant diseases of genitourinary tract with other etiologies. *Genitourinary complications.* The spread of infection on to above areas of genitourinary tract leads to the development of complications, among which a special place is occupied by an inflammation of prostate gland. *Prostatitis* in most cases occurs as primary chronic process. There are four symptoms of this complication - *painful, dysuric, sexual, reproductive.* Each of these symptoms may be the only symptom or manifestation of primary disease. Pain is localized in the perineal area, and irradiation may be in the rump, anus, suprapubic area, urethra, testicles. The intensity of pain varies from paresthesia to the feeling of heaviness and pressure followed by severe pain. Pain may increase with prolonged sitting, bumpy ride, defecation etc.

	Dysuric symptoms include pollakiuria, dysuria, nocturia, sluggish stream of urine, sometimes strangury. Given the fact that all of these symptoms can be observed in BPH, it is necessary to carry out differential diagnostics of this disease in men aged 45 years and older. Dysuria with prostatitis is often caused not only by urethra inflammation, but also it involves bladder neck in the process. The emergence of sexual dysfunction in some patients with changes in the prostate depends on the involvement of adjacent organs (seed tubercle, seminal vesicles) into inflammation process. In this case, even the most minor infractions as rapid ejaculation and certain unpleasant feelings can cause neurotic disorders in the patient, which, in its turn, closes the circle of neurotic symptom complex. Sometimes, chronic prostatitis is latent, when there are no symptoms for years, and the disease manifests itself in copulatory and reproductive dysfunctions or only one of them. This can occur on the background of genetically determined congenital hypoandrogenism. It may however be caused by prolonged inflammation in prostatic acini. Common symptoms of chronic prostatitis include fatigue, weakness, low-grade fever, which is probably due to intoxication and hormonal disorders. Long-term course of the disease, the presence of multiple symptoms, excessive focus on existing problems lead to the development of neurotic disorders. Vegetative local and general reactions lead to the appearance of paresthesias in patients, anorectal itching, perinea sweating. Prostatitis is characterized by alternation of active phases with periods of remission. Without treatment, chronic chlamydia prostatitis can continue for an indefinite period of time. The result of this process depends on its form, state of macroorganism and therapy effectiveness. At a superficial inflammation of the prostate and early treatment a patient can recover with full restoration of function, while in case of late treatment parenchyma is replaced with scar tissue. The spread of contagious agent from the back of urethra through spermduct to the epididymis leads to the development of *epididymitis. Acute epididymitis* is manifested by intense pain in the corresponding part of the scrotum, the skin of which is congested, swollen and hot to the touch. The body temperature rises to 39 ° C. When examining epididymis by touch it is defined as a helmet, covering the bottom and the back surface of the testis. The appearance of serous effusion in the egg shell *(periorchiepididymitis),* involvement of testis into the process *{orchiepididymitis)* result in palpation of scrotum organs as a single conglomerate, in which it is difficult to distinguish the egg from epididymis. Spermduct can also be involved into pathological process *{vasitis),* which is palpable in

	the form of a painful cord. Spread of inflammation to the surrounding tissue of spermatic cord leads to its inflammation *(juniculitis).* Without treatment, within 2-3 days, all the painful events are increasing, and in the next 2-3 weeks they gradually subside, the effusion between shells resolves, but may remain scars in the tail of epididymis, which disrupts the patency of ductuli efferentes. However, impairment of fertility is not always associated with mechanical causes. Immune mechanisms of self-aggression may play one of the important roles in development of infertility. *Extragenital complications*. Among the most frequent extragenital complications are ophtalmochlamydia infection, reactive arthritis that form the symptomatic complex of Reiter's disease, to include pharyngitis and proctitis. A serious complication of chlamydia infection is Reiter's syndrome *{syndromum urethrooculosynoviale).* The disease develops in individuals with a genetic predisposition; it often affects HLA B27 antigen carriers. Men suffer 20 times more often than women. The disease is characterized by combined lesion of urinary organs (genitourinary prostatitis, xerotica balanitis), eyes (conjunctivitis), joints - by type of asymmetric reactive arthritis and skin (psoriasiform rash, keratoderma of palms and soles). The disease usually occurs with repeated attacks and remissions. C. *trachomatis* or its antigens are found in synovial fluid samples obtained from diseasedjoints. ***Clinical picture of genitourinary chlamydia infection in women.*** Chlamydia infection in women is associated with impaired reproductive function and infectious complications in the form of inflammatory diseases of pelvic organs, tubal infertility and ectopic pregnancy. Clinical manifestations of genitourinary chlamydia infection vary from expressed inflammation events to asymptomatic carriage. They single out the affection of the lower region of genitourinary tract (endocervicitis, urethritis, paraurethritis and bartholinitis) and ascending infection (endometritis, salpingitis, salpingoophoritis, pelviperitonitis, perihepatitis). The spread of chlamydia from foci located in the lower genitourinary tract, promote abortion and other operations, including extragenital. Often, the process becomes complicated and manifests by the development of infertility. Chlamydia infection is multifocal. In the vast majority of cases, the process is asymptomatic or with poor clinical symptoms and is often associated with other genitourinary infections. In women cervical canal is most often affects than urethra. *Urethritis* in women occurs less often than in men, and due to their anatomy, it is accompanied by less severe symptoms, including slight leukocytosis in microscopy of scrapings from urethra. *Bartholinitis* is an inflammation of large vestibule glands. It is often manifested in the form of catarrh with lesions on mouth ducts only. The development of an acute process with a fever, severe pain and formation of abscess in big vestibule gland, is possible only with concomitant

