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**INFLAMMATORY GYNECOLOGIC
DISEASES**

*Study textbook for independent work to senior students of medical faculty and
medical interns specialized in Obstetrics and Gynecology*

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The textbooks in gynecology used by the senior medical students according to the curriculum and study hours include the chapters about gynecologic diseases requiring emergency. The independent training with the textbook proposed now will help the senior students, obstetricians and gynecologists in future to learn this pathology better.

The interns specialized in Obstetrics and Gynecology within the period of their studies during three years have to master their knowledge and skills based on the advanced studies of gynecologic emergencies. The data exposed in this textbook are aimed at this goal. The textbook is recommended to be used by the obstetricians and gynecologists in their practice.

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ABBREVIATIONS

AB - antibiotics
BP - blood pressure
BV - bacterial vaginosis
IUD - intrauterine contraceptive
HBO - Hyperbaric oksihenatsiya
AA - "acute abdomen"
HS - hemorrhagic shock
DIC - disseminated intravascular coagulation
ART - assisted reproductive technology
STD - infection, sexually transmitted infections
COC - combined oral contraceptives
CFU - colony forming units
ICD-10 - International Classification of Diseases – 10
BCC - blood volume
EP - ectopic pregnancy
TD - trophoblastic disease
HCG - human chorionic gonadotropin
HC - horionkartsynoma
HR - heart rate

1. INTRODUCTION

Inflammatory female genitals' diseases represent one of the main problems in medicine influencing considerably the health of million women in reproductive age. 60 – 70% amid gynecologic patients in out - patients centers for women are those who suffer from inflammatory genitals' diseases. The rate for occurrence in inflammatory diseases of female genitals is increasing in all countries. Thus, within recent decade acute inflammatory diseases occur more frequently for 13% in the whole population and for 25% in women using intrauterine contraceptives.

Due to their wide spreading and severity of complications infections of fertile system are considered as serious problem for the health of women. According to the data of WHO more than 33 million of recent cases for curable diseases in fertile system are recorded every year.

As incurable infections including AIDS more than 5 million persons have been infected by this agent during one year.

Infections of reproductive system produce many severe complications for women, such as: inflammatory diseases of pelvic organs, infertility, extrauterine pregnancy, syndrome of chronic pelvic pain. Within pregnancy they may lead to intrauterine infection and complicated pregnancy. The processes interrelated which start with acute inflammation and finish with destructive changes are in the basis of development and progressing inflammatory diseases.

Unfortunately, a lot of infectious diseases have no clinically evident symptoms and they are diagnosed when function of reproductive system is damaged. Opportune diagnosis and adequate treatment have to assure auspicious consequences.

2. PECULIARITIES OF MICROBIOCENOSIS FOR GENITAL SYSTEM IN HEALTHY WOMEN

Bacterial invasion is the main starter for inflammatory process in genitals. The development of inflammation is determined by bacterial agent. Development of inflammation is determined by bacterial agent.

From the point of view for biology female genital system may be considered as aggregation of several biotopes. Biotope is a medium site for one or several species of microorganisms. It is characterized by the following parameters: species and concentration of nourishing substances, pH, oxidation-reduction potential, temperature, hydration, hormonal and oxygenous rates and other parameters.

Vagina, vaginal microflora and vaginal medium form a single harmonious biotope protecting genital system against pathogenic agents and preventing dissemination of infection on internal genitals.

Female reproductive system (vulva, vaginal parietes, vagina, cervical canal, uterus, uterine tubes) from the point of view for anatomy represent succession of caval organs which are in direct or indirect contact with external world. The developed system of protecting mechanisms is in female fertile tract for resistance against pathogenic infectious agents.

It must be mentioned:

- Features of anatomic structure;
- Permanent renewal of epithelial cover in vagina accompanied by recurrent desquamation of superficial cells
- Physical and chemical peculiarities of vaginal content which is secretum of cervical glands, endometrium and endosalpinx, transudate of circulatory and lymphatic vessels;
- Cyclic menstrual apoptosis;
- Acid vaginal medium and its microflora.

All factors mentioned above in total ensure colony resistability so called.

Vagina is covered with multiple strata of flat epithelium (30 – 40 cells layers) which is never submitted to keratinization. Capacity to independent detachment and

renovation is a peculiarity of vaginal epithelium. The cells detached are very important in forming acid medium of vaginal content that is necessary for monitoring vaginal flora.

Cyclic changes occur in vaginal epithelium in response to influence of reproductive hormones. The number of glycogene accumulated in superficial cells is a significant index for vaginal epithelium resistance. The superficial cells are permanently desquamated and submitted to cytolysis, in consequence of it glycogen is released ensuring nutrient substrate for standard vaginal microflora.

Hormonal status for different phases of menstrual cycle affects glycogen content and its fermentation in women of fertile age. Considerable content of glycogen in vaginal epithelium is result of estrogens circulating in blood and its maximum at the moment of ovulation.

Glycogen is decomposed under lactobacilli influence to lactic acid that ensures acid medium of vaginal content (pH 3.8 – 4.5). Thus, protection for this biotope is ensured and vaginal microecosystem is formed.

Vaginal microflora acts in vaginal secretion. It is composed of transudate, released from capillaries in vaginal mucosa, secretum from glands in cervicovaginal canal and Bartholin's glands, leucocytes and epithelium desquamated. Vaginal secretion contains water, inorganic salts, mucin, proteins, fatty acids, urea, lysozyme.

Amount of vaginal secretion varies within menstrual cycle because it depends on estrogens' activity. It is maximum (to 5ml for 24 hours) within ovulation. For secretory phase (yellow body phase) when progesterone predominates and formation of cervical mucus is hampered the amount of vaginal secretion decreases.

Vaginal microflora is composed both of microorganisms forming standard resistant microflora and transitory bacterieas accidentally brought from environmental medium of 3 groups: nonpathogenic, conventionally pathogenic and pathogenic. These transitory microorganisms aren't apte to be in genital tract for a long time and they don't promote development of pathologic syndrome till immune and non immune protective agents are able to realize their impeding function. These agents prevent reproduction for exogenic microorganisms and their penetration on

vaginal mucosa with following dissemination into genital and other organs and tissues.

It has been proved that vaginal microflora contains both aerobic and anaerobic microorganisms. Specific composition for microflora in genital tract of healthy women in fertile age is presented in the table 1. It is from the textbook “Infections in Obstetrics and Gynecology” edited by prof. Chayka

Table 1

Specific composition for microflora in genital tract of healthy women in fertile age

	Obligate anaerobic microorganisms	Optional anaerobic and aerobic microorganisms.
Gram-positive		
bacilli	Lactobacillus spp. Bifidobacterium spp. Eubacterium spp. Propionibacterium spp.	Corynebacterium spp. Acinetobacter spp. Mobiluncus spp. Gardnerella vaginalis
cocci	Peptococcus spp. Peptostreptococcus spp.	Staphylococcus: S. epidermidis S. aureus Streptococcus: S. agalactiae (rp. B) Enterococcus
Gram-negative		
bacilli	Bacteroides spp. Prevotella spp. Fusobacterium spp. Campylobacter spp.	Enterobacteriaceae: Echerichia Klebsiella spp. Enterobacter Proteus Acinetobacter Pseudomonas
cocci	Veillonella spp.	-
Other microbes	Candida spp. M. hominis M. fermentas M. genitalium. U. urealyticum	

According to the table there are two species of anaerobes:

- Optional ones: can exist both in the medium free of oxygen and when the oxygen is available;

- Obligate ones: can exist in the medium free of oxygen only. Vaginal secretion in women of reproductive age as standard, contains 10^8 - 10^{10} microorganisms for 1ml, the ratio between anaerobic and aerobic bacteria is from 2:1 to 10:1. Lactobacteria and bifidobacteria predominate in vaginal microbiocenosis, but those producing hydrogen peroxide exceed.

Table 2

Microorganisms composing microflora for genital tract in healthy women of reproductive age.

Microorganisms	Occurrence (rate in %)	Concentration In UFC/ml
Lactobacillus spp.	95-98	$10^{5,34} - 10^8$
Bifidobacterium spp.	<10	$10^{5,45}$
Eubacterium spp.	+	10^3
Clostridium spp.	<10	10^3
Propionibacterium spp.	25	10^3
Corynebacterium spp.	6-7	$10^3 - 10^4$
Bacteroides spp.	55	till 10^4
Prevotella spp.	55-61	till 10^4
Fusobacterium spp.	<10	10^3
Leptotrix spp.	+	
Campylobacter spp.	+	
Enterobacteriaceae:		$10^{3,57}$
E. coli	<10	10^3
Klebsiella spp	+	10^3
Enterobacter spp.	+	10^3
Proteus spp.	+	10^3
Acinetobacter spp.	+	10^3
Mobiluncus spp.	+	10^3
Pseudomonas	+	10^3
Gardnerella vaginalis	>10	
Staphylococcus spp.:	+	+
Staphylococcus epidermidis	+	+
Staphylococcus aureus	<10	+
Micrococcus spp.	35	+
Enterococcus spp.	10	$10^3 - 10^4$

Streptococcus spp.	<10	$10^5 - 10^{5.2}$
Streptococcus gp. B	5-25	$10^4 - 10^5$
Peptococcus spp.	40-90	$10^3 - 10^4$
Peptostreptococcus spp.	<10	10^4
Veillonella spp.	5-25	10^3
Candida spp.	<10	10^4
M. hominis	15	10^4
M. fermentas	<10	10^3
M. genitalium	<10	10^3
U. urealyticum	<10	10^3
Actinomyces spp.	<10	10^2

Notes: 1) UFC – units forming colonies

3. PHYSIOLOGIC BARRIERS FOR SPREADING INFLAMMATORY PROCESSES IN FEMALE BODY.

Lactobacilli with concentration $10^6 - 10^9$ UFC/ml in 95 – 98% cases take a leading part in vaginal microbiocenosis. All researchers recognize that lactobacilli have a protecting effect against dissemination of pathogenic bacterias.

Nowadays it is observed some aspects of their protecting function for vaginal ecosystem.

1) Formation of lactic acid and other organic acids maintains low pH in vaginal medium by lactobacilli. Standard pH of vaginal content is 3.8 – 4.5 for oppression of anaerobe's growth, Gardnerella vaginalis.

2) Formation of hydrogen peroxide by lactobacilli oppresses biotope colonization with pathogenic microflora habitat.

3) Following lactobacilli's high adhesive capacity to superficial epithelial cells they prevent adhesion of other microbes (Gardnerella vaginalis, Candida albicans and Mycoplasma hominis) to epithelial receptors and ensure colonization resistance.

4) Lactobacteria influence on different links of immune system and regulate non specific and specific cellular and humoral immunity.

5) Lactobacteria produce bacterial substances like proteins, namely, bacteriocines participating in regulation of colonization resistance in vaginal microbiocenosis by oppressing growth of other microorganisms.

Specific content of lactobacteria for vaginal biotope is proper for individual woman and often it is represented by some species. More than 150 lactobacteria are classified, different researchers report that vagina of healthy women can contain from 6 to 15 species. The most frequent are: *Lactobacillus acidophilus*, *Lactobacillus paracasei*, *Lactobacillus crispatum*, *Lactobacillus brevis*.

V.O. Potapov refers (2014) conditions decreasing the amount of lactobacteria and disturbing microbiocenosis that may lead to bacterial vaginosis, nonspecific vaginitis or vulvovaginal candidosis. These conditions are:

- Taking medicines (antibiotics, hormones);
- Contraceptives (spermicides, condoms, combined oral contraceptives (COC), endometrial contraceptives (EMC));
- Sexual dissipation and risky sexual behaviour;
- Infections sexually transmitted (IST)
- Pregnancy;
- Extragenital somatic diseases (obesity, diabetes, gastrointestinal diseases)
- Chronic stress;
- Synthetic clothes;
- Diet, lack of intimate hygiene;
- Abusing hygienic means (pads, tampons, lotions, irrigations and so on);
- Self-treatment;
- Surgery.

Bifidobacteria are the most important amid microorganisms protecting genital tract against environmental aggression. They are anaerobes observed in 10% healthy women. The most spreading are: *B.bifidum*, *B.breve*, *B.longum*, they oppress the growth of *Escherichia*, *Clebsiella*, *Gardnerella*, *Streptococci* due to their capacity to form acid and powerful adhesion to epitheliocytes in genital tract.

Biphidobacteria are able to produce bacteriocines, lysozyme, aminoacids and vitamins which being in the low segments of female genital tract influence on support for normal microbiocenosis.

Peptostreptococci are species for normal microflora in genital tract of healthy woman equally with lacto- and biphidobacteria. They are observed with the rate from 40% to 90% in $10^3 - 10^4$ UFC/ml of examined material. At the same time it must be noted that peptostreptococci are revealed in case of different pyo-inflammatory diseases in female genitals organs. They are observed associated with other anaerobic bacteria in concentration 10^5 UFC/ml and higher of examined material in vaginal vaginosis. Model species for normal microflora in female genital tract is propionobacterium *P. acnes* revealed with a rate till 25% of examined material in number that doesn't exceed 10^4 UFC/ml. Propionobacteria possess immunosimulating properties and take part in supporting colonization resistance on account of organic acids produced by them.

Vaginal microflora contains obligate anaerobic gram-negative bacilli species *Bacteroides*, *Prevotella*, *Fusobacterium* and *Porphyromonas*. They are isolated in 85 – 90% healthy women in a number $10^3 - 10^4$ UFC/ml in examined material. The significance of these microorganisms is not studied yet, but it is known that some species possess pathogenic properties. As standard being part in vaginal flora they can cause acute inflammatory genital diseases. These microorganisms are revealed in high concentrations in bacterial vaginosis.

Bacteroides urelyticum revealed in 55% healthy women is the most frequent amid vaginal bacteroides.

The rate of revealing species *Prevotella* in vaginal tract of healthy women reaches 60% cases, but their number doesn't exceed 10^4 UFC/ml in material examined. The most frequently revealed are *P. bivia* and *P. disiens* (61%).

Porphyroonas asacharolitica is model species *Porphyromonas* in vaginal secretum. Quantitative level of these bacteria doesn't exceed 10^3 UFC/ml in material examined, but they are revealed in 31% cases.

As compared with other strictly anaerobic bacteria colonizing vagina fusobacteria occur seldom less 8% in concentrations not higher than 10^3 UFC/ml of material examined.

Gardnerellae are optional anaerobic bacteria. *G. Vaginalis* is the sole specimen of this species occurring in 60% sexually active women, its concentration often reaches 10^6 UFC/ml of material examined. These microorganisms possess pronounced capacity to adhesion for superficial vaginal epitheliocytes. They are able to produce mucolytic enzymes and hemolysines and take an essential part in development of bacterial vaginosis.

Corynebacteria are gram-positive aerobes or optional anaerobic polymorphic bacteria. In vagina of healthy women they are revealed in 6 – 7% cases in a number $10^3 - 10^4$ UFC/ml of vaginal secretum. Mostly revealed are *C.minutissimum*, *C.eguli*, *C.aquaticum*, *C. xerosis*. The other corynebacteria are revealed utterly seldom.

Mycoplasmas are relatively pathogenic microorganisms. Mostly revealed in women sexually active are: *M. hominis*, *M. genitalium*, *U. urealyticum*.

As standard **Ureaplasma urealyticum** is isolated in 6 – 7% women with amount $10^3 - 10^4$ FCU/ml of material examined, *M.hominis* is isolated in 2 – 15% women, their amount is 10^3 FCU/ml of material examined. In bacterial vaginosis the essential one is *M. hominis* which is observed with the rate to 30%, quantitative level increases to 10^5 UFC/ml of material examined it is associated with other obligate anaerobes, gardnerellae.

Staphylococci are gram-positive optional anaerobic cocci. In vagina of healthy women epidermal staphylococci are the most spreading (*S.epidermis*). Their number forms $10^3 - 10^4$ CFU/ml of vaginal secretum. Goldish staphylococci coagulopositive (*S. aureus*) colonize vagina transitionally and are revealed only in 5% cases. Microorganisms of this species are able to produce toxin that may result in toxic shock syndrome.

Streptococci are optional anaerobic gram-positive cocci. As standard in vagina of healthy women the following streptococci are observed: streptococci viridans, streptococci of serologic group B – *S. agalactiae* and specimen *Enterococcus*. The

revealing rate and their amount vary considerably: from 5 to 55% occurrences and to $10^4 - 10^5$ UF/ml of material examined concerning amount.

Enterobacteria are gram-negative optional anaerobic bacilli. The most spreading specimen is E coli of species Escherichia. The rate of their secretions is 9 – 25% and amount forms $10^3 - 10^4$ CFU/ml of material examined.

Amid other bacteria in the family **Enterobacteriaceae**, **Klebsiella** and **Enterobacter** can be isolated from vagina of healthy women as well, but only in 2 – 4% cases. Enterobacteria may cause inflammatory diseases in urogenital tract.

Yeast fungi Candida are revealed in vagina of healthy women with the rate to 10% and amount to 10^4 UFC/ml of material examined. In this concentration they don't cause pathologic process and can't disturb leucocytes' viability and function. C. albicans is the most frequently revealed.

Vaginal microflora depends on female hormonal state (physiologic changes within menstrual cycle, pregnancy, puberty and climacteric periods). Estrogens cause enlargement for vaginal epithelium and glycogen accumulation, which stimulate virulent activity for pathogenic microorganisms. More microorganisms are revealed in the first phase of menstrual cycle than in the second one. Larger amount for gram-negative bacteria are observed in proliferative phase. Seven days before menstruation microorganisms number sharply decreases.

Staphylococci, diphtheroids, bacteroids predominate in virgin's microflora in prepubertal period. Within pregnancy content for microflora is permanently changed, anaerobes number is decreased, but lactobacteria are increased. After delivery, surgical operations proliferation of microorganisms is common that leads to infectious complications.

Conclusions:

1) Vagina, vaginal microflora and vaginal medium form common, harmonious biotope ensuring protection against pathogenic agents in genital system and preventing infectious process in internal female genital organs.

2) Standard microflora in female genital organs is composed of saprophytic and relatively pathogenic aerobic and anaerobic microorganisms, which are in biologic balance and take's part in one among unspecific resistance agents in the body.

3) Relatively pathogenic microorganisms (RPM) under certain conditions (associated other infectious agents, changes for physiologic and immune state and so on) can realize their pathogenic capacities and cause inflammatory diseases in female genital organs.

Protective barriers, ensuring dynamic balance in vaginal content form humoral agents (complement system, lysozyme, opsonin, fibropectin, beta-lysins, so on) in complex with cellular agents (macrophages, polymorphonuclear leucocytes, T-lymphocytes) and agents for topic immunity (Ig A-antibodies).

Deterioration in any link in the system result in disturbance of its balance and cause pathologic state.

Decreasing lactobacilli number leads to the sharp increase in number for pathogenic microorganisms, change for ratio of anaerobes and aerobes (10000:1) that is microorganisms' concentration approaches to their concentration in intestine. Epithelial desquamation begins that favours ascending infection.

Microorganisms permanently existing in genital tract may become virulent under certain conditions. Physiologic protective mechanisms impeding microorganisms activation and their participation in inflammation are:

1. Physiologic desquamation and cytolysis of superficial cells in vaginal epithelium under influence of ovarian hormones.

2. Unspecific antimicrobial mechanisms on cellular level, phagocytosis assisted by microphages.

Unspecific humoral agents:

- Transferrin, plasma binding protein, iron necessary for bacteria's growth;
- Opsonins boosting cellular phagocytic activity;
- Lysozyme-peptide with antibacterial activity;
- Lysin, secreted by thrombocytes in focus of inflammation.

3. There are immune protecting mechanisms against fungal and viral infections with endo-cellular bacterial parasites (T-lymphocytes, immunoglobulins, complementary system).

4. Protective mechanisms in cervical canal and endometrium are very important in upper segments of genital system.

Uterine cervix for women in fertile age is of cylindrical shape, but for virgin and infantile women it is in shape of cone.

Vaginal part in uterine cervix is covered with flat epithelium multistratified where within menstrual cycle the changes occur as in vaginal mucosa.

Endocervix is covered with one layer of high cylindrical epithelium with nuclei placed basally. Endocervical flat cellular epithelium matures during four days. Mucous secretum in the cells for endocervical epithelium is gel. Excessive mucus secretion (alkaline) is observed within follicular phase especially, in preovulatory period. After ovulation secretion decreases and becomes acid. Secretum is of great importance for impregnation and is a barrier for infection. Mucous congestion possesses bactericide, proteolytic activity on account of lysozyme, lactoferin and all immunoglobulins.

Endometrium realizes protective function for uterus, it prevents from microorganisms penetration due to recurrent rejection of functional layer within menstruation. Leucocytes execute protective function as well, they infiltrate endometric basal layer. Uterine blood supply ensures it with humoral agents.

Microorganisms penetrate into upper genital canals by means of spermatozoa, trichomonas, microorganisms' passive transport, through blood and lymph.

Agents disturbing protective mechanisms for access of infection into genital tract and initiating inflammatory processes are:

1. Labour trauma in perineum further penetration for pathogenic microorganisms into vagina from external genital organs.

2. Uteroptosis.

3. Mechanical, chemical, thermic deteriorations for vaginal epithelium (neglecting hygiene, frequent irrigations, using chemical contraceptives in vagina).

4. Ruptures in cervix of uterus.

5. Parturitions, abortions, menstruation, gynecologic operations.

6. Endouterine contraceptives (EUC). Inflammation when using EUC is boosted by inflammatory reaction around contraceptive, endometrial erosion, increasing fibrinolytic activity and synthesis of prostaglandins.

7. When tampons, particularly "Tampax" are used. Adsorbing blood they form the best conditions for fast reproduction of pathogenic microorganisms and oppression for vaginal protective mechanisms.

8. Endouterine procedures (intubations, hysterosalpingography and other).

4. CLASSIFICATION FOR INFLAMMATORY DISEASES IN FEMALE GENITAL ORGANS

There are many classifications for inflammatory diseases in female genital organs. Depending on the way of transmission infections for reproductive tract are divided into three types:

- Sexually transmitted infections (STI);
- Endogenic infections;
- Iatrogenic infections.

Agents for sexually transmitted infections are virus, bacteria and other microorganisms which don't belong to standard vaginal microflora. They can enter through urethra, vagina, oral cavity and anus.

Wide diversity of clinical manifestations is observed in such infection. It causes genital ulcerations, genital verruca, pathologic discharges from genital tract and inflammatory processes in small pelvis; infertility due to the lesion in uterine tubes, uterine cervical cancer, ectopic pregnancy, spontaneous abortion and other complications.

Endogenic infections are consequence of intensified reproduction for microorganisms colonizing standard vagina. Candidosis is one of their manifestations. It develops secondary to intensified growth of fungi *Candida* after recent antibiotic therapy, usage progesterone containing oral contraceptives, or in immune suppression. It isn't proved yet if *Candida* is sexually transmitted. Dysbalance for standard vaginal microflora can be resulted in bacterial vaginosis.

Iatrogenic infections occur when microorganisms penetrate to upper part of reproductive female tract (foremost, into sterile uterine medium) within medical procedures (e.g. while inducing EMC). Such infections can be severe danger for the health and even for the life of women.

By their localization infections in female genital organs are divided into two groups: infections for lower part in reproductive tract (vulva, vagina and uterus cervix) and infections for upper part in reproductive tract (uterine body, uterine tubes

and ovaries). In severe cases infection can pass to pelvis and abdominal cavity and even initiate bacterial shock.

With regard for large diversity of etiologic agents it is advisable to divide inflammatory diseases in female genital organs under influence of infectious agents into two large groups: specific and unspecific etiology.

Inflammatory processes due to staphylococci, colon bacillus, streptococci, blue pus bacillus and so on belong to the first group. The second group includes inflammatory processes caused by trichomonas, gonococci, candida, mycoplasmas, chlamidia, tuberculosis, viri.

It must be noted that inflammatory processes in almost all patients are caused by several infectious agents. So, associations of different microorganisms take part in development of inflammatory processes.

The most widely spread classifications consider the following divisions for inflammatory diseases in female genital organs:

For clinical course

- I. Acute processes.
- II. Subacute processes
- III. Chronical processes

For degree of severity

- I. Mild form
- II. Intermediate form.
- III. Severe one.

For localization

I. Inflammation in lower part of genital organs:

- 1) vulva (vulvitis);
- 2) large parietal vaginal gland (bartolinitis);
- 3) vagina (colpitis);
- 4) cervix of uterus:
 - a) cervicitis (inflammation in vaginal part of uterus covered with flat multistratified epithelium);

b) endocervicitis (inflammation of mucosa transferring to cervical canal in uterus and is covered with cylindric epithelium).

II. Inflammations in upper part of genital organs (organs in small pelvis):

1) uterine body;

a) endometritis (inflammation in mucosa of uterine body);

b) metroendometritis (inflammation in mucosa and muscles of uterine body);

2) adnexitis;

a) salpingitis (inflammation in uterine tubes);

b) oophoritis;

c) salpingo-oophoritis;

d) inflammatory tuboovarian tumor;

e) hydrosalpinx (accumulation of serous liquid in tubal lumen);

f) pyosalpinx (pus tube);

g) pyoovarium (ovarian abcess);

3) parametritis (pelvic cellulitis) lateral, anterior, posterior;

4) pelvic peritonitis.

5. INFLAMMATORY DISEASES IN LOWER PART OF GENITAL SYSTEM.

The lower part of female genital organs includes vulva, large vaginal parietal gland (bartholinic one), vagina and cervix of uterus. According to DIC-10 the inflammatory processes are registered as follows:

- N 76.2 – acute vulvitis;
- N 76 – chronic vulvitis;
- N 75 – bartholinitis;
- N 75.1 – bartholinic abcess;
- N 76.0 – acute vaginitis;
- N 76.1 –chronic vaginitis;
- N 72 –inflammatory diseases in uterine cervix;
- N72.0 – cervicitis.

5.1. VULVITIS

Vulvitis is inflammation for large and small genital lips, clitoris and vaginal paries. Primary and secondary vulvites are distinguished. The primary vulvitis occurs in infantile or prepubertal age. Its development is favoured by inobservance for individual hygiene, mechanical irritation or damage for vulva in itch of external genital organs. The itch may be caused by diabetes, enterobiosis, some cutaneous diseases, urinary incontinence.

The secondary vulvitis is observed more frequently in adult women. It occurs in inflammation for internal genital organs. In this case pathologic secretions damage vulval epithelium and form conditions for microorganisms' penetration.

Ovarian hypofunction promotes both primary and secondary vulvites. Bacteria and fungi are the most significant in ethiology for primary vulvites (to 70% cases). 50% cases are caused by associations of microorganisms. Ethiology for secondary vulvites is the same as in vulvivaginitis.

Symptoms in acute vulvitis. The patients complain of itch and burnings in external genital organs, particularly, after uresis, pain in moving, purulent secretions.

Within examination swollen tissues, vulval hyperemia, large secretions are observed. Sometimes hyperemia is transferred to inguinofemoral part.

In chronic stage hyperemia, edema, exudation are decreased. Itch is soothed, but constantly reoccurs.

Analysis of patient's complaints, anamnesis, data of examination, results of bacterioscopic and bacteriologic investigations further accurate diagnosis.

The treatment for vulvitis is carried out in conformity with its cause. In some cases only hygienic measures are needed, in others strengthening therapy is used. Specific therapy is recommended in diphtheria and diabetes

Topic treatment includes irrigation for external genital organs with solution of potassium permanganate (1:10 000), infusion of matricary or eucalyptus. Half bath are prescribed with matricary infusion or potassium permanganate solution (1:10 000) 2 – 3 times a day within 10 minutes. In bacterial fungal parasitogenic vulvitis terjanin for 10 days is indicated.

CANDIDIASIS VULVITIS – is inflammation for mucosa in genital lips, clitoris and vaginal paries, caused by yeast like fungi. In certain patients candidiasis vulvitis is extended to skin in inguinofemoral folds and in perianal area. More often this process is observed in patients suffering from chronic candidiasis secondary to diabetes, myxoedema, hypofunction of ovary.

Candidiasis vulvitis if urinary tract is intact is characterized by the following symptoms: decreased secretions from vagina, itch, sensation of burning or irritation in external genital organs, sensitivity of mucosa to water and urine.

There are the following clinical types for candidiasis vulvitis:

1. Solitary follicles with small erythema show vesicular type. As cracking they form erosions punctate or confluent with polycyclic borders and around epithelium rejected.

2. Diffuse – eczematous type is displayed by edema, reddening and desquamation inside focus of damage. Some days later this form is transformed in vesicular one.

3. Follicular type, when papules and pustules are revealed.

4. Granular type (chronic mycosis) starts in infantile period and then is manifested as granular inflammation with forming hyperkeratosis nodules.

Therapy for candidiasis vulvitis is the same as for candidiasis vaginitis because they occur simultaneously.

5.2. Bartholinitis

Bartholinitis is inflammation of Bartholin's gland in vaginal paries.

Etiology. Staphylococci, streptococci, colon bacillus, gonococcus, trichomonas are agents for inflammatory process. In inflammation of vaginal paries bacteria penetrate into ducts of bartholinic gland, provoking inflammation, that is, canaliculites.

Reddening is observed around external foramen of excretory duct. Obstruction for excretory duct results in false abcess; when inflammation passes to surrounding fat true abcess appears.

Symptoms. In canaliculites the patients complain of ache in external genital organs, discharges from genital tract. Patients' general condition is satisfactory. Within examination hyperemia around excretory duct is revealed, in pressing this area the pus can be excreted. It is necessary to do bacterioscopic investigation.

In false abcess of Bartholin's gland the patients complain of increasing temperature, sharp pain in area of external genital organs when walking. Some days later false abcess is lanced itself, chiefly in upper- internal part of small genital lip. Incomplete pus discharge from the focus of inflammation results in relapse for abcess or chronic bartholinitis.

Patients' general condition is rather satisfactory. In examination swelling for genital lips is revealed, sharp tenderness is perceived in palpation and opening of vulvar slit. Skin above tumour is hyperemic, swelling, but lively.

True abcess in Bartholin's gland occurs when purulent microorganisms penetrate into its parenchyma. Gland's parenchyma is completely melt in this case. The patients complain of increasing body temperature to 39 C, permanent sharp pain in area of external genital organs even at rest, sharp pain when walking. General

condition of patient is severe. Hyperemia, swelling in area of genital lips, tenderness at palpation, increasing inguinal lymphatic node are observed at examination.

Therapy. In acute stage of disease bed rest, antibiotics, analgetics, cold on perineum are administered. Antibacterial therapy is administered according to microorganisms' sensitivity. If condition is not improved during 2 – 3 days, procedures aimed at more rapid abscess formation (heat, Vishnevsky linimentum) are administered. When fluctuation appears, abscess is lanced.

Treatment of false and true abscesses consists in opportune, wide opening and drainage of abscess. Antibacterial and detoxical therapy are administered, drainage and antibiotics are introduced into abscess.

Retentional cyst in Bartholin's gland and recurrent false abscesses are submitted to surgical treatment, gland removal is carried out. It is recommended to operate in cold season.

Incision of abscess. Operative area is processed by routine method. Longitudinal incision is made along internal surface of large genital lip in the site of the most evident fluctuation (for preventing deformation in external genital organs). Incision is extended to lower pole in abscess for preventing sacs formation. (fig.1). Abscess cavity is emptied from the pus and it is drained. Drain is changed 1- 2 times daily. It is necessary that cavity gradually fills with granulations.

Removal of Bartholin's gland. Operative area is processed. Longitudinal or ellipsoid incision is made along internal surface of small genital lip. Excretory duct of gland is situated in center of ellipsoid incision therefore it allows to seize skin and fix tissues to remove. Gland is ablated carefully with scalpel or scissors. Arterial ramifications inside the wound are damaged and they must be ligated. The wound is sutured with catgut.

Abscess in glands of urethra duct more often appears in gonorrhoea inflammation of these glands, crypta and gaps in vaginal paries. False abscess occurs when excretory ducts for glands are damaged. When pathologic process extends to cellular tissue surrounding paraurethral meatus true abscess develops. Swelling and abscess are located in vaginal-urethral septum, mucosa around it is hyperemic, fluctuation is observed. When pressing the pus is discharged from urethra duct.



Fig.1. Incision of abscess in Bartholin's gland:

a) incision, b) introduction of gauze tampon.