	infection with gonococci and pyogenic microbes. *Colpitis* is rare as chlamydia do not breed in the stratified squamous epithelium, and outside the cells, they are sensitive to vagina acidic reaction. Primary colpitis is possible only in case of change in endocrine profile, in women of post-menopausal period, pregnant women and girls. C. *trachomatis* can cause urethral syndrome, characterized by dysuria, pain in the urethra, and sometimes pain in the lower back. Often chlamydia cervical lesions, morphologically, are characterized as follicular cervicitis and erosive affection of cervix. *Endocervicitis* is frequent, the most typical and common manifestation of genitourinary chlamydia infection in women, it is its most common clinical form. However, chlamydia can attack vulva in newborn girls, and vaginal vault in women who have undergone hysterectomy. *Chlamydia cervicitis* is the main source of infection for men and children. Clinical manifestations of cervicitis occur in about 3-4 weeks after infection and are accompanied by dysuric disorders. Some women complain of itching and burning in the perineal area, whites and lower abdominal pain. Cervicitis runs with scant mucous purulent discharge, the appearance of inflammatory halo around the external os to form a peculiar lymphoid follicles in the external os *(follicular cervicitis)* and light vulnerability of this site. The discharge from cervical canal macerate stratified squamous epithelium of vaginal part of cervix, thus causing its partial desquamation. The cervix becomes edematous and so-called hypertrophic ectopia appears. A kind of an infected cervix can vary from clinically normal to erosive, with thickened edematous mucosa and lots of mucous purulent discharge. Likewise in other genitourinary infections, genitourinary chlamydia infection in women, besides cervix, affects urethra and paraurethral ducts, to include rectal mucosa. *Proctitis symptoms* develop less than urethral syndrome. Proctitis is characterized by rectal bleeding and absence of diarrhea. Approximately in two thirds of women proctitis is caused by passive dissemination of vaginal discharge, and in a third - because of anogenital contact. Bartholinitis of chlamydia etiology occurs relatively rare. Characteristic of *chlamydia cervicitis* symptoms that can