Treatment. Emergency care consists in longitudinal incision of abscess along the most convex site of tumour with following drainage.

5.3. Colpitis

Colpitis (vaginits). It is inflammation in vaginal mucosa. This disease is caused by ovarian hypofunction, mechanical or chemical damage for epithelium, acute infectious diseases (measles, scarlet fever, diphtheria), dysnutrition.

Ethiology. Staphylococci, streptococci, colon bacillus, proteus, enterobacteria, gardnerellae, mycoplasmas, actinomycetes, filterable virus, yeast fungi, chlamydia are vaginitis agents.

Symptoms. The patients complain of discharges from genital tract, itch, burning in vagina and in vulva, dull pain and burning in urination. In some cases dull pain in the bottom of abdomen is observed.

To diagnose it is necessary as certain vaginitis ethiology because efficiency of treatment depends on it.

Ordinary (seropurulent) vaginitis. In etiology of disease staphylococci, streptococci, colon bacillus and proteus are of importance. At examination with specula vaginal mucosa is hyperemic, swollen covered with serous or seropurulent coating.

In granulocellular vaginitis small-grained granularity is revealed, it is formed by tiny infiltrates in subepithelial layer. In chronic stage changes in vaginal mucosa are insignificant, leucorrhoea are slight.

Therapy. Etiotropic therapy is carried out with antibiotics after determining agents' sensitivity. Duration for antibacterial therapy is 7 – 10 days. Vaginal suppositories, irrigation with antiseptic solutions are administered as well. Hygienic procedures for external genital organs and anal area are obligatory before using antibiotics. Pimafucin (0,1g), Hexicon (16 mg), Tergynan, Myramistinum (solution 0,01%, liniment 0,5%), Polymixin M are medicines of choice.

Atrophic (senile) vaginitis occurs in women in menopause. In this case atrophic changes are in vaginal mucosa, glycogene amount is decreased in epithelium, acidity for vaginal secretum is reduced and pathogenic microflora starts its development. The course for disease is flabby, free of complaints. Sometimes complains of burning and itch occur. In case of discharges sanguineopurulent it is necessary to except malignant processes.

Therapy. Topic treatment includes: half bath (with matricaria, calendula, eucalyptus, urtica); irrigations with disinfectant, astringent, antiseptic, deodorant solutions; standardization for vaginal biocenosis, tampons with medicinal ointments; vitamins A, E apilac, propocean, actovegin, aloe, kalanchoe, cod liver oil, panthenol, so on.

Estrogens Ovestin or estriol – 1tablet (0.5mg) 1tablet a day. Duration for therapy is 2 – 3 weeks. Then, one tablet twice a week.

Diphtheric (gangrenous) vaginitis occurs as complication after diphtheria. Examination reveals coatings as necrotic films on vaginal mucosa. Pronounced epithelial desquamation accompanied by adhesion complete or partial or vaginal stenosis is significant feature for gangrenous vaginitis. The severity of disease depends on local or general response of organism.

Therapy. Antitoxic antidiphtheric serum is introduced, antibacterial, roborant therapy is administered, vagina must be treated with antibacterial ointments.

Candidiasis vaginitis is the most spreading diseases. It is mycotic damage not only for vaginal mucosa but for vaginal segment in cervix of uterus.

It forms 6 – 30% among vaginites. Pregnancy, diabetes, prolonged therapy with wide-range antibiotics and some antitumoral medicines promote this disease. In women urogenital candidiasis process is localized mainly in area of external genital organs and vagina. Vaginal candidiasis occurs in menstruant women and may have more or less pronounced clinical symptoms, e.g. itch.

Classification for candidiasis vaginitis according to severity of disease:

1. Vaginal colonization. No complains. There are blastospores and no leucocytes in native culture from vagina. Culture is positive. Clinical signs are vague.

2. Latent vaginal candidiasis is revealed in patients suffering from candidiasis vaginitis even only once. No complaints. There are blastospores and no leucocytes in native culture from vagina. Culture is positive. Clinical signs are unclear.

3. Mild candidiasis vulvitis. The patients complain of itch and burning. Native culture from vagina is characterized by unspecific flora, blastospores, free of inflammation. Culture is positive. Free of vaginitis symptoms.

4. Midsevere candidiasis vulvitis. The most spreading complaints (especially premenstrual) are: itch, burning. Native vaginal material contains unspecific flora, increased number for leucocytes, lactobacilli, blastospores and (or) pseudomicels. Culture is positive. Signs for inflammation, e.g. papillary colpitis, are revealed.

5. Severe vaginal candidiasis. The patients complain of itch. There are signs of inflammation (great leucocytes' number) in vaginal secretum. Culture is positive. Necrotic colpitis is diagnosed at examination.

6. Persistent vaginal candidiasis.

Despite antimycotic treatment vaginal candidiasis persists (blastospores are present, clinic symptoms).

7. Recurrent vaginal candidiasis. It relapses in 4 – 12 weeks after antimycotic therapy.

8. Chronic recurrent candidiasis. It is characterized by relapses 4 times a year after antimycotic therapy.

Such clinical course is observed in vulvocandidiasis. Vulva and vagina are affected in this disease.

Fungi Candida possesses allergenic effect. Sensibilization develops in Candida carrying as well. Clinically allergenization is displayed by itch in area of external genital organs and perineum. Swollen vaginal mucosa, erosions that aren't healed for long time, ulcerations are observed at examination.

Diagnosis in candidiasis vaginitis.

Diagnosis is based on anamnesis and data of investigation. To confirm diagnosis cultural investigation for vaginal discharges is performed. Bacterioscopy reveals micellar threads, yeast cells, pH for vaginal content is 4.0 – 4.5. In alkaline reaction mixed infection is typical for bacterial vaginitis.

To diagnose candidiasis vaginitis it needs to assess in complex all kinds of examinations. It is necessary to take in account that fungi Candida are peculiar to standard vaginal microflora and they can hide their pathogenic properties and behave themselves as saprophytes and so their presence in smears don't testify the disease. It is necessary to take account the presence in smears only vegetative specimen of yeast fungi and considerable growth for culture.

Therapy for candidiasis vaginitis

Therapy for patients, especially for those suffering from chronic form is complicated. Recent time sensitivity of Candida to antimycotic medicines is decreased. Therapy for patients suffering from mycoses must be complex and be based on the principles of stepped therapy. It must cover the following aspects:

1. Influence on etiologic agent with antimycotic medicines.
2. Eliminating or softening effect of pathogenic agents.
3. Decreasing allergy and autoallergy.
4. Increasing specific and nonspecific immunologic reactivity of organism.

To treat acute forms of disease topical treatment with cream, ointments vaginal tablets, suppositoria is practised. Medicines of group imidazole: miconazole (Gynezol) cream 45g, butocanazole (Gynofort) -cream 20 g once a day for 3-5 days),

econazole (gyno-pevaril) vaginal tablets: 1 tablet a day for 3 days. Clotrimazole (cream 1% once a day for 7 – 14 days, vaginal tablets 500 mg three times a day for 7 – 10 days).

Imidazole group medicines suppress ergosterol production, which is an important element for fungus, their membrane becomes accessible to intracellular components. Imidazoles cooperate with enzymes inactivating hydrogen peroxide. Its intrauterine concentration leads to death for cells due to autolysis.

Antifungal antibiotics: natamycin: Pimafulin (vaginal suppositories 0.6 for 6 days, or cream); Nystatinum (pills 500 000AU 4 times per day for 10-14 days); Levorinum (pills 500000 AU twice or three times a day for 10-14 days); amphotericin (ointment 2 – 3 times a day for 7 – 14 days).

For peroral therapy of acute genital candidiasis in nonpregnant women are administered: Itraconazole (200mg once a day for 3 days); ketoconazole (200mg twice a day for 5 days); fluconazole (capsules 150 mg once per os or 50 mg during 1 week).

To treat chronic urogenital candidiasis general therapy is practiced as well as topical one. In such cases the treatment is more prolonged.

Simultaneously with etiologic therapy in urogenital candidiasis background disease is treated and medicines for stimulating body resistance are administered accordingly to indications. Eubiotics like as “Vagilac” (mixture of lactobacteria 4 billion), lactobacterin, bifidobacterin are used. These medicines are advisable after antimycotic therapy.

The patients suffering from urogenital candidiasis must be informed about compulsory investigation or if it is necessary treatment for their sexual partners. It is recommended sexual continence till recovery or in recurrence.

Vaginitis caused by chemical substances

Chemical or contactant vaginitis is resulted from irritant effect of soap, detergents and deodorants. Frequent irrigations promote this affection.

Symptoms. The patients complain of discharges from genital ducts and itch. Hyperemia, swollen vaginal mucos and vulva are revealed at examination. If disease isn't treated recurrent infection is feasible. When contact with agent is ceased, annoying manifestations disappear quickly.

Treatment. **Antihistaminic medicines** are administered (suprastin, tavegil, pipolphen). Creams or liniment with corticosteroids are used topically.

5.4. Bacterial vaginitis (BV)

Bacterial vaginitis (vaginal dysbacteriosis) it is dysbiosis for vaginal biotope. It is characterized by high concentration for obligate and optional anaerobic relatively pathogenic microorganisms and sharp decreasing number or lactobacteria absence in vaginal discharges. In bacterial vaginitis proliferation of relatively pathogenic flora is observed: Bacteroides peptococcus, Peptostreptococcus, Mobiluncus spp, Mycoplasma hominis, Gardnerella vaginalis. Bacterial vaginitis is diagnosed in 35% women in reproductive age suffering from gynecologic pathology.

Risk factors for bacterial vaginitis are:

- Inflammatory processes in small pelvis in anamnesis;
- Background diseases in cervix of uterus;
- Disorders for menstrual cycle;
- Using hormonal and intrauterine means for contraception;
- Great number for sexual partners.

50% women who apply intrauterine contraceptives suffer from BV. The rate of this pathology forms 40 – 50% of all infectious vaginal diseases.

Etiology. Microbiology in bacterial vaginitis has been studied, range for BV-associated microorganisms has been established. This range includes obligate-anaerobe bacteria species Bacteroides, Fusobacterium, Peptococcus, Mobiluncus and microaerophils Gardnerella vaginalis and Mycoplasma hominis. It is impossible to isolate dominant agent, because in average vaginal microcenosis is composed of 5 – 6 associates.

Gardnerella vaginalis has been described for the first time in 1953. It is optional anaerobe, stiff, polymorphic, gramvariable. It grows in nourishing media,

enriched with CO₂ at temperature 35⁰C. It is indole-, nitrate-, ureas are negative. Gardnerella vaginalis doesn't form capsules, that is why it is vulnerable to unfavourable agents, it is free of antigenic structures, possesses poor immunogenicity; it is deprived of opsonization with antibodies, phagocytosis and it can't cause acute inflammatory response of macroorganism.

Gardnerella vaginalis by fermentation produces acetic acid, sometimes, lactic, formic, succinic acids from glycogene of vaginal epithelium. These organic acids aren't aggressive to macroorganism and in physiologic concentrations aren't dangerous to tissues. Gardnerella vaginalis doesn't produce catalase and oxidase, so, it is vulnerable for hydrogenium peroxide and activated oxygen, doesn't form spores (it isn't able to resist unfavourable environmental conditions and exist out body).

All these facts show that Gardnerella vaginalis is an element of standard microflora, so the macroorganism doesn't strive against it as with foreign infectant agent.

However, in decreasing proliferation Gardnerella vaginalis secondary to oppression of its main antagonists, namely, lactobacteria it acquires pathogenic capacities which produce by some strains spalydasum, that is enzyme active for globular glycoproteids in vaginal mucosa.

This enzyme is like similar enzyme of certain pathogenic bacteria. This enzyme is responsible for substantial homogeneous, sometimes, foamy discharges. Foaming is caused by CO₂, forming in fermentation of glucose and glycopyurate into acetic acid. This reaction is exothermic, so itch and burning in vaginal mucosa are observed in some patients.

Producing microcapsule in association with ureaplasms that promotes reservation and reproduction for Gardnerella vaginalis intracellularly in epithelial cells and trichomonas is also pathogenic agent for gardnerella vaginalis. **Mycoplasma hominis**. It occurs in 24% - 75% women suffering from bacterial vaginitis and in 13 – 22% free of it. **Mobiluncus** – it is species of vaginoassociated flora which has been recently recognized. They are bent, gramvariable and lively microorganisms. Mobiluncus grows slowly on nourishing media and requires enriched medium for growth. It is indole-, catalase-, oxidase- and H₂S-negative, it

doesn't produce spores. The growth in broth is stimulated by horse or rabbit sera and carbohydrates fermentation such as maltose or glycogene. Mobiluncus is one of infectant agents for bacterial vaginitis.

Anaerobic bacteria. The data about role of anaerobic bacteria in pathogenesis for bacterial vaginitis were reported in 1978. Their species are: Bacteroides, Peptostreptococcus, Prevotella bivia, Prevotella disiens, Porphyromonas spp. Specific odour for discharges and increasing pH for vaginal content in bacterial vaginitis are caused by polyamines.

Diagnosis and symptoms. Incubative period is 7 – 10 days, but it can vary from 5 days to 21 days. The patients complain of substantial discharges from genital ducts (20 – 50 ml a day, when standard is 8ml), discharges are grey or white, often with disagreeable odour, especially after menstruation. Discharges are foamy or homogeneous, sticky. They are spread evenly on vaginal paries. The other symptoms such as itch, dysuria or dyspareunia are uncommon.

Symptoms of inflammation are seldom observed. Colposcopy shows colpitis as tiny focus.

Two clinical types for bacterial vaginitis are recognized:

- asymptomatic one; it is characterized by absence of clinical manifestations for disease and positive laboratory investigations. According to the data of foreign researchers more than half of women suffering from bacterial vaginitis have no clinical symptoms.

- disease with clinical manifestations characterized by prolonged considerable discharges and pathologic alterations in cervix of uterus with recurrences.

To diagnose bacterial vaginitis pH-metry for vaginal content, amino-test with 10 % solution of potassium hydroxide are used. Standard pH in vagine for healthy, nonpregnant women varies from 3.8 to 4.5. In bacterial vaginitis due to decreasing number of lactobacteria pH changes to alkaline medium (pH is more 4.5). Measurement for pH in vaginal content is performed with paper indicators. Accuracy in this technique is 90 %.

Positive amino-test is carried out in unpleasant odour “putrid fish” when mixing vaginal discharges and 10 % solution of potassium hydroxide in equal

proportions. Disagreeable odour shows the presence of volatile amines which are products of activity for anaerobes nonforming spores. Gardnerella vaginalis furthers increasing anaerobes' growth.

In microscopy of smear painted by Gram the following studies are performed: assessment for general bacterial dissemination, recognition of morphologic bacterial species, assessment for amount of leucocytes, phagocytes, epithelium condition and presence of key cells.

Key cells. They are surface cells of vaginal epithelium, covered with a lot of gramvariable bacilli, where Gardnerella vaginalis and vaginal anaerobes are predominant. Mobiluncus, which is less than trichomonas, but moving with the same velocity is observed in wet specimen. Key cells are revealed in smear for 90 % women suffering from bacterial vaginitis; lactobacilli and polymorphonuclear leucocytes are absent.

Microbiologic investigations microorganisms' cultivation with aerobic and anaerobic techniques and assessment for associated microorganisms as a part for microcenosis are important criterium for diagnosis.

Criteria for diagnosis of bacterial vaginitis are:

1. Specific homogeneous, adhesive, grey or grey-yellow discharges with disagreeable odour;
2. Alteration of pH in vaginal content;
3. Positive amino-test;
4. "Key cells" in native

Diagnosis is confirmed by presence of not less than 3 criteria. Microscopic study is the most appreciable (94.4%) and specific (100%) for diagnosis of bacterial vaginitis. Diagnosis is based on evidence in native smear "key cells", that is, epithelial cells, which after gardnerella adhesion to them have granular aspect.

Complementary diagnostic techniques are: assessment for acidity in vaginal content and amino-test (in interaction of vaginal secretum with 10% solution of potassium hydroxide a specific odour appears). Therapy for bacterial vaginitis has to be carried out in 2 stages. The medicines of choice for etiotropic therapy in bacterial vaginitis are antianaerobic antibiotics used as the first stage of therapy.

Nowadays the most effective medicines are: metronidazole and clindamycin. Efficiency is 80 – 90%, but some authors report about 98.8%. Metronidazole (clion) as a drug of first choice is administered perorally by 400 – 500mg twice a day within 5 – 7 days, or 2g at once.

It is important to know side effects of metronidazole: gastrointestinal disorders, metal smack in mouth, allergic reactions. Due to its teratogenic effect it is contraindicated in pregnancy.

Recent time effective treatment has been performed with clindamycin. This medicine is used per os by 0.3 twice a day during 7 days or intravaginal suppositoria: one suppositorium every day within 7 – 10 days.

It is advisable to use vaginal cream with 2% clindamycin (dalacin). It is applicated intravaginally once a day (5gr) for 3 – 7 days.

Occasionally for treating bacterial vaginitis ampicillin is administered by 500mg 4 times a day (per os or vaginally) combined with doxacyclin (amoxicyclin) by 0.2 gr a day (first day) and then 0.1gr a day for 7 – 10 days.

But clindamycin is rather drug of choice, because of its efficiency regarding anaerobes.

It should be kept in mind that in treating with metronidazole or clindamycin reccurences occur within the first year in a half of patients.

Thus, for replenishment of standard vaginal biocenosis probiotics are recommended at the second stage of therapy. Probiotics includes pharmacologic drugs or biologically active nutritional substances, containing vivid strains of standard human microflora, as a rule, microorganisms- producents of lactic acid. This term was proposed by F. Vergin in 1954 to define auspicious effect of useful bacteria on microflora contrary to harmful effect of antibiotics and other antibacterial medicines.

The main point of this process has been discovered by Ilya Mechikov at the beginning of 20-th century. He supposed, that bacteria Streptococcus thermophilus and Lactobacullus bulgaricus isolated from acidolactic products are able to influence on colon microflora and it is possible to use them instead of harmful microorganisms.

The notion “probiotics” (“eubiotics”) was brought into use in 2002 at Conference of WHO and means drugs containing sufficient amount of vivid bacterial cultures keeping microbiotic balance and optimal health for bearer. Besides notion “probiotics” “prebiotics””, “symbiotics” and “synbiotics” are rather often used.

Prebiotics are substances or products for functional feeding containing substances indigested in the upper part of gastrointestinal tract. In intestine they become nutrition for biphido-and lactobacteria.

Synbiotics are substances obtained after rational combination of probiotics and prebiotics. Symbiotics are combinations for two or more probiotics in drugs, biologically active supplements and food for boosting their therapeutic effect.

Depending on medicinal form probiotics may be dry or fluid, but depending on way for introduction into body they are divided in topical (vaginal or rectal) and peroral forms. Topical (vaginal) probiotics as suppositories containing lactobacteria are the most widespread in gynecology.

Independently on probiotic kind its positive effect is conditioned by specific mechanisms for action which are proper to probiotic microorganisms’ strains composing it.

Probiotics as well as standard vaginal microflora possess following properties:

- They promote replenishment for protective function in vaginal paries;
- They possess antagonistic activity against pathogenic and relatively pathogenic microorganisms;
- They produce bacteriocines and biosurfactants counteractive to adhesion of pathogenic microorganisms;
- They stimulate production of lysozyme and a range of peptides with antibacterial activity;
- They form acid vaginal medium, which is favourable for optimizing standard vaginal biocenosis.

According to Classification elaborated by V.O. Potapov probiotics are divided in 8 groups (table 2).

1. Monocomponents probiotics are considered classic ones. They include only one concrete strain, as usually, from obligate intestinal microflora.

2. In gynecologic practice monocomponent probiotics are inferior to polycomponent probiotics or symbiotics containing some bacterial strains of sole or some species intensifying effect of each other.

3. Combination from probiotics and prebiotics presents combined probiotics (synbiotics).

4. When manufacturing recombined probiotics (genetic engineering), useful genes are implanted into bacteria and they bring new capacities to colonies. For example, medicine “Subalin” contains bacteria able to produce interferon.

5. Self-eliminating (sporulating) antagonists contain microorganisms unusual to human biotope. Antagonists

6. Absorptive probiotics contain vivid bacteria immobilized on sorbent.

7. Metabolic ones present products after vital function of probiotic strains.

8. Multiprobitics are composed of 7 or more symbiotic bacterial strains.

In Ukraine for second stage of therapy in bacterial vaginitis such probiotics as gynoflor, vagisan, vagilac, lactoginal and others are widely used. V. O. Potapov considers that up-to-date probiotic must contain effective combination of microorganisms with reliable probiotic properties. Multiprobitics (Symbiter – 2, Probis- Femina) are more effective in renewal for vaginal biotope than monocultures.

5.5. Endocervicitis (cervicitis)

Etiology. Agents for endocervicitis are staphylococci, streptococci, colon bacillus, enterococci, Mycoplasma and so on. This disease is favoured by laceration in uterine cervix within parturition, traumatism in uterine cervix in abortion, such diseases as vaginitis, salpingoophoritis, erosion in cervix of uterus and others.

Symptoms. The patients complain of mucopurulent discharges, dull pain in lower part of abdomen. Hyperemia, turbid (sometimes purulent) discharges from cervical canal are observed around external fauces at examination with specula. Hypertrophy in cervix of uterus develops in prolonged course of disease

Therapy in cervicitis is carried out simultaneously with treatment in background and precancerous affections in uterine cervix. The aim for therapy is: elimination of unspecific inflammatory process, increasing number for agents in specific and unspecific resistance of body, metabolic normalization, stimulation for regenerative processes.

Antibacterial therapy is administered depending on agent in acute stage. Topical procedures are contraindicated (risk for spreading infection), they are performed only after extinction of process: vaginal irrigation, applications with chlorohexidine, recutan, dimexide. In case of endocervicitis secondary to uterine cervix lacerations cervicoplasty (by Emmet or by Shturmdorf) is indicated after anti-inflammatory therapy.

Erosion on uterine cervix.

Cervicitis, endocervicitis and concomitant discharges result in maceration and desquamation in multistratified flat epithelium and genuine erosion (lack of covering epithelium on uterine cervix) is formed. Genuine erosion is of irregular shape, it is brightly red and it bleeds when it is touched. It exists for 10 – 14 days. Then the genuine erosion epithelizes or transforms into pseudoerosion (defect in multistratified flat epithelium is substituted by cylindric epithelium from uterine cervix duct).

Therapy. Vaginal sanitation is performed with antiseptic solutions. Diathermocoagulation or cryocoagulation is carried out after colposcopic and histologic examinations. Nowadays the most effective method for treatment is distraction of erosion in uterine cervix with laser (laserocoagulation). This operation doesn't form scars on uterine cervix; it's especially important for women never being parturient.

6. INFLAMMATORY DISEASES FOR UPPER PART OF GENITAL ORGANS

Inflammatory diseases for upper part of genital organs are:

- inflammatory diseases in uterine body: endometritis, metroendometritis, panmetritis;
- inflammatory diseases of uterine appendages: salpingitis, oophoritis; salpingoophoritis (adnexitis), inflammatory tumours (adnextumours), hydrosalpinx, pyosalpinx;
- inflammation in perimetric cellulitis: parametritis, pelviocellulitis;
- pelvioperitonitis;
- diffused peritonitis.

According to International Classification of Diseases 10 are mentioned:

N 71.0 – acute metritis;

N 71.1 – chronic metritis;

N 70.0 –acute salpingoophoritis;

N 70.1 – chronic salpingoophoritis;

N 73 – acute parametritis;

N 73 1 –chronic parametritis;

N 73.3 –acute pelvioperitonitis;

N 73.6 –adhesive process in small pelvis.

6.1. Endometritis

6.1.1.Acute endometritis

Acute endometritis and endometritis form 0.9% among inflammatory diseases in internal genital organs. When internal uterine surface is infected, the basal endometric layer is damaged.

Etiology.Acute process is generated in ascending infection through uterine cervix canal. More often bacteria penetrate into endometric and myometric tissues

when cervical barrier is damaged in the course of spontaneous or artificial abortion. Blood, residues of decidual tissue, fetal egg promote increasing bacterial flora.

Integrity of cervical barrier is damaged in diagnostic curettage for cervical canal mucosa and uterine body, introduction of intrauterine contraceptives and other intrauterine interventions. Therefore, endometritis is generated after abortions, parturitions, diagnostic curettage and introduction of intrauterine contraceptives.

Hematogenic, lymphogenic and contact spreading of infection to tissues in uterine paries are rare. In these cases endometritis is combined with inflammations in internal genital organs of other localization.

It is caused by gonococci, chlamydia, streptococci, staphylococci, mycoplasmas, colon bacillus, enterococci, proteus and by association of 3 – 4 anaerobes with 1 – 2 aerobes and bacteroides. Chlamydial and herpetic genital infections have significant influence.

The symptoms of acute endometritis after abortion are typical. Affection begins sharply 3 – 4 days after contamination by fever with chill. General condition is midsevere or severe. Pain with irradiation to groin appears in lower abdominal segments.

A patient complains of purulent or sanguineopurulent discharges from genital ducts (abundant purulent, mucopurulent, saniopurulent discharges assume probable chlamydian infection; putreficative aspect in fluid purulent, sometimes, foamy discharges show probability of anaerobic flora). Abundant bleeding may occur in fetal residues.

General condition depends on degree of intoxication and loss of blood. More often skin is of common colour, tongue is wet. Pulse rate depends on body temperature, blood pressure is unchanged. Skin palor, pronounced tachycardia and hypotonia result from substantial loss of blood. Grey colour for skin shows intoxication. Abdomen is soft, painful in palpation of lower segments.

General blood test shows leukocytosis and formula's deviation to left, increasing ESR.

Gynecologic examination reveals uterus slightly increased, painful in palpation, especially laterally (along great lymphatic vessels). If residues of fetal egg

of early pregnancy are in uterine cavity external fauces in cervical duct is half-open, for late abortion a finger crosses easily cervical uterine canal, behind internal fauces tissue of fetal egg and blood clots are palpated.

Uterine body has bullet-shaped, its involution is delayed greatly. Uterine appendages and parametria are free of pathologic changes. Discharges are seropurulent, often, they are sanious for prolonged time, it is connected with mucosa regeneration delayed. Ultrasonic diagnosis of patient suffering from postabortive endometritis reveals the following changes (fig.2): uterine cavity is dilated; narrow echopositive strip of low, middle or high echodensity is visualized in center, it is restricted by fading echonegative area whose thickness is to 15mm (from central echopositive inclusion to myometrium). Morphologic substrate for echonegative area is inflammatory infiltrate with perifocal hydrops.

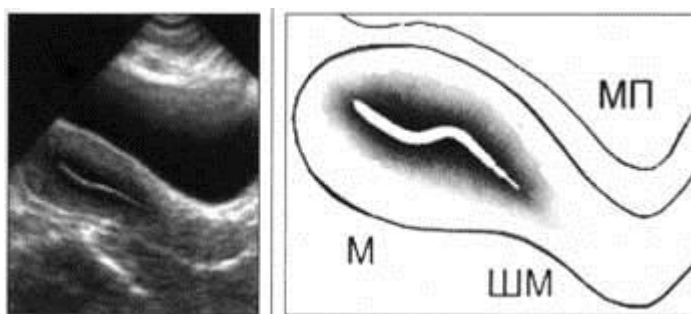


Fig.2. Postabortive endometritis (USD)

Before administration for antibacterial therapy in the first examination it is obligatory to take material for isolation of pathogenic agent. Acute stage in endometritis is for 8 – 10 days, disease ends by recovery in adequate treatment, seldom it transforms in subacute or chronic forms. As a rule endometritis being complication after spontaneous or artificial abortion performed in medical center may have positive progress of the illness in opportune and adequate therapy, but it's necessary to keep in mind occasional spreading of infection and progressing such severe complication as septic (bacterio- toxic) shock. Endometritis after illegal abortion is more severe, that it is connected with massive uterine infecting, probable mechanical or chemical damage for its parietes, toxic effect of substances used for

abortion and late requesting medical assistance. All these factors may promote progressing infection even to its generalization and doctors must operate urgently using all necessary techniques and therapeutic methods.

Intrauterine contraceptives form favourable conditions for bacteria transcervical passage-way and response of tissues around contraceptive futhers acute progressing inflammatory process followed by fast abcess formation. Danger for progressing inflammation in internal genital organs is high in first month and two years after intrauterine contraceptive introduction. Clinical symptoms for disease are: fever, abdominal pains in lower parts, purulent or sanguineopurulent vaginal discharges.

Particular form in uterine inflammatory process is pyometra. It is uterine secondary purulent damage resulted from stenosis in isthmus or cervical duct with malignant tumour, uterine myoma or endometritis. In climacteric period stenosis in cervical canal may be resulted from senile atrophy.

Retention of purulent exudate is often asymptomatic. Women are hospitalized with complains of fever, abdominal pains in lower parts. Purulent discharges from genital ducts may be absent or poor because of hampered outflow from uterine cavity. Gynecologic examination shows: uterine cervix is atrophic or standard; uterine body is increased, round, consistency soft or densoelastic.

Diagnosis for pyometra is confirmed when after surmounting with uterine probe obstacles in cervical canal or isthmus purulent outflow is observed. To diagnose oncologic diseases it is obligatory to take material for histologic study with curette. Taking purulent discharges for bacteriologic study and determining sensivity to antibiotics is obligatory too.

Therapy. Acute endometritis is treated at hospital. Therapy must be opportune and adequate. Efficiency of therapy depends on these factors.

Approaches to the treatment of patients for acute endometritis and endomyometritis are similar. Treatment must be complex, based on etiology and pathogenetics and must be individual. The patient must keep bed rest for the whole period of fever.

Infection for endometritis is quickly spreading from uterus to appendages, parametral cellulite and peritoneum of small pelvis. All it requires early antibacterial therapy. Identification for agent and antibioticogram promote correction in therapy administered. Various associations of gram-negative and gram-positive aerobes and anaerobes, chlamydia and gonococci represent large scale for agents of acute endometritis.

Systemic antibiotics are administered in acute stage. Therapy depends on scale of agents suspected (gram-positive cocci, enterobacteria, anaerobes non-sporulating and others) and their sensitivity to antibacterial medicines.

Adequate choice for antibacterial drug depends on preceding antibioticotherapy. The drugs belonging to the same group are inexpedient in progressing infectious complication in patient submitted to antibacterial therapy for another case.

Combined antibioticotherapy is used more frequently due to polymorphic etiology of disease. It is recommended to administer combination of cephalosporins (3 – 4 generations) and metronidazole, lincosaminides as well as aminoglycosides (2 – 3 generations).

Required antimicrobial effect may be assured by combination of benzylpenicillin-natrium or carbanicillin-dinatrium with gentamycin sulfate or with lincosamin hydrochloride, clindamycin phosphate. Antibacterial therapy should be administered accounting sensitivity to antibiotics (table 4).

Monotherapy is realized with aminopenicillins inhibitorprotected.

When chlamydia infection is suspected doxycycline or macrolides are administered in addition. Severity of disease determines dosages and duration for antibioticotherapy. If it is required infusive, desensitizing and strengthening therapy should be carried out.

Permanent irrigation for uterine cavity is performed with antiseptics (furacilin, chlorhexedin) in association with antibacterial medicines.

In acute endometritis resulted from late abortion uterotonics are administered, they improve drainage from lochia, decrease endometrial injury and resorption for

substances of microbic and tissue lysis. To improve drainage from lochia it is obligatory to associate uterotonics and spasmolytics.

It is difficult to correct uterine contractile activity because uterotonics' effect is powerful, quick, but transitory and can cause thrombosis. It is recommended to use medicines with prolonged effect, for example, desaminooxitocin 50 AU 3 – 4 times a day buccally.

When endometritis is complication after late abortion it would be expedient to perform intrauterine lavage with various antiseptic solutions (dioxidine, nitrofurantoin).

Diet should be rich in vitamins, easily assimilable and don't cause intestinal disorders. Recurrent cold applications to lower abdominal part have anti-inflammatory, analgetic and hemostatic effect.

Local hypothermia promotes decreasing hyperemia and hyperhydration in focus of inflammation, local decreasing metabolic processes and oxygen consumption, ceasing allergic reactions, strengthening antibiotics' effect.

Acupuncture and other reflexotherapeutic procedures are administered as well. Electrophoresis with zinc by diadynamic current is used, it possesses not only contractile effect but has anti-inflammatory effect. Spasmolytics (no-spa 2% 1 – 2 ml 2 – 3 times a day) are administered simultaneously.