	be minimally expressed include: cervical contact bleeding, mucous-purulent discharge from the cervix and pseudoerosions. Due to the close connection of cervical duct and uterine, inflammatory lesions of cervix are almost always accompanied by the appearance of processes covering the endometrium. *Chlamydia endometritis* may occur in acute and chronic forms, when the latter is accompanied by uterine bleeding. Chlamydia endometritis develops slowly. Postpartum and post-abortion periods contribute to the emergence of chlamydia endometritis. Chronic chlamydia endometritis in its pure form is rare; it is often accompanied by chronic salpingitis or salpingoophoritis. Among all of chlamydia genital lesions *salpingitis* attracts due to the frequency of the disease and pronounced clinical symptoms. *Acute salpingitis* is a severe systemic disease. Clinical diagnosis of acute salpingitis is relatively simple it is established on the basis of severe pain in the abdomen, pain on palpation, increased body temperature, high leukocytosis, accelerated erythrocyte sedimentation rate. *Inflammatory diseases of pelvic organs* are a group of independent clinical entities, witnessing of the presence of bottom-up process including any combinations of endometritis, salpingitis, oophoritis, tubo-ovarian abscess and pelvic peritonitis. The spread of chlamydia infection in the peritoneal cavity leads to the development of perihepatitis known as Fitz-Hugh-Curtis syndrome. Peritonitis and perihepatitis complicate genitourinary chlamydia infection predominantly in young women. The onset of the disease is sudden; there are sharp pains in the abdomen and in the right upper quadrant extending into the right shoulder blade and shoulder, positive peritoneal signs, fever and intoxication. Fitz-Hugh-Curtis syndrome can occur after such interventions as hydrotubation. Genital chlamydia infection can also occur in pregnant women. The likelihood of adverse pregnancy outcomes and fetus affection in pregnant women depend on the severity of genitourinary chlamydia infection, duration of the disease and the adequacy of treatment. Clinical picture of genitourinary chlamydia infection in pregnant women is the same as that of non-pregnant. ***Clinical picture of chlamydia infection in children.*** A common clinical form of chlamydia in newborns is conjunctivitis (so called *conjunctivitis with inclusions)*, non-severe disease, which does not cause much anxiety in neonatologists. The disease is characterized by diffuse conjunctival hyperemia, bonding of eyelids after sleep, no copious purulent discharge. However, in line with conjunctivitis or later in infancy, there appear other clinical

	forms of chlamydia infection acquired before birth or during the passage via birth canal. These include pharyngitis, pneumonia, vulvitis and vulvovaginitis, urethritis, which are asymptomatic in most cases. **Diagnostics of chlamydia infection.** *Laboratory Diagnostics* of chlamydia infection is of paramount importance because clinical manifestations are non- pathognomonic and much common are atypical and asymptomatic forms of the disease. The development of laboratory methods for diagnostics of genitourinary chlamydia infection is directly associated with understanding of biological characteristics of chlamydiae, their antigenic structure, the pathogensis of infection process caused by this agent, to include the overall progress in the field of diagnostics of infectious diseases. The quality of diagnostics of genitourinary chlamydia infection depends on the correctness of taking clinical material, the compliance with terms of its delivery to the laboratory and use of high-quality diagnostic tests. To isolate chlamydiae you should investigate biological materials from different sources, often scrapping smears from men's urethral mucosa and women's cervix and urethra. With the introduction of molecular biology techniques it became possible to study non-invasive clinical samples, such as the first portion of freely released urine in men and vaginal discharge in women. If necessary, study material is taken from the rectum, nasopharynx and lower eyelid conjunctiva. In children you should explore the discharge from lower lid conjunctiva, the rear wall of pharynx, vulva in girls. Based on clinical signs, you may study biopsy and surgical materials. Such methods of diagnostics as culture, immunofluorescence test, PCR, ELISA test are used to study materials obtained from the cervix, urethra, rectum, nasopharynx, conjunctiva, biopsy and surgical materials. To study the first portion of urine and vaginal secretions only PCR is used. Cytoscopical method of diagnosing genitourinary chlamydia infection involves the study of Romanowsky-Giemsa stained biological materials using light microscopy. The criterion for detecting chlamydiae in this case is the presence of Halberstadt-Provazek corpuscles in the cytoplasm of infected cells. Light microscopy makes it possible to find large blue-violet vegetative forms (RC) and small pink infectious forms (EC). Cytoscopical method is widely available, but is effective only for acute forms of infection and requires qualified assessment of cytological picture. In genitourinary chlamydia infection the frequency of detection of Halberstadt-Provazek corpuscles in scrapings from urethra and cervix usually does not exceed 10-12%.