Surgical care. Surgical intervention is indicated when improvement in patient's condition is not observed within 72 hours after starting therapy for acute endometritis. In intrauterine contraceptive it is required to remove contraceptive, simultaneously it needs to take material for inoculation, bacteriologic and cytologic study.

If there are fetal egg residues infected after artificial abortion surgery consists in instrumental revision for uterine cavity (vacuum-aspiration of fetal corpuscles in first 3 – 4 days, for later terms it is recommended to use ovum forceps and curette by fixation uterine cervix with bullet forceps, avoiding uterus removal, if it is possible)

Physiotherapy and sanitary-resort care are advisable in subsequent management for preventing complications in endometritis, improving regenerative capacity for endometrium and menstrual cycle recovery.

Acute period for endometritis is for 8 – 10 days. In adequate therapy process ends, sometimes it transforms in subacute or chronic forms.

6.1.2. Chronic endometritis

Morbidity rate is 2 – 4 % in average – 14 %. Recent years the frequency for chronic endometritis is progressing due to wide intrauterine contraceptives usage, to increasing number for abortions and other intrauterine procedures, including endoscopic methods of investigation. Chronic endometritis is a sequel after acute undercured endometritis postpartum or postabortive.

Hysterorrhagia is the main symptom for chronic endometritis. Deficiency in endometrial transformations disturbs the processes of desquamation and regeneration in functional layer and causes post- and premenstrual discharges. Intermenstrual bloody discharges are associated with increased permeability in endometrial vessels within ovulation.

Similar transformations are observed in healthy women as well, but diapedesis in blood cells is clinically imperceptible. Decreasing uterine contractile capacity, damages for thrombocytes' aggregational properties cause uterine hemorrhages. The patients may complain of serous or seropurulent discharges from genital ducts and dull pain in lower part of abdomen. Increasing and induration for uterus are observed in vaginal examination.

Diagnosis in chronic endometritis is based on anamnesis data, clinical symptoms, on findings after histologic study of endometrial scrape. To get maximum information diagnostic scrape is performed in first phase (8 – 10 day) of menstrual cycle. Hysteroscopy shows atrophic variety for chronic endometritis.

Therapy. In aggravation for inflammatory process the treatment is analogous to therapy in acute endometritis. Antibiotics are administered with regard to agent's sensitivity: semisynthetic penicillins, kanamycin, gentamycin and others. Dosages and duration of treatment depend on severity for disease. If it is required infusive, desensitizing and strengthening therapy should be carried out

Physiotherapy and sanitary-resort treatment are advisable in remission.

6.2. Parametritis

Parametritis is inflammation in parametrium. This disease occurs seldom; it is resulted from infection in parametrium due to uterine cervix damage, as a rule, in lacerations of internal fauces for cervical canal.

Inflammatory process spreading to parametrium is localized in one of lateral parts of pelviocellulate (dextrolateral or sinistrolateral parametritis, anterior parametritis, posterior parametritis), if inflammation embraces the whole pelviocellulate it is pelviocellulitis.

Etiology. Postpartum or postabortion infection, various intrauterine interventions (probing biopsy, hysterosalpingography), spreading infection through lymphogenic way from uterus, uterine tubes, intestine or through blood (for flu, typhus, tuberculosis) are causes for this disease. Parametritis is diffuse inflammation for parametrium enriched with venous and lymphatic vessels. Perivascular pronounced edema is evident, lymphatic vessels are dilated, filled up with thrombus and purulent mass. Exudate in cellulite is serous, seropurulent or purulent.

Symptoms. Persistent ache at the lower part of abdomen intensifying with progressing process is the first sign for disease. Body temperature is 38 – 39 C, sometimes with chill. Emiction is accelerated, painful; dyspeptic disorders (constipation, diarrhea, tenesmus are noted. At initial stage when palpating anterior abdominal wall practically isn't painful, percutaneous sound isn't dull. In bimanual examination (rectal examination is recommended too) laterally, in front or behind of uterus painful infiltration with faint contours is revealed.

Subsequently this infiltration is extended to pelvic walls, up and down to inguinal and pubic parts smoothing or sagging out uterine fornix. In suppuration fluctuation in center of infiltration is revealed. Uterus is dislocated to healthy side.

For posterior parametritis infiltration surrounds rectum in front and laterally, it is evident in rectal examination accompanied by tenesmus and mucosal discharges from anus. When inflammatory infiltration is resolved patient's condition becomes better and temperature is normalized.

More prolonged course for disease is associated with infiltration's suppuration. Patient's general condition becomes worse, temperature curved line is remittent,

chills, dysuric symptoms are proper for anterior parametritis, tenesmus and pus and blood in feces are in posterior parametritis. When pus breaks through bladder or rectum general condition for patient becomes better, temperature is normalized, pus appears in feces or urine. But if narrow orifice is closed after break for abscess, patient's condition becomes worse.

Diagnosis for parametritis in standard cases isn't complicated. Diagnose should be based on clinical symptoms, data after bimanual and rectoabdominal examination. In extensive pelvic abscess parametritis should be differentiated from pyosalpinx, suppurative hematoma in tubal abortion, pelvic peritonitis.

Treatment in acute period includes the following: rest, cold to the bottom of abdomen, antibacterial therapy, for pains analgetics are administered. In suppurating parametral infiltration (in sagging out posterior fornix) opportune dissection should be done.

6.3. Salpingoophoritis

Among gynecologic diseases acute salpingo-oophoritis is the most frequent. Isolated inflammation for fallopian tube in medical practice occurs seldom. Simultaneous inflammation for fallopian tubes and ovaries is more frequent. It may be associated with uterine inflammation.

Etiology. Salpingo-oophorites are caused by pathogenic and relatively pathogenic microorganisms: staphylococci, streptococci, colon bacillus, chlamydia. Anaerobes, such as, bacteroides and peptococci may be agents for inflammation or secondary associated infection. More often flora is mixed. Inflammation starts with endosalpingitis: it is revealed hyperemia, microcirculation disorders, exudation, perivascular infiltrations. In extending inflammation to muscular membrane fallopian tube is thickened and lengthened, becomes edematous, sharply painful when palpating.

Some main pathogenic mechanisms for infecting uterine appendages and spreading pathogenic and relatively pathogenic agents to upper parts of genital organs are mentioned nowadays.

Transcanalicular way (through cervical canal along endometrium to fallopian tubes and ovaries) is one of the ways for infection. Gradually all parts of genital duct and parietal peritoneum. Pathologic process progresses on both sides simultaneously.

Transcanalicular mechanism for damaging genital organs occurs mostly in young women. Most of bacteria and viruses reach the upper parts of genitalia with spermatozoides. Progressing inflammation for pelvic organs is observed secondary after direct contact of abdominal organs damaged by inflammation.

Inflammatory diseases for tubes and ovaries have similar pathogenesis and symptoms. Microorganisms with fallopian content penetrating through abdominal end damage its serous layer (perisalpingitis), ovarian epithelium and peritoneum surrounded (perioophoritis). After follicule laceration its granulosis membrane is infected and inflammatory process in ovary develops.

Tumours with serous (hydrosalpinx) or purulent (pyosalpinx) content emerge in ampullar part of tube due to adhesion of phimbria and adhesions. Inflammations in ovaries (cysts, abcess) and hydrosalpinx or pyosalpinx form tuboovarian “tumour” (tuboovarian abcess).

6.3.1. Acute salpingo-oophoritis

Symptoms and diagnosis. The patients complain of fever, clinical deterioration, violent pain in lower part of abdomen, rigor, dysuric disorders. Examination reveals abdomen rigid, painful when palpating. In gynecologic investigation the pain is intensified; contours for uterine appendages are indistinct due to edema and perifocal processes, they are increased, pastous with mobility limited.

Changes in nervous and vascular systems are caused by pronounced intoxication. Clinical symptoms for acute salpingo-oophoritis depend on degree for pathogenity of microorganism, intensity for inflammatory reaction and content's species (serous, purulent).

Blood analysis shows: deviation of leucocytic formula to left, increasing blood sedimentation rate; globulin fractions are prevalent in proteinogram, increasing C-reactive protein in blood.

In opportune adequate treatment acute inflammatory process may result in complete recovery, but it can transform in subacute or chronic process for months, even, for years.

To diagnose the patients suffering from acute inflammation of internal genital organs it is advisable to use laparoscopy.

The following cases are suitable for laparoscopy:

- 1) acute catarrhal salpingitis;
- 2) catarrhal salpingitis with signs for pelvioperitonitis (table 3);
- 3) acute purulent salpingo-oophoritis with signs for pelvioperitonitis or diffuse peritonitis;
- 4) purulent inflammatory tubo-ovarian formation;
- 5) laceration in pyosalpinx or purulent tubo-ovarian formation, generalized peritonitis.

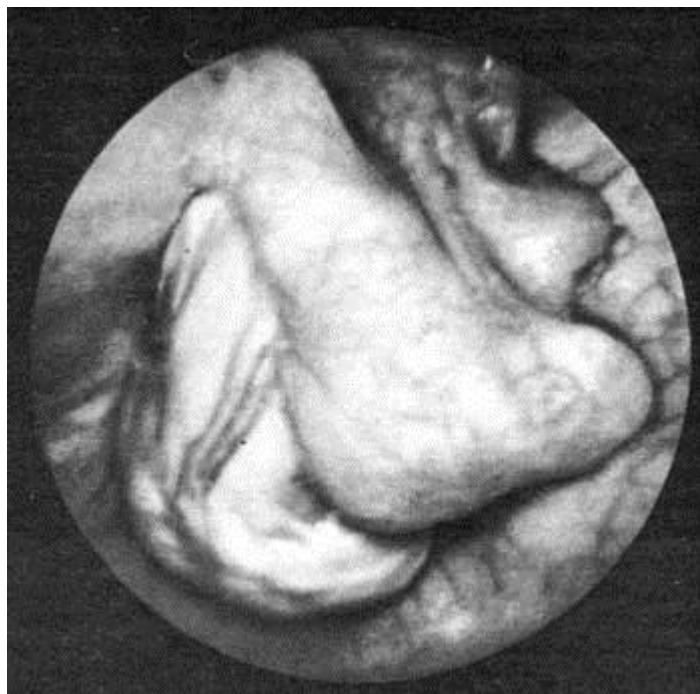


Fig. 3. Acute purulent salpingo-oophoritis, pelvioperitonitis (laparoscopic image)

Acute purulent salpingo-oophoritis begins sharply with fever, rigors, pains in the lower part of abdomen, abundant purulent discharges; symptoms of purulent intoxication are evident. At the beginning of disease the pain is distinctly localized. When pelvioperitonitis is progressing the pain is diffused all over the abdomen. In this case differential diagnosis with acute surgical diseases for abdominal cavity is indicated.

Diagnosis for acute purulent salpingitis is based on three criteria: abdominal pain, sensitivity in movements of uterine cervix, sensitivity in appendages associated with if only one criterion: fever, leukocytosis more than 10 000, increasing sedimentation rate, evident inflammatory formations in bimanual examination or in USD (fig. 4), pus after puncture for posterior vaginal vault (fig. 5).

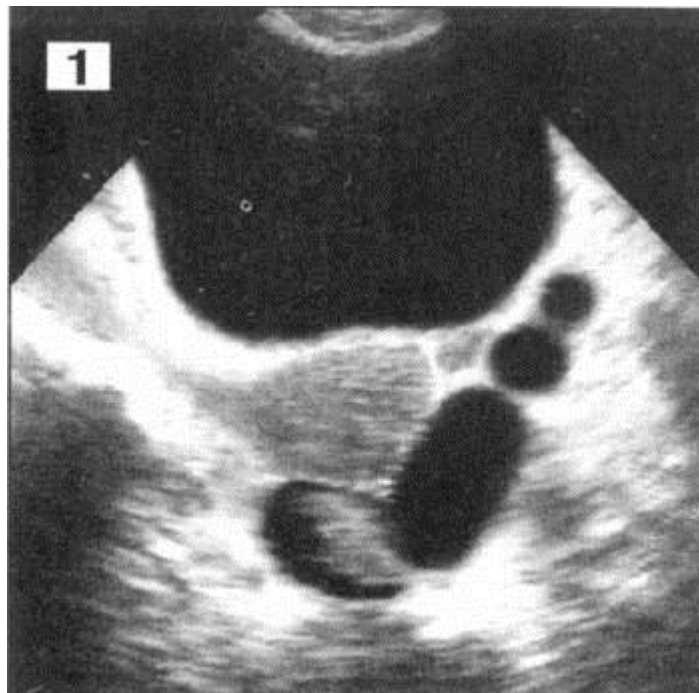


Fig. 4. Multi-cellular exudative purulent salpingitis (USD)

Sonogram in left parametrial part shows that liquid formation of irregular prolonged shape (97 and 38mm) is observed; the cavity for this formation is divided in 4 unequal cells. The content in 3 upper cells is homogenous, but there are numerous echopositive inclusions of mean density, linear and punctiform shape in lumen of lower cell.

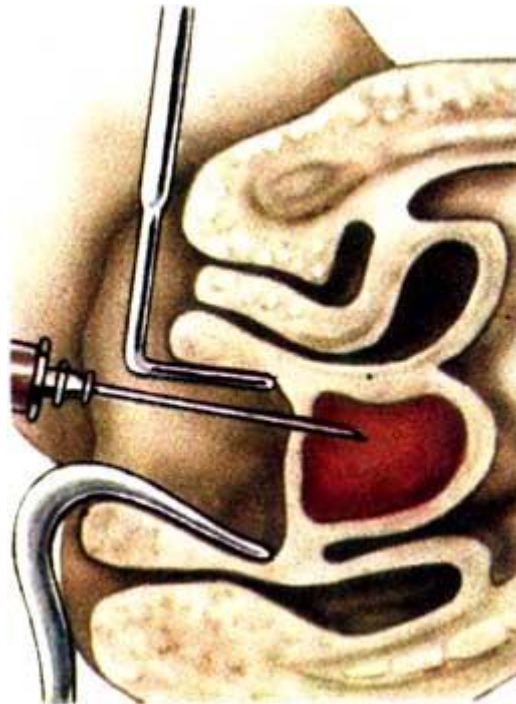


Fig.5. Puncture of abdominal cavity through vaginal posterior vault.

The treatment of patients in acute stage is carried out at hospital. They are ensured physical and mental comfort, permanent monitoring for condition and functioning their organs.

Antibacteril therapy is administered regarding agent and sensivity to antibiotics. Antibiotics with long half-life period should be administered. Paramount schema includes combination 3-rd generation cephalosporins (cephotxim, cephtriaxon) with metronidazole and aminopenicillins inhibitorprotected (amoxicillin/clavulonic acid).

As alternative, lyncosamides in combination with aminoglycosides, phtorchinolons (ciprofloxacin, ofloxacin) with metronidazole and carbopens may be administered. It is advisable to administer simultaneously doxycyclin or macrolides taking into consideration the high risk for chlamyidian infection.

As a rule antibacterial therapy in small pelvis diseases begins with intravenous administration of drugs followed later on by oral use (stepped therapy).

In severe cases in association for gram-negative and gram-positive flora and when anaerobic flora is suspected various antibiotics' combinations are expedient: (clindamycin with chloramphenicol; gentamycin with levomycetin, lyncomycin clindamycin.

In bacterial associations (streptococci, staphylococci enterobacteria) and anaerobes (bacteroides, peptococci, paptostreptococci) the treatment begins with cephtriaxon 1,0 2 times per day, simultaneously aminoglycosides are administered (canamycin 0,5gr intramuscularly 2 times a day or gentamycin 1mg/kg intravenously 3 times a day).

When clinical effect is absent within 72 hours clindamycin is associated 600mg intravenously 4 times a day till decreasing temperature and abolition for symptoms of peritoneal irritation.

Then, cephtriaxon and aminoglycosides are taken perorally during 5 days. The following therapeutic schema is practiced:

- Cephtriaxon 1000mg intramuscularly or intravenously 1 dosage + doxicyclin 100mg 2 times a day perorally for;
- ofloxylin 400 mg 2 times a day perorally;
- clindamycin 300-450 mg 4 times a day perorally;
- metronidazole 500 mg 3 times a day perorally for 10-14 days.

Combination undermentioned is recommended: cephoxitin 2 gr intramuscularly+ probenecid 1 gr perorally + doxicyclin 100mg perorally for 14 days.

In severe case, in purulent salpingo-oophoritis are administered: При важкому перебігу, гнійному сальпінгоофориті призначається:

- cephoxitin 2 gr intravenously every 6 hours;
- cephotetan 2 gr intravenously every 12 hours, in combination with doxicyclin 100 mg intravenously or perorally every 12 hours;
- clindamycin 600 – 900 mg intravenously + gentamycin or ampicillin/sulbactam or ticarcillin/clavunat or piperacillin-tasobactam.

When anaerobic flora is suspected metronidazole is administered due to its bactericide effect on obligate anaerobes and infections caused by *B. fragilis*. It is administered in severe cases intravenously 1 – 1.5 gr a day with a rate 5 ml/min for 5 – 8 days; in less severe cases the drug is recommended perorally 400 – 500 mg a day for 7 – 8 days, but, if necessary, duration of treatment may be increased.

In intoxication infusive therapy is used: solution 5 % with glucose, polyglucin, rheopolyglucin, proteinic medicines (total amount for liquid is 2 – 2.5l a day). If necessary, vitamins are added to infusive liquid as well as 4 - 5 % solution of natrium bicarbonate (500 – 1000 ml for correction of acido-alkaline condition, antihistaminic medicines (dimedrol, suprastin).

It is recommended to use cold to the lower part of abdomen. Cold affects skin receptors, alleviates pain, possesses anti-inflammatory and hemostatic effect. It is used every 2 hours with 30 minutes interval.

In mild cases for inflammatory diseases of small pelvis organs the patients may be treated in out-patients conditions. In these cases oral medicines of high bioavailability are preferable. Antibiotics are associated with detoxicant therapy: intravenous administration of saline solutions, 5% glucose solution, rheopolyglucin, hemodesum, polydesum, maphusol, vitamins, protein drugs and others.

If indicated, analgetics, local anti-inflammatory medicines are administered as suppositiria, cold to venter. When general condition is stabilized and acute process is alleviated ionophoresis with calcium, copper and magnesium (depending on phase of cycle is carried out.

In subacute stage autohemotherapy, aloe, physiotherapy is administered: UV rays, electrophoresis with medicines (potassium, magnesium zinc), vibromassage, ultra-high-frequency therapy.

The pronounced inflammatory process in uterine appendages often requires surgical treatment. Diagnostic laparoscopy is expedient. For acute inflammation antibiotics' solution is introduced (for example, ampicillin 1gr for 20ml physiologic solution).

Later on surgical treatment is required when therapeutic effect after conservative treatment is failed and tubo-ovarian formations are revealed.

For laparoscopy, depending on pathologic changes, tubes irrigation with isotonic solution containing antiseptics and antibiotics, incision for new adhesions, removal phimbria from tubes and ovaries are carried out.

In progressing purulent processes (pyosalpinx, pyovarium) surgical intervention is indicated.

6.3.2. Chronical salpingo-oophoritis

Chronical salpingo-oophoritis may be resulted from undertreated acute process, but it can progress without acute stage. Chronical stage is characterized by evident infiltrations, physiologic functions for mucous and muscular uterine membranes failed, emerging connective tissue, narrowing vascular lumen, sclerotic processes.

In prolonged course for disease often obstruction in uterine tubes accompanied by forming hydrosalpinx and adhesions around ovaries are observed. Peritubal and periovarian adhesions blocking to impregnation may be formed.

Symptoms. Patients' complaints are resulted from nevrosis observed in prolonged period of chronical salpingo-oophoritis and frequent recurrences of disease. The main complaint is dull pain intensifying in cooling, intercurrent diseases and before and within menstruation. Specificity consists in pain reflected, emerging by mechanism for viscerosensory and viscerocutaneous reflexes.

Pains in lower abdominal parts, in inguinal and pubic parts, in vagina cause inconveniences. Often, pain becomes aware along pelvic nerves (pelvioneuralgia, vegetative ganglioneuritis resulting from chronical inflammatory process).

Menstrual disturbances (polymenorrhea, oligomenorrhea, algodysmenorrhea) are observed in 40 – 55% patients and are connected with ovarian disturbances (hypofunction, anovulation). More often menstrual disorders are revealed in patients suffering from chronical salpingo-oophoritis and ovarian hypofunction.

Infertility is caused by anatomic and functional changes in uterine tubes and ovarian hypofunction. Disturbances for sexual function (painful coitus, decreasing or libido failed) are observed in 35 – 40% patients. Secretory function disturbances (leucorrhoea) resulted from concomitant colpitis and endocervicitis are frequent.

The course for chronic salpingo-oophoritis is long. It is characterized by aggravation of painful sensations and deteriorating general condition under influence of cooling and other factors.

Diagnosis. It is made on the basis of anamnesis: it should be taken in consideration inflammatory process in uterine appendages after abortion, parturition complicated, intrauterine diagnostic procedures.

In bimanual examination it is important to find out uterus position, mobility, conditions of appendages, sacrouterine ligaments and pelvic parietes. USD reveals hydro-or pyosalpinx (fig.6). Hysterosalpingography reveals changes in fallopian tubes.

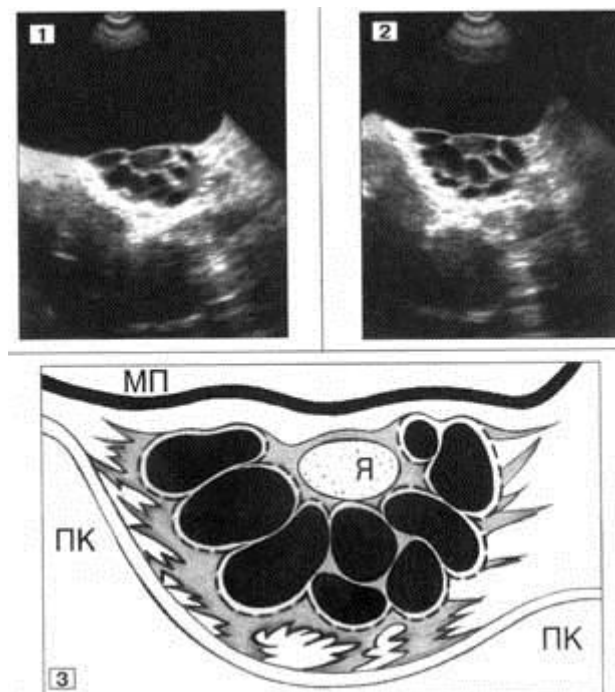


Fig.6. Tuboovarian formation (by left) as sequel of chronic recurrent exudative salpingitis and adhesive disease

Left uterine tube filled with homogenous liquid is seen around left ovary (it is poly-cell hydrosalpinx). Ovarian capsule is thickened owing to adhesions. Total dimensions for tuboovarian formation are 65 for 40mm. Ovary is 27 for 40mm of standard microfollicular structure.

Rehabilitation. There are three rehabilitative levels in chronic salpingo-oophoritis:

Prime level – clinical recovery (improvement for general condition, abolition of painful syndrome), anatomic changes resuscitation in uterine appendages, normalization for blood composition;

Second one – recovery for endocrinic function in genital system (resuscitation for menstrual cycle, normalization in functional diagnosis data, improvement for hormonal correlations);

Third one – restoration for adaptational and protective mechanisms and reproductive function (normalization for sympathicoadrenal system, pregnancy); it is necessary to observe succession for stages and therapeutic measures according to rehabilitative level. Rehabilitative treatment for persistent remission is based on physiotherapeutic effect taking in consideration cyclic character of menstrual function in body (table 3).

Table 3

Cyclic physiotherapeutic rehabilitation for chronic salpingo-oophoritis

(K.I. Malevich, P.S. Rusakevich)

Menstrual cycle phase	
Prime(proliferations)	Second (secretions)
Aim of therapy	
Activation for parasympathic link of vegetative nervous system	Activation for sympathetic part of vegetative nervous system.
Procedures	
Electrophoresis with novocain to cervicofacial part	17-th – 26-th day of cycle: endonasal electrophoresis with vitamin B1
10 th – 24 th day of menstrual cycle: electrostimulation for uterine cervix (by S.N. Davydov) or progressive laserostimulation with helium-neon laser.	5 th – 25 th day of cycle: progressive gynecologic massage for from 5 to 20 minutes.

6.3.3. Tuboovarian purulent inflammatory formations

Etiology is multimicrobial for formation of inflammatory process in uterine appendages, intracanalicular way of infection from lower part of genital organs is predominant in pathogenesis. More often inflammatory diseases for uterine appendages start with endometritis, then inflammatory process by intracanalicular way transform in purulent salpingitis.

Ovarian germinal protective integumentary epithelium set up powerful barrier and so the ovary is damaged only in cystic formations. Therefore inflammatory process in ovaries more often occurs as inflammatory adnextumor. Pelvic abscesses or gynecologic peritonitis is observed in perforation of abscesses, but sometimes without it.

Thus, forming tuboovarian purulent abscesses occur after aggravation for chronic salpingoophoritis. Infection is spread by canalicular way from chronic endometritis (for uterine hemorrhages, abortions, intrauterine interventions...) to purulent salpingitis and oophoritis.

Clinical manifestations depend on complexity for process. Acute salpingitis is free of complications, but all encapsulated inflammatory adnextumours (purulent tuboovarian formations) are complicated forms.

Clinical signs for purulent inflammatory process are various. Purulent salpingitis and purulent tuboovarian formations in acute stage have similar clinical manifestations and are like symptoms for gonorrhoea.

Purulent salpingitis begins sharply with fever, tachycardia, acute local pains in left or right hypogastral part irradiating to loin, rectum and thigh by side damaged. Diffused character for pains (by the whole abdomen) is observed in patients suffering from pelvioperitonitis.

Persistent symptom for purulent salpingitis is abnormal character for vaginal discharges. They are excreted not only from vagina, but from urethra and cervical canal accompanied by dysuric disturbances.

Often it is impossible to determine in gynecologic examination formations in area of uterine appendages due to sharp tenderness and peritoneal symptoms.

Tenderness for dislocation of uterus and in palpating posterior and lateral vault is the most typical sign for this process.

Firstly, the painful syndrome is localized, then in progressing disease sore area is extended over. Increasing pains testify to probable perforation of purulent tumour with the most severe complications as diffused purulent peritonitis, interintestinal abscesses and fistulas.

The main manifestations for infectious syndrome are: fever, deterioration of general condition, increasing leukocytic index for intoxication. Moderate leukocytosis, deviation of leukocytic formula to left and increasing erythrocytes sedimentation rate are observed in blood.

Syndrome for inflammation of adjacent organs is accompanied by spreading infectious process to adjacent organs and tissues with forming secondary appendicitis, pelvic abscesses, secondary changes in intestinal loops (sigmoiditis, rectitis) in omentum (omentitis), in parametric cellulitis (parametritis) and others. Symptoms stratification of main disease and processes in adjacent organs are going on.

Syndrome for metabolic disturbances is evinced by hypoproteinemia, dysproteinemia, electrolytic disorders to acidosis, hypoxia and disorders in cellular metabolism. Clinically metabolic disturbances are revealed in progressing polyorgans insufficiency both before operation and after it.

To diagnose tuboovarian formations transvaginal echography, diagnostic (therapeutic) laparoscopy, diagnostic puncture of abdominal cavity through posterior vaginal vault are used.

Differential diagnosis should be done in case of acute appendicitis, cholecystitis, intestinal impaction, surgical peritonitis, oncologic process.

Therapy

Medicamentous treatment is used with the aim to be confined to conservative anti-inflammatory treatment. It is realized in cases of diagnostic difficulties between purulent process and routine inflammation free of abscess. Furthermore this treatment may be performed as preoperative arrangements.

When acute purulent process is exactly diagnosed antibiotics, antiprotozoal and antifungal drugs are administered. Disintoxicative therapy is used for infusive correction in volemic and metabolic disturbances, antihistaminic drugs are administered as well.

Surgical treatment is the main stage for tuboovarian abscess. It is chosen individually in each case: from abscess puncture, laparoscopic drainage to panhysterectomy.

Laparoscopy is considered as the most effective and advanced method to treat pyosalpinx, py ovaries and tuboovarian formations if they persist for 2 – 3 weeks. It should be used if case isn't clear and differential diagnosis is awkward, especially in young women.

Laparotomy should be performed for encapsulated tuboovarian formations and their complications: secondary appendicitis, rectitis, sigmoiditis, omentitis and peritonitis. Adequate anesthesia and skilled surgeon are very important for successful operation.

Postoperative treatment envisages antibacterial and other therapy. The measures used are aimed to standardization for digestive motility, hyperbaric oxygenation, haemosorption or plasmapheresis. Therapeutic complex includes hepatotropic therapy, immunostimulating therapy (UV-rays, laser irradiation for blood, immunocorrection) antianemic therapy, parenteral feeding, gymnastics and other.

Patients submitted to surgery for tuboovarian formations require subsequent out-patient monitoring. Women with residual scars and adhesions need medical examination twice a year and adequate diet with vitaminotherapy.

Unparturient women submitted to tubectomy may be sent to extracorporal impregnation after rehabilitation course.

6.4. Pelvioperitonitis and peritonitis

In gynecologic practice inflammation of small pelvis peritoneum, that is, pelvioperitonitis is local restricted inflammatory process but peritoneal diffused inflammation is diffused or disseminated peritonitis.

6.4.1. Pelvioperitonitis

Mostly pelvioperitonitis is secondary one. Inflammatory diseases of uterus and fallopian tubes extend to peritoneum of organs and result in perimetritis and periadnexitis in progressing inflammation the whole small pelvis peritoneum is involved in the process and thus pelvioperitonitis emerges.

Etiology. OcPrime etiologic agent is bacterial flora penetrated to abdominal cavity (staphylococci, streptocci, gonococci, colon bacillus and so on). The following factors promote penetrating infection: abscess laceration in pyosalpinx, small pelvis cellulitis abscess, uterine perforation, hydrotubation, hysterosalpingography, introduction of chemical substances to uterine cavity aimed to abortion, in some cases, surgical interventions on small pelvis organs.

Symptoms. There are three stages for peritonitis: 1) acute; 2) subacute; 3) chronic.

The disease begins with fever, chill, violent pain in lower abdominal part, nausea, vomiting, meteorism, intestinal atony, constipation or diarrhea, sometimes, painful emiction. Abdominal paries is strained, sharply painful in lower abdominal parts is observed in percussion. Symptom Shchetkin – Blumberg is positive. Topic diagnosis is awkward for gynecologic examination due to strain in anterior abdominal paries and violent tenderness.

Blood test displays: leukocytosis, neutrophilia, lymphopenia and increasing erythrocytes sedimentation rate.

By symptoms acute stage for pelvioperitonitis is like diffused peritonitis one.

The second subacute stage for pelvioperitonitis occurs after restricting inflammatory process with adhesions. Patient's general condition becomes better, free of nausea, vomiting and abdominal irritation; flatulence and tenderness are retained in lower abdominal parts when palpating; body temperature is dropped,

pulse becomes standard. In vaginal investigation exudate is revealed in recto-uterine hollow, it removes uterus forward and up and protrudes posterior fornix. The upper border for exudate is revealed above pubis.

In auspicious conditions exudate is resolved, interorgans adhesions are remained, displace and restrict motility for pelvic organs.

In unfavourable conditions the process transforms in chronic stage which is characterized by prolonged slow course, limpness, recurrent aggravation. Within aggravation abcess can be formed and break through rectum, bladder, vagina or abdominal cavity. After abcess drainage by puncture (fig.5) or colpotomy (fig.7, 8, 9) patient's condition becomes better. In spontaneous abcess lancing into bladder or abdominal cavity disease is complicated by ascending infection for urinary ducts or diffused peritonitis.