	Immunomorphological methods (Immunofluorecsence test, ELISA test) are based on detection of chlamydia antigenic substance in the epithelium and other tissues. The sensitivity of immunofluorecsence test is 80-90%, while specificity-98- 99%. According to various researchers, the sensitivity of ELISA test ranges from 60 to 80%. Immunofluorecsence test and ELISA test are not suitable for the study of materials obtained from rectum, nose, throat, and urine samples. One of the most objective methods of chlamydia infection laboratory diagnostics is the isolation of the pathogen from affected tissues in McCoy cells culture *{culture method)*. This method is labor intensive, besides from 1 to 4 weeks is required to get the response. It has been recognized as gold standard all over the world and has one hundred percent of specificity. It should be stressed that, being the gold standard for specificity, the culture method is inferior to the rest by sensitivity. The sensitivity of this method in the study of a sample from cervix makes 75-80%. *Amplification tests* aimed at identifying nucleic acids of chlamydiae, such as polymerase chain reaction (PCR), have a sensitivity of at least 93-96%. Now the most promising and highly sensitive are molecular and biological methods chlamydiae detection i.e. PCR, hybridization reaction, RNA detection, SDA, NASBA, etc. *Serological studies* relate to the subsidiary methods of diagnostics of genitourinary chlamydia infection. They can detect IgM, IgA, IgG in the serum, which is especially important for chlamydia detection in children, in complicated process in adults when the use of other methods of detecting the pathogen or its antigen is impossible, and in mass epidemiological studies. Thus, it should be noted that today there are many different methods for chlamydia infection diagnostics. It is important to choose the most reliable, allowing setting correct diagnosis and to timely carry out and monitor the specific therapy. **Treatment.** The treatment of patients with genitourinary chlamydia infection is subject to general principles of management of infectious patients. The therapy should be integrated and etiologycally, pathogenetically and symptomatically differentiated according to the clinical form of inflammatory process, the nature of affection, the severity and duration of illness. Causal treatment of genitourinary chlamydia infection must meet a number of requirements, in particular to high degree of penetration of antichlamydia drug into the cell, its accumulation and to the ensurance of inhibitory concentration at the point where the agent is localized. To treat patients with chlamydia infection they use such antibiotics as tetracyclines, macrolides and fluoroquinolones. The *group of tetracyclines* includes antibiotics that are congenial by chemical structure, antimicrobial spectrum and mechanism of

	action and that are assigned according to the following schemes: Tetracyclinum and Oxytetracycline orally after meals, 500 mg 4 times per day for 7 or 14 days; Metacyclinum 300 mg four times a day for 7-10 days; Doxycyclinum 100 mg 2 times a day for 7-14 days. *Macrolides* are broad-spectrum antibiotics, which are characterized by the presence of macrocyclic lactone ring in their molecule. There are known natural (Erythromycinum, Oleandomycinum, Josamycinum and Spiramycinum) and semisynthetic (Azithromycin, Roxithromycine, Clarithromycin etc.). Such drugs as Azithromycin Josamycinum, Clarithromycin penetrate well into the various tissues and biological fluids, thus creating high and stable concentration of the drug, which is much higher than in serum. Macrolides are referred to the safest antibiotics due to a minor number of possible side effects. *Fluoroquinolones* are fluorinating derivatives of nalidixic acid, the spectrum of activity of which involves mainly the action of gram-negative bacteria. ***Treatment for pregnant women.*** In compliance with applicable guidelines, the following treatment is proposed: Erythromycinum 500 mg orally four times daily for 7 days; Erythromycinum 250 mg orally 4 times a day for 14 days; Amoxicillin 500 mg orally 3 times a day for 7 days; Azithromycin 1,0 g orally once; Josamycinum 750 mg orally 2 times a day for 7 days. Standard local measures traditionally involve washing, douching (of urethra, vagina) with a solution of potassium permanganate (1:8000) in alternation with instillation of 1.2% Protargolum solution, 1 -2% Collargolum solution during 10-15 days. Officinal local media that have antichlamydia activity include vaginal suppositories and Betadine cream as well as Erythromycinum and Tetracyclinum ointment. **Follow-up examination.** After the treatment of patients with genitourinary chlamydia infection follow-up examination should be carried out not earlier than in 3- 4 weeks. PCR study examination conducted earlier than 10-14 days after antibiotic therapy, can give false positive results. To control the cure it is desirable to use two methods (culture in combination with PCR or immunofluorescence test in conjunction with PCR), when, at the same time, you may use RT-PCR method and real-time NASBA. Detection of chlamydiae one month after the treatment requires the appointment of a repeated course of therapy with other groups of drugs, the duration of which shall not exceed 7-10 days. **Prevention.** Prophylaxis of chlamydia infection is not significantly different from prophylaxis of other sexually transmitted diseases. First of all prophylaxis shall involve comprehensive and timely treatment of patients, elimination of infection in asymptomatic carriers of the pathogen, detection and qualitative examination of patients -sexual partners, administration of prophylactic treatment, use of condoms, health education

	of the population and especially high-risk groups.