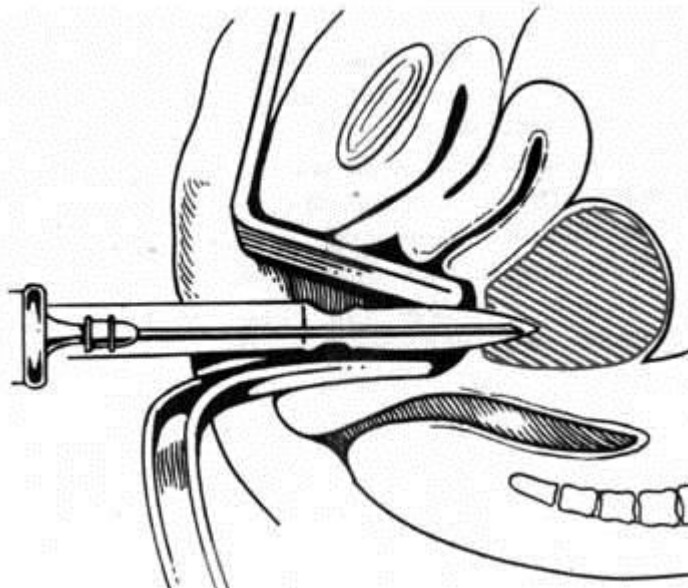


Fig.7. Incision of posterior vaginal fornix with scalpel (scalpel acute borger is turned to uterine cervix)

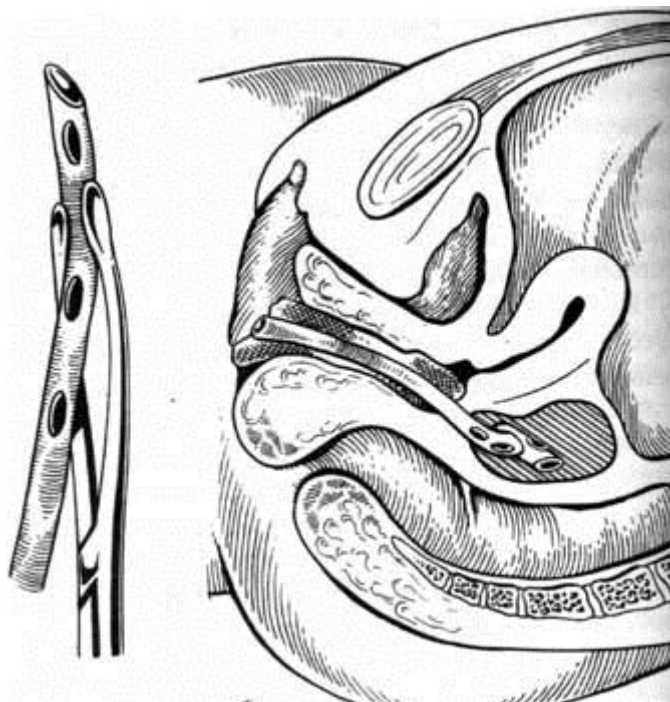


Fig.8. Abscess drainage: polychlorviinyl drainage tube is seized with dressing forceps

Diagnosis is based on anamnesis data, data of vaginal examination and laboratory investigations. In acute stage differential diagnosis with diffused peritonitis should be done. In patient with pelvioperitonitis within intensive therapy tendency to restricting process and improvement for general condition is observed, but progressing process supposes diffused peritonitis.

Differential diagnosis with extrauterine pregnancy is based on findings posterior fornix puncture.

Therapy depends on stage for pelvioperitonitis. In initial acute stage therapy is aimed to restricting inflammatory process: strict bed regimen, cold to lower abdominal part, complex drug therapy: antibacterial drugs (antibiotics, nitrofurans and sulfanilomides) (table 2); detoxicant therapy, antihistaminic medicines and analgetics.

Posterior vault puncture is indicated to determine exudate nature and for therapeutic goal. Punctate should be sent for bacteriologic study.

6.4.2. Peritonitis

Gynecologic peritonitis is secondary disease occurring mostly in postoperative period (after cesarean and gynecologic operations), in rupture for purulent inflammatory tumours (pyosalpinx, purulent parametritis, in spreading infection by lymphagenic or hematogenic ways from inflammation focus. Sometimes, peritonitis is a sign for septic infection generalized.

Classification. There are primary and secondary peritonitis. Primary peritonitis emerges after penetrating infection from vagina to uterus, fallopian tubes and to abdominal cavity by canalicular way or by lymphagenic or hematogenic ways. Secondary peritonitis is resulted from penetrating infection from abscesses in internal genital organs.

Course of disease. There are three phases for peritonitis: reactive, toxic and terminal.

Reactive phase is superimposed on disease, which caused peritonitis. The patients complain of abdominal pains, thirst, dyspnoe, eructation, nausea and vomiting. Tongue is furred and dry. General weakness, abdominal flatulence and pectoral respiration are observed. Then, straining abdomen muscles, exudate to abdominal cavity, positive symptoms Shchetkin – Blumberg, persistent vomiting and dehydration become evident. Symptoms for general intoxication are observed: dryness in mouth without thirst, excitation, tachycardia and tachypnoe.

Reactive phase for gynecologic peritonitis is often characterized by fading topic symptomatics, acute beginning with fever (38-40C), pulse accelerated and dyspnoe. Simultaneously meteorism moderate is observed, symptom Shchetkin – Blumberg is poor, stools are normal. In these cases it is obligatory to control pulse rate (120 – 140 beats/min.), intestinal paresis and if there is exudate to abdominal cavity.

Reactive phase transforms in toxic one, which is characterized by progressing intoxication accompanied by excitation, disorientation, further by adynamia transforming in prostration. Patient's habitus is exhausted, skin cyanosis, hypotension and high leukocytosis are observed, pulse is frequent and filariform.

Toxic phase transforms in the last terminal phase for peritonitis. It is characterized by such main symptoms as adynamia, retardation, total disorientation and Hippocratic face. Intestinal paresis, metabolism abruptly disturbed is observed, body wastes proteins, salts, liquid, diuresis is abruptly decreased. Vegetative disturbances as accelerated pulse and respiration, hypotension are intensified. For this stage prognosis is unfavourable.

Diagnosis for peritonitis is based on clinical symptoms and data after laboratory blood investigation. When early diagnosis is awkward X-ray investigation is expedient: Horizontal lines are proper for liquid accumulated in intestinal loops, these lines don't change their site due to peristalsis failed (Kleiber dishes). Vaginal posterior fornix puncture is advisable for unclear cases.

Treatment. General and topic therapeutic methods are distinguished. The first is performed by correction for fluid and-electrolyte balance, acid-alkali balance, protein metabolism, resuscitation for damaged function of internal organs and abolition of infection and intoxication. Topic therapy includes early removal of infection focus and recovery of paralytic intestinal impaction. Treatment for peritonitis is stepped and includes preoperative measures, operative intervention and intensive therapy for postoperative period.

When diffused peritonitis is finally diagnosed surgical intervention is only the method for treatment in this disease. It is aimed to removal of origin for infection (purulent tumours in ovaries and fallopian tubes, abscesses in parametral cellulitis, hysterectomy); removal for toxic substances from abdominal cavity. To prevent intestinal paresis it is recommended method for permanent resorption if intestinal content with transnasal probe introduced through stomach.

Preoperative measures. Preparation for surgical intervention has to last for not more than 1.5 – 2 hours. It includes puncture and catheterization for subclavicular vein and intravenous administration of antibiotics and complete transfusional therapy. While preparing for operation the patient needs as minimum 1200ml of liquid transfused including 400ml rheopolygluchinum or hemodesum, 400ml plasma or albuminum and 400ml saline solution while carrying out transfusional therapy within anesthesia and intensive therapy in postoperative period.

Antishock therapy consists in emergent renewal for circulation (administration of plasma, albuminum, rheopolygluchinum and so on). Endotracheal narcosis with muscular relaxant should be used for anesthesia. Any operation in peritonitis resulted from inflammatory formations in uterine appendages is atypical, because of difference for adhesive process in small pelvis and abdominal cavity, significant changes in tissues surrounded appendages damaged and specificity for adhesions. They may be friable or dense. For last case recovery is long, sometimes, tissues surrounded and organs are traumatized and it requires supplementary interventions. Thus, these operations are prolonged and traumatic. For complete revision of abdominal cavity incision on anterior abdominal paries should be located on lower-middle part.

To prevent bacterio-toxic shock intraoperative administration of antibiotics (intravenously, at the moment of skin incision) is indicated to all patients (table 4). Antibiotic maximum concentration in blood is observed 15 – 30 minutes after intravenous administration, that is, at the moment when purulent tuboovarian content is discharged from adhesions. It is expedient to use cephalosporins of 2-nd generation (cephuroxim, zinaceph) associated with metronidazole or cephalosporins of 3-rd generation (fortum or clarofan). Antibacterial intravenous therapy has to go on in postoperative period.

An important moment for surgical treatment in peritonitis consists in complete recovery for standard anatomic relations among organs in small pelvis, abdominal cavity and tissues surrounded. It is obligatory to perform revision in abdominal cavity, to determine appendix condition and except interintestinall abcesses for purulent inflammatory process in uterine appendages.

Scope for surgical intervention depends on undermentioned aspects:

- 1) peculiarity of process;
- 2) concomitant pathologies in genital organs;
- 3) patient's age

Surgical tactic should be based on the following: operation for elder patients should be more radical, despite difficulty in surgical intervention. Postoperative

period for elderly women is longer than for young ones and depends on outflow from abdominal cavity. Operation consists in hysterectomy.

Main advices for surgical treatment of patients suffering from peritonitis:

1. Operation should be finished by complete removal of destructive focus. Operative scope depends on peculiarity and severity for main disease, concomitant pathology for internal genital organs and patient's age.

2. Within operation revision for abdominal organs and urinary ducts should be done. When there are signs of secondary appendicitis, interintestinal or subphrenic abscesses, occlusion for urinary ducts operative scope must be extended to remove this pathology.

3. Optimization for surgical treatment consists in correct and consecutive execution of various techniques and stages, radical removal for all destructive focus and scarry tissues, accurate commissure for borders of wound free of tension, adequate choice for sutural material, thorough sanitation for abdominal cavity and adequate drainage.

4. Sewing for anterior abdominal paries should be accurate and firm with using up-to-date materials.

Abdominal drainage. Drainage is performed for right and left subphrenic spaces, perinephric area, right and left glomal areas.

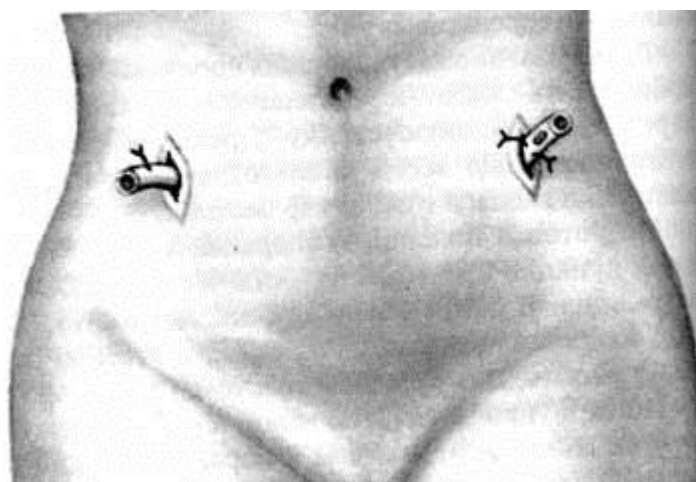


Fig. 9. Abdominal drainage

Abdominal sanitation is carried out in postoperative period with furacin aqueous solution (1: 5000), sterile saline solution with supplementary antibacterial drugs.

Dyalis is performed for 3 days, on the 4-th day drains are removed. In postoperative period intensive therapy: infusional, antibacterial (table 4) strengthening ones goes on

Principles of intensive therapy in patients with postoperative peritonitis:

1. Use adequate pain relief in postoperative period. The best is the use of prolonged epidural. Epidural anesthesia - is not only a way of anesthesia, but a therapeutic method that allows you to keep independent breathing in full, reduces spasm of peripheral vessels, improves blood circulation in the kidneys, stimulates diuresis, improves motor-evacuation function of the gastrointestinal tract, improves psycho-emotional The state is the prevention of thrombosis of blood vessels of small pelvis and lower extremities and thromboembolic complications.

If there are contraindications to the use of the method of epidural anesthesia is necessary to conduct narcotic analgesics during the first three days of their introduction through various time intervals (4-6-8-12 hours). For potentiation of and reducing the need for narcotic drugs they should be combined with antihistamines and sedatives.

2. Antibiotic therapy (tab. 4). Preparations should be broad spectrum and affect aerobic and anaerobic pathogens.

3. Infusion therapy. Number of introduced liquid should be at 35-40 ml / kg body weight per day. Introduced to 400-1000 ml / day of colloidal solutions, protein drugs with the calculation 1-1.5 g of native protein per 1 kg, crystalloid. The total number of introduced liquid is 2.5-3 liters.

4. Stimulation of the intestine (epidural anesthesia, adequate fluid resuscitation, use of drugs metoclopramide).

5. Heparin therapy in high daily dose of 20 thousand. OD with a gradual reduction and discontinuation of therapy.

6. Continues imunokoryhuyuchoyi of therapy.

7. A correction ecological community.
8. Treatment hepatotropic and cardiac drugs.
9. Symptomatic therapy.
10. According Show - extracorporeal detoxification methods (hemisorbtion, limfosorbtsiya, plasmapheresis, UV, HBO).

Postoperative rehabilitation of patients is to eliminate the remaining infiltrative inflammatory changes, prevention of relapse of purulent process, restoring hormonal funktsiyi body or its replacement after radical surgery.

6.5. SEPTIC SHOCK

Cod by ICD-10 A48.3

By definition, a clinical protocol "Septic shock", approved by the Ministry of Health of 31.12 2004 № 676, this state is defined as a clinical syndrome that occurs in the systemic inflammatory response to infection and manifest violation of the body's ability to maintain homeostasis and hemodynamics as a result of inadequate oxygenation of tissues and circulatory disorders.

Since the concept of sepsis, septic shock, and systemic inflammatory response often identified, particularly in clinical practice, in 1992, experts from different disciplines were elaborated a new definition for sepsis and its effects.

Definitions of sepsis, severe sepsis, septic shock

SYSTEMIC INFLAMMATORY RESPONSE SYNDROME (SYSTEMIC INFLAMMATORY RESPONSE SYNDROM-SIRS)

- systemic inflammatory response to a variety of severe clinical damage that manifests two or more of the following manifestations:

- 1) body temperature over 38 ° C or below 36 ° C;
- 2) heart rate over 90 beats / min.;
- 3) respiratory rate over 20 min or Pa CO₂ below 32 mmHg;
- 4) the number of leukocytes over 12000/mm³, less than 400/mm³ or more then 10% of young forms.

SEPSIS

- systemic inflammatory response to infection reliably detected in the absence of other possible causes for these changes, which are characteristic of SIRS.

Clinical manifestation includes two or more of the following symptoms:

- 1) body temperature over 38 ° C or below 36 ° C;
- 2) heart rate over 90 beats / min.;
- 3) respiratory rate over 20 min or Pa CO₂ below 32 mmHg;
- 4) the number of leukocytes over 12000/mm³, less than 400/mm³ or more then 10% of young forms.

SEVERE SEPSIS / SIRS

Sepsis, which is accompanied by dysfunction of organs, hypoperfusion or hypotension. Hypoperfusion and perfusion disturbances may include (but are not limited to) the acidosis resulting from accumulation of lactic acid, oliguria or acute

impairment of mental status. Sepsis - Induced Hypotension: systolic blood pressure below 90 mmHg or decrease in blood pressure by 40 mm Hg. century. from baseline in the absence of other causes for hypotension.

SEPTIC SHOCK (SIRS - SHOCK)

This complication of severe sepsis and is defined as sepsis - induced hypotension that is not subject to correction by adequate fluid replenishment; perfusion abnormalities that may include (but are not limited to) the acidosis, oliguria or acute impairment of mental status.

Patients receiving inotropes or vasopressors may not be hypotension, but still retain features hipoperfuziynyh disorders and dysfunction of organs that belong to the manifestations of septic shock.

Promote the emergence of shock:

- the presence of foci of infection (septic abortion endomyometritis, horionamnionit, lohiometra, the remains of the ovum and others);
- reducing the overall resistance of the organism;
- the possibility of penetration of pathogens or their toxins in the bloodstream.

In the development of septic shock distinguish two stages:

- Hyperdynamic - reducing peripheral resistance, reflex increases heart function, ie cardiac output;
- Hipodynamic - violation of perfusion and oxygenation secondary in relation to regional vasoconstriction and cardiac dysfunction.

Diagnosis. In case of severe sepsis there is also a number of changes that take place and: in septic shock. These are:

- thrombocytopenia $< 10\ 100/L$, which is not explained by other causes;
- increase the level of C-reactive protein;
- improving prokaltsytoninu $> 6,0\ ng / ml$;
- positive blood cultures with the detection of circulating microorganisms;
- positive test for endotoxin (LPS-test).

The diagnosis of septic shock is established, if listed above clinical and laboratory signs of joining:

- hypotension (systolic pressure less than 90 mmHg. Cent., Or lowered more than 40 mm Hg.C. from baseline);
- tachycardia over 100 beats / min.;
- tachypnea over 25 min;
- impairment of consciousness (less than 13 points on a scale of Glasgow);
- oliguria (urine output less than 30 ml/h);
- hypoxemia (Pa O₂ less than 75 mm Hg. C. while breathing ambient air);
- Sp O₂ < 90%;
- increase of lactate over 1,6 mmol/l;
- petechial rash, necrosis of the skin.

It is necessary to carry out the following activities:

- 1) monitoring of hemodynamic parameters: blood pressure, heart rate, central venous pressure;
- 2) control the parameters of the respiratory system (counting respiratory rate, blood gases, Sp O₂);
- 3) hourly monitoring of urine output;
- 4) measurement of rectal temperature at least 4 times a day for comparison of body temperature in aksilyarnyh areas;
- 5) urine, blood and secretions from the cervical canal;
- 6) determination of the acid-base balance of blood and tissue oxygen saturation;
- 7) platelet count and determine the content of fibrinogen and fibrin monomer (soluble fibrin).

For a complete picture of the idea seems besides these clinical and laboratory examinations must additionally hold:

- ECG - to identify the metabolic disturbances or myocardial ischemia;
- Ultrasound of the abdomen to detect possible hematogenic abscesses;
- X-ray examination of the chest cavity to confirm the acute respiratory distress syndrome or pneumonia.

Laboratory analysis show the presence of severe inflammation and degree of organ failure:

- In most cases, common anemia;
- neutrophilic leukocytosis with a shift to the left;

- leukocytosis > 12000/ml in some cases may be marked leukemoid response of leukocytes to the number of 50-100 thousand and above. Sometimes leukopenia may occur;

- morphological changes of neutrophils include toxic granularity, the appearance of cells fate and vacuolation;

- thrombocytopenia, lymphopenia.

The degree of intoxication reflects leukocyte *index of intoxication* , which is calculated by the formula:

$LI = (S + 2SL + 3Y + 4Mi) (PC - 1) / (Mo + Ly) (E + 1)$, where:

S - segmented neutrophils, SL - stab leukocytes, Y - young white blood cells,

Mi - myelocytes, PC - plasma cells, Mo - monocytes, Ly – lymphocytes, E - eosinophils.

LI in normally equal to one.

Increasing the index to 2-3 indicates limited inflammation/

Increasing to 4-9 - a significant component of the bacterial endogenous intoxication.

Leukopenia with high LI are poor prognostic sign for patients with septic shock.

BASIC PRINCIPLES OF INTENSIVE THERAPY OF SEPTIC SHOCK:

1. Immediate admission to the intensive care unit.
2. Correction of hemodynamic disturbances through inotropic therapy and adequate fluid therapy with constant monitoring of Geodynamics.
3. Maintain adequate ventilation and gas exchange.
4. Surgical rehabilitation of the site of infection.
5. The normalization of bowel function and early enteral nutrition.
6. Timely correction of metabolism under constant laboratory control.
7. Antibiotic therapy under constant microbiological control.
8. Antymediatorna therapy.

The main goal of fluid therapy in septic patients is to maintain an adequate blood supply to the tissues. The volume of infusion therapy in case of septic shock is determined by a comprehensive assessment of hemodynamic responses to infusion

reaction (blood pressure, particularly pulse blood pressure, central venous pressure, heart rate, speed of diuresis). Of particular importance in these cases is the definition of CVP dynamics. The benchmark is to test for dose-response CVP input fluid (test with a load volume). Patients within 10 minutes of injected test dose of liquid (see Table 8) and evaluate hemodynamic response.

Table 8.

SAMPLE WITH VOLUME LOADING

Output level CVP	Volume of fluid injected
8 cm of water	century and less than 200 ml
8-10 cm of water	century. 100 ml
14 cm of water	century. 50 ml

The reaction hemodynamics assessed as follows: if CVP increased by more than 5 cm of water century, the infusion is stopped and produce inotropic support, but if CVP increased no more than 2 cm of water century then continue infusion therapy without inotropic support.

Recommended following program infusion therapy in case of septic shock. Initially, the fluid is injected at 10 ml/min. for 15-20 min., and then - in a normal pace, depending on hemodynamic, respiratory, urine output and so on.

For the infusion used derivatives hidroksyetylkrohmalju (venofundyn, Refortan, HAES - steril) and crystalloids (0,9% sodium chloride solution, Ringer's solution) at a ratio of 1:2. Unlike other colloid solutions hidroksyetylkrohmalju reduce the extent of damage to the endothelium of the capillaries, improves lung function and reduces symptoms of systemic inflammatory response .

To correct hypoproteinemia appointed concentrated solutions of albumin - 20-25%. The use of 5% albumin at critical conditions improves mortality of patients.

The inclusion of transfusion media glucose inappropriate. Purpose of glucose in critically ill patients increases the production of lactate and CO₂, and increases ischemic brain damage and other tissues. Glucose infusion is justified only in cases of hypoglycemia and hypernatremia .

The structure of infusion environments should include fresh frozen plasma (600 - 1000 ml), a donor of antithrombin. Antithrombin inhibits leukocyte activation and prevents damage to the endothelium of blood vessels, thereby decreasing the manifestations of systemic inflammatory response and endotoxemia. In addition, the introduction of fresh frozen plasma is necessary for the treatment of DIC, which usually develops during the progression of septic shock.

Inotropic support. If after CVP normalize blood pressure is low, the dopamine administered at a dose of 5-10 mg/kg/min. (up to 20 mcg/kg/min.) or dobutamine, which is introduced at 5-20 mcg/kg/min. If this therapy is not conducive to a stable increase in blood pressure, the introduction of complementary therapy sympathomimetic norepinephrine hydrotartrate at a speed of 0,1 - 0,5 mg/kg/min. while reducing the dose of dopamine to the "kidney" (2-4 mg/kg/min.).

Given the role of beta-endorphin in the pathogenesis of septic shock with sympatomimetykamy justified concomitant use of naloxone and 2,0 mg, which improves blood pressure.

In case of failure of complex hemodynamic therapy can be applied corticosteroids. Equivalent dose (in terms of hydrocortisone) is 2000 mg/day. Its introduction, in order to prevent erosive lesions of the stomach, it is necessary to combine with H2-blockers (ranitidine, famotidine).

Maintain adequate ventilation and gas exchange. In severe cases, respiratory failure against the background of the progression of multiple organ dysfunction should immediately decide on the transfer of the patient to the ventilator.

Indications for mechanical ventilation:

- Pa O₂ < 60 mm Hg;
- Pa CO₂ > 50 mm Hg or < 25 mm Hg;
- Sp O₂ < 85%;
- Respiratory rate over 40 per minute.

The flow of oxygen should be minimal, providing Ra O of 80 mm Hg respiratory therapy of septic shock should also include positive pressure mode and at the end of exhalation (3-6 cm water column), but with adequate recovery BCC.

Surgical rehabilitation of the site of infection

Indications for laparotomy and hysterectomy with uterine tubes are:

- No effect of intensive care;
- the presence of pus in the uterus;
- uterine bleeding;
- purulent lesions in the region of the uterus;
- detection by ultrasound presence of residual ovum.

The normalization of bowel function and early enteral nutrition is one of the important tasks in patients with sepsis and septic shock, as the recovery of barrier function of the intestine is the key to further microbial translocation into the bloodstream and decrease of systemic inflammatory response.

This is achieved by enteral drip of 0,9% sodium chloride or carbonated mineral water 400-500 ml per day via gastric tube or nipple duodentalnyy followed by an increase in the volume of injected fluid and the expansion of food preparations provided normalization of peristalsis in the "nutritional factor", corresponding to 2000 - 4000 calories a day.

It is also appropriate concomitant use of prokinetics (metoclopramide) and glutamic acid, as the latter normalizes metabolism in the gut villi.

After stabilization of the patient for further prevention of bacterial translocation may conduct selective decontamination of the gut. For this purpose, 4 times a day in the intestine injected mixture polymyxin - 100 mg tobramycin - 80 mg and amphotericin B - 500 mg.

One of the highlights in the treatment of septic shock is antibiotic therapy. Given that today is almost impossible microbiological rapid diagnosis, during antibiotic therapy it is advisable to stick tactics deeskalatsiynoyi empirical antibiotic therapy. After identification of microorganisms and to determine its sensitivity to antibiotics are transferred to antibiotic therapy according antybiotykoqramy.

Antymediatorna therapy based on current knowledge of the pathogenesis of septic shock and is very promising. There is strong evidence bahatoklonalnyh use of antibodies in combination with pentoxifylline. Given the lack of Ukraine bahatoklonalnyh antibodies, appropriate use in the treatment of septic shock pentoxifylline. With the same purpose justified the use of dipyridamole.

Application of extracorporeal detoxification only after stabilization of the patient, as in the expanded picture of multiple organ failure they increase the mortality of patients.

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8. ADDITION

PROBIOTICS IN GYNECOLOGY

Tabl. 1

The main species composition of microflora vaginal biotope

The species composition of microflora	number, CFU / ml
Microaerophilic bacteria	
<i>Lactobacillus spp.</i>	$10^7 - 10^{10}$
<i>G.vaginalis</i>	till 10^6
Obligate anaerobic Gram + bacteria	
<i>Lactobacillus spp.</i>	$10^7 - 10^9$
<i>Bifidobacterium spp.</i>	$10^3 - 10^7$
<i>Clostridium spp.</i>	till 10^4
<i>Propionibacterium spp.</i>	till 10^4
<i>Mobiluncus spp.</i>	till 10^4
<i>Peptostreptococcus spp.</i>	$10^3 - 10^4$
Obligate anaerobic Gram - bacteria	
<i>Bacteroides spp.</i>	$10^3 - 10^4$
<i>Prevotella spp.</i>	до 10^4
<i>Porphyromonas spp.</i>	до 10^3
<i>Fusobacterium spp.</i>	до 10^3
<i>Veilonella spp.</i>	до 10^3
Fakultatyvno- anaerobic Gr + bacteria	
<i>Corinebacterium spp.</i>	$10^4 - 10^5$
<i>Staphylococcus spp.</i>	$10^3 - 10^4$
<i>Enterobacteriaceae</i>	$10^3 - 10^4$
<i>Streptococcus spp.</i>	$10^4 - 10^5$
<i>Mycoplasma hominis</i>	10^3
<i>Ureaplasma urealyticum</i>	10^3
<i>Mycoplasma fermentus</i>	till 10^3
Fungus	
<i>Candida</i>	till 10^4

Genus and species of microorganisms that are part of probiotics

Genus	Species
<i>Lactobacillus</i>	<i>L. acidophilus. L. bulgaricum. L. casei. L. crispatum. L. fermentum. L. gasseri. L.gohnsonii. L. lactis. L. paracasei. L. plantarum. L. reuteri. L. rhamnosus. L. salivarius</i>
<i>Bifidobacterium</i>	<i>B. animalis. B. adolescentis. B. bifidum. B. breve. B. infantis. B. lactis. B. longum</i>
<i>Lactococcus</i>	<i>L. spp.cremonis. L. lactis. spp. lactis</i>
<i>Escherichia</i>	<i>E. colli</i>
<i>Streptococcus</i>	<i>S. thermophilus. S. cremoris. S. lactis, S. intermedius. S. diacetylactis</i>
<i>Enterococcus</i>	<i>E. faecium. E. faecalis</i>
<i>Saccharomyces</i>	<i>S. boulardii. S. cerevisiae</i>
<i>Propionibacterium</i>	<i>P. acnes</i>
<i>Bacillus</i>	<i>b. subtilis, B. Cereus/B. licheniformis</i>

Probiotic strains in preparations, which have indications for use in urogenital diseases

Probiotic strain	CFU / ml	Preparation	Producer
Vaginal forms of probiotics			
<i>Lactobacillus acidophilus</i>	$1,0 \times 10^9$	<i>Hinoflor</i>	<i>Medinova Ltd, Switzerland</i>
<i>Lactobacillus rhamnosus</i> ; <i>Streptococcus thermophilus</i> ; <i>Lactobacillus delbrueckii (Bulgaricus)</i>	$1,6 \times 10^9$ $1,6 \times 10^9$ $0,2 \times 10^9$	<i>Fermalak</i>	<i>Institut Rossel Inc., Canada</i>
<i>Lactobacillus rhamnosus</i> , штам GR-1; <i>Lactobacillus reuteri</i> , штам RC-14	$1,0 \times 10^9$ $(5,0 \times 10^9)$	<i>Vahisan (Laktovah)</i>	<i>Yardan, Croatia</i>
<i>Bacillus</i> штам 35	$1,0 \times 10^9$	<i>Laktozhynal</i>	<i>Laboratoires LYOCENTRE SAS, France</i>
<i>Lactobacillus plantarum</i> , штам P17630	$>1,0 \times 10^9$	<i>Hinolakt</i>	<i>Teva, Israel</i>
<i>Lactobacillus delbrueckii (Bulgaricus)</i>	$4,0 \times 10^9$	<i>Vahilac</i>	<i>Fharmascience</i>
<i>Bifidobacterium bifidum</i>	$1,0 \times 10^7$	<i>Bifidumbacterin</i>	<i>Biomed, Russia</i>
<i>Lactobacillus acidophilus</i> , штам NK, 100 і КСІІ24	$1,0 \times 10^7$	<i>Atsilakt</i>	<i>Vytafarm, Russia</i>
<i>Lactobacillus acidophilus</i> , штам LaCH-2	$>1,0 \times 10^8$	<i>Ekofemin</i>	<i>Vinci A/S, Dania</i>
<i>Lactococcus spp.</i> <i>Lactobacillus spp.</i> <i>Bifidobacterium spp.</i> <i>Propionibacterium</i> Містить 24 штами корисних бактерій	$5,0 \times 10^{10}$ $1,0 \times 10^9$ $10,0 \times 10^9$	<i>Symbiter 2</i>	<i>Пролісок, Ukraine</i>

Oral forms

<i>Lactic Acid Bacillus</i> (<i>Bacillus coagulans</i> (<i>Lb. sporogenes</i>) <i>Lactobacillus</i> <i>rhamnosus</i> , <i>Lactobacillus reuteri</i> , <i>umam RC-14</i>	$1,2 \times 10^8$ $1,0 \times 10^9$ $(5,0 \times 10^9)$	<i>Laktovit forte</i> <i>Vahisan</i>	<i>Mili Healthcare, United Kingdom</i> <i>Yardan, Croatia</i>
<i>Bifidobacterium</i> <i>bifidum</i> , <i>Lactobacillus</i> <i>acidophilus</i> , <i>Lactobacillus</i> <i>salivarius</i> , <i>Lactobacillus casei</i> , <i>Lactobacillus</i> <i>plantarum</i> , <i>Lactobacillus</i> <i>rhamnosus</i>	$1,0 \times 10^7$	<i>Femi Biokap</i>	<i>Про-Фарма, Ukraine</i>
<i>Lactobacillus</i> <i>acidophilus</i> <i>Lactobacillus</i> <i>rhamnosus</i> <i>Lactobacillus casei</i> <i>Lactobacillus reuteri</i> <i>Lactobacillus</i> <i>plantarum</i> <i>Lactobacillus</i> <i>fermentum</i> <i>Bifidobacterium</i> <i>bifidum</i>	$2,0 \times 10^9$ $2,0 \times 10^9$ $1,0 \times 10^9$ $2,0 \times 10^9$ $1,0 \times 10^9$ $1,0 \times 10^9$ $1,0 \times 10^9$	<i>Probiz Femina</i>	
<i>Lactobacillus spp.</i> <i>Lactococcus spp.</i> <i>Bifidobacterium spp.</i> <i>Propionibacterium</i>	$5,0 \times 10^{10}$ $1,0 \times 10^{10}$ 10.0×10^{10}	<i>Symbiter</i>